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INTERNATIONAL JOURNAL OF PROGRESSIVE RESEARCH IN ENGINEERING MANAGEMENT AND SCIENCE (IJPREMS)
(Int Peer Reviewed Journal)
Vol. 05, Issue 03, March 2025, pp : 1897-1906

e-ISSN : 2583-1062
Impact Factor : 7.001

POISON ON THE PLATE: A REVIEW OF NATURAL TOXIC SUBSTANCES IN FOOD

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DOI: https://www.doi.org/10.58257/IJPREMS39272

ABSTRACT

Food is essential for human survival but, some edible substances contain toxic compounds that can be detrimental to our health. Natural toxic substances are present in various foods, posing potential health risks to consumers. This review aims to summarize the current knowledge on natural toxic substances in food, including their sources (plants, animals, and fungi), mechanisms of toxicity and potential health effects. We discuss the toxic compounds found in plant-based foods, such as lectins, saponins, and glycoalkaloids, those found in animal-based foods, like biotoxins in seafood, as well as those found in mushrooms including amatoxins, phallotoxins, and gyromitrin. We also examine the factors that influence the levels of these toxic substances in food and discuss strategies for minimizing exposure. It is essential to be aware of these potential food poisons and take steps to prevent and treat food borne illnesses.

1. INTRODUCTION

Food is needed for human survival, but it can also contain natural toxic substances that pose health risks. Naturally occurring toxins are harmful substances produced by living organisms, including plants, animals, and microorganisms [1]. While these toxins are not detrimental to the producing organisms, they can be poisonous to other species, including humans, if ingested [2]. These toxic compounds exhibit a wide range of chemical structures and vary in their biological functions and levels of toxicity. Certain plants synthesize toxins as a self-defense mechanism to deter herbivores, insects, and pathogens, thereby protecting themselves from harm [3]

The presence of these naturally occurring toxins in food has been a concern since the dawn of agriculture. Ancient civilizations undoubtedly encountered poisonous plants, animals and fungi, leading to the development of empirical knowledge regarding safe food selection and preparation. Early toxicology, while not formally established as a scientific discipline, was intertwined with traditional medicine and culinary practices. The identification and characterization of specific toxins, however, emerged much later. The study of plant toxins, or phytotoxins, gained significant traction with the advancement of analytical chemistry in the 19th and 20th centuries. Pioneering work by scientists like Claude Bernard (1813-1878) on the physiological effects of plant extracts laid the foundation for future research [4]. Later advancements in analytical techniques like chromatography and mass spectrometry revolutionized the understanding of food borne toxins [5].

2. TOXIC COMPOUNDS IN PLANT-BASED FOODS

2.1. Solanine

Solanine is a toxic glycoalkaloid compound found in various Solanaceous plants, including potatoes (Solanum tuberosum), tomatoes (Solanum lycopersicum), and eggplants (Solanum melongena). While solanine is present in small amounts in these plants, ingestion of high amounts can cause a range of adverse health effects, including nausea, vomiting, diarrhea, and abdominal pain [5].

Mechanism of Action

The mechanism of action of solanine involves its ability to disrupt cellular membranes and interfere with normal cellular function. The key steps involved in this process include:

1. **Inhibition of cholinesterase-** Solanine inhibits the enzyme cholinesterase, which breaks down the neurotransmitter acetylcholine. This leads to an accumulation of acetylcholine in the nervous system, causing overstimulation of muscles and glands [5]
2. **Disruption of cellular membranes-** Solanine disrupts the integrity of cellular membranes, allowing ions and other molecules to leak out. This can lead to changes in cellular function and even cell death (6-7).
3. **Interference with normal cellular function-** Solanine can also interfere with normal cellular function by inhibiting the activity of certain enzymes and receptors. For example, solanine has been shown to inhibit the activity of the enzyme Na^+/K^+-ATPase, which is essential for maintaining proper ion balance in cells [8].

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Toxic Effects

The toxic effects of solanine can vary depending on the amount and duration of exposure. Some of the possible toxic effects exhibited by solanine include:

1. **Gastrointestinal symptoms**
   Nausea, vomiting, diarrhea, and abdominal pain are common symptoms of solanine toxicity [9].
2. **Neurological symptoms**
   Headache, dizziness, and confusion can occur due to the accumulation of acetylcholine in the nervous system [5].
3. **Cardiovascular symptoms**
   Solanine can cause changes in heart rate and blood pressure, leading to cardiovascular symptoms such as arrhythmias and hypertension [9].

Prevention

To prevent solanine toxicity, it is essential to handle and store Solanaceous plants properly by observing the following precautions

1. **Choose healthy plants**: Select plants that are free of signs of disease or damage.
2. **Store plants properly**: Store plants in a cool, dry place to prevent moisture accumulation.
3. **Cook plants properly**: Proper cooking of plants can help reduce the levels of solanine.

Treatment

If solanine toxicity occurs, treatment typically involves supportive care, such as:

1. **Fluid replacement**: Replacing lost fluids and electrolytes.
2. **Rest**: Providing rest and relaxation.
3. **Medications**: Administering medications to manage symptoms such as nausea and vomiting. [10-11]

2.2. Amygdalin

Amygdalin is a naturally occurring cyanogenic glycoside found in various fruits, including bitter almonds (Prunus dulcis), apricot kernels (Prunus armeniaca), and other members of the Rosaceae family [12]. While amygdalin may have potential therapeutic applications, its ingestion can lead to the release of cyanide, a potent toxin that can cause severe health consequences, including respiratory failure, cardiac arrest, and death [13].

Structure and Mechanism of Action

Amygdalin is a glycoside composed of a cyanide group, a glucose molecule, and a benzaldehyde group [14]. When ingested, amygdalin is hydrolyzed by the enzyme beta-glucosidase, which is present in the gut and liver, releasing cyanide (CN-) and benzaldehyde [12].

Toxicity and Health Consequences

Cyanide is a potent toxin that can cause severe health consequences, including:

1. **Respiratory Failure**: Cyanide binds to cytochrome c oxidase, inhibiting cellular respiration and leading to tissue hypoxia [13].
2. **Cardiac Arrest**: Cyanide can cause cardiac arrhythmias, leading to cardiac arrest [15].
3. **Neurological Symptoms**: Cyanide exposure can cause headache, dizziness, nausea, and vomiting, as well as seizures and coma in severe cases [13].
4. **Death**: Ingestion of large amounts of amygdalin can lead to fatal cyanide poisoning [12].

2.3. Oxalic Acid

Oxalic acid also known as Ethanedioic acid or Oxiric acid is a naturally occurring compound found in various foods, such as spinach, beets, rhubarb, and strawberries [17]. While it is generally considered safe in moderate amounts, excessive consumption of oxalic acid can cause several health issues, including kidney damage, kidney stones, and bladder blockage [18].

Structure and Properties

Oxalic acid is a dicarboxylic acid with the chemical formula $C_2H_2O_4$ [19]. It is a colourless, crystalline solid that is highly soluble in water. Oxalic acid is a strong acid and can react with minerals such as calcium, magnesium, and iron to form insoluble compounds.

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Dietary Sources and Intake

Oxalic acid is found in various foods, including:

- Leafy greens: spinach, kale, collard greens
- Root vegetables: beets, rhubarb, carrots
- Fruits: strawberries, cranberries, raspberries
- Legumes: beans, lentils, peas

The average dietary intake of oxalic acid varies widely depending on the population and their dietary habits. However, a typical Western diet may contain around 200-300 mg of oxalic acid per day. The estimated daily oxalate intake ranges from 150-500 mg, with a mean intake of 200-250 mg. Individual variations in oxalate intake can be significant due to differences in food choices and consumption patterns [17].

Health Risks Associated with Excessive Oxalic Acid Consumption

While moderate amounts of oxalic acid are generally considered safe, excessive consumption can cause several health issues, such as:

1. **Kidney Damage Due to Kidney Stones Formation**: Oxalic acid can increase the risk of kidney damage due to the formation of insoluble calcium oxalate crystals in the kidneys [18].
2. **Mineral Deficiencies**: Oxalic acid can bind to minerals such as calcium, magnesium, and iron, making them unavailable for absorption and potentially leading to mineral deficiencies [20].
3. **Gastrointestinal Issues**: Excessive consumption of oxalic acid can cause gastrointestinal issues such as diarrhea, abdominal pain, and nausea [21].

2.4. Lectins

Lectins are a type of carbohydrate-binding protein found in various plant-based foods, particularly in legumes such as beans, lentils, and peas. While lectins can provide beneficial effects, such as antimicrobial and anti-inflammatory properties, they can also cause adverse health effects, including nausea, vomiting, and diarrhea.

Mechanism of Action

The mechanism of action of lectins involves their ability to bind to specific carbohydrate molecules on the surface of cells, particularly in the gastrointestinal tract. This binding causes a series of events that can lead to toxicity.

1. **Binding to cell surface carbohydrates**: Lectins bind to specific carbohydrate molecules, such as galactose, mannose, and N-acetylglucosamine, on the surface of cells [22].
2. **Activation of cell signaling pathways**: The binding of lectins to cell surface carbohydrates activates various cell signaling pathways, including those involved in inflammation, immune response, and cell death [23].
3. **Disruption of gut epithelial barrier**: Lectins can disrupt the integrity of the gut epithelial barrier, allowing toxins and undigested food particles to pass through the gut wall and into the bloodstream [24].
4. **Induction of apoptosis and inflammation**: Lectins can induce apoptosis (programmed cell death) and inflammation in the gut, leading to symptoms such as nausea, vomiting, and diarrhea [25].

Toxicity

The toxicity of lectins can vary depending on the type and amount consumed. Some of the toxic effects of lectins include:

1. **Gastrointestinal symptoms**: Lectins can cause nausea, vomiting, diarrhea, and abdominal pain [22].
2. **Inflammation and immune response**: Lectins can activate the immune system, leading to inflammation and potentially exacerbating conditions such as irritable bowel syndrome [23].
3. **Interference with nutrient absorption**: Lectins can interfere with the absorption of nutrients, including proteins, carbohydrates, and fats [25].

2.5. Linamarin and Lotaustralin

Cassava (Manihot esculenta), a staple food in many tropical regions, contains naturally occurring cyanogenic glycosides, particularly linamarin and lotaustralin. Linamarin is the main cyanogenic glycoside present in cassava together (93%) while the lotaustralin content is 7% [26].

These compounds can release cyanide, a potent toxin, when ingested [27].

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Mechanisms of Cyanide Toxicity

Acute Cyanide Toxicity:

1. **Inhibition of Cellular Respiration**: Cyanide binds to the iron atom in cytochrome c oxidase (CcOX), a key enzyme in the mitochondrial electron transport chain. This inhibits cellular respiration, preventing cells from producing ATP (adenosine triphosphate), the primary energy currency of the cell [28].
2. **Disruption of Oxidative Phosphorylation**: Cyanide's inhibition of cytochrome c oxidase disrupts oxidative phosphorylation, leading to a decrease in ATP production and an increase in lactate production [29].
3. **Lactic Acidosis**: The decrease in ATP production and the increase in lactate production lead to lactic acidosis, a condition characterized by an excessive accumulation of lactate in the body [30]. High levels of lactate can cause a decrease in blood pH, leading to lactic acidosis. This can result in respiratory distress, cardiac arrhythmias, hypotension, organ failure [31 – 36]. Elevated lactate levels can impair muscle function, leading to muscle fatigue, weakness and cramping [37-38]. High lactate levels can also cause gastrointestinal symptoms such as nausea, vomiting and abdominal pain [39]. Severe lactic acidosis can lead to respiratory failure [40]. This can occur in several ways:

a. **Respiratory Depression**: Severe metabolic acidosis can lead to respiratory depression, characterized by slow and shallow breathing. This can impair the body's ability to remove carbon dioxide, leading to respiratory failure (41).
b. **Compensatory Mechanisms**: In an attempt to compensate for the acidosis, the body may increase respiratory rate and depth. However, this can lead to respiratory muscle fatigue, ultimately resulting in respiratory failure (42).
c. **Pulmonary Edema**: Severe metabolic acidosis can cause pulmonary edema, a condition characterized by fluid accumulation in the lungs. This can impair gas exchange and lead to respiratory failure (43).

   A study published in the Journal of Critical Care found that metabolic acidosis was an independent predictor of respiratory failure in critically ill patients (41).

4. **Neurological Damage**: The brain is particularly vulnerable to cyanide toxicity due to its high energy demands. Prolonged exposure to cyanide can lead to neurological damage, including seizures, coma, and even death [28].

Chronic (Long-term) Effects of Cyanide Exposure

1. **Konzo**: Chronic exposure to cyanide from cassava consumption can lead to konzo, a neurological disorder characterized by spastic paralysis, muscle weakness, and tremors [44].
2. **Neurological Impairment**: Long-term exposure to cyanide can also lead to neurological damage, including cognitive impairment, memory loss, and Parkinson's disease-like symptoms [45].

Factors Affecting Cassava Toxicity:

1. **Variety**: Some cassava varieties contain higher levels of toxic compounds than others [46].
2. **Soil and Climate**: Environmental factors, such as drought or poor soil quality, can increase the concentration of toxic compounds in cassava [47].
3. **Preparation and Processing**: Improper preparation, such as not soaking or cooking cassava thoroughly, can lead to higher toxin levels [27].
4. **Consumption Patterns**: Frequent or excessive consumption of cassava can increase the risk of toxicity [28].

Safe Consumption of Cassava

1. **Proper Preparation**: Soak, cook, or ferment cassava properly to reduce toxin levels [27].
2. **Choose Low-Cyanide Varieties**: Opt for cassava varieties that are known to have lower toxin levels [6].
3. **Consume in Moderation**: Limit cassava consumption to recommended levels to avoid excessive toxin intake [28].
4. **Diversify Diet**: Balance cassava consumption with other nutrient-rich foods to minimize reliance on a single, potentially toxic food source

2.6. Hypoglycin

Ackee, Jamaica's national fruit contains a toxin called hypoglycin [48]. Hypoglycin can cause vomiting, seizures, and even death if ingested in large amounts. The toxin is particularly concentrated in the fruit's arils and seeds [48].

Mechanisms of Action of Hypoglycin

Hypoglycin is a non-proteinogenic amino acid and a potent inhibitor of the enzyme acyl-CoA dehydrogenase, which plays a crucial role in the metabolism of fatty acids (beta-oxidation of fatty acids in the mitochondria) [49].This

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inhibition leads to a decrease in the production of acetyl-CoA, a key intermediate in the metabolism of carbohydrates, fats, and proteins.

Disruption of Energy Metabolism

The inhibition of acyl-CoA dehydrogenase by hypoglycin disrupts energy metabolism, leading to a decrease in the production of ATP [50]. This decrease in ATP production can cause a range of symptoms, including vomiting, seizures, and even death.

Hypoglycemia and Hypoketonemia

Hypoglycin also causes hypoglycemia (low blood sugar) and hypoketonemia (low levels of ketone bodies in the blood) by inhibiting the production of glucose and ketone bodies [50]. This can lead to a range of symptoms such as confusion, dizziness, and loss of consciousness.

2.7. Anthraquinone Glycosides (Rhein and Emodin)

Rhubarb (Rheum rhabarbarum) a perennial plant and a popular ingredient in pies and tarts contains two main toxic compounds:

1. Anthraquinone glycosides particularly rhein and emodin [51].
2. Oxalic acid which are in high concentrations in the leaves [52]. Hence, the stalks are usually consumed as the leaves are considered toxic due to the high oxalate concentrations.

Toxic Effects

Ingestion of rhubarb can cause:

1. Gastrointestinal symptoms: Nausea, vomiting, diarrhea, and abdominal pain [51].
2. Kidney damage: Oxalic acid in rhubarb can increase the risk of kidney stones and eventual kidney damage [52].
3. Cardiovascular effects: High levels of anthraquinone glycosides can cause arrhythmias and other cardiovascular problems [53].

Clinical Manifestations

1. Seizures: High levels of anthraquinone glycosides can cause seizures and other neurological symptoms [51].
2. Coma: In rare cases, rhubarb poisoning can cause coma and even death [53].

Treatment

1. Patients may require hospitalization and supportive care, including hydration and monitoring of vital signs [52].
2. Administration of activated charcoal may help reduce the absorption of toxic compounds [51].

2.8. Phytohemagglutinin (PHA), Phytoalexins and Saponins

Kidney beans (Phaseolus vulgaris) a staple legume in many cuisines worldwide while providing essential nutrients and health benefits, also contain naturally occurring toxic compounds that can pose health risks if not properly prepared or consumed in excess. These toxic compounds found in kidney beans include:

1. **Phytohemagglutinin (PHA)**

   Phytohemagglutinin (PHA) is a glycoprotein that can cause nausea, vomiting, diarrhea, and abdominal pain if ingested in large amounts [54]. PHA can also agglutinate red blood cells, leading to haemolysis [55].

2. **Phytoalexins**

   These are defense compounds produced by the plant in response to stress or infection and they can be toxic to humans and animals if ingested in large amounts. Symptoms of toxicity can include acute and short-term digestive problems and nausea, cognitive disorders like dizziness and confusion, and long-term symptoms like intestinal permeability, chronic inflammation, nutrient deficiencies, pulmonary complications, and death [56].

3. **Saponins**

   These are glycosidic compounds that can cause haemolysis and other adverse effects if ingested in excess [57].

Mechanisms of Toxicity

The toxicity of kidney beans is attributed to the following mechanisms:

1. **Inhibition of protein synthesis**

   PHA can inhibit protein synthesis in the gut, leading to malabsorption and gastrointestinal symptoms [58].

2. **Disruption of cell membranes**

   Saponins and other toxic compounds in kidney beans can disrupt cell membranes, leading to haemolysis and other adverse effects [57].

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Precautions

To minimize the risks associated with kidney bean toxicity, the following precautions should be observed:

1. **Proper cooking**: Kidney beans should be cooked thoroughly to inactivate PHA and other toxic compounds [54].
2. **Soaking and boiling**: Soaking and boiling kidney beans can reduce PHA content by up to 90% [55].
3. Kidney beans should be consumed in moderation, as excessive consumption can lead to adverse effects [58].

3. ANIMAL-BASED FOOD POISONS

3.1. Tetrodotoxin

Tetrodotoxin (TTX) is a naturally occurring compound that has been extensively studied due to its unique mechanism of action and potential applications in medicine and research. It is a powerful neurotoxin found in various marine animals, including pufferfish (Takifugu rubripes) where it is abundant in the ovaries, liver, and skin, blue-ringed octopuses (Hapalochlaena maculosa) (abundant in the salivary glands), and other marine animals, including sea slugs, starfish, and sea urchins [59].

Properties

TTX is a non-proteinaceous, low-molecular-weight compound with a molecular formula of $C_{11}H_{17}N_3O_8$ [59]. It is a highly potent toxin, with an LD50 (the dose required to kill 50% of test subjects) of approximately 8 µg/kg in mice [60].

Mechanism of Action

TTX acts as a potent blocker of voltage-gated sodium channels (Nav channels) in neurons, preventing the influx of sodium ions and subsequent depolarization of the cell membrane [61]. This blockade leads to a rapid onset of paralysis, respiratory failure, and eventually death.

Toxicity and Symptoms

Ingestion of TTX can cause a range of symptoms, including:

1. Paralysis: TTX can cause rapid onset of muscle paralysis, resulting to respiratory failure and eventual death [60].

2. Respiratory Failure: Blockade of Nav channels in the diaphragm and other respiratory muscles can lead to respiratory failure [61].

3. Cardiovascular Collapse: TTX can also cause cardiovascular collapse due to its effects on the heart and blood vessels. Cardiovascular reactions of intoxicated animals are a reduced blood pressure, bradycardia, and in few cases, reduced ventricular force and stroke volume [60].

3.2. Ciguatoxin

Ciguatoxin (CTX) is a potent neurotoxin found in certain species of fish, including moray eels (Muraenidae) and barracudas (Sphyraenidae)[62]. Primarily, CTX is produced by dinoflagellates, such as Gambierdiscus toxicus, which are ingested by herbivorous fish [63]. The toxin is then accumulated in the fish's flesh and can cause severe food poisoning in humans who consume contaminated fish.

Structure and Properties

CTX is a polyether compound with a molecular formula of $C_60H_{86}O_{19}$ [64]. It is a highly potent toxin, with an LD50 of approximately 0.35 µg/kg in mice [62].

Mechanism of Action

CTX acts on the voltage-gated sodium channels (Na_v channels) in neurons, causing a prolonged opening of the channels and leading to an influx of sodium ions [63]. This results in a depolarization of the cell membrane, leading to the activation of pain receptors and the transmission of pain signals to the brain.

Toxicity and Symptoms

1. **Gastrointestinal Symptoms**: Nausea, vomiting, diarrhea, and abdominal pain are common symptoms of ciguatera fish poisoning [63].
2. **Neurological Symptoms**: CTX can cause numbness, tingling, and paresthesia (abnormal sensations) in the mouth, throat, and extremities [62].
3. **Cardiovascular Symptoms**: In severe cases, CTX can cause bradycardia (slow heart rate), hypotension (low blood pressure), and cardiac arrhythmias [63].

4. DIAGNOSIS AND TREATMENT

Diagnosis of ciguatera fish poisoning is typically based on the presence of characteristic symptoms and a history of consuming fish from a tropical region [62]. Treatment is primarily supportive, with a focus on relieving symptoms and managing pain. In severe cases, hospitalization may be necessary to monitor and treat cardiovascular and neurological symptoms.

Prevention

Prevention of ciguatera fish poisoning involves avoiding consumption of fish from tropical regions, particularly those known to contain CTX [63]. Fishers and seafood suppliers should also take steps to minimize the risk of CTX contamination, such as avoiding areas with high levels of dinoflagellates.

Fungal food poisons

Edible fungi, including mushrooms, have been consumed for centuries and are considered a delicacy in many cultures. However, some edible fungi may contain toxic compounds that can cause adverse health effects (64). The toxicology of edible mushrooms is a complex field that requires understanding of the various toxins present in mushrooms, their mechanisms of action, and the clinical manifestations of mushroom poisoning.

Toxins in Mushrooms

Mushrooms contain a wide range of toxins, including amatoxins, phallotoxins, and gyromitrin (65). Amatoxins are the most toxic and are found in species of the genus Amanita, including the notoriously poisonous death cap mushroom (Amanita phalloides). Amatoxins can cause liver and kidney failure (66). Phallotoxins are also found in Amanita species and can cause severe gastrointestinal symptoms (67). Gyromitrin is a toxin found in species of the genus Gyromitra and can cause neurological symptoms (68).

Mechanisms of Action

The toxins in mushrooms can cause a range of effects on the human body. Amatoxins, for example, inhibit RNA polymerase II, leading to cell death and organ failure (65). Phallotoxins cause similar damage to the liver, while gyromitrin can cause neurological symptoms by inhibiting the enzyme monoamine oxidase (68).

Clinical Manifestations

The clinical manifestations of mushroom poisoning can vary depending on the type and amount of toxin ingested (69). Gastrointestinal symptoms such as nausea, vomiting, and diarrhea are common, as well as neurological symptoms such as confusion, tremors, and seizures.

Treatment

Treatment of mushroom poisoning depends on the specific toxin involved and the severity of symptoms (70). Supportive care is essential, including measures to manage fluid and electrolyte imbalances, maintain cardiovascular stability, and provide respiratory support. Specific antidotes are limited, and early diagnosis is critical to guide appropriate medical interventions.

Prevention

Mushroom toxicity is a serious health risk that can be avoided by being aware of the toxic species, taking precautions when foraging or consuming mushrooms, and seeking medical attention immediately if symptoms occur. Prevention is the best way to avoid mushroom poisoning (64). This includes correctly identifying mushrooms before consumption, avoiding consumption of mushrooms that are past their prime or have an off smell, and cooking mushrooms thoroughly before consumption.

5. CONCLUSION

While certain food items contain toxic compounds, they can still be safely consumed if properly prepared and cooked. It is essential to be aware of the potential health risks associated with these foods and take necessary precautions to minimize exposure to their toxic compounds. Further research is needed to fully understand the mechanisms of toxicity and to develop effective strategies for mitigating the risks associated with edible toxins.

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