Chapter 4

CHEMISTRY OF TOXICANTS
(a.) Chemistry of Toxicants

Glyphosate

Glyphosate is a systemic herbicide that can control most annual and perennial plants. It controls weeds by inhibiting the synthesis of aromatic amino acids necessary for protein formation in susceptible plants. This compound was first synthesized in 1955 by E. Beriger of CIBA limited Basle, Switzerland. Glyphosate is strongly adsorbed to soil particles, which prevents it from excessive leaching or from being taken-up from the soil by non-target plants. It is degraded primarily by microbial metabolism, but strong adsorption to soil can inhibit microbial metabolism and slow degradation. Photo- and chemical degradation are not significant in the dissipation of Glyphosate from soils. The half-life of Glyphosate ranges from several weeks to years, but averages two months. In water, Glyphosate is rapidly dissipated through adsorption to suspended and bottom sediments, and has a half-life of 12 days to ten weeks. Glyphosate by itself is of relatively higher toxicity to fishes and other aquatic organisms.

Glyphosate, also known by the trade names Roundup for agricultural use, is a broad-spectrum translocated herbicide, used primarily in agricultural applications and for vegetation control in non-crop areas. It is used for aquatic weed control in fish-ponds, lakes, canals, slows running water, etc. (USDA 1984). Formulations of glyphosate include round up have been extensively investigated for their potential to produce adverse effects in non-target organisms. Glyphosate is soluble in water, and tends to bind tightly to sediment, suspended particulates, organic matter and soil, becoming essentially
unavailable to plants or other aquatic organisms. Since glyphosate developed in the 1970’s, there have been much documented cases of adverse effects on fishes and aquatic invertebrates associated its use for the control of aquatic weeds (Giesy et al. 2000).

Glyphosate is perhaps the most important herbicide ever developed. Literature of toxicological and ecotoxicological properties of glyphosate is extremely sparse, considering its importance as herbicide. Generally, glyphosate is slightly toxic to plant, but it may have an impact on the aquatic environment and also on the other aquatic organisms especially on fishes life. Due to this, its toxicity investigation is very important. The study of lethal and sublethal effects is of special importance for toxicological evaluation of compound. It occurs in two isomeric forms α and β in the ratio of 3:7. Its insecticidal property is mostly due to α isomer. It is used as systemic and also as stomach poison.

IUPAC name: N-(phosphonomethyl) glycine
Molecular formula: C₃H₈NO₅P
Structural formula:

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\begin{center}
\begin{tikzpicture}
  \node (A) at (0,0) {O};
  \node (B) at (1,0) {O};
  \node (C) at (1,1) {\text{HO}--\text{C}--\text{CH}_{2}--\text{N}--\text{CH}_{2}--\text{P}--\text{OH}};
  \node (D) at (2,1) {\text{OH}};
  \node (E) at (2,0) {\text{H}};
\end{tikzpicture}
\end{center}
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Its trade names are mostly Roundup® Armada; Kleenup® (isopropyl ammonium); Spasor®; Squadron etc.
Physical Properties: -

Pure Glyphosate is a colourless, odourless, crystalline solid with a melting point of 185 °C and decomposes at 187 °C producing toxic fumes including nitrogen oxides and phosphorus oxides. Solutions of the Glyphosate salts are corrosive to iron or galvanized steel. Pure Glyphosate is slightly soluble in water (12 g/litre at 25 °C), and is practically insoluble in most organic solvents. The alkali-metal and amine salts are readily soluble in water.

Mode of action: -

Glyphosate penetrates the plant leaf cuticle shortly after contact and begins a cell by cell migration to the phloem, from which it is transported throughout the plants. The herbicidal action usually occurs within 7 days and up to 30 for woody plants (McLaren and Hart 1995; Monsanto, 1985) Glyphosate's primary herbicidal mode of action is to block the synthesis of aromatic amino acids and the metabolism of phenolic compounds by disrupting the plant's shikimic acid metabolic pathway, leading to the inability of the plant to synthesize protein and produce new plant tissue. This is the only herbicide known to interfere with this particular pathway (McLaren and Hart 1995). A secondary mode of action affects the photosynthetic process, respiration and synthesis of nucleic acids by interacting with a complex series of enzymes which control synthesis of important molecules such as chlorophyll.

Phosphamidon

Phosphamidon is one of the versatile oreganophosphorus
pesticide extensively used an agricultural operation all over the world since 1956. Phosphamidon was first synthesized in 1955 by E. Benger of CIBA limited, Basle, Switzerland. Technical grade material is dark brown and commercial product is bright violet due to addition of a dye. It occurs in two isomeric forms a and b in the ratio of 3:7. Its insecticidal property is mostly due to b-isomer. It is a systemic poison and also acts as stomach poison. It has relatively low contact action. It is commonly used against insects possessing piercing and sucking, and chewing mouth-parts. It is widely used for the control of yellow borer and other sucking pests. There are mainly three routes of entry into water sources. One is from industrial waste or effluent discharged directly into water. A second is by seepage from buried toxic wastes into water supplies. Thirdly, contamination of running water directly or from run-off during spraying operations can occur. Mostly due to rainfall and drainage the pesticide residue reach the water bodies of many non target organism causing toxic effect on fishes.

**Primary Use: Insecticide**

<table>
<thead>
<tr>
<th>IUPAC</th>
<th>2-chloro-2-diethylcarbamoyl-1-methylvinyl dimethyl phosphate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Common name</td>
<td>Phosphamidon</td>
</tr>
<tr>
<td>Molecular formula</td>
<td>C₁₀H₁₉ClNO₃P</td>
</tr>
<tr>
<td>Structural formula</td>
<td>![Structural formula image]</td>
</tr>
</tbody>
</table>

Trade names : Phosphamidon, Famfos, Dimecron etc.

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*Studies on the effect of some pesticides on blood parameters in fresh water fishes of Bundelkhand region* 43
Physical properties: -

Phosphamidon is a pale yellow to colorless oily liquid with a faint odour. It has a boiling point of 162°C at 1.5 mmHg. Phosphamidon exists as a mixture of 70% cis-isomer and 30% trans-isomer and is corrosive to iron, tinplate and aluminum and it is miscible with water. It is soluble in aromatic hydrocarbons but insoluble in non polar aliphatic hydrocarbons.

Mode of action: -

Phosphamidon poisoning inhibits the activity of superoxide dismutase and increases the lipid peroxidation in several regions in the central nervous system (Brain O’ 1987). Delayed neuropathy is initiated by an attack on a nervous tissue system. The target has esterase activity and is called neuropathy target esterase (NTE). The disorder develops not because of loss of esterase activity, but because of a change brought about in the protein molecule that results from the process of ageing of inhibited NTE. The catalytic activity of NTE appears in the nervous tissue, even during the period of development of neuropathy (Edward et al., 1991). Investigations conducted by Extoxnet (1985); Matsumura et al., (1985); Rend G.M. and S.R., Petrocelli (1985) in fishes showed that phosphamidon is potentially neurotoxic because of its ability to inhibit brain NTE activity.

Organophosphorus pesticides exert their acute effects by inhibiting acetyl cholinesterase in the nervous system with subsequent accumulation of toxic levels of acetylcholine. They may also inhibit butyl cholinesterase as well as other esterase. The function of butyl
cholinesterase is unknown, but its inhibition can provide an indication of exposure to an organophosphate.

**Metasystox**

This pesticide was evaluated in 1965 by Joint Meeting of the FAO Committee on Pesticides in Agriculture and the WHO Expert Committee on Pesticide Residues (FAO/WHO, 1965) under the name of Oxydemeton-methyl. (ODM) Oxydemeton-methyl, S-[2-(ethylsulfinyl)ethyl], which is most commonly sold under the trade name of Metasystox R, is a systemic contact insecticide with approximately 120 tones applied in the states of India during 2004. Approximately 50% of the total was applied to broccoli, and 20% was applied to cauliflower during the year. It was assumed that ODM would be detected in air samples near application sites due to its relatively low vapor pressure (3.80 k Pa), but would not be found long distances from application sites. Dioxydemeton-methyl is a potential transformation product of ODM in air samples.

**Primary Use**: Insecticide, Oxydemeton-methyl

**IUPAC name**: O,O-dimethyl-S-2-(ethyl-sulfinyl)-ethyl / phosph orothioate

**Molecular formula**: \( C_9H_{15}O_4PS_2 \)

**Structural formula**: 

```
          O       O
CH_3O   "     "
     "     "
   \"     "
P-S-CH_2-CH_2-S-C_2H_5
/     
CH_3O
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*Studies on the effect of some pesticides on blood parameters in fresh water fishes of Bundelkhand region* 45
Tread name : Metasystox

Physical properties

The molecular formula of metasystox is C₅H₁₂O₄PS₂ and its molecular weight is 246.3. It is clear amber-colored liquid (pure compound). The melting point is 10 °C (pure compound), boiling point is 106 °C/0.01 mm Hg (pure compound) and it is miscible in water.

Mode of action

Oxydemeton-methyl (Metasystox R) is an organophosphate pesticide. The target organ is nervous system and inhibiting cholinesterase enzyme. It binds to the enzyme that is normally responsible for breaking down Ach. When an insect has been poisoned by a cholinesterase inhibitor, the cholinesterase is not available to help breaking down the ACh, and the neurotransmitters continue to cause the neuron to "fire" or send its electric charge. This causes over stimulation of the nervous system, and the insect die.

Imidacloprid

Imidacloprid was discovered in 1984 at Nihon Bayer Agrochem in Japan by screening novel synthetic compounds for a high affinity to the insect nicotinic AChRs receptors, but with low toxicity to vertebrate species (Kagabu 1997). Its molecule includes the insecticidal N-(3pyridinyl) methyl group of nicotine and a nitroimine moiety, Because of their structural similarity to nicotine, imidacloprid and related insecticides (acetamiprid, thiacloprid, thiamethoxam and nitenpyram) were termed neonicotinoids (Tomizawa and Yamamoto, 1993). Imidacloprid was first registered in U.S. in 1994 as a pesticide
and developed for commercial use. Its major manufacturer is Bayer Corporation that markets imidacloprid products with the brand names Admire, Advantage, Confidor, Gaucho, Supreme etc.

Imidacloprid is a systemic chloronicotinyl insecticide that enters the target pest via ingestion or direct contact. In the environment, the principal routes of dissipation for imidacloprid are aqueous photolysis, microbial degradation and uptake by plants. The major degradation product of imidacloprid in the environment is desnitro-imidacloprid. Imidacloprid is used for controlling sucking insects, soil insects, termites, and some chewing insects.

Primary Use : Insecticide

IUPAC name : 1-(6-chloro-3-pyridylmethyl)-N-nitroimidazolidin-2-ylideneamine) is a synthetic active ingredient used in various insecticide

Structure formula

\[
\text{C}_9\text{H}_{10}\text{ClN}_{5}\text{O}_2
\]

Physical properties:-

It is a colourless crystalline solid, has the molecular formula
C₉H₁₀ClN₅O₂ and a molecular weight of 255.7 (Tomlin 2000). Imidacloprid is soluble in water, relatively non-volatile.

**Mode of action:**

Imidacloprid, and other insecticides in the nicotinoid chemical family, are “similar to and modeled after the natural nicotine [a tobacco toxin]. Because of their molecular shape, size, and charge, nicotine and nicotinoids fit into receptor molecules in the nervous system that normally receive the molecule acetylcholine. Acetylcholine carries nerve impulses from one nerve cell to another or from a nerve cell to the tissue. Imidacloprid and other nicotinoids irreversibly block acetylcholine receptors. It is used to treat seeds, soil, crops and structures, and is a flea control treatment on domestic pets (Meister, 2000). The toxicity of imidacloprid is based on interference of the neurotransmission in the nicotinic cholinergic nervous system. Imidacloprid binds to the nicotinic acetylcholine receptor (nAChR) at the neuronal and neuromuscular junctions in insects.

**(b.) Physical properties of water:**

The chlorine free tap water was used through out the course of the experiment. The physiological characters of water sample like as the temperature of the test medium, pH, dissolve oxygen, alkinity, hardness and specific conductivity were tested in the Zonal laboratory U.P. Jal nigam Babina. Analyses of water have done during all three seasons.
### Table: Physical properties of water

<table>
<thead>
<tr>
<th>S. No.</th>
<th>Water Parameters</th>
<th>Rainy</th>
<th>Summer</th>
<th>Winter</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Dissolve oxygen mg/liter</td>
<td>7.6</td>
<td>6.2</td>
<td>8.9</td>
</tr>
<tr>
<td>2</td>
<td>Alkinity as CaCO3 to methyl orange</td>
<td>326</td>
<td>320</td>
<td>308</td>
</tr>
<tr>
<td>3</td>
<td>Hardness as Ca</td>
<td>120</td>
<td>111.7</td>
<td>128.7</td>
</tr>
<tr>
<td>4</td>
<td>Specific conductivity micro mho</td>
<td>792</td>
<td>765</td>
<td>782</td>
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<tr>
<td>5</td>
<td>Water temperature °C</td>
<td>27</td>
<td>42</td>
<td>14</td>
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<tr>
<td>6</td>
<td>pH value</td>
<td>7.4</td>
<td>6.8</td>
<td>7.2</td>
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</tbody>
</table>