Lecture

Inhibition of Photosynthesis
Inhibition at Photosystem I

1. General Information

The popular misconception is that susceptible plants treated with these herbicides "starve to death" because they can no longer photosynthesize. In actuality, the plants die long before the food reserves are depleted.

The photosynthetic inhibitors can be divided into two distinct groups, the inhibitors of Photosystem I and inhibitors of Photosystem II.

Both of these groups work in the energy production step of photosynthesis, or the light reactions. Light is required as well as photosynthesis for these herbicides to kill susceptible plants.

Herbicides that inhibit Photosystem I are considered to be contact herbicides and are often referred to as membrane disruptors. The end result is that cell membranes are rapidly destroyed resulting in leakage of cell contents into the intercellular spaces.

These herbicides act as "electron interceptors" or "electron thieves" within Photosystem I of the light reaction of photosynthesis. They divert electrons from the normal electron transport sequence necessary in Photosystem I. This in turn inhibits photosynthesis. The membrane disruption occurs as a result of secondary responses.

Herbicides that inhibit Photosystem I are represented by only one family, the bipyridyliums. See chemical structure shown under herbicide families.

These molecules are cationic (positively charged) and are therefore highly water soluble. Their cationic properties also make them highly adsorbed to soil colloids resulting in no soil activity.

2. Mode of Action

See Figure 7.1 (The electron transport chain in photosynthesis and the sites of action of herbicides that interfere with electron transfer in this chain (Q = electron acceptor; PQ = plastoquinone). Review Photosystem I and II.

The herbicide moves through the cuticle into the cell and into the chloroplast where photosystem I is occurring. The bipyridyliums inhibit photosystem I by intercepting electrons (electron diversion) from one of the iron-sulfur protein electron acceptors in the normal electron transport sequence. The specific mechanism of action is as follows:

Addition of the electron changes the divalent paraquat/diquat cation (+2 charge) to an unstable "free radical" (the cation is reduced). This free radical is not toxic to the plant.

Since the molecule desires to become stable (i.e. the normal cationic state), the free radical is re-oxidized (autooxidation) in the presence of oxygen and water to yield the original ion (see top left hand corner of Figure 7.1). During this oxidation process, electrons (e^-) are transferred to molecular oxygen (oxygen is reduced) and superoxide anion radicals (O_2^-) are produced.

Superoxide radicals are then enzymatically altered (superoxide dismutase / SOD enzyme) to form hydrogen peroxide (H_2O_2). Superoxide radicals and hydrogen peroxide react together to produce hydroxyl radicals (•OH).

These unstable products (in particular hydroxyl radical) interact with fatty acids in membranes (lipid peroxidation) causing membrane disruption/leakage (membrane integrity destroyed).

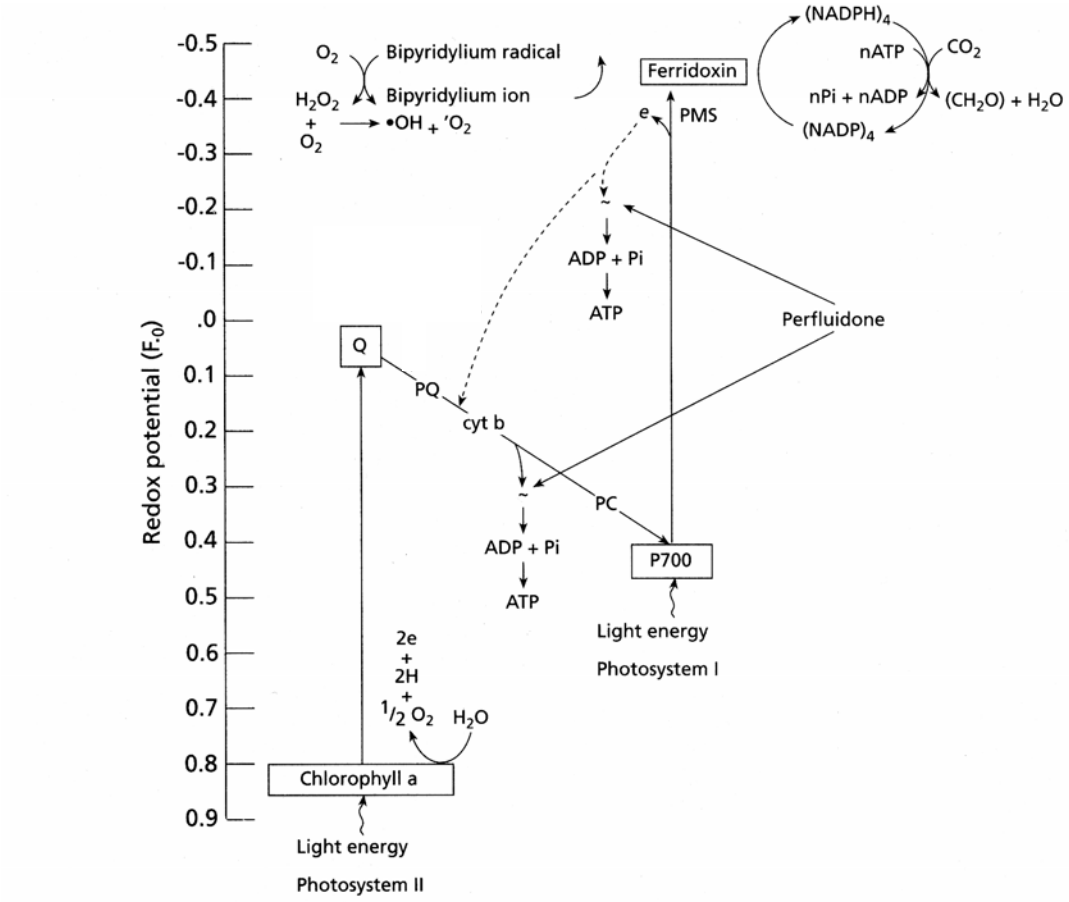


FIGURE 7.1 ■ The electron transport chain in photosynthesis and the sites of action of herbicides that interfere with electron transfer in this chain (Q = electron acceptor; PG = plastoquinone).

SOURCE: F. M. Ashton and A. S. Crafts. 1981. *Mode of action of herbicides,* 2nd ed., p. 55. New York: John Wiley & Sons, Inc. Reprinted with permission of John Wiley & Sons, Inc.

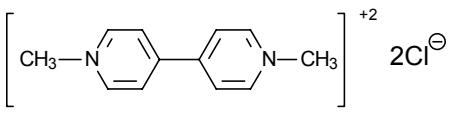
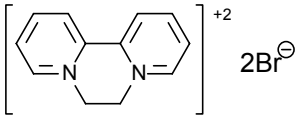
3. Site of Action

The bipyridyliums inhibit Photosystem I by intercepting electrons from one of the iron-sulfur protein electron acceptors, most likely F_AF_B. F_AF_B is located on the stroma side of the thylakoid membrane, the site of action.

4. Symptoms

- contact type injury – herbicide is not extensively translocated and injury occurs on plant tissue contacted
- injury consists of a water soaking appearance within minutes followed by chlorosis, wilting, and desiccation within hours after application in full sunlight
- translocation can be increased in absence of light – this is why better activity can be seen following late evening application
- complete foliar necrosis occurs within 1-3 days

5. Herbicide Family

Bipyridyliums	
Examples	 paraquat (Gramoxone Extra/Starfire)  diquat (Diquat/Weedtrine-D)
	two pyridine rings (6 member ring consisiting of 5-C's and 1-N); this makes the herbicide a heterocyclic (more than one type of atom in the ring); for paraquat, the N is in the "para" position; notice that the molecules are cationic (+ charged) which affects their reaction with soil and their subsequent interaction with other compounds in photosystem I; they are formulated as salts (dichloride or dibromide)
Metabolism	plant: not metabolized in higher plants soil: half-life of 1,000 days, residues tightly adsorbed and biologically unavailable in soil (both herbicides)
Absorption & Translocation	absorption by leaves, no root uptake; translocation limited to localized transport only in apoplast; if applied under low light conditions, localized transport is increased and in some cases weed control can be enhanced
Selectivity	Non selective, but effective only on herbaceous plants; grass activity is reduced when plants are large

Herbicide Use	paraquat controls grass and broadleaf weeds; used as a preplant burndown in numerous crops (stale seedbed/no-till/reduced till), postemergence directed for general weed control, and as a desiccant/harvest aid in numerous crops; used for marijuana control in Mexico several years ago highly toxic to humans oral LD_{50} of 40-150 mg/kg; emetic and stench added to formulation to prevent ingestion; soil as antidote; used in past to commit suicide; pulmonary fibrosis fb liver/kidney damage; off target drift problems due to physical drift not volatility; Restricted Use Pesticide (EPA concern that a herbicide can cause significant injury to humans and/or the environment); past problems with grower error resulting in contamination of water supply in New Roads, LA) diquat applied POST (spray to wet) to control cattails; applied to ponds, lakes, drainage ditches for algae control, submersed aquatic weeds such as bladderwort, coontail, and Elodea, and floating aquatics such as pennywort, salvinia, and water hyacinth

6. General Comments

Both paraquat and diquat were first synthesized in 1955 by Dyestuffs Division of ICI, Ltd. They were initially used in 1959 as growth regulators, and later as herbicides. A nonionic surfactant or oil adjuvant is required for their activity.

Paraquat is sometimes referred to as methyl viologen in the literature and the chemical family is sometimes called the quaternary ammonium herbicides.

7. References

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