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Mode of Action of commonly used Herbicides and their Impact on Crops and the Environment

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A B S T R A C T

Commercial herbicides exhibit many different mechanisms of action. Several enzymes involved in biosynthesis of amino acids are sites of action for herbicides. Many different herbicide classes inhibit photosynthesis by binding to the quinone-binding protein, D-1, to prevent photosynthetic electron transfer. Modern herbicides are often synthetic mimics of natural plant hormones which interfere with growth of the target plants. At low doses, it promotes plant growth while at high doses it drives plant overgrowth causing stunting and twisting of stems, and general abnormal growth in plants. Farmers quickly adopted glyphosate for agricultural weed control after the development of glyphosate resistant crops that kills weeds without killing the crops. Some herbicides are known to have more than one site of action. Virtually all knowledge of herbicide structure-activity relationships is semiempirical. In addition to site of action structure-activity relationships, herbicide structure and chemical properties also strongly influence absorption, translocation, bioactivation, and environmental stability. The use of adjuvants may enhance the activity of herbicides.

Introduction

Weed management will be of crucial importance, given that crop yield losses caused by weeds (about 32%) are higher than those caused by either pests (18%) or pathogens (15%) (Oerkeand Dehne, 2014). Herbicides are the weed killers that prevent plant growth and killing them outright, they are broad spectrum as they can kill wide range of weeds without affecting many crops. Weeds germinating in the top layers are killed due to incidental absorption of herbicides.eg. triazines, ureas and anilide (Baisha *et al.*, 2017). Herbicides kill plants by disrupting an essential physiological process. This is

accomplished by the herbicide specifically binding to a single protein for many herbicides. The target protein is referred to as the herbicide "site of action." Herbicides in the same family generally have the same site of action (Rana, 2016).

Absorption of applied herbicides occurs through shoot and root tissue. To be effective in killing weeds, herbicides applied postemergence must move through the leaf surface to the living parts (symplast) of plant cells (Monaco *et al.,* 2002).

Use of adjuvants, other than as formulation agents, is primarily restricted to post emergence herbicide applications (Kirkwood, 1994).

Herbicide MOAs can be classified by the plant process affected, e.g. photosynthesis, cell division or specific enzyme targets. The most commonly used herbicides that are specifically used in agricultural sectors are: 2,4-D (Growth regulator), Atrazine (photosystem II inhibitor), Glyphosate (Amino acid inhibitor) and Clomazone (Carotenoid biosynthesis inhibitor).

Growth regulators

2,4-D [(2,4-dichlorophenoxy) acetic acid] is a selective herbicide that kills dicots without affecting monocots and mimics natural auxin at the molecular level. Physiological responses of dicots sensitive to auxinic herbicides include abnormal growth, senescence, and plant death (Yaling Song, 2013) (Fig. 1).

The difference in vascular tissue structure between dicots and monocots may contribute to the selectivity of auxinic herbicides. In monocot stems, the vascular tissues are scattered in bundles, and lack a vascular cambium; in dicot stems, the vascular tissues are formed in rings and possess a cambium (Wright *et al.,* 2010). At low doses, it promotes plant growth while at high doses it drives plant overgrowth, including cupping and stunting of leaves, brittleness, stunting and twisting of stems, and general abnormal growth (Grossmann, 2009) (Fig. 2).

At low concentrations of auxin, Aux/IAA binds to ARF, thus repressing the expression of auxin inducible gene; at high auxin concentration, auxin serves as "molecular glue" that brings Aux/IAA protein to F-box protein TIR1 and mediates the degradation of Aux/IAA proteins. Thereby ARF is alleviated from Aux/IAA allowing the homo-dimerization of ARFs, and binding to AuxREs, and the subsequent activation of auxin response genes (Tan *et al.,* 2007). Moreover, 2,4-D is also recognized by AUXIN BINDING PROTEIN1 (ABP1), which acts as a PM auxin receptor, suggesting there are multiple pathways to sensor 2,4-D (Sauer and Kleine-Vehn, 2011; Simon and Petrasek, 2011).

In rice, over expression of two *Aux/IAAs* (*OsIAA1* and *OsIAA4*) exhibits significant resistance to 2,4-D (Song and Xu, 2013).

Low doses of 2,4-D induced a strong defensive reaction upstream of the jasmonic acid and ethylene pathways, and significantly increased trypsin proteinase inhibitor activity and volatile production (Xin *et al.,* 2012).

Mode of action of photosynthesis inhibitors

Light energy from the sun is utilized by the light-harvesting pigments (chlorophylls and carotenoids) of green plants to produce reducing power and O2. Initially, the light energy that is captured by pigments catalyzes reactions resulting in the release of an electron and O2 from water. Second, the captured light energy excites the available electron and, through a series of interactions with

specialized light-harvesting pigments, produces the reducing power necessary for use in plant growth in PS I and the dark reactions of photosynthesis. The actual process is shown in Figure 3. The excited electron is transferred from P680 to pheophytin and then to a plastoquinone molecule QA. QA passes two electrons (one at a time) to QB (a protein-bound plastoquinone). Once two electrons are passed from QA to QB, the fully reduced QB molecule becomes protonated (two hydrogen ions are added from the stroma) to form a bound plastohydroquinone (PQH2) molecule. PQH2 has a lowered binding affinity for its protein binding site, so it is easily displaced by an oxidized QB (PQ), and the process of PS II is repeated. PQH2 can now transfer its electrons to the cytochrome b6f complex, and eventually the electrons are transferred to PS I via plastocyanin. Herbicides that inhibit photosystem II bind to a protein on the binding niche for QB, called the D-1 protein.

These herbicides compete with QB, for the binding niche in the D-1 protein. This competition can lead to displacement of the QB and stop electron flow through PS II so that no reduced QBH2 (PQH2) forms and therefore no reducing power is generated in photosynthesis (Tikhonov, 2014)

Glyphosate is one of the few herbicides that have been shown to cross the plasma membrane using a carrier protein. Reports of early mechanism-of-action research with glyphosate indicated that levels of the aromatic amino acids phenylalanine and tyrosine were reduced in treated tissue.

A reduction in amino acids can reduce protein synthesis and subsequently cause an inhibition of plant growth (Monaco *et al.*, 2002)**.** Shikimate builds up in glyphosate-treated plants because S-3-P cannot be converted to EPSP, and because S-3-P is unstable, it is rapidly converted to the more stable shikimate, which accumulates (Fig. 4).

The mechanism for generating glyphosate-resistant crops being explored is glyphosate degradation. An amine oxidase enzyme [termed glyphosate oxidoreductase (GOX) by Monsanto] that converts glyphosate to aminomethyl phosphonate + glyoxylate has been isolated from bacteria, and this gene is then coupled to the resistant CP4 EPSPS gene for insertion into plants (Zhou *et al.*, 1995).

Glyphosate-resistant sugar beet currently in development contains both the GOX gene and the resistant CP4 EPSPS gene (Mannerlof *et al.*, 1997).

Clomazone

Clomazone is used for preemergence weed control in soybeans, cotton, tobacco, pumpkins, peppers, and squash.

Clomazone is relatively immobile in the soil, and the principal path of breakdown is microbial decomposition. Its degradation is more rapid in sandy loam soils than in silt loam and clay loam soils. Degradation appears to proceed via binding to the soil matrix and mineralization to carbon dioxide, and its half-life ranges from 24 to 80 days, depending on microbiological activity and environmental conditions. The most striking symptom resulting from treating plants with herbicides that inhibit carotenoid biosynthesis is the totally white foliage which is sometimes termed “albino growth.” The white foliage is the result of a primary inhibition of carotenoid biosynthesis coupled with a secondary destruction (photooxidation) of chlorophyll as it is formed and, to some extent, an inhibition of chlorophyll biosynthesis. These herbicides are also called “bleaching herbicides” or “bleachers.”

Fig.1 A proposed model of the molecular mechanism on 2,4-D works as herbicides

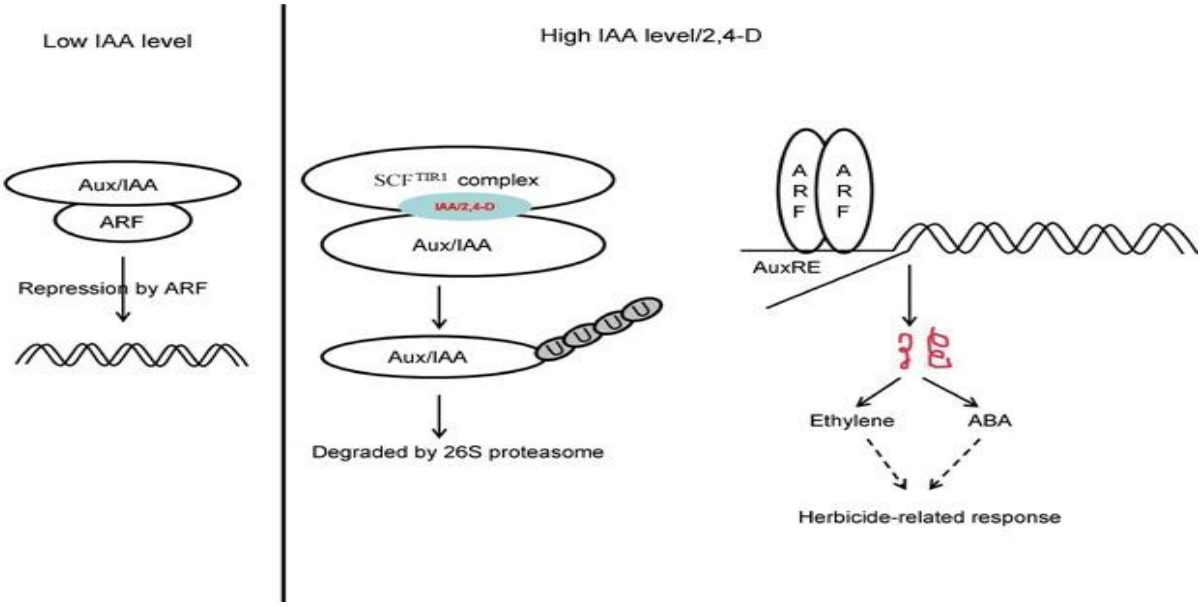


Fig.2 Injury symptoms of 2,4-D(Stem twisting and epinasty)

Fig.3 Process of electron transfer in PS II and the action of atrazine herbicide

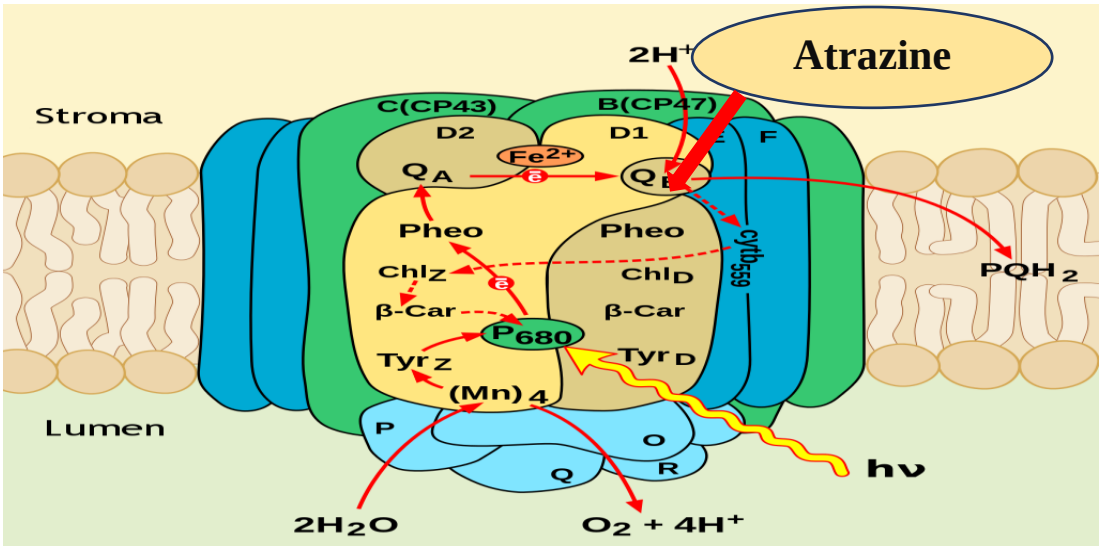
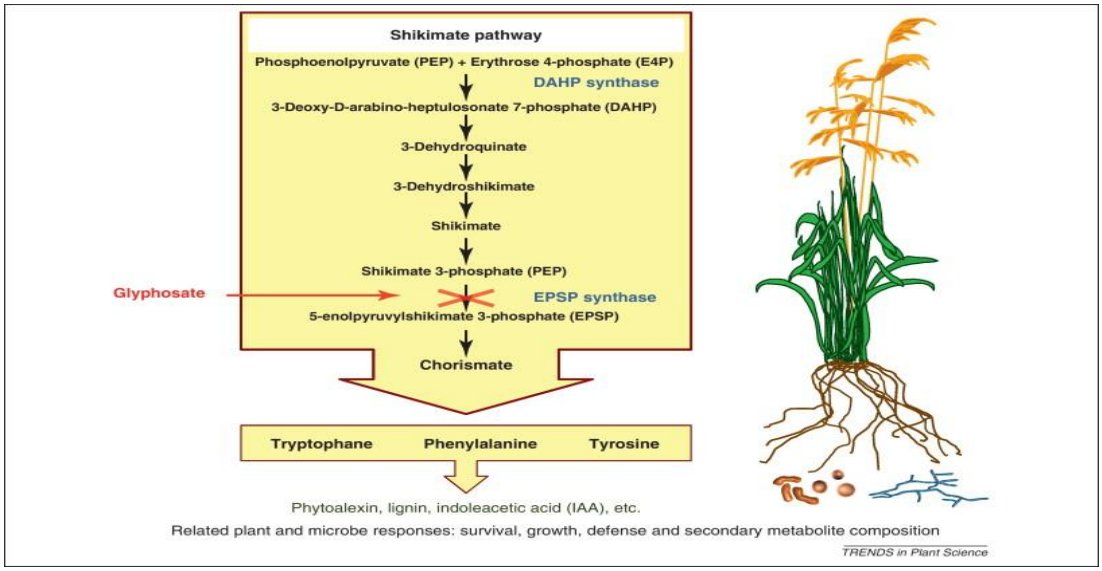


Fig.4 Shikimic acid pathway and the action of glyphosate



Future perspective

Herbicides are a critical component to managing undesirable species in natural areas. Selective herbicides are proved beneficial to plants as they do not kill the desired crops such as glyphosate and 2,4-D and control a vast range of annual and perennial weeds. Soil type and pre-emergent herbicides has better application than foliar sprayed as they translocated from the xylem to other parts of weeds and killing them. Inhibitors of electron transport from PSII block photophosphorylation and starve the cell of the energy normally produced by photosynthesis in grasses and broad weeds. Clomazone is highly soluble in water, but it has a moderate tendency to adsorb to soil particles. It therefore has a low to moderate potential to contaminate

groundwater. It is less toxic to human as compared to other herbicides. Use of GM crops through genetic engineering gives better tolerance to crops from weeds. Development of new herbicide mode of action is critically important for weed management. The auxinic herbicides are very important agrochemicals for selective weed management in grass and cereal crops and are generally phytotoxic to broadleaf plants, causing little or no damage to monocots.

Further study on the auxin-signaling transduction pathway will help elucidate the complete molecular mode action of 2,4-D. Unraveling the molecular mechanism of 2,4-D will not only contribute to research on how hormones work at a molecular level, but will also provide basic information for better use of this herbicide in agriculture.

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