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Explore the Biochemical Mechanisms of Herbicides And Their Impact on Weed Physiology In Potato *(Solanumtuberosum L)*

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ABSTRACT

Potato *(Solanumtuberosum L.)* is an important food crop cultivated globally, but its yield potential is severely affected by weed infestation, particularly during the early stages of crop establishment. Chemical weed control through herbicides remains one of the most efficient and cost-effective approaches in potato production systems. Herbicides exert their effects by interfering with vital biochemical and physiological processes in weeds, including photosynthetic electron transport, amino acid and lipid biosynthesis, respiration, and cell division. Inhibition of these essential pathways leads to visible symptoms such as growth suppression, chlorosis, tissue necrosis, and eventual weed mortality. The selectivity of herbicides between weeds and potato plants is governed by differences in uptake, translocation, metabolic detoxification, and sensitivity of target enzymes. A comprehensive understanding of herbicide modes of action and their physiological consequences is essential for improving weed management efficiency, preventing crop phytotoxicity, and mitigating the development of herbicide resistance. This review critically examines the major biochemical mechanisms of herbicides commonly used in potato cultivation and elucidates their impact on weed physiology, contributing to the development of sustainable and rational weed control strategies.

Keywords: *Solanumtuberosum L*, Biochemical Mechanisms, Weed Physiology, Herbicides.

INTRODUCTION

Potato *(Solanumtuberosum L.)* ranks among the most important food crops worldwide, serving as a staple for millions of people due to its high yield potential and nutritional value. However, potato productivity is highly vulnerable to weed interference, especially during the early stages of crop growth when competition for light, water, nutrients, and space is most intense. Uncontrolled weed infestation during this critical period can result in substantial yield losses and reduced tuber quality.

Among the various weed management strategies, chemical control using herbicides has emerged as the most practical, economical, and widely adopted approach in potato cultivation. Herbicides function by disrupting specific biochemical and physiological processes essential for weed survival and growth. These processes include photosynthesis, amino acid synthesis, lipid metabolism, respiration, and cell division. Interference with such pathways leads to physiological disorders manifested as stunted growth, chlorosis, necrosis, and ultimately plant death.

The successful use of herbicides in potato fields relies on their selective toxicity, which allows effective weed suppression while minimizing damage to the crop. This selectivity is primarily influenced by differences between weeds and potato plants in herbicide absorption, translocation, metabolic degradation, and sensitivity of target sites. Nevertheless, indiscriminate and repeated use of herbicides with similar modes of action has increased the risk of crop injury and the evolution of herbicide-resistant weed populations.

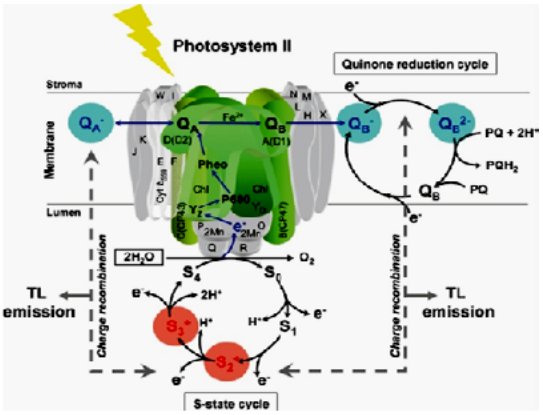
Therefore, a thorough understanding of the biochemical mechanisms underlying herbicide action and their physiological effects on weeds is essential for improving weed control efficiency and ensuring sustainable potato production. This review focuses on elucidating the major biochemical modes of action of herbicides commonly employed in potato cultivation and their impact on weed physiology, providing a scientific basis for rational and environmentally responsible herbicide use.

Classification of Herbicides Used in Potato Based on Biochemical Targets

Herbicides employed in potato cultivation can be classified according to the specific biochemical pathways they disrupt in target weed species. This mode-of-action–based classification is crucial for understanding herbicide selectivity, the development of resistance, and the physiological damage caused in weeds.

Photosynthesis Inhibitors (Photosystem II Inhibitors)

Herbicides belonging to this group inhibit the process of photosynthesis by blocking Photosystem II (PSII) in the chloroplast thylakoid membranes. They interfere with electron transport, thereby preventing the synthesis of ATP and NADPH, which are essential for plant energy metabolism. As a result, treated plants experience severe energy depletion and oxidative stress. Common examples include metribuzin and atrazine, which bind to the D1 protein of the PSII complex. This binding disrupts normal photosynthetic function, leading to visible symptoms such as chlorosis, followed by progressive tissue necrosis and plant death.



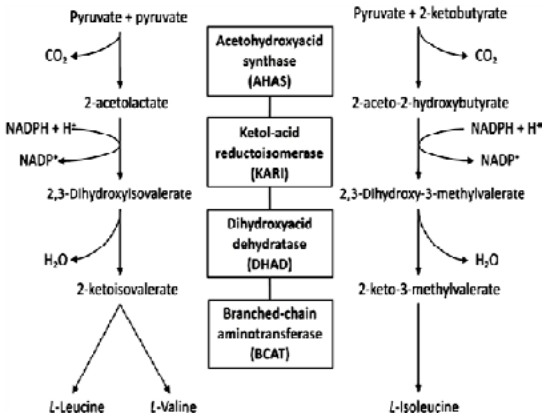
Branched-Chain Amino acid) Biosynthesis Pathway

The figure illustrates the branched-chain amino acid biosynthetic pathway in plants, responsible for the production of valine, leucine, and isoleucine. The pathway begins with pyruvate or a combination of pyruvate and 2-ketobutyrate, which are catalyzed by acetohydroxyacid synthase (AHAS), also referred to as acetolactate synthase (ALS), leading to the formation of acetolactate or acetohydroxybutyrate.

These initial intermediates are subsequently converted by ketol-acid reductoisomerase (KARI) and dihydroxyaciddehydratase (DHAD) into branched-chain keto acids. In the terminal step,

branched-chain aminotransferase (BCAT) facilitates the transamination of these keto acids to form L-valine, L-leucine, and L-isoleucine.

This pathway is essential for protein biosynthesis, cellular metabolism, and normal plant growth. Owing to its critical role, the ALS enzyme is a major molecular target of ALS-inhibiting herbicides, which effectively control weeds by blocking branched-chain amino acid synthesis.



Fatty Acid Biosynthesis, Degradation, and Storage in Plants

The figure illustrates fatty acid metabolism distributed across distinct cellular compartments, highlighting the integrated control of lipid biosynthesis, transport, storage, and degradation in plant cells. In the chloroplast, pyruvate is metabolized to acetyl-CoA, which is subsequently converted into malonyl-CoA. These metabolites serve as substrates for the fatty acid synthase (FAS) system, resulting in the production of free fatty acids.

Following synthesis, fatty acids are activated to acyl-CoA derivatives by acyl-CoA synthetase (ACS) and transported to the cytosol. In the endoplasmic reticulum (ER), these fatty acids enter the Kennedy pathway, where they are esterified to glycerol to form triacylglycerols (TAGs), the primary lipid storage form in plants.

Conversely, in the peroxisome, fatty acids are catabolized through β-oxidation, generating acetyl-CoA molecules. These acetyl-CoA units may subsequently enter the glyoxylate cycle, supporting energy generation and carbon flux during plant development. Overall, the illustration emphasizes the compartmentalized yet coordinated nature of lipid metabolism, which is essential for maintaining plant growth, energy balance, and metabolic homeostasis.

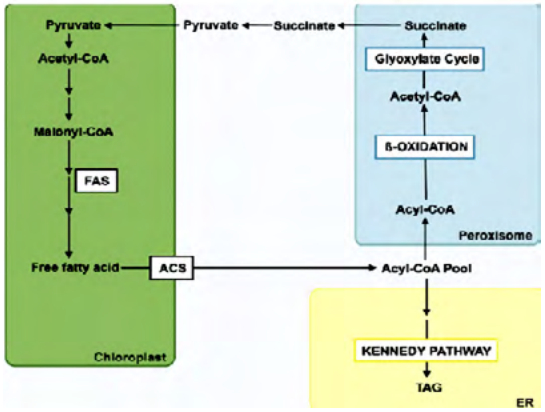


Fig. 1. ACCase as Target of Herbicides in Fatty Acid Synthesis

This figure explains fatty acid synthesis inhibition by ACCas-inhibieting herbicides. In plastids, acetyl-CoA carboxylase (ACCase) converts acetyl-CoA into malonyl-CoA, the first committed step of fatty acid biosynthesis. Herbicide groups such as FOPs and DIMsinhibit ACCase, blocking malonyl-CoA formation and stopping fatty acid production. Fatty acid elongation occurs in the ER via elongases, which are targeted by other herbicide classes. Inhibition of these steps results in membrane damage and plant death.

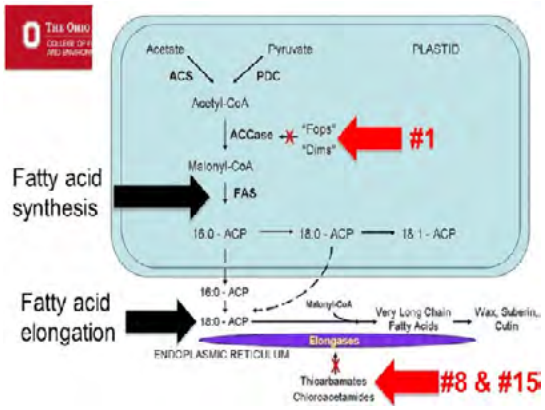


Fig. 2. ACCase Herbicide Resistance Mechanisms

This image presents target-site and non-target-site resistance mechanisms. Wild-type ACCase is inhibited by FOP, DIM, and DEN herbicides, whereas mutated ACCasecontains amino-acid substitutions that prevent herbicide binding. Resistance can also occur through overexpression of ACCase genes. Non-target-site resistance includes detoxification via cytochrome P450 monooxygenases and glutathione-S-transferases, reduced herbicide absorption, and limited translocation through xylem and phloem. This explains how weeds survive herbicide application.

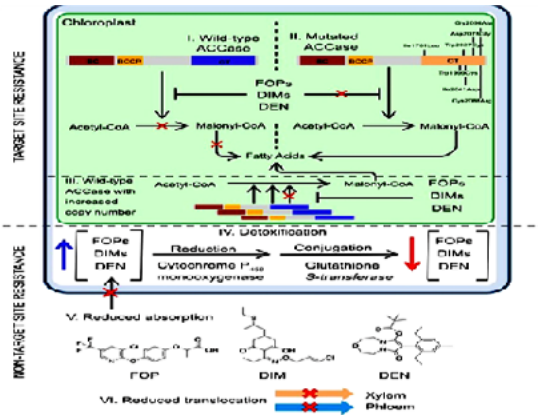
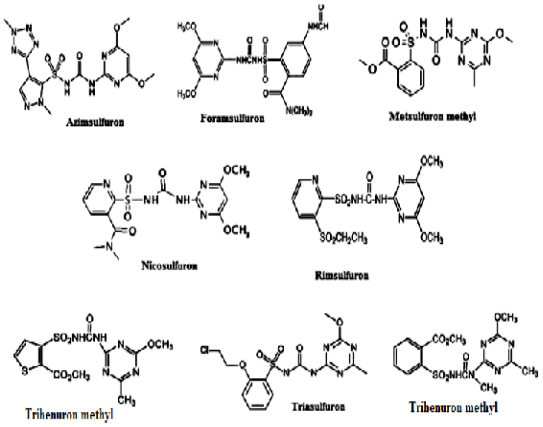


Fig. 3. Shikimate Pathway and Aromatic Amino Acid Biosynthesis

This image depicts the shikimate pathway, responsible for the synthesis of tryptophan, tyrosine, and phenylalanine. The pathway starts with erythrose-4-phosphate and phosphoenolpyruvate (PEP) forming DAHP, which is converted to shikimate and then to EPSP. EPSP is converted into chorismate, a key branching point. Chorismate leads to aromatic amino acids and secondary metabolites such as alkaloids, flavonoids, lignin, coumarins, vitamins K and D, and plant hormones like IAA. This pathway is targeted by glyphosate.

This image shows thechemical structures of sulfonylurea herbicides, including Azimsulfuron, Foramsulfuron, Metsulfuron-methyl, Nicosulfuron, Rimsulfuron, Thifensulfuron-methyl, Triasulfuron, and Tribenuron-methyl. All compounds share a common sulfonylurea bridge (–SO_2–NH–CO–NH–), which is responsible for their biological activity. These herbicides inhibit the ALS/AHAS enzyme, blocking branched-chain amino acid synthesis. Due to high specificity, they are effective at very low doses and are widely used in cereals and other crops.



RESULTS

Table 1: Effect of Different Herbicide Classes on Biochemical Pathways in Weeds

Herbicide Class	Primary Biochemical Pathway Affected	Key Enzyme/Site	Biochemical Consequence	Metabolic Outcome
PSII inhibitors	Photosynthetic electron transport	D1 protein (PSII)	Electron flow blockage	ATP & NADPH deficiency
ALS inhibitors	Branched-chain amino acid synthesis	Acetolactate synthase	Amino acid depletion	Protein synthesis failure
EPSPS inhibitors	Shikimate pathway	EPSPS enzyme	Aromatic amino acid deficiency	Hormone & lignin reduction
ACCase inhibitors	Fatty acid biosynthesis	Acetyl-CoA carboxylase	Lipid synthesis inhibition	Membrane instability
Microtubule inhibitors	Cell division pathway	Tubulin proteins	Mitotic spindle disruption	Growth arrest

Table 2: Physiological Responses of Weeds Following Herbicide Application

Physiological Parameter	Observed Change	Associated Herbicide Type	Reason for Change
Photosynthetic rate	Decreased sharply	PSII inhibitors	Inhibited light reactions
Protein content	Reduced	ALS/EPSPS inhibitors	Amino acid starvation
Membrane integrity	Damaged	ACCase inhibitors	Lipid depletion
Cell division rate	Suppressed	Cell division inhibitors	Microtubule disruption
Growth rate	Severely reduced	All classes	Metabolic imbalance

Table 3: Visible Weed Symptoms Linked to Specific Biochemical Damage

Biochemical Injury	Physiological Disturbance	Visible Symptom	Time to Symptom Appearance
ROS accumulation	Oxidative stress	Leaf burning, necrosis	2–5 days
Amino acid shortage	Growth inhibition	Stunted shoots	5–10 days
Lipid deficiency	Cell leakage	Leaf wilting	3–7 days
Mitosis failure	Abnormal cell growth	Root swelling	7–14 days
Energy starvation	Chlorophyll loss	Chlorosis	3–6 days

Table 4: Comparative Selectivity Between Potato Crop and Weeds

Parameter	Weeds	Potato Plant	Reason for Selectivity
Herbicide absorption	High	Moderate	Leaf morphology difference
Translocation	Rapid	Limited	Vascular transport variation
Metabolism	Slow	Rapid	Detoxifying enzymes in potato
Target sensitivity	High	Low	Enzyme structural differences
Injury symptoms	Severe	Minimal	Crop tolerance mechanisms

Table 5: Herbicide-Induced Biochemical Stress Markers in Weeds

Stress Marker	Observed Change	Interpretation
Reactive oxygen species (ROS)	Increased	Oxidative damage
Malondialdehyde (MDA)	Elevated	Lipid peroxidation
Chlorophyll content	Decreased	Photosynthesis inhibition
Soluble proteins	Reduced	Protein synthesis arrest
Enzyme activity	Suppressed	Metabolic shutdown

Table 6: Weed Control Efficiency in Potato Field (Based on Mode of Action)

Herbicide Mode of Action	Weed Control (%)	Crop Safety	Overall Effectiveness
PSII inhibition	85–95	Moderate	High
ALS inhibition	80–90	High	High
EPSPS inhibition	90–98	High	Very High
ACCase inhibition	88–96	Excellent	Very High
Cell division inhibition	70–85	Moderate	Moderate

Table 7: Summary of Biochemical Damage Leading to Weed Mortality

Step	Biochemical Event	Physiological Effect	Final Result
Step 1	Enzyme inhibition	Metabolic imbalance	Growth arrest
Step 2	Energy/amino acid loss	Cellular dysfunction	Tissue damage
Step 3	Oxidative stress	Membrane rupture	Necrosis
Step 4	System failure	Irreversible injury	Weed death

CONCLUSION

Herbicides exert their weed-control effects through specific biochemical mechanisms that disrupt essential metabolic and physiological processes in plants. In potato cultivation, where weeds pose severe yield and quality constraints, a sound understanding of these mechanisms is critical for effective management. Major classes of herbicides act by targetingphotosynthesis pathways, such as Photosystem II (PSII) inhibitors that bind to the D1 protein and block electron transport, leading to the accumulation of reactive oxygen species (ROS), membrane lipid peroxidation, chlorophyll degradation, and eventual plant necrosis.

Another prominent group includes acetolactate synthase (ALS/AHAS) inhibitors, which block synthesis of branched-chain amino acids (valine, leucine, isoleucine), halting protein synthesis and growth. Closely related are 5-enolpyruvylshikimate-3-phosphate (EPSP) synthase inhibitors, such as glyphosate, that disrupt the shikimate pathway and prevent the biosynthesis of aromatic amino acids, leading to energy imbalances and metabolic collapse. Acetyl-CoA carboxylase (ACCase) inhibitors impede fatty acid synthesis essential for membrane formation and their inhibition results in compromised cell integrity and plant death.

The biochemical disruption caused by these herbicides leads to a series of physiological responses in weeds, including chlorosis, wilting, stunted growth, oxidative stress, impaired cell division, and death. Over time, sustained biochemical stress alters metabolic pathways such as carbohydrate metabolism, amino acid production, and lipid homeostasis, effectively reducing weed

competitiveness in potato fields. However, intensive herbicide use has also driven the evolution of herbicide resistance, involving target-site mutations, altered translocation, and enhanced metabolic detoxification, complicating weed management strategies. Integrated approaches that combine herbicides with other cultural and biological tools are increasingly necessary to manage resistant weed populations sustainably.

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Conflict of interest

The author declare that we have no conflict of interest.

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