

# Paraquat: An Oxidative Stress Inducer

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## 1. Introduction

Paraquat (1,1-dimethyl-4,4-bipyridinium dichloride), is a foliar-applied and non selective bipyridinium herbicides, and it is one of the most widely used herbicides in the world, controlling weeds in a huge variety of crops like corn, rice, soybean, wheat, potatoes; major fruits: apples, oranges, bananas; beverages: coffee, tea, cocoa; and processed crops: cotton, oil palm, sugarcane and rubber.

For a foliar absorbed herbicide to completely kill a plant, it must be capable of accessing the whole plant, as growing leaves and newly emerging roots. This often means that the herbicide not only needs to damage at the point of its absorption, but must also be translocated to parts of the plant not contacted by the herbicide during application.

Paraquat is a cation formed by two pyridine rings, each having a quaternary amine and thus charged 2+. Although the majority of herbicides are passively transported as noionic molecules, paraquat cation movement by diffusion across membrane lipid bilayer is unlikely. Transporter studies to explain paraquat compartment were made using several systems. ABC transporters, large membrane proteins which use ATP for the active transport of several compounds including paraquat have been described. Other groups of transporters are small antiporter proteins which exchange protons for some other molecules using the proton electrochemical potential gradient (Morymio et al., 1992, Yerushalmi, et al., 1995). In animal tissues it has been shown that paraquat transport occurs by carriers that also function as carriers of other molecules such as polyamines (Rannels et al., 1989, Jóri et al., 2007). Hart et al. (1992a 1992b) demonstrated that paraquat movement across plasma membrane root epidermal and cortical maize cells has a concentration-dependent kinetic and that the herbicide binds to cell wall, and its transport is facilitated by a carrier that normally functions in the movement of molecules that has a similar chemical structure or similar charge distribution such as diamines like putrescine and cadaverine. Using maize protoplast Hart et al. (1993) showed that paraquat uptake has similar concentration-kinetic to that observed in intact cells and the accumulation inside cells increase in a time-dependent manner and is saturated after 10 min, although 50% of uptake occurs during the first 10 s. The saturable  $K_m$  for paraquat uptake in maize cells and protoplasts was determined at 90  $\mu\text{M}$  and 132  $\mu\text{M}$  respectively, similarly the  $K_m$  in rat lung was 70  $\mu\text{M}$

suggesting in both animal and vegetal tissues a carrier-mediated process (Rannels et al., 1985).

In order to investigate paraquat uptake, compartmentation and translocation, maize plantlets with their root immersed in paraquat solution for several loading periods were used (Hart et al., 1993). The lack of chloroplasts in roots provides a system to minimize the short-term phototoxic effect. The paraquat accumulation in the root vacuole was linear over a 24 h loading period. The vacuolar paraquat content, with respect to the total accumulated increased from 15% to 42% after 2 h and 24 h loading period, respectively. In contrast to the vacuole, total cytoplasmic paraquat content appeared to approach saturation whereas paraquat associated with the cell wall fraction remained relatively constant, suggesting that this phase is rapidly saturated. Even though paraquat is considered to be relatively immobile, linear paraquat (PQ) translocation occurred from roots to shoots and was estimated that approximately 50% of the paraquat effluxing from roots started translocation to shoots 5 h after the beginning of loading period (Hart et al., 1993b).

Paraquat acts as a redox cyler with a great negative reduction potential ( $E_0 = -0.446$  V). This feature restricts its interaction with strong reductant compounds. When dication of paraquat ( $PQ^{2+}$ ) accepts an electron from a reductant form the paraquat monocation radical ( $PQ^+$ ), which then rapidly reacts with oxygen ( $O_2$   $E_0 = 0.16$  V) to initially produce superoxide radical ( $O_2^{\cdot-}$ ) ( $k 7.7 \times 10^8$   $M^{-1} s^{-1}$ ) and subsequently the other reactive oxygen species (ROS) such as hydrogen peroxide ( $H_2O_2$ ) and hydroxyl radical (OH).

In plants, paraquat is principally reduced within chloroplasts, where it acts as an alternative electron acceptor taking electron from Fe-S proteins of photosystem I; inhibiting the ferredoxin reduction, the NADPH generation, and also the regeneration of ascorbic acid. In consequence, paraquat is a potent oxidative stress inducer, because it greatly increases the ROS production and inhibits the regeneration of reducing equivalents and compounds necessary for the activity of the antioxidant system.

Paraquat also induces the increase of superoxide radical production in mitochondria, where complexes I and III are the major electron donors. For this reason paraquat has been widely used to induce mitochondrial oxidative stress in many experimental systems such as isolated mitochondria, cultured cells, and whole organisms including plants, *Saccharomyces cerevisiae*, *Caenorhabditis elegans*, *Drosophila melanogaster* and rodents (Cocheme & Murphy, 2008).

## 2. Generation and role of ROS

Superoxide radical ( $O_2^{\cdot-}$ ), singlet oxygen ( $^1O_2$ ) hydrogen peroxide ( $H_2O_2$ ) and hydroxyl radical (OH) are highly reactive compounds that induce protein and pigment degradation, lipid peroxidation, nucleic acid damage, affecting key components of plant cell metabolism that can finally lead to cell death. These deleterious reactions triggered by ROS are known as oxidative stress phenomenon (Casano et al., 1994, 1997; Lascano et al., 1998, 1999).

Even though all ROS are highly reactive compounds their effects and plant responses depend on the ROS in question as well as on its concentration, site of production, interaction with other stress molecules and on the developmental stage and plant cell previous history.

In green tissues under light, chloroplasts are the main intracellular source of ROS (Asada, 1999) and peroxisomes, through photorespiration, are other important ROS producers (del Río et al., 2006). While mitochondria, are the principal source of ROS in darkness and non green tissues. On the other hand, the NADPH oxidase complex, peroxidases and amino oxidases are major sources of apoplasmic ROS (Sagi & Fluhr, 2006).

Primarily, the chloroplasts mainly produce  $O_2^{\cdot-}$  at photosystem I (PSI) and  $^1O_2$  at photosystem II (PSII), and the mitochondria produce  $O_2^{\cdot-}$  at complexes I and III (Asada, 1999). The peroxisomes produce  $H_2O_2$  as byproduct of photorespiratory glycolate oxidase reaction, fatty acid  $\beta$ -oxidation and reaction of flavin oxidase, and  $O_2^{\cdot-}$  is generated by xanthine oxidase and by electron transport chains in the peroxisomal membrane (del Río et al., 2006).

Various interconverting reactions occur among different ROS. Superoxide is spontaneously or enzymatically converted to  $H_2O_2$  by disproportionation mechanism and  $H_2O_2$  and  $O_2^{\cdot-}$  can interact to produce  $\cdot OH$  through the Fenton reaction catalyzed by free transition metal ions (Fridovich, 1986).

Different ROS have different features. Hydrogen peroxide is a non radical, apolar molecule and, in consequence, it is a relatively stable compound with half-life around 1 ms. In plant tissues, its concentration could be in the micro to millimolar range. The half-lives of the other ROS are very short, ranging from nano to micro second, and then they are present at very low concentrations (Asada, 1999).

Reactive oxygen species also have different reactivities. Hydrogen peroxide (Eo 1.77 V), not a highly reactive ROS *per se*, mainly oxidizes thiol groups, in presence of transition metal ions it catalyzes  $\cdot OH$  generation by Fenton reaction. Superoxide radical (Eo -0.33V) oxidizes ascorbate and NADPH, reduces metal ions and cytochrome C and reacts with protein Fe-S centers. Singlet oxygen is particularly reactive with conjugated double bonds of polyunsaturated fatty acids. Whereas  $\cdot OH$  (Eo 2 V), the most oxidant ROS, reacts with all types of macromolecular cellular components. The differential ROS reactivity means that they leave different footprints in the cell in the form of different oxidatively modified components (Moller et al., 2007).

Cellular membranes are the principal targets of ROS. Peroxidation of polyunsaturated fatty acids (PUFAs) is a common oxidative stress effect. Linoleic acid (18:2) and linolenic acid (18:3) are major fatty acid present in galactolipids of thylakoids and phospholipids of all membranes. PUFAs peroxidation generates mixtures of lipid hydroperoxides several aldehydes, e.g., 4-hydroxy-2-nonenal (HNE) and malondialdehyde (MDA), hydroxyl and keto fatty acids and oxidative modification in membrane protein. The consequences over the membrane function are the fluidity and selectivity decreases (Halliwell et al., 1999; Halliwell, 2006). Some of the PUFA peroxidation products act directly or after enzymatic modification as secondary messengers either, e.g. oxylipins (Muller et al., 2004).

ROS induce mainly irreversible covalent modification on proteins. The reversible modifications on sulfur containing amino acid are very important in the redox or oxidative signaling. Cystein thiol groups are initially oxidized to disulfide and in further oxidation to sulfenic and sulfinic acid. The highest level of cysteine oxidation, cysteic acid seems to be irreversible and damaging (Ghezzi & Bonetto; 2003). Nitrosylation and glutathionylation are

other cystein thiol modification mediated by nitric oxide, reactive nitrogen species (RNS) and glutathione. RNS are generated by the interaction between nitric oxide and ROS. (Costa et al., 2003; Halliwell, 2006). Carbonylation, a common oxidative protein modification affecting particularly Arg, His, Lys, Pro, Thr, and Trp; and conjugation with peroxidation PUFA products, mainly with HNE, are other oxidative protein modifications (Shacter, 2000; Winger et al., 2005).

The generation of 8-Hydroxyguanine is the most common DNA modification induced by ROS. The nucleotide bases are attacked by  $\cdot\text{OH}$  and  $^1\text{O}_2$  while  $\text{H}_2\text{O}_2$  and  $\text{O}_2^{\cdot-}$  do not react at all (Wiseman & Halliwell, 1996). Chloroplastic and mitochondrial DNAs are into the two major source of ROS where potentially high rates of modification might occur (Thorslund et al., 2002). Another indirect oxidative modification to DNA is the conjugation of MDA with guanine (Jeong, 2005). The DNA oxidative modification could induce changes in cytosines methylation patterns, and then in the regulation of gene expressions. ROS-induced DNA modification seems to be a not completely random process (Halliwell, 2006).

Carbohydrates can be oxidatively modified by  $\cdot\text{OH}$ , being the formic acid the main breakdown product of sugar oxidation (Isbell et al., 1973).

In spite of its toxic effects, increasing evidence indicates that ROS are signaling molecules that participate in many processes, such as cell cycle, cell elongation, cell death, plant growth and development, senescence, hormone signaling, responses to biotic and abiotic stress and in symbiotic interaction with microorganisms (Bustos et al., 2008; Mittler et al., 2004; Muñoz et al. 2011, submitted; Rodriguez et al., 2010). The  $\text{H}_2\text{O}_2$  molecular properties make it a good second messenger that could cross membrane by diffusion or aquaporins. However, all ROS can act as signaling molecules directly or by oxidized product. NADPH oxidase complex, the main source of apoplasmic ROS, has a key role in oxidative signaling (Sagi & Fhlur, 2004).

The dual role of ROS, as toxic or signaling molecules, depends on the ratio and subcellular location of its generation, thus the tight regulation of the steady-state level of ROS in different subcellular compartments has both signaling and oxidative damage protection purposes. The function of ROS as signaling molecules is intrinsically related to the interaction with non-enzymatic antioxidants, such as ascorbate and glutathione, which are redox buffers and also signal molecules *per se* (Foyer & Noctor 2005 a, 2005b).

The relationship among ROS, antioxidants, reducing equivalents, sugars, the redox state of chloroplastic and mitochondria electron transport chains are major determinants of the cellular redox state, which has a critical function in the environmental perception and modulation of defense, acclimation and tolerance responses (Foyer & Noctor, 2005 a; 2005b; Lascano et al., 2003; Melchiorre et al., 2009; Robert et al., 2009).

### 3. Antioxidant system in plants

Plants have evolved a complex antioxidant system composed by both non-enzymatic and enzymatic components, to prevent the harmful effects of ROS.

Low-molecular-mass metabolites soluble in both aqueous and lipid phases lipid with high ROS reactivity such as ascorbate, glutathione tocopheroles, flavonoids, alkaloids, carotenoids, proline and amines, form non-enzymatic part of the antioxidant system (Apel & Hirt, 2004; Sharma and Dietz, 2006).

Superoxide dismutase (SOD) (E.C.: 1.15.1.1), ascorbate peroxidase (APX) (E.C.: 1.11.1.11), catalase (CAT) (EC 1.11.1.6), and glutathione reductase (GR) (E.C.: 1.6.4.2) are key antioxidant enzymes that modulate the concentration of two of the Haber/Weiss and Fenton reaction substrates,  $O_2^{\cdot-}$  and  $H_2O_2$ , preventing the formation of the highly toxic  $\cdot OH$  radical (Asada, 1999). Approximately, 80% of SOD, GR, and APX activity is located in the chloroplast (Asada, 1999). CAT activity is located in peroxisomes and mitochondria (Scandalios, 1994). SOD catalyses the disproportionation of  $O_2^{\cdot-}$  to  $H_2O_2$ , and is present in multiple isoforms: copper/zinc (CuZn-SOD), iron (Fe-SOD) and manganese (Mn-SOD) (Bowler et al, 1992). In most plants, CuZn-SOD and Fe-SOD are present in the chloroplasts, CuZn-SOD in the cytosol and Mn-SOD in mitochondria (Casano et al., 1997; Scandalios, 1993). Degradation of  $H_2O_2$  in the chloroplasts and in the cytosol is carried out by the ascorbate-glutathione cycle, which involves APX and GR activities (Lascano et al., 1999, 2003). APX has chloroplastic and cytosolic isoforms, and catalyses the conversion of  $H_2O_2$  to water using ascorbate as electron donor (Asada, 1999).

Reduced glutathione (GSH) and ascorbic acid are the most important soluble non-enzymatic antioxidants and in chloroplasts they are present at millimolar concentrations (Noctor & Foyer, 1998). Ascorbate acts as a ROS quencher and it is involved in the regenerations of tocopherol and violoxanthine deoxidase activity of xanthophylls cycle (Noctor & Foyer, 1998). Reduced glutathione is a tripeptide  $\gamma$ -glutamylcysteinyl glycine ( $\gamma$ -Glu-Cys-Gly) involved in: direct reaction with ROS, the regeneration of the ascorbate pool and as electron donor of glutaredoxins which are linked to type II peroxiredoxin activity. Likewise, GSH participates in the glutathionylation, a post-transcriptional modification of protein thiols groups that regulates the function of proteins like glyceraldehyde-3-phosphate dehydrogenase and thioredoxin activities (Michelet et al., 2005; Zaffagnini et al., 2007). The reduction of oxidized glutathione is NADPH-dependent and carried out by GR, a ubiquitous flavoenzyme with many isoforms, located in chloroplasts, cytosol, and mitochondria (Lascano et al., 2001; Tanaka et al., 1994).

Other more recently identified components of enzymatic antioxidant system are peroxiredoxins and glutathione peroxidase, non-heme-containing peroxidase which activity depend on cystein residues (Bryk et al., 2000; König et al., 2003).

#### 4. The use of paraquat in stress response studies

Plants as sessile organism are permanently exposed to changing environment that become stressful conditions affecting their growth, development and productivity. Tolerance to environmental stress is a major selection criterion in plant breeding. The cellular and molecular tolerance mechanisms of plants to different stresses have been intensively studied.

Reactive oxygen species are produced as byproduct of normal aerobic metabolism and the life under aerobic conditions is strictly dependent on the presence of antioxidant system. Nowadays, it is widely accepted that the generation of ROS is enhanced under abiotic and biotic stress conditions. Depending on stress intensity and its associated-ROS levels the plant responses range from tolerance to death.

Likewise, the positive response of the antioxidant system correlates, in part, with the tolerance to many different environmental stress conditions. ROS and antioxidant system

are central components of the cross-tolerance phenomenon which states that a tolerant genotype to one stress condition could be also tolerant to other kinds of stress.

Paraquat treatments have been frequently used, as a potent oxidative stress inducer, in many different basic studies like: oxidative stress tolerance and cross tolerance responses associated with the antioxidant system responses (Lascano et al., 1998, 2001, 2003), forward and reverse genetic approaches to study the function of different antioxidant system components (Melchiorre et al., 2009 and references therein), ROS signaling (Robert et al., 2009), ROS and NO-induced cell death (Tarantino et al., 2005), and to mimic the drought effect on carbon and nitrogen metabolism of nodules (Marino et al., 2006).

Several attempts made to enhance tolerance photooxidative stress conditions have been tested with paraquat treatments. These have involved the overexpression of enzymes associated with the Asada-Halliwell pathway including SOD (Arisi et al., 1998; Bowler et al., 1991; McKersie et al., 1999; Melchiorre et al., 2009; Perl et al., 1993; Pitcher et al., 1991; Sen Gupta et al., 1993; Tepperman et al., 1990; Van Camp et al., 1996), and GR (Aono et al., 1991, 1993; Creissen et al., 1995; Foyer et al., 1991, 1995; Melchiorre et al., 2009). The tolerance to different oxidative stress conditions was dependent on the copy numbers and overexpression levels; the isoform overexpressed; the subcellular location where the overexpressions were targeted; and the induction of other antioxidant enzymes. The results of chloroplasts-targeted Mn-SOD or GR overexpression in wheat chloroplasts, suggest that antioxidant enzyme overexpression effects on tolerance response not only depend on their antioxidant capacities but also on their effects on the cellular redox state, which modulates the responses to photooxidative stress in a pathway where apoplastic superoxide generation could be involved (Melchiorre et al., 2009). The photooxidative activations of NADPH oxidase complex, the main source of apoplastic ROS, can be mimicked by paraquat treatment (Robert et al., 2009).

Paraquat has also been used as an efficient inducer of cell death in both animal and plant cells (Dodge, 1971; Suntres, 2002). The cell death processes in plants are major regulatory mechanism of growth, development, and responses to biotic and abiotic stresses (Lam et al., 2001; Pennel & Lamb, 1997). Environmental or developmental conditions where cellular redox balance is disturbed and significant ROS accumulation occurred, could lead to the induction of cell death processes (Dat et al., 2000). In this context, two type of ROS-associated stress intensity-dependent death can be defined: Ordered or Programmed Cell Death (PCD) when the cell maintains the membrane and energy generation systems, and Disordered or Necrosis, when these systems are overwhelmed by the oxidative burst. Continuous or transient light-dependent H<sub>2</sub>O<sub>2</sub> accumulation, provoke necrosis or PCD, respectively indicating the existence of a ROS levels threshold below which PCD is triggered and above which necrotic cell death prevail (Montillet et al., 2005).

Programmed Cell Death in plant cells shares some similarities with that of animal cells, like organelle degeneration, nuclear condensation, nuclear DNA fragmentation and eventually cell shrinkage. Interestingly, animal anti-apoptotic protein (Bcl-2, Bcl-xL, and CED-9) expressed in plant, prevented apoptosis-like death mediated by chloroplasts photooxidative stress induced by paraquat (Chen & Dickman, 2004; Mitsuhara et al., 1999).

The *in vivo* relationship between ROS-associated to environmental stress condition like drought and biological nitrogen fixation (BNF) inhibition in the legume-Rhizobium

symbiosis were studied using different dose of paraquat to induce oxidative stress in nodules. Paraquat produced cellular redox imbalance leading to an inhibition of biological nitrogen fixation (BNF). The low paraquat dose provoked BNF decline, preceded by a decrease in sucrose synthase gene expression protein content and activity, while high paraquat induced a faster and more pronounced BNF inhibition, coinciding with a decline in sucrose synthase and also with a reduction in leghaemoglobin content. These results support the occurrence of two regulation pathways for BNF under oxidative stress, one of these involving carbon shortages and the other involving leghaemoglobin /oxygen flux (Marino et al., 2006, 2008).

#### 4.1 Paraquat resistant mutants

To date, several mutants, ecotypes, and biotypes with paraquat resistance have been characterized in a few plant species. Paraquat-resistant mutants have been shown to be cross tolerant to other oxidative stress conditions and have been used to study the tolerance to other photooxidative stress condition (Tsugane et al., 1999).

There are several paraquat-resistant *Arabidopsis* mutants. Photoautotrophic salt tolerate 1 (*pst1*), an *Arabidopsis* mutant that can grow under high salt concentrations, is nearly 10 times more tolerant to paraquat than wild-type seedlings. This mutant, which is also tolerant to high light intensities exhibits higher SOD and APX activities under paraquat, salt, and high light intensities treatments (Tsugane et al., 1999).

The paraquat-resistant *Arabidopsis thaliana* mutant, allelic to the ozone sensitive mutant *rcd1-1* (radical-induced cell death1-1) (Overmyer et al., 2000), called *rcd1-2*, is also tolerant to UV-B and freezing. The tolerance in this mutant is also related to higher levels of the ROS-scavenging enzymes, particularly chloroplastic CuZn-SOD and APX, and also with an increased accumulation of flavonoids (Fujibe et al, 2004). *Arabidopsis* Cvi ecotype also shows a higher resistance to paraquat, which seems to be determined by a new allele of plastidic CuZnSOD (Abarca et al., 2001).

*Gigantea*, a late-flowering *Arabidopsis* mutant, is resistant to paraquat (Kurepa et al., 1998), however, the resistance mechanism remains unknown (Huq et al., 2000). In the broadleaf weed *Archoteca calendula* (L) paraquat tolerance has been associated with increases in antioxidant defense. This species also exhibit cross tolerance to other stress conditions (Soar et al., 2003).

*Arabidopsis paraquat resistant2-1* (*par2-1*) mutant show an anti-cell death phenotype. Paraquat treatment induce similar superoxide production in *par2-1* and wild-type plants, suggesting that PAR2 acts downstream of superoxide to regulate cell death. *par2-1* encode a S-nitrosoglutathione reductase (GSNOR) that catalyze a major biologically active nitric oxide species, S-nitrosoglutathione. Compared to wild type, *par2-1* mutant showed higher nitric oxide level, suggesting that nitric oxide level and nitrosylation protein modification regulates cell death in plant cells (Chen et al., 2009).

Other paraquat-resistant genotypes have also been reported; like the grass weed *Hordeum glaucum* (Lasat et al., 1997) and *Conyza bonariensis* (Fuerst et al., 1985; Norman et al., 1994). The resistance mechanism seems to be related to a higher herbicide compartmentalization in root vacuoles of the resistant biotype than in the susceptible one. On the contrary, the amount of paraquat accumulated in the cytoplasm of the susceptible biotype was double that found in the resistant biotype.

Additionally, paraquat tolerance has been associated with the expression of transporters able to carry molecules with similar chemical structure or charge distribution to paraquat, like polyamines (Tachihara et al., 2005). Pharmacological treatments with blockers of proton pump ATPases, such as nitrate, carbonyl-cyanide-m-chlorophenylhydrazone (CCCP) and N4N1- dicyclohexylcarbodiimide (DCCD) were used in order to study their effects on paraquat moving into inactive compartments in *C. canadensis* (Jóri et al., 2007). Recovery after paraquat treatment in tolerant biotypes was strongly inhibited by nitrate, as nitrate selectively blocks ATPases in the vacuoles -responsible for energy supplies to vacuolar membranes- the results suggested that paraquat sequestration uses energy from the proton gradient (Jóri et al., 2007).

Regarding the relationship between paraquat tolerance and leaf age, some studies have shown that young leaves are more tolerant than mature ones (Kuk et al., 2006; Ohe et al., 2005), it is worth nothing that responses are closely related with detoxify mechanism and antioxidative responses as well as with morphological leaf characteristics such as epicuticular wax content and leaf cuticle development which is the first and most significant barrier for foliar-applied chemicals. Although damage originated by paraquat treatment in *Cucurbita spp* varied among cultivars, the injury provoked by herbicide application was lower in younger leaves than in older ones as it was observed by lesser conductivity values and malondialdehyde production which indicate membrane damage with cellular leakage and membrane lipid peroxydation respectively. These responses correlated also with higher antioxidant activity and increases in ascorbate content as well as with higher epicuticular wax in young leaves (Yeol Yoon , 2011).

## 5. Conclusion

Paraquat is potent oxidative stress inducer, which beyond the widely use as desiccant herbicide, it has been a very useful tool in plant biology basic research. Many aspect of oxidative stress in plants, the toxic and signaling roles of ROS, the native and transgenic plant tolerance/susceptibility responses to many environmental stress conditions, the cross tolerance phenomenon and different cell death processes have been studied using paraquat treatments.

## 6. References

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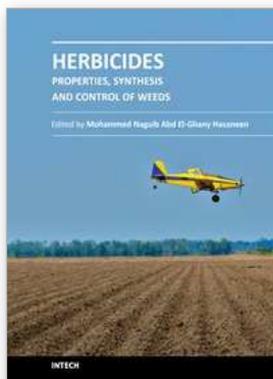
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## **Herbicides - Properties, Synthesis and Control of Weeds**

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This book is divided into two sections namely: synthesis and properties of herbicides and herbicidal control of weeds. Chapters 1 to 11 deal with the study of different synthetic pathways of certain herbicides and the physical and chemical properties of other synthesized herbicides. The other 14 chapters (12-25) discussed the different methods by which each herbicide controls specific weed population. The overall purpose of the book, is to show properties and characterization of herbicides, the physical and chemical properties of selected types of herbicides, and the influence of certain herbicides on soil physical and chemical properties on microflora. In addition, an evaluation of the degree of contamination of either soils and/or crops by herbicides is discussed alongside an investigation into the performance and photochemistry of herbicides and the fate of excess herbicides in soils and field crops.

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