J. Pesticide Sci. 24, 299-307 (1999)

Commentaries

Biochemical Mechanisms of Action of Herbicides and the Impact of Biotechnology on the Development of Herbicides

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(Accepted May 20, 1999)

INTRODUCTION

German chemists, during the 1800s, published on the synthesis of a large number of organic compounds, some of which eventually became pesticides or served as precursors of pesticides. Examples, with the year of synthesis noted parenthetically, include organophosphates (1820), lindane (1825), urethane (1834), DNP and PCP (1843), phenylurethane (1870), DDT (1874), and simazine (1885). During these years, German physiologists and biochemists also studied the effects of many organic compounds, including some of the above, on animals and plants.

Phenols (Fig. 1)

Phenols were among the earliest compounds that found many uses as pesticides, but were highly toxic to most organisms as reflected in the LD_{50}s which range from 25 to 200 mg/kg. PCP (pentachlorophenol) was introduced initially as an insecticide, for the preservation of wood, but was used later as a selective herbicide. However, when used to control grasses in transplanted rice, was toxic to fish and shellfish. DNOC (2-methyl-4, 6-dinitrophenol) was patented by Bayer in Germany (1892) for use as an insecticide, but was subsequently developed for use in Europe and Great Britain as a selective herbicide (1932-1935). Dinoseb or DNBP (2-*sec*-butyl-4,6-dinitrophenol) was developed later for the U.S. markets by Dow (1945). Bromoxynil (3,5-dibromo-4-hydroxybenzonitrile) was the last of the phenols to be introduced to the U.S. markets. It still is of commercial importance because of the development of bromoxynil-tolerant cotton.

In studies with isolated rat liver mitochondria, phenols, with DNP as the prototype, were shown to uncouple the electron transport pathway from the energy generating pathway in 1948. In the presence of uncouplers, respiration (oxygen uptake) is increased (stimulated), but no adenosine triphosphate (ATP) is generated. However, it was not until 1967, some 20 years later, that plant biochemists were able to isolate tightly coupled mitochondria from mung bean hypocotyls (*Phaseolus aureus* renamed *Vigna radiata*). The action of phenols on plant mitochondria was shown to be the same as that expressed on animal mitochondria.[1] Those of us working with plant tissue envied the animal biochemists because of the ease with which mitochondria could be isolated from mammalian livers. The phenols also inhibit photosynthetic electron transport and uncouple photophosphorylation in chloroplasts. Both effects may contribute to their herbicidal action.[2]

Carbamates (Fig. 1)

A second group of organic compounds that became herbicides and insecticides were carbamates: R-NH-COOR′. In the herbicidal carbamates, R is generally an aryl or aromatic group and R′ a short chain alkyl group. These are essentially non-toxic to animals with rat oral LD_{50}s around 5000 mg/kg. In the insecticidal carbamates, the substitutions are reversed with R′ being an aryl (aromatic) group and R being a short chain alkyl group, frequently just a methyl moiety. These are essentially non-toxic to plants, but are very toxic to animals with LD_{50} values around 800 mg/kg.

Urethane (ethyl carbamate) and phenylurethane (ethyl carbanilate) were the earliest of the carbamates to be studied with plant systems. The phenylcarbamates are toxic to grasses, but not to broadleaved weeds. They retard germination, produce morphological aberrations and affect mitosis[3,4] much like colchicine through effects on microtubule-organizing centers (MTOCs). Other herbicides (dinitroanilines and phosphoramides, Fig. 1) were subsequently introduced that also inhibit mitosis in plants, but not in animals, by preventing the polymerization of tubulin subunits into microtubules.[5,6] Whereas the phenylcarbamates are primarily recognized as being inhibitors of mitosis, they also inhibit photoinduced electron transport in isolated chloroplasts. The dinitroanilines also inhibit electron transport and phosphorylation in chloroplasts and mitochondria.[7]

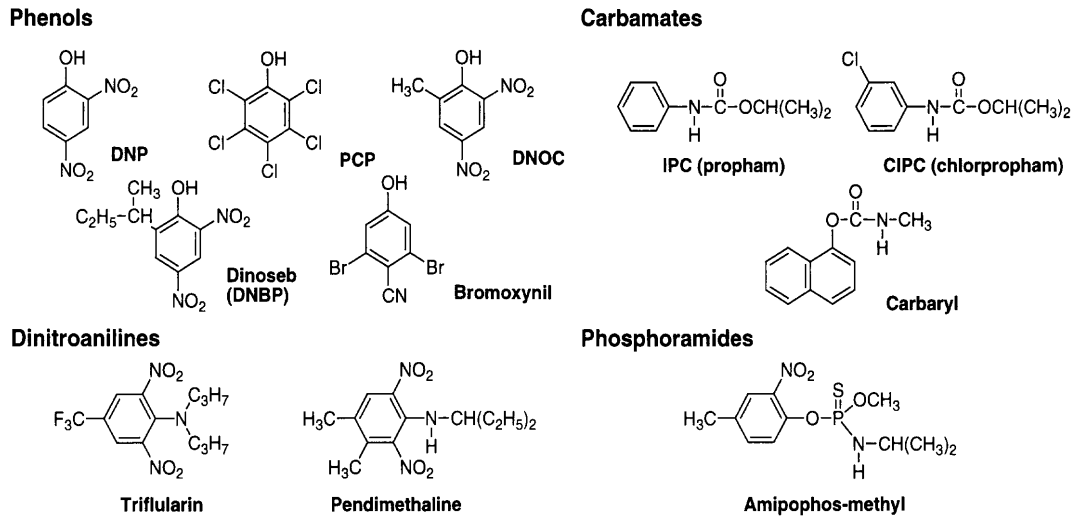

Fig. 1 Phenols, carbamates, dinitroanilines, and phosphoramide herbicides.

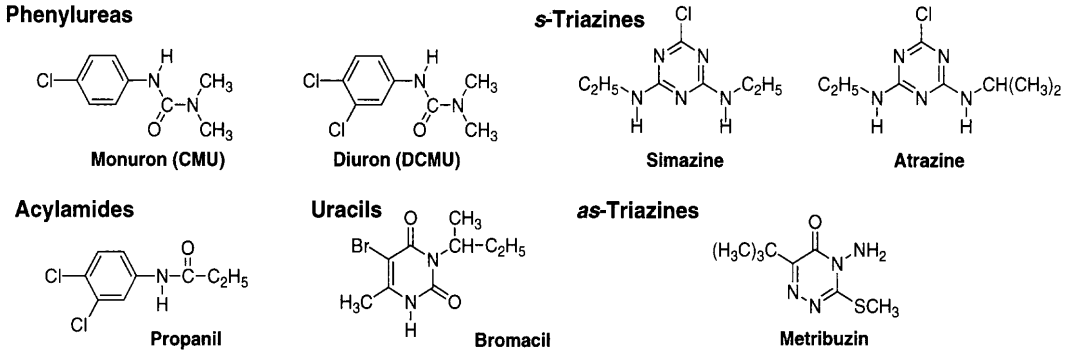

Fig. 2 Inhibitors of photosynthesis.

Carbamate insecticides were developed in parallel with the development of carbamate herbicides. Around 1947, Geigy in Switzerland began studies that led to the insecticidal carbamates. Carbaryl was introduced as an insecticide by Union Carbide in 1956. By 1978, more than 39 insecticidal carbamates had been developed with over 50 others having been patented, but not fully developed. All inhibit cholinesterase activity like the organophosphates, a process unique to animals, hence are not toxic to plants.

Robin Hill, an English plant biochemist, observed in 1937 that isolated chloroplasts evolved oxygen when illuminated in the presence of an electron acceptor.[8] Subsequently in 1949, the carbamates that Warburg had reported in 1920 to inhibit photosynthesis in *Chlorella* cells,[9] were shown to inhibit the Hill reaction.[10] These studies extrapolated the light reactions of photosynthesis from intact cells to the organelle level. In 1956, results of the earliest structure/activity studies on inhibition of the Hill reaction were published that involved arylcarbamates and phenylureas.[11]

Inhibitors of Photosynthesis (Fig. 2)

DuPont first reported on the herbicidal activity and introduced the phenylureas. In 1952, monuron (CMU), the 4-chloro derivative, was introduced commercially followed by diuron. Through analog synthesis, many other companies including Ciba, Hoechst, Sandoz, FMC, and Bayer commercialized over 25 additional substituted ureas. Over 20 symmetrical triazines, with simazine being the prototype, were commercialized. Most of the early patents were held by Geigy, but Gulf Oil, Shell, Ciba, and Monsanto also produced candidate compounds. Various acylanilides (at least 8 compounds) were developed by Bayer, Rohm & Haas, Monsanto, FMC, Schering, Gulf Oil (Spencer), and 3M. The best known in rice-growing areas is propanil. The substituted uracils were patented by DuPont in 1962. Three were developed commercially. The synthesis of metribuzin was reported in 1964 with the first German patent being issued in 1966 to Bayer. It was tested in the U.S. by both Mobay and DuPont.

The photosynthesis-inhibiting herbicides developed during the mid-1950s and 1960s continue to be associated with weed control strategies for most of our major crops. With experience being gained with the pre-emergence herbicides, a need was soon recognized for herbicides that could be used post-emergently, *i.e.*, after the extent of

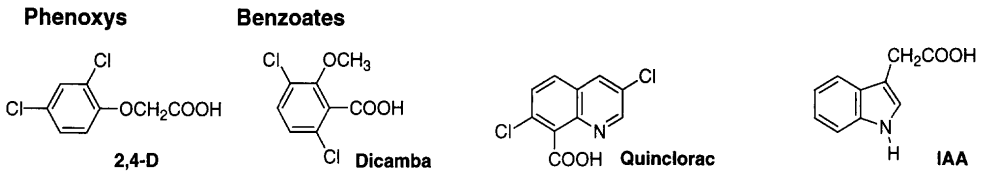

Fig. 3 Auxin herbicides.

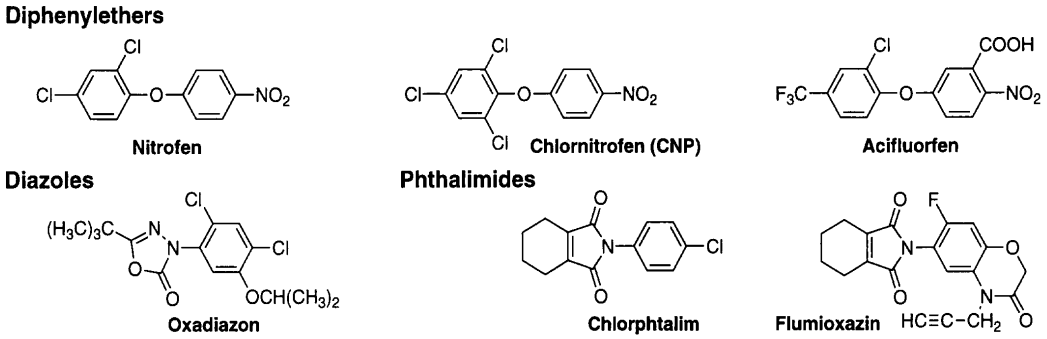

Fig. 4 Protox inhibitors.

the weed problem could be identified.

Auxin Herbicides (Fig. 3)

Phenoxyalkanoates, which were introduced in the mid to late 1940s, represented by 2,4-D (2,4-dichlorophenoxyacetic acid) and other herbicides that possess auxin activity (benzoic acids and quinoline carboxylic acids) have been used extensively for the control of dicot weeds in cereal crops for over 50 years. These herbicides mimic, in many ways, lethal doses of the plant hormone auxin (indole-3-acetic acid, IAA). After many years of study, the precise mode of action remains to be established. In a recent study, quinclorac was shown to stimulate ethylene biosynthesis by inducing the activity of 1-aminocyclopropane-1-carboxylic acid (ACC) synthase.[12] In susceptible dicots, increased levels of ethylene trigger an accumulation of abscisic acid (ABA). In susceptible grasses, the level of tissue cyanide (HCN), a co-product formed during ethylene biosynthesis, increased. These increases in ethylene, ABA, and HCN cause epinasty of leaves, growth retardation, and senescence. However, not all researchers are in agreement about the association between plant sensitivity to the auxin type herbicides and the increase in ethylene production.[13]

Protox Inhibitors (Fig. 4)

The diphenylethers of which nitrofen is the prototype was patented in the U.S. in 1955 and introduced in 1961 by Rohm & Haas. It was registered for use in Japan in 1963. Mitsui, in 1966, provided chlornitrofen (CNP) which had a third chlorine substituted at ring position 6, and registered it for use in Japan. Several other diphenylethers were commercialized by other companies including Ciba and Mobil Chem. Co. By 1974, diphenylethers were used on about 96% of the cultivated rice paddy fields in Japan. The compounds are not toxic to fish or shellfish. Matsunaka provided evidence that light was required for the diphenylethers to become phytotoxic.[14] The oxadiazoles were introduced by Rhone Poulenc in 1969, and were effective, in rice culture, when applied in a granular formation or as an emulsifiable concentrate.[15] The diphenylethers, diazoles, and phthalimides inhibit the activity of protoporphyrinogen oxidase (Protox).

In the presence of Protox inhibitors, tetrapyrroles, especially protoporphyrin IX (proto IX), accumulate. Protox inhibition leads to the accumulation of its substrate protoporphyrinogen, which is readily oxidized to proto IX by oxidative enzymes. Proto IX is a quite effective photosensitizer and in the light it transfers absorbed energy to molecular oxygen to form singlet oxygen. The singlet oxygen peroxidizes lipids leading to the destruction of cellular membranes.[16]

Bleaching Herbicides (Fig. 5)

Both norflurazon, a pyridazinone which was introduced by Sandoz in 1967–70 for general selective weed control, and fluridone which was introduced by Elanco in 1976 for the control of aquatic weeds, inhibit the biosynthesis of carotenoids and induce chlorosis. Carotenoids are terpenoids synthesized by the isoprenoid pathway. Carotenoid biosynthesis, in higher plants, occurs only in plastids. Carotenoids protect chloroplasts against photooxidation. Chlorosis occurs when the synthesis of carotenoids is inhibited. In most cases, phytoene desaturase is inhibited by these herbicides.[17] Transgenic plants conferring resistance to the phytoene desaturase inhibitors have been obtained by introducing the gene encoding the herbicide insensitive phytoene

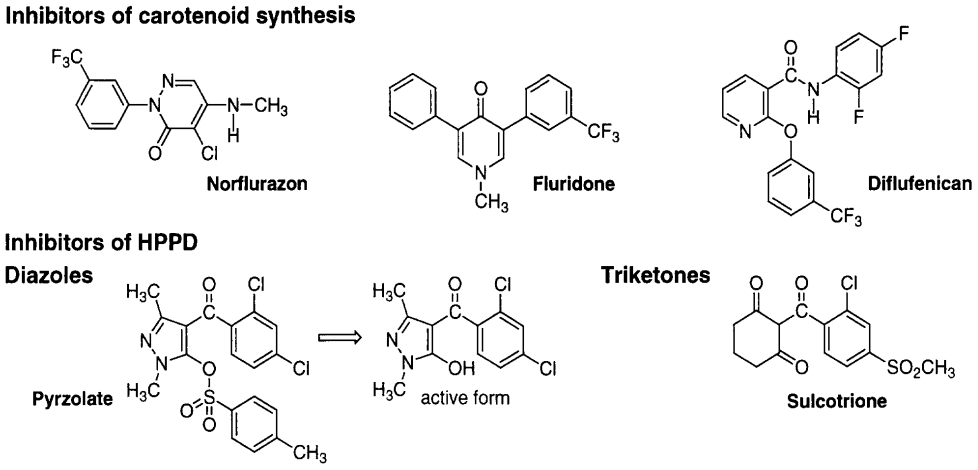

Fig. 5 Bleaching herbicides.

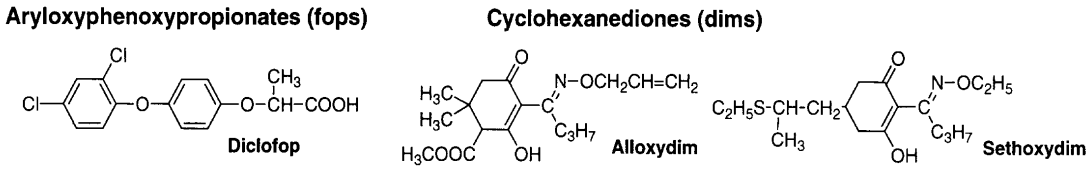

Fig. 6 ACCase inhibitors.

desaturase from *Erwinia uredovora*.[18,19] There are bleaching herbicides with a different inhibition site. Triketones and pyrazole herbicides block biosynthesis of plastoquinone, an important cofactor for phytoene desaturase, by inhibiting *p*-hydroxyphenylpyruvate dioxygenase (HPPD).[20]

Inhibitors of Fatty Acid Biosynthesis (Fig. 6)

Aryloxyphenoxypropionates (fops) block the *de novo* synthesis of fatty acids by inhibiting the activity of acetyl-CoA carboxylase (ACCase). Diclofop-methyl is the prototype and was commercialized by Hoechst in 1971. In higher plants, the activity of ACCase is strongly enhanced by light. The propionate free acids are the herbicidally active forms. These also contain an optically active carbon close to the carboxyl group. Only the *R*-enantiomer is herbicidally active. Cyclohexadiones (dims), such as sethoxydim which was introduced by Nippon Soda around 1977, are also potent inhibitors of ACCase activity. The ACCase inhibitors are applied post-emergently to control grasses. Plants contain, in their plastids and cytoplasm, two general types of ACCase, referred to as the eucaryotic and procaryotic forms. Grasses possess the eucaryotic form, whereas dicots have both forms.[21,22] The eucaryotic form is far more sensitive to these herbicides than the procaryotic one. The action of both the fops and dims has been reviewed recently by Burton.[23]

Inhibitors of Amino Acid Biosynthesis (Figs. 7 and 8)

In 1966, the herbicidal properties of sulfonylureas were reported. DuPont received their first patent in 1977 followed in 1982 by a Ciba-Geigy patent. By 1987, over 230 U.S. patents had been issued to about 18 companies. The imidazolinones were introduced around 1986 by American Cyanamide. Both groups of compounds inhibit the activity of acetolactate synthase which is also called acetohydroxyacid synthase and are frequently called ALS or AHAS inhibitors.[24] The herbicides block the biosynthesis of the branched-chain amino acids leucine, isoleucine, and valine. The pathway is present in plants, but not in animals. All three amino acids are nutritionally essential for animals and must be obtained by animals through their food chain. The triazolopyrimidines and pyrimidinyl(thio)oxybenzoates are also ALS inhibitors.

There are two other herbicides, glyphosate and glufosinate, that inhibit biosynthesis of amino acids in plants. They are general, non-selective herbicides, but are playing a pivotal role, at least in U.S. agriculture, especially in the development of herbicide-tolerant crops.

Glyphosate, a broad spectrum herbicide, was introduced by Monsanto in 1971. It inhibits the biosynthesis of the aromatic amino acids tryptophan, phenylalanine, and tyrosine by blocking an enzyme referred to as EPSPS or EPSP synthase (5-*enol*pyruvylshikimate 3-phosphate synthase).[25] Animals are unable to synthesize these essential amino acids, but must obtain them from plants in their food chain. Hence, glyphosate is not toxic to animals. A three-dimensional structure of the enzyme from *Escherichia coli* has been determined by crystallographic techniques.[26]

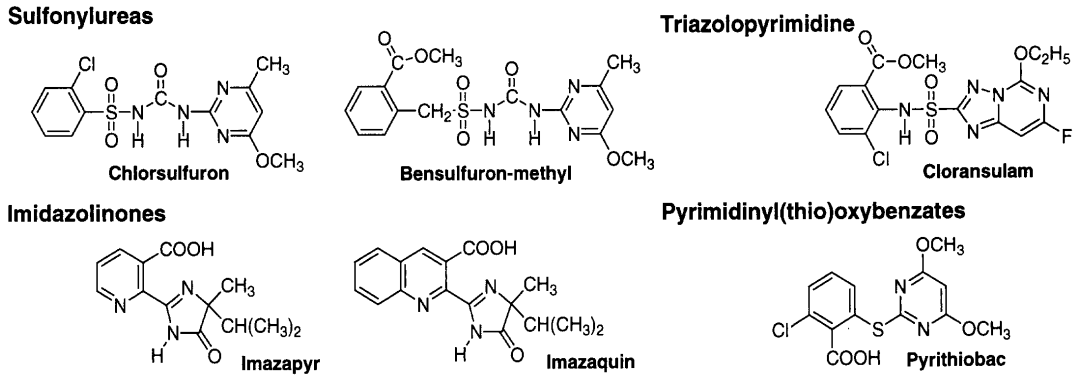

Fig. 7 ALS inhibitors.

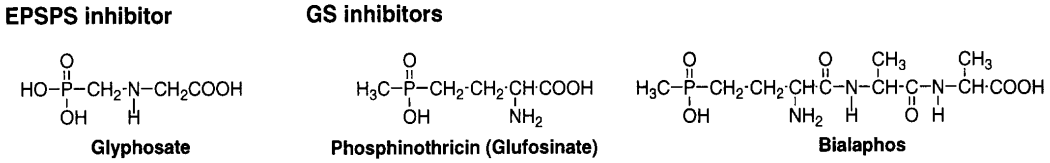

Fig. 8 EPSPS and GS inhibitors.

Marked weed resistance to glyphosate has not developed over the past 20 years, except for two ryegrass populations in Australia.[27] It is considered to possess a low risk for the development of weed resistance. Glyphosate has only limited residual activity in the soil and is relatively quickly metabolized by microorganisms.

Bialaphos is a fermentation product of *Streptomyces* spp. It was structurally identified independently by both Japanese and German scientists in 1972–73 as a tripeptide (L-phosphinothricyl-L-alanyl-L-alanine). It is metabolically hydrolyzed at the first peptide linkage to yield phosphinothricin which inhibits glutamine synthetase (GS), hence, can be classified as a proherbicide.[25] Bialaphos was introduced commercially around 1976 by Meiji Seika Kaisha. Phosphinothricin was subsequently produced synthetically and marketed as glufosinate by Hoechst (Hoe 39866) in 1981. Only the L-isomer is herbicidally active. The GS reaction is one of the few reactions through which inorganic nitrogen is converted into an organic form by organisms. Inhibition of GS results in an accumulation of ammonia to toxic levels. At one time, this was considered to be the cause of phytotoxicity. However, some investigators have suggested that inhibition of GS causes the concentration of glyoxylate to elevate which inhibits RuBP carboxylase, the first enzyme involved in carbon fixation. GS has been crystallized from *Salmonella typhimurium* and an atomic model has been determined by X-ray diffraction.[28]

As discussed previously herein, the biochemical mechanisms through which toxicity is expressed have been identified for most of the major groups of herbicides. However, there are still some herbicides, including the phenoxys and chloroacetamides, whose mechanisms continue to challenge investigators. Mechanism studies were primarily laboratory curiosities through the 1980s. However, the studies have found a meaning and are being used to develop: (a) strategies for delaying the appearance of herbicide-resistant weeds, and (b) herbicide-tolerant cultivars of crop plants.

Resistance

Resistance to a pesticide becomes evident when some individuals of a sensitive species are no longer killed by the chemical at doses that were previously toxic. Unfortunately, the terms *resistance* and *tolerance* are used interchangeably by many individuals. We tend to say that weeds are *resistant* to a herbicide when they are no longer killed by a given rate of the chemical. However, when a crop plant is genetically altered so that it is not killed by a given rate of a herbicide, we tend to say that the plant *tolerates* the herbicide. One of the major problems that has become associated with the use of pesticides is the development of resistance by the target organism.

Resistance of insects originally showed up with inorganic treatments during the late 1800s and early 1900s, and increased in importance with the introduction of organic pesticides. DDT was the first organic insecticide to be used on a world-wide basis (1942–45). Resistance to DDT was identified within a year or two after its introduction (1946–47) in house flies in Sweden and Italy.

Those of us involved with herbicides were aware of the insect resistance phenomenon, but were naively confident that we had nothing to worry about because of the longer life cycles associated with plants. We were surprised with the first published reports of resistance to the tri-

azines in 1970.[29]

Elucidation of the molecular basis for triazine resistance has provided an understanding of the photosynthetic reaction center in chloroplasts. Crystallization of the photosynthetic reaction center from *Rhodopseudomonas viridis* and elucidation of its X-ray structure led to the award of a 1988 Nobel Prize in Chemistry to 3 German scientists.[30]

The D-1 protein consists of 353 amino acids. Triazine resistance was shown to be associated with a replacement of serine (S) by glycine (R) at position 264, *i.e.*, susceptible plants had a serine at this position and resistant plants had a glycine.

The development of herbicide resistance has become of great concern to the users of herbicides. Heap, in 1997, reported, based on a 1995–96 International Survey that there were 183 herbicide-resistant weed biotypes (124 different species) in 42 countries and that approximately 9 new observations of resistance were being reported worldwide each year.[31]

To prevent or slow down the development of herbicide resistance in weeds, farmers are being advised to use herbicides with different mechanisms of action in mixtures, sequences, and rotations. Herbicides have been classified, by their mechanisms of action, by Herbicide Resistance Committees (HRC) for use in the U.S.[32] in Europe[33] and in Australia.[34] In Australia, a code for the mechanism of action has been added to the labels on the containers of herbicides.

Cytochrome P450 Monooxygenases

The most common biochemical mechanisms associated with resistance are an increased rate of metabolism of the parent compound to non-toxic forms, or a decreased affinity of the target site for the toxicant. The microsomal cytochrome P450 monooxygenase system is the main oxidative degrading system for xenobiotics in all organisms. The single amino acid substitution in the D-1 chloroplast protein associated with triazine resistance is one example of decreased binding affinity.

The P450 system provides intermediates for a large number of biosynthetic pathways and metabolizes a very large number of exogenous compounds including pharmaceuticals, insecticides, fungicides, and herbicides. The system has been studied more intensively in animals than in plants because of the high concentration of the system in mammalian livers, and its ease of isolation.

Those of us who work with plants have long envied the success that mammalian and insect physiologists and biochemists have enjoyed in isolating and characterizing the P450 system of mammals, insects, and microorganisms. For many years, investigators have experienced problems in isolating catalytically active microsomes from higher plants. There are a number of reasons for this:

1. Plants have tough cell walls that have to be mechanically disrupted. This releases vacuolar contents which tend to denature the P450.
2. Plant cells also contain endogenous compounds that inhibit the activity of the P450 enzymes.
3. Pigments in plant cells, especially chlorophyll, interfere with spectrophotometric determinations.
4. When successful, the P450/protein ratios are quite low. Most crude rat liver preparations average around 1 nmole P450/mg protein, whereas some of the better plant preparations will range around 100 to 150 pmoles P450/mg protein.

The metabolism of more than 20 herbicides by plant microsomes including sulfonylureas, imidazolinones, substituted ureas, chloroacetanilides, diclofop, bifenox, flumetsulam, and bentazon has been reported.[35]

Modulation of the Activity of the P450 System

Chemicals can be used to modify the activity of the P450 system. The rate of metabolism of a pesticide can be decreased by compounds called *synergists*, or increased by chemicals referred to as *inducers* in animal studies, but as *safeners* in plant studies.

The major chemical strategy for increasing the toxicity of insecticides in dealing with resistant populations is to add a synergist. Synergism becomes evident when there is an increase in toxicity of a given pesticide after exposure of an organism to a second, mostly non-toxic, chemical. The net effect is a decrease in tolerance of the pest for the pesticide, *i.e.*, concentrations of the pesticide that were previously non-toxic to the pest are now toxic. Synergists were used commercially before their mode of action was known. In mammals, most synergists are substrates for the P450 system, hence, they act as competitive substrates. Many synergistic inhibitors of the animal P450 system also inhibit some plant P450s. However, the intentional use of synergists with herbicides has not received as much attention as that for insecticides.

Synergism between two herbicides became evident with tridiphane and atrazine. Tridiphane was introduced by Dow as a post-emergence grass herbicide, but was not a very strong herbicide. Atrazine is rapidly degraded by panicoid grasses. The addition of tridiphane slowed down the rate of metabolism in the grasses and the concentration of atrazine could be reduced by a factor of 10, *i.e.*, from 4.7 kg/ha to 0.45 kg/ha. Hence, the addition of tridiphane increased the toxicity of atrazine to the panicoid grasses. In the panicoids, atrazine is efficiently conjugated by glutathione (GSH). The reaction is catalyzed by glutathione *S*-transferases (GSTs) which are inhibited by tridiphane.[36]

Synergisms became a problem when some insecticidal organophosphates and carbamates were used with certain herbicides. Propanil, in rice and other tolerant plants, is hydrolyzed by an aryl acylamidase. The synergistic

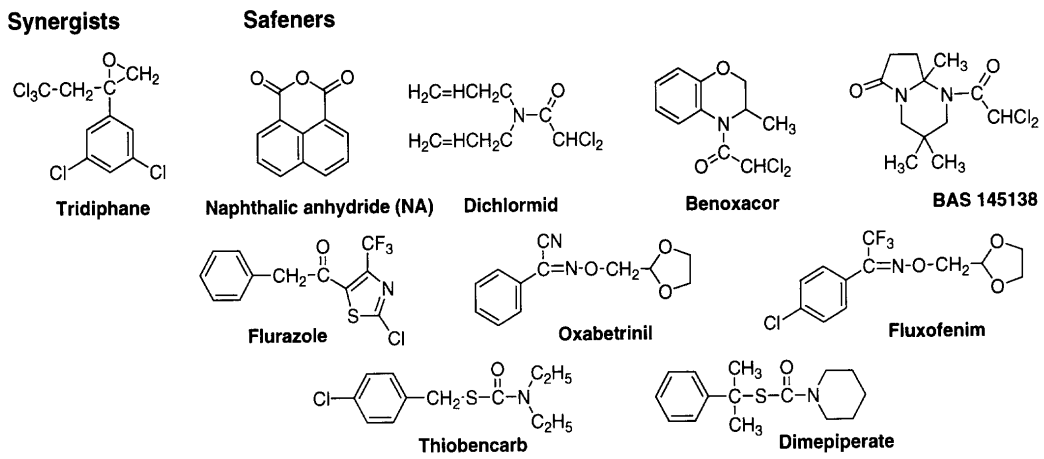

Fig. 9 Synergists and safeners.

insecticides are considered to act by inhibiting the activity of the acylamidase, hence, propanil becomes toxic to rice. The original observation by Matsunaka[37] involved the insecticide carbaryl. Synergism again presented a problem, in rice and corn production, when the insecticides were used in combination with sulfonylurea herbicides.[36]

Tolerance Based on Enhanced Metabolic Inactivation (Fig. 9)

Herbicide safeners are used to increase the tolerance of crop plants to herbicides. They have also been referred to as antidotes, protectants, or antagonists. The use of the word "safeners" seems to have become of general use and avoids confusion over other types of activity.[38]

Metabolism of pesticides initially involves an oxidation or hydrolytic reaction which provides a functional group suitable for subsequent conjugation. The conjugates are generally more hydrophilic and less mobile than the parent compound. Glutathione is one of the major conjugates in both plants and animals. Conjugates are also formed with glucuronic acid, catalyzed by glucuronidases, in animals and glucose, catalyzed by glucosidases, in plants. Energized transporters transfer the conjugates to the excretory system in animals or to the vacuole in plants. Safeners, applied as seed treatments or in combination with the herbicide in field applications, enhance metabolism by increasing the activity or concentration of all the components involved in the above reactions.

Otto Hoffman with Spencer Chemical first began reporting on chemicals that could be used to protect plants from the toxic effect of herbicides in 1962.[39] Naphthalic anhydride (NA) was marketed as Protect by Gulf Oil in 1972 and dichlormid was patented as Eradicane by Stauffer Chemical in 1971. Both compounds were marketed as maize seed treatments for protection against injury from thiocarbamate herbicides. BASF introduced BAS 145138 in 1984. Several safeners were developed to protect grain sorghum seed from injury by chloroacetamide. These included Screen (flurazole) by Monsanto in 1980, oxabetrinil by Ciba Geigy in 1982 and fluxofenim in 1986. Benoxacor was introduced as a corn treatment.

Bensulfuron-methyl (Fig. 7) was introduced by DuPont in 1984 for use in direct-seeded and transplanted rice. Some injury to *Japonica* rice varieties was observed when the herbicide was used at high rates (100 g/ha). Injury could be reduced when thiocarbamate herbicides including thiobencarb and dimepiperate were applied in combination with bensulfuron-methyl. The safeners were shown to increase the rate at which bensulfuron was metabolized to a non-toxic form.[40] Metabolism involved *O*-demethylation of one of the methoxy groups to a hydroxy group, conceivably a P450-mediated reaction, followed by conjugation with glucose.

Safeners have been developed primarily for use with monocot crops (maize, sorghum, wheat, and rice) to increase tolerance to sulfonylureas, imidazolinones, aryloxyphenoxypropionates, cyclohexanediones, thiocarbamates, and chloroacetamides.

Herbicide-Tolerant (Genetically Modified) Crops

Over the past 10 or so years, the introduction of new herbicide chemistry has slowed down. During this same time interval, advances in biochemistry, physiology, and molecular biology have enabled the development, production, and commercialization of several herbicide-tolerant crops (maize, soybean, canola, cotton, sugar beet, wheat, and others).[41]

The tolerant cultivars have been developed by: 1) altering the architecture of the target site, 2) overexpressing a target enzyme, or 3) increasing the plant's capacity to detoxify the herbicide. Tolerant cultivars have been produced through traditional breeding, cell and tissue culture selection, mutation, and transformation. Genes that confer tolerance of higher plants to herbicides have mainly been transferred from microbial sources. How-

ever, Ohkawa and his associates at Kobe University have transferred a human P450 gene into tobacco, rice, and potato.[42]

In some crops, the Bt gene has been inserted in addition to the one that confers herbicide tolerance. BT transgenic plants contain a gene from the soil bacterium *Bacillus thuringiensis* that enables the plant to produce a protein toxin that kills many types of caterpillars. In the U.S., EPA approved insertion of the gene into potatoes in 1995 and later to maize and cotton. The toxin does not injure most insects or warm-blooded animals including man. Because the toxin is produced by the plant throughout the growing season, there is a real risk for the development of resistance to the Bt toxin.

Development and Trends in the Introduction of Herbicide-tolerant Crops

The engineering of tolerance to existing herbicides in crops seems to represent an attractive option to industry. By emphasizing the introduction of herbicide-tolerant crops, the large costs associated with discovery, registration, and introduction of new herbicides have been eliminated. More effort is being made by the companies to expand the use of existing products.[43,44] There is no research being supported by the U.S. Department of Agriculture to create herbicide-tolerant crops for commercial purposes.

Commercial development in the U.S. and Canada has largely focused on the non-selective, environmentally and toxicologically safe herbicides, glyphosate and glufosinate. Development and introduction of herbicide-tolerant crops in the United Kingdom, Europe, and Japan has been limited by a number of issues that have technical, political, ethical, and moral implications.

The Future of Chemical Weed Control

What does the future hold for chemical weed control? Are there undiscovered, sensitive, biosynthetic pathways in plants that can serve as targets for new herbicide chemistry? The use of glyphosate and glufosinate with crop seed tolerant to them is rapidly becoming the foundation for chemical weed management in the U.S. Is too much reliance being placed on just two herbicides with this technology? Will the combinations work consistently under all environmental conditions? What impact will this new practice have on the chemical industries that developed other herbicides for use in the same major crops? Will weeds now controlled by glyphosate and glufosinate eventually develop tolerance to the chemicals? Answers to these and other concerns should be provided over the next few years after farmers have had time to adjust their management practices to the new materials and to test responses under a variety of environmental conditions.

The number of companies involved in herbicide discovery, development, and marketing has decreased significantly over the past several years. In the U.S. there are only about one-half the number of companies that there were 15 or 20 years ago. Some of this decrease has been caused by mergers. The rate of attrition, in the U.S., has been projected to continue. In Europe, the attrition rate has been much slower. Little or no attrition seems to have occurred in Japan.

CONCLUSION

At this time, no one can predict what genetic engineering will mean to us in the future. I have only mentioned its role with regard to herbicide tolerance in plants. Additionally, there is a tremendous amount of effort being expended to increase yields; to improve the quality of the consumed product; to manipulate constituent biochemicals; to increase tolerance to insects, pathogens, drought, and frost; and even to add plant-produced vaccines to our diets. We are just beginning to understand the power of biotechnology. Some writers postulate that biotechnology will have the same impact on agricultural technology in the 21st century that plant breeding, organic pesticides, and inorganic fertilizers had in the 20th century.[45]

ACKNOWLEDGMENTS

This paper is based on a presentation delivered at the 24th Annual Meeting of the Pesticide Science Society of Japan held in Utsunomiya March 24-27, 1999. The author is most appreciative of having had this opportunity and would especially like to thank Prof. Toshio Shono, retiring President, Dr. Isamu Yamaguchi, incoming President, and Prof. Yasutomo Takeuchi, Chairman of the Organizing Committee. The author is also grateful for the invaluable suggestions, critical reading, and assistance in the preparation of the manuscript provided by Prof. Koichi Yoneyama.

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