Pesticide Removal from Water

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Executive Summary

Commercial pesticide use has the potential to contribute to significant human symptomatic illnesses, both acute and toxic. These may result from direct pesticide contact during application as well as from mobile or persistent pesticides that remain in the soil which can over time enter groundwater and drinking water supplies. Pesticide persistence or mobility in the environment varies with a multitude of conditions, including surrounding soil and temperature conditions as well as the solubility, degradation kinetics, and other properties of the pesticide. The EPA, European Union, and other international organizations set standards for safe pesticide use as well as maximum levels of pesticides that may be present in drinking water. These and other regulations attempt to minimize detrimental effects of pesticides and their degradation byproducts on humans, wildlife, and the ecosystem.

Water treatment facilities employ a number of treatment techniques to reduce potentially harmful and disruptive contaminants, such as pesticides. Some techniques include, but are not limited to, adsorption to activated carbon, reactions with oxidants such as potassium ferrate and chlorine, and free radical degradation using ozonation and UV exposure. The goal of this project was to test these techniques for their effectiveness and their respective feasibility of implementation. In order to test these techniques, three pesticides were chosen for evaluation: glyphosate, alachlor, and atrazine. These are three commonly used pesticides which have been linked to detrimental health effects.

Standard solutions were prepared for each chemical, and concentrations were correlated to UV absorbance in a spectrophotometer. This enabled measurement of pesticide concentrations in solutions after treatment. Granular activated carbon was contacted with pesticide solutions with varied mass ratios relative to the mass of pesticide in water: from 1:1 to 50:1, at a pH of 7. Removal was measured after 24 hours of contact, followed by centrifugation. Aqueous calcium hypochlorite was added in molar ratios from 1:1 to 25:1 and agitated for 24 hours, at pH 4 and 9. Potassium ferrate was added in molar ratios from 1:1 to 25:1 and agitated for 24 hours at pH 3. UV radiation was supplied by a low-pressure 254 nm wavelength lamp for residence times ranging from 5 to 90 minutes, with and without hydrogen peroxide addition in molar ratios from 1:1 to 100:1. Ozone gas was applied to solutions for contact times of 5 to 90 minutes. All three pesticides were treated with activated carbon and UV. Alachlor and atrazine were also treated with chlorine and ozone. None of the three pesticides were effectively treated with ferrate since no feasible method for removal of the insoluble byproducts could be developed.

Activated carbon and UV with hydrogen peroxide were most successful at reducing pesticide concentrations. Activated carbon at mass ratios of 25:1 for alachlor, 18:1 for atrazine, and 15:1 for glyphosate removed 98, 90, and 51%, respectively, after 24 hours. UV without hydrogen peroxide led to significant byproduct formation and persistence for alachlor and glyphosate, but removed 87% of atrazine in 75 minutes according to first order degradation. UV with 25:1 molar ratios of hydrogen peroxide led to byproduct formation as well but with continued contact removed 95% of alachlor and 52% of glyphosate after 90 minutes, and 92% of atrazine after 120 minutes with a molar ratio of 100:1. Removal of alachlor and atrazine was first order; removal of glyphosate was zero order. Chlorine (hypochlorite ion) at 25:1 molar ratios removed a maximum of 59% of alachlor and 11% of atrazine at pH 9. Ozonation led to significant byproduct formation for alachlor, tested for 90 minutes, but removed 17% of atrazine in 30 minutes.

Byproduct formation was observed with UV exposure and ozonation for all tested pesticides, though the identities of the byproducts could not be determined using the UV spectrophotometer detection method. Byproducts then degraded with continued treatment but experiments were not conducted to attempt to determine the time to significantly reduce byproducts since large contact times would become impractical for use in a treatment facility.

Activated carbon adsorption and UV with hydrogen peroxide are therefore recommended as effective treatment techniques that can be implemented, and are already commonly used, in large-scale treatment facilities. Both were tested at pH 7. Chlorine use would likely require pH adjustment to maximize removal with the hypochlorite ion above the pK_a of 7.6. Recommendations for further research include hydrogen peroxide addition during ozonation. Further work to develop a method for quantifying concentration reduction using ferrate is required. Identification and monitoring of byproducts is recommended to develop further understanding of reaction kinetics.

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Introduction

A pesticide is any substance, chemical, biological or otherwise, that is used for the purpose of preventing, destroying, or controlling pests. Pests may mean any species of plants or animals that interferes with the desired plants' growth and harms its production, processing, storage, transport, or marketing. Pesticides also include substances that are used before or after the desired plant is harvested to protect it during storage and transport.[1] Ideally, an applied pesticide would target only the specific pest that is bothersome. This would be a narrow-spectrum pesticide. However, most pesticides are broad-spectrum and their effects cannot be limited to target individual pests. Beneficial organisms may be damaged by pesticides as well.

Pesticides can have many benefits and lead to greater crop harvests. They can also help to limit the health dangers that insects carrying diseases pose to humans. However, there are major problems with some pesticides also. Some pesticides' usefulness deteriorates over time when the pests they are targeting develop resistances to them. In these cases, larger concentrations of pesticides or applications of stronger, more toxic pesticides must be used. In some extreme cases, pests can develop resistances to all types of pesticides legally permitted for their treatment. Furthermore, the introduction of pesticides into the environment can lead to the imbalance of ecosystems if certain species' populations are altered in significant ways, the effects of which may be more widespread than considered since broad-spectrum pesticides can eliminate multiple species rather than merely the targeted species.

The detrimental environmental and health effects of pesticides on humans have been documented in the past decades. Prior to many studies, highly toxic pesticides were used in large quantities and in sensitive areas with great environmental and human exposure. For example, large-scale spraying of trees and plants in the 1960's was common. One pesticide used for this was DDT (dichlorodiphenyltrichloroethane) which has been shown to have significant health consequences.[2]

To humans specifically, pesticides can pose a threat when they leach into groundwater and enter into drinking water supplies. Degradation may occur during the time between application to crops and when the water enters a drinking water treatment facility. However, degradation may be a slow process and large amounts of the base compound may remain. The byproducts themselves may be more toxic than the base compound. Treatment facilities may not always feature adequate treatment methods to reduce pesticide concentrations. For example, in agricultural areas, heavy pesticide application may lead to difficult removal from water while in other areas, fewer pesticides may be present in water but they may be of more persistent nature. Treatment facilities not targeting specific pesticides in their water supplies (which may also vary seasonally or if accidental spills occur) may need additional water treatment techniques to reduce concentrations to a safe level.

The purpose of this research was to investigate a wide range of treatment methods for removing pesticides from E-Pure water, which is reagent-grade bacteria free water. This end was accomplished by experimenting with techniques that range from new technologies to traditional methods used in the water treatment industry. Common pesticides alachlor (trade name Lasso), atrazine, and glyphosate (trade name Roundup) were used as examples from the organochloride and organophosphate families of pesticides, to study the effectiveness of treatment methods for multiple types of pesticides. The data acquired for each treatment method was then evaluated against each other to determine the relative effectiveness of each method, in addition to inferring reasons for a methods success or failure.

Background

The following chapter presents a background on pesticide usage, the three specific pesticides studied here, and the five treatment methods studied here are established. The health and environmental impacts are stressed to demonstrate that pesticide contamination of water supplies is a current, significant problem for which continued data regarding new treatment techniques should be gathered.

Generations of Pesticides

First generation pesticides refer to the pesticides commonly produced and used prior to the 1940's. These first generation pesticides were organic pesticides, naturally-occurring, typically withdrawn from plant compounds. When drawn from plants, pesticides are called botanicals. They do not persist in the environment and are easily degraded, but can be very toxic to aquatic life before they have degraded.

Second generation pesticides refer to synthetic pesticides produced after the 1940's, which are modified forms of botanicals to have more targeted effects on pests. These can be more poisonous than first generation pesticides and are more likely to persist in the environment. Their persistence depends on their class and type of pesticide. Currently, over 2,000 types of pesticide products are commercially available.[2]

Types of Pesticides

There are many types of pesticides that target different types of pests: insecticides to kill insects, herbicides to kill harmful vegetation, rodenticides to kill rodents, fungicides to kill funguses, and so on. Pesticides may employ a number of different mechanisms to eliminate harmful pests. The types of pesticides used, classified by their treatment methods, include: chemical pesticides, biological pesticides, antimicrobials, and pest control devices. The major groups of chemical pesticides include organophosphates, carbamate pesticides, organochloride pesticides, and pyrethroid pesticides. They vary in the mechanism that targets and inactivates or inhibits pests.

Organochloride Pesticides

Organochloride pesticides were used heavily in the 1940's-1960's but are not as widely used today since they have a high potential for chronic health effects and they persist in the environment for months or even years. These chlorinated hydrocarbons are broad-spectrum. They are primarily used as insecticides. They can include chlorinated ethane derivatives such as DDT, cyclodienes, and hexachlorocyclohexanes.[3] Some that remain in use today include alachlor, atrazine, lindane, and methoxychlor.

The most famous type of organochloride insecticide is DDT (dichlorodiphenyltrichloroethane), perhaps one of the most well-known of all pesticides. The wide-spread toxic effects of DDT were studied by Rachel Carson and published in her 1962 book *Silent Spring,* which revealed the detrimental effects of pesticides on bird populations, particularly eagles and others at the top of the food chain, and the significant weakening of their eggs' shells. This book is sometimes credited with helping to truly launch the environmental movement and was published prior to the formation of the US Environmental Protection Agency in 1970.[2] DDT also has effects on the human immune system.

Organophosphates

Organophosphates (OPs) are insecticides that contain phosphorous and kill insects by targeting an enzyme that regulates the neurotransmitter acetylcholinesterase, disrupting brain function. Following the decreased usage of organochloride insecticides, organophosphates have become the most widely used today. They were originally developed during WWII. Some organophosphates are highly poisonous, comparable to poisons such as arsenic and cyanide. However, they degrade in the environment readily and do not have long-term environmental effects.[4] Because of this dichotomy, many organophosphates are used in large-scale agriculture settings but are not available on smaller scales because of their highly toxic properties. Some examples of organophosphates include glyphosate, dimethoate, and malathion.[2]

Carbamate Pesticides

Carbamate pesticides are insecticides that were derived from carbamic acid and functions in a way similar to organophosphates, inhibiting the cholinesterase enzymes. They were first introduced in the 1950's and remain widely used because of their relatively low toxicity compared to other insecticides, particularly the organophosphates. Like the other types of insecticides, these can affect the human nervous system with routes similar to those that affect the target insects. Respiratory problems result from poisoning, but the inhibition of acetylcholinesterase is reversible so short-duration exposure may not be extremely detrimental.[5] Two common carbamates are carbaryl and aldicarb.

Pyrethroids

Pyrethroids were synthesized to have the same effects as the naturally-occurring pesticide pyrethrum, extracted from the chrysanthemum flower, but be increasingly stable without persisting in the environment.[4] They are widely used. An example of a pyrethroid is cypermethrin. However, the effects of pyrethroids on the human immune system have not been extensively studied since they were developed relatively recently.[3]

There are hundreds of types of each of these 4 types of chemical pesticides.

Legislation

The US federal government has passed many laws surrounding the use of pesticides. After pesticides' application, laws also govern acceptable residue limits found on food and the allowable contaminant levels found in drinking water and surface water bodies. Internationally, the World Health Organization and divisions of the United Nations work to maintain standards for pesticide use as well, in addition to foreign governments. The European Union also sets standards to regulate concentrations in water and on foods.

Domestic Legislation

Some of the major laws governing pesticide use within the US are the Food, Drug, and Cosmetics Act (FDCA), the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA), the Food Quality Protection Act, the Clean Water Act, and the Safe Drinking Water Act. The Environmental Protection Agency (EPA) is empowered by these laws to monitor pesticide registration, use, and concentrations in foods and water supplies.

Food, Drug, and Cosmetics Act (FDCA)

This act, originally passed in 1938, was amended in 1954 to allow for the establishment of standards for acceptable and unacceptable levels of pesticides found in food. This was the first means for regulating pesticide levels in foods. With a later amendment called the Delaney Clause added in 1958, it also specifies that no processed foods can contain any pesticides that have been shown to cause cancer in animals during laboratory tests. However, this clause did not cover raw foods such as vegetables, meats, or milk, and also was difficult to enforce since not a lot of data was available at the time to link specific pesticides to cancers.[2]

Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA)

The Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) passed in 1947 and amended in 1983 and 1988 requires the registration of all pesticides used in the US and sets standards for their distribution, sale, and use. This act was passed to help prevent the use of pesticides that were no longer usable and would instead cause damage to users or to the environment. When a pesticide is registered, the EPA investigates the type of pesticide, the area it is intended to be used and in what quantities, and the storage and disposal methods. The pesticide must meet the standards set up the FDCA in order to be granted registration.[6] However, some critics are dissatisfied that FIFRA does not require pesticide manufacturers to disclose all the inert ingredients in the pesticides, only the active ingredients, when inert ingredients may pose health and environmental dangers as well since some common inert ingredients include toxins such as benzene, lead, and formaldehyde.[2] For instance the surfactant POEA in commercial formulations of glyphosate such Round Up has been shown to be much more acutely toxic to humans and wildlife than glyphosate alone. The lethal dose of POEA is less than a third of the lethal dose of glyphosate.[7]

Evaluation of Carcinogenic Potential

Carcinogenic potential is one important part of the hazard assessment that the EPA does. This involves laboratory testing with rats and mice. After research is conducted, the Cancer Assessment Review Committee assigns each active ingredient within the pesticide a cancer classification, which the EPA then uses to determine regulations surrounding the pesticide's use.

Based on the research conducted, pesticides can be assigned one of five different levels of carcinogenicity.

Table 1: Levels of Carcinogenetic[8]	
1	Carcinogenic to humans – though studies are based on animals, similar mechanisms observed between animals and humans can suggest conclusions that some compounds are likely to be carcinogenic in humans. The only pesticides of this group that are registered for legal use are arsenicals, but the use of these, typically for wood treatment, is strictly monitored and has been reduced significantly as of 2003.
2	Likely to be carcinogenic to humans – this classification results from data that demonstrates carcinogenic potential. An example from this group is imazalil, a fungicide used in citrus agriculture.
3	Suggested evidence of carcinogenicity, but not sufficient to asses human carcinogenic potential – further studies are required to determine the true human carcinogenic potential in this case. Pyrethrins fall into this category.
4	Data are inadequate for an assessment of human carcinogenic potential – there is a lack of sufficient data, or different studies may contradict one another. Further studies are required. Pyraclostrobin, used for fruits and vegetables, is an example from this category.
5	Not likely to be carcinogenic to humans – studies have revealed that the pesticide does not pose a threat to humans. This may be deduced from studies of human exposure or from animal studies that are shown to be relevant to humans. An example would be glyphosate.

Food Quality Protection Act

Passed in 1996, this Food Quality Protection Act amended both the FDCA and FIFRA to set pesticide residue limits for all foods—raw or processed. It also refined the pesticide limits to include all health risks rather than simply cancer, and to take into consideration the higher risks that children and infants face. This act also sharply reduces the amount of time between when a pesticide is banned to the time it must be removed completely from use (from 10 years to 14 months).[2]

Clean Water Act

The Clean Water Act (CWA), originally passed in 1948 and expanded in 1972 with amendments in 1977 is the primary federal law monitoring water quality which sets the structure for regulating concentrations of pollutants in surface water supplies. Ground water is not described in this law. It protects "navigable" water bodies by limiting point source discharges that manufacturers and other facilities may make into surface water bodies with the goal of making them safe for fishing and swimming.[9] Non-point sources are more difficult to monitor and regulate, and the approach for this involves education, technical assistance to manufacturers, and similar approaches. Water quality standards specify water quality standards (WQS) for allowable pollutant levels that water bodies must meet, involving total maximum daily loads (TMDLs).[10]

The basis of these standards is the protection of aquatic life as well as human health. Aquatic life must maintain a certain quality of water, including sufficient oxygen and nutrient levels, limits on alkalinity, dissolved solids, turbidity, dissolved metals, and other potential pollutants. pH must be in the range of 6.5 – 9. Temperature is species dependent for the aquatic life criteria. Human life criteria consider pollutants effects upon humans and the environment.[9]

Safe Drinking Water Act

This act was first passed in 1974 and amended in 1986 and 1996. It regulates drinking water quality in public water supply systems as well as their sources. The EPA sets primary and secondary drinking water standards that water treatment facilities must comply with before discharge to public water distribution systems. These standards involve treatment processes that must be included as well as permissible contaminant levels in the plant's effluent. States may also set their own drinking water standards as long as they are at least as stringent as the national standards.[11]

Under this portion of the law, both drinking water health regulations and advisories are made. The National Primary Drinking Water Regulations (NPDWRs) specify maximum contaminant levels (MCLs) of a contaminant that is the highest permissible and safe concentration in water discharged to public water systems. These contaminants include microorganisms, disinfectants and byproducts, inorganic chemicals, organic chemicals, and radionuclides. Many pesticides have specified MCLs, including alachlor (0.002 mg/L), atrazine (0.003 mg/L) and glyphosate (0.7 mg/L).[12] Potential health effects are included in this analysis. Table 2 lists some of the organic chemicals' MCLs listed by the EPA. The full list is shown in Appendix A. Maximum contaminant level goals (MCLGs) are non-enforceable recommendations below which there is no expected health effects.[12]

Secondary standards also exist which encompass aesthetic aspects or cosmetic effects. Drinking water advisories are made, which provide information and about contaminants' health effects upon humans.[12]

Table 2: Organic Chemicals' MCL's (Incomplete)[12]

Contaminant	MCLG[1] (mg/L)[2]	MCL or TT[1] (mg/L)[2]	Potential Health Effects from Long-Term Exposure Above the MCL (unless specified as short-term)	Sources of Contaminant in Drinking Water
Acrylamide	zero	TT[8]	Nervous system or blood problems; increased risk of cancer	Added to water during sewage/wastewater treatment
Alachlor	zero	0.002	Eye, liver, kidney or spleen problems; anemia; increased risk of cancer	Runoff from herbicide used on row crops
Atrazine	0.003	0.003	Cardiovascular system or reproductive problems	Runoff from herbicide used on row crops
Benzene	zero	0.005	Anemia; decrease in blood platelets; increased risk of cancer	Discharge from factories; leaching from gas storage tanks and landfills
Benzo(a)pyrene (PAHs)	zero	0.0002	Reproductive difficulties; increased risk of cancer	Leaching from linings of water storage tanks and
Carbofuran	0.04	0.04	Problems with blood, nervous system, or reproductive system	Leaching of soil fumigant used on rice and alfalfa
Carbon tetrachloride	zero	0.005	Liver problems; increased risk of cancer	Discharge from chemical plants and other industrial
Chlordane	zero	0.002	Liver or nervous system problems; increased risk of cancer	Residue of banned termiticide

Monitoring Foods for Quality

The Food and Drug Administration (FDA) and US Department of Agriculture (USDA) enforce the tolerance limits for residues of pesticides left on foods that are sold for human consumption. The limits are set by the EPA. If the residue concentrations are found to be greater than the allowable standards, the food must be destroyed, which provides incentive for farmers to abide by the limits.[13] More recently, the EPA has increased the number of tests conducted on foods commonly eaten by infants and children in attempts to more closely protect the more susceptible younger populations.[14]

International Legislation

Internationally, the International Code of Conduct on the Distribution and Use of Pesticides was passed in 1985 by the United Nations' Food and Agriculture Organization (FAO) and revised many times since then, the latest in 2002, to set voluntary standards for the use of pesticides. Though countries are not obligated to abide by these standards, they help raise awareness of the potential consequences associated with use of pesticides and serve as a reference that is considered the "globally accepted standard for pesticide management".[1] National legislatures maintain the standards that each country must abide by but in some cases, particularly in developing countries, monitoring compliance with laws is difficult.

European Union Legislature

More formally, the European Union (EU) has detailed legislation surrounding the use of pesticides in member nations. In doing this, the EU has separated its legislation into two main divisions: the classification and usage of pesticides, and the official maximum residue level for each compound. These two divisions work together to set a standard for pesticide restriction in order to keep the general public safe.

Classification and Usage

The first initiative in which the European Union started to standardize the restriction and legislation of pesticides occurred in 1993 when Directive 91/414 was passed by the EU. This directive stated that over the next 14 years a council was to be created to review all pesticides, their uses and the products in which they were found. The review would establish whether the pesticides and products were harmful to the community and would either allow continued production of the pesticide or ban the product and/or pesticide. This stemmed from concern regarding the effects of pesticides on the food market both within and outside the European Union. [15]

The classification of the pesticide would be based on information from manufacturers, regarding pesticide efficiency, main purpose, and potential harm to humans and the environment. The classification would simply list the chemicals followed by this information. The EU would then make a decision to either ban or allow the continued production of this pesticide. This process, otherwise known as risk assessment, took into account the possible damage the pesticide could present should there be a contamination in the local water system. Risks to humans as well as the possible risks to the environment and wildlife were of paramount importance. In December of 2008, the review process was extended until 2010 and then later until 2012. Prior to this directive the legislature on pesticide restriction was dealt with on the national level only.[15]

The next directive, Directive 2009/128/EC, was passed in November 2009 and states that each national community will develop and/or adopt a National Action Plan. This plan would be used on the national level to reduce the risk to human and environmental life when dealing with pesticide usage. The hope of this Directive is to focus on the pesticide concentration in food products. The plan was passed in hopes to advocate the research and development of new techniques and delivery methods.[15]

In May 2011, the Plant Protection Products Regulation Act established a list of approved chemicals and products for use in the EU. This list shows the accepted purity, date of approval, as well as the expiration of approval. Once a pesticide's approval expires, the review committee reevaluates the chemical and may either continue to approve the usage or ban the pesticide. Coinciding with Directive 91/414, a list was also established which lists banned substances in the European Union.[16]

Maximum Residue Level

The second division of the European Union's pesticide legislation involves the investigation of each pesticide in order to establish a maximum residue level. This maximum residue level is the maximum concentration that is found in local crops. The European Union defines these levels as the "highest possible level of a pesticide residue that is legally authorized in food and feed." In September 2008, the EU passed Regulation 396/2005. This regulation set a standard for maximum residue levels in all EU governed states. This regulation was to ensure that a product would not be legal in one country and yet above the maximum residue level in another community.[17]

Health Impacts and Toxicity

In addition to the desired effects on targeted pests, pesticides can also have detrimental effects on human health. Acute effects occurring within minutes or hours after a single exposure, as well as chronic effects spanning multiple exposures and weeks to years can be sustained. Various mechanisms of contact with the pesticide can lead to different symptoms. Chronic toxicity is much more difficult to monitor and observe since the effects are widespread, and depend on individuals' various degrees of contact with the substance and the individuals' own health affected by genetics and various other factors, and may have synergistic effects with other substances or lifestyles the individual was exposed to. Chronic effects include birth defects, tumor formation, cancer, blood disorders, and nerve disorders. Both long and short-term effects are tested by subjecting test animals to concentrations of pesticides to simulate human effects, either for short exposures or, in chronic tests, the equivalent of extended exposures.

Toxicity Information

Dermal contact is responsible for the majority (~90%) of pesticide poisonings, typically during pesticide application, handling, or other routine uses.[13] Ingestion and inhalation are the other means. The seriousness of dermal exposure and the degree of the effects then depend on the rate of absorption of the substance through the skin, the size of the area of skin exposure, the length of contact time, the number and concentration of the substances that contacted the skin, and of course, the level of toxicity of the pesticide(s). Pesticides that volatilize can be inhaled from the atmosphere. Eye irritation can also result from direct contact with pesticides.

Acute toxicity levels are measured by the half lethal dose, or LD_{50}: the dosage at which 50% of animals exposed to the substance were killed. The lower the LD_{50} is for a particular pesticide, the greater the toxicity. This acute toxicity level determines the type of labeling required for pesticide containers, to help warn users of their dangers. Table 3 shows the acute toxicity level standards. Highly toxic pesticides must have the words "danger" and "poison" displayed on them, as well as the universally understood skull and crossbones picture. Only a few drops of highly toxic pesticides could be fatal for a 150-pound person. Moderately toxic pesticides have "warning" labels and either slightly toxic or relatively non-toxic pesticides read "caution." Even pesticides classified as relatively non-toxic can still be hazardous if proper care if not taken to use them as directed and avoid excessive exposure.[18]

Table 3: Acute Toxicity Measures and Warnings[13]

		LD_{50}	LD_{50}	LC_{50}	
Categories	**Signal Word**	**Oral mg/kg**	**Dermal mg/kg**	**Inhale mg/l**	**Oral Lethal Dose***
I Highly Toxic	DANGER, POISON (skull & crossbones)	0 to 50	0 to 200	0 to 0.2	a few drops to a teaspoonful
II Moderately Toxic	WARNING	50 to 500	200 to 2,000	0.2 to 2.0	over a teaspoonful to one ounce
III Slightly Toxic	CAUTION	500 to 5,000	2,000 to 20,000	2.0 to 20	over one ounce to one pint
IV Relatively Non-toxic	CAUTION (or no signal word)	5,000+	20,000 +	20 +	over one pint to one pound

* Probable for a 150 lb.-person.

To avoid excessive exposure and help protect against pesticide-induced health risks when handling them, manufacturers recommend some levels of minimum person protective equipment (PPE) which typically includes long pants and sleeves, shoes, gloves, and possibly safety glasses and a face mask for more toxic pesticides.

Table 4 lists some common herbicides in use in the US, their active ingredients, and the acute oral and dermal LD_{50} values. REI, or restricted-entry interval, is the amount of time necessary between the application of the pesticide to crops and the safe reentry of humans into the area is permitted and is also listed. This is a partial excerpt taken from a list of 77 pesticides for which LD_{50} values were reported.[18]

Table 4: LD_{50} Concentrations and Restricted-Entry Intervals for Selected Herbicides[18]

		LD_{50} Values (mg/kg)		**REI**
Active Ingredient, Trade Name	**Use Category**	**Oral**	**Dermal**	**(hours)**
Acetochlor, Degree	R	2,148	4,166	12
Acifluorfen, Blazer	G	2,025	>2,000	48
Alachlor, Lasso, Partner	R-12	Tech 930-1,350	13,300	12
Ametryn, Evik	G	1,950	■	12
Asulam, Asulox	G	>5,000	>2,000	12
Atrazine, AAtrex	R	1,869	>3,100	12
Bensulide, Prefar	G	Tech 271-1,470	■	12
Bentazon, Basagran	G	2,063	>6,050	12
Bromoxynil, Brominal, Buctril	G	Tech 260	>2,000	12
Butylate, Sutan +	G	4,500	>4,640	2
Carfentrazone-ethyl, Aim	G	5,143	>4,000	12

Health Effects

Organisms that come into contact with pesticides typically suffer negative health effects, either acute or chronic, that have the potential to be very severe. As desired, pesticides are acutely toxic to pests and work to inactivate them; thus this highly toxic nature can be expected to have similarly detrimental effects on both animals and humans, varying with the length of the exposure and the dose of the pesticide. These health effects can result from direct contact with pesticides, but also indirect contact when an individual drinks contaminated water or consumes contaminated foods.

Bioaccumulation and Biomagnification

Persistent pesticides that do not readily degrade can be stored within plants and later consumed by and transferred to animals and humans. Since many of these pesticides can be fat-soluble, they accumulate within fatty tissue of animals and humans. As subsequent animals consume the previous animals, the pesticide components remain within the system. High concentrations of the pesticide are stored. Moving up the food chain, organisms closer to the top have higher concentrations of these pesticides within their tissues. This phenomenon of greater concentrations of pesticides with successively higher levels on the food chain is called biomagnification. Population groups that consume large amounts of fish and wildlife may be at increased risks of health consequences due to bioaccumulation of toxic pesticide compounds.[2]

A group within the EPA focuses on persistent, bioaccumulative, and toxic (PBT) pesticides' effects and monitoring, since these pose serious health issues that remain for years.[19] These Level 1 PBT pesticides are aldrin, dieldrin, chlordane, DDT, mirex, and toxaphene; all highly chlorinated which degrade very slowly.[20]

Short-Term Effects

Humans that are exposed to large doses of pesticides even for short periods of time can experience serious health effects. These may range from nausea and vomiting, too much more serious consequences, even death, depending on the type of pesticide and the dosage. The individuals' prior health also plays a significant factor but high doses of pesticides can be fatal. Organophosphates, in particular, tend to have extreme acute effects. According to the World Health Organization, 300,000 people die annually from pesticide poisoning worldwide, while a total of four million suffer other health effects from some form of poisoning. This is often due to improper handling of pesticides and not necessarily only due to the transport and transfer of pesticides in the environment.[2] Neurological disruptions including headaches, dizziness, nausea, vomiting, confusion, tremors, and convulsions are possible, as well as eye, nose, mouth, and throat irritation.

Long-Term Effects

Chronic exposure to pesticides can have a wider range of health effects. Pesticides have been linked to many different types of cancers including lymphoma, leukemia, and brain, lung, and testicular cancer. Breast cancer may be linked to pesticides since they tend to bioaccumulate within breast tissue but further research must be conducted to definitively prove the correlation. Long-term exposure is also responsible for causing sterility both in humans and in other animals. Miscarriages also have been linked to pesticide exposure. Another disease that may be related to contact with pesticides is Parkinson's disease. With each of these long-term illnesses, identifying a definitive correlation between exposure to a specific pesticide and the illness can be difficult since large sample populations may not be available, and many other factors could play into the individual's illness.[2]

Level 1 pesticides are all classified as probably carcinogens. Some have been linked to central nervous system damage and neurological system disruption, damage to the liver, kidney, thyroid, reproductive system, and digestive system. Some may cause neurological disorders in children whose mothers are exposed during nursing or before giving birth. Many are suspected endocrine disruptors.[20]

Endocrine Disruptors

Many different types of pesticides have been shown to affect the endocrine hormones, such as estrogen and testosterone, as well as alter the reproductive systems or organs of animals. These endocrine disruptors can affect many different types of organisms, such as fish, amphibians, birds, reptiles, laboratory rats, and even humans. For example, male frogs that were exposed to atrazine were found to turn into females from lack of testosterone. 75% of male frogs were emasculated and 10% were turned into females. When these atrazine-induced females then mated and produced offspring, all offspring were male, skewing the sex-ratio of frogs in that population.[21]

Research into the possible endocrine disruption in humans has yet to determine the long term potential effects that these pesticides could be having. This could take decades to observe the long-term effects and understand the root causes. Table 5 lists some commonly used pesticides that have been shown in laboratory research with animals to be endocrine disruptors, and whether or not they are still used within the US.[2]

Table 5: Known Endocrine Disruptors Used in the US[2]

Pesticide	General Information
Atrazine	Herbicide; still used
Chlordane	Insecticide; banned in US in 1988
DDT (dichlorodiphenyltrichloroethane)	Insecticide; banned in US in 1972
Endosulfan	Insecticide; still used
Kepone	Insecticide; banned in US in 1977
Methoxychlor	Insecticide; still used

Health Effects on Young Children

Since infants and children have much less developed immune systems and much greater cell division rates as they grow, they are more susceptible to the dangers posed by pesticides. Their biochemical and physiological functions are largely immature compared to those of grown adults, in addition to smaller proportions of organs, muscles, bones, and brains. Therefore, toxicity of pesticides can be increased due to different absorption, transport, and metabolism rates. In the beginning months of pregnancy, toxic compounds can have permanent damage to fetuses but many dangers still exist after birth. During the continual development of the central nervous system, pesticides that have neurotoxic effects (such as organophosphates, organochlorides, and carbamates) can be particularly toxic even in low doses [13].

In Garry's 2004 review paper considering previous studies of the health impacts upon children, he notes that some studies have shown altered sex ratios (more females than males), increased occurrence of miscarriages, and "significantly increased numbers of birth defects"[22] compared to children either living in non-agricultural communities or not exposed to pesticides in direct studies. In particular, he points to one study from the Minnesota Red River Valley farm community where increase birth defects were observed in families where fumigant phosphine and herbicide glyphosate were used. Since the sample population was only around 1500 children, he calls for further studies to support this. From the same review paper, he concludes that childhood cancers are "weakly but consistently associated with pesticide use and in particular paternal pesticide use."[22] Neurodevelopmental studies indicate lower short-term memory and deficient motor skills in children with multiple pesticide exposures. Linkage to attention deficient disorders or hyperactivity has been theorized but has not been supported by studies yet.[22]

Overall Illnesses Caused by Pesticides, 1996

Table 6 lists the relative number of illness that each type of pesticide was responsible for in the US in 1996. This includes minor, moderate, major, and fatal illnesses but only those which were reported to a poison control center so it cannot be considered completely comprehensive. According to the American Association of Poison Control Centers, many of these illnesses could have been avoided with proper treatment after the exposure such as dilution of the pesticide with sufficient water. However, some of the more toxic pesticides would require medical attention to remediate the effects. This list includes organophosphates, pyrethrins/pyrethroids, hypochlorite disinfectants, carbamantes, organochlorides, phenoxy herbicides, and anticoagulant rodenticides.[13]

Table 6: Pesticides Most Often Implicated in Symptomatic Illnesses, 1996* [13]

Rank	Pesticide or pesticide class	Child <6 years	Adults and those 6 – 19 years	Total*
1	Organophosphates	700	3,274	4,002
2	Pyrethrins and pyrethroids**	1,100	2,850	3,950
3	Pine oil disinfectants	1,336	903	2,246
4	Hypochlorite disinfectants	808	1,291	2,109
5	Insect repellents	1,081	997	2,086
6	Phenol disinfectants	630	405	1,040
7	Carbamate insecticides	202	817	1,030
8	Organochloride insecticides	229	454	685
9	Phenoxy herbicides	63	387	453
10	Anticoagulant rodenticides	176	33	209
	All other pesticides	954	3,604	4,623
	Total all pesticides and disinfectants	7,279	15,015	22,433

*Totals include a small number of cases with unknown age.

**Rough estimate: includes some veterinary products not classified by chemical type.

Source: American Association of Poison Control Centers, Toxic Exposure Surveillance System, 1996 data.

Fate in the Environment

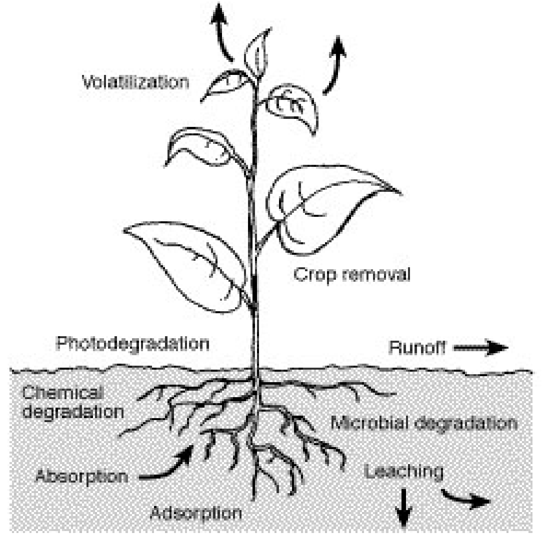

Figure 1: Pesticides Fate in the Environment [23]

When pesticides are introduced into the environment, they may either be degraded over time or they can remain in the environment. If they persist, they may be adsorbed into the soil or transported through water flow. Figure 1 shows the various fates of pesticides present in the environment.

The accumulation or transport of pesticides in the soil determines possible groundwater leaching which can directly affect human toxicity issues. Individuals who come in contact with treated soil are also susceptible to health issues, such as children that are playing on lawns or in fields that have been treated.

Degradation in Water or Soil

Pesticides may persist in the environment for extended periods of time, ranging from weeks to years. They undergo degradation as they are metabolized and oxidized into potentially less-toxic substances, in some cases into only carbon dioxide and any other elements in the base compound. Pesticides that readily undergo degradation are called non-persistent. Recalcitrant pesticides are persistence and do not readily degrade. Some of the most persistent pesticides include chlorinated hydrocarbons.[2]

Degradation can occur through three primary means. Microorganisms, particularly bacteria and fungi within the soil or in water supplies can degrade compounds. Microorganisms harvest the carbon and other organic components in pesticides as substrate and degrade the compounds through simple chemical reactions. The rate of this degradation depends on a number of factors, including temperature, oxygen content, pH, the amount of organics present in the oil, and the size of the microorganism population. This degradation can be quite rapid, since microorganism populations can grow exponentially in favorable conditions when substrate is available to them.[9] Degradation can also occur through chemical reactions independent of microorganisms. Chemical oxidation can also occur in the absence of microorganisms or sufficient oxygen. One of the most common mechanisms of chemical degradation is hydrolysis, or the breakdown when in contact with water.[23] UV radiation from the sun can also degrade compounds in surface water supplies or on the topmost layer of soil, called photodegradation. This is limited to exposed areas, however, and occurs more commonly on leaves or trees rather than when the pesticide is applied directly to the soil.[24]

Eventually, pesticides form residues when all of their original carbon has been oxidized to form CO_2, a process called mineralization. Secondary components formed during degradation are called metabolites, and are specific to each type of pesticide. These metabolites that may persist longer than the parent pesticide, may have lower activities than the parent and be less harmful. In other cases, however, the byproducts formed may be more harmful and more persistent. These metabolites are the compounds that are typically tested for in humans and other animals to determine pesticide presence.[23]

The commonly used pesticide atrazine provides a good example of the possible long-term effects of pesticides in the environment. According to USEPA, the overall half-life of atrazine is 608 days, the water half-life is 578, and the sediment half-life is 330 days. Field half-lives have been found to be between 13 and 261 days; variances were attributed to temperature differences and the assumption was made that atrazine could remain longer in colder temperatures without degrading. A study in Oregon found the half-life for atrazine on exposed soil was 87 days, in foliage was 13 days, and on leaves and leaf waste was 66 days. On turf, the half-life is shorter (between 5 to 10 days). Since atrazine has a low adsorption coefficient with many plants, it is likely to be washed into water sprays and may leach downward into groundwater supplies. Transport into surface water supplies has been shown to be possible also. Atrazine is mobile and persistent.[17]

Adsorption in Soil

In general, pesticides can either accumulate in the top layers of soil if they have a high affinity for the soil or are not highly water soluble, or they can leach downward and potentially into groundwater if they are more water soluble or mobile. Highly mobile pesticides will have a lower residence time in the soil, but then may be more likely to transfer to water supplies or contaminate other soil areas if they are persistent. Bonding between soil and pesticide particles is dependent on the chemical and physical properties of each species such as the charge of the pesticide as well as the charge of the soil.[23] The organic content of the soil is the greatest determining factor affecting the adsorption of pesticides in the soil and may range from 0.1% to 90%, from desert sand to more organic soils.[25] Sandy soils are less likely to retain pesticides than other soils with high concentrations of organic matter or clay since organic soils have greater surface areas and therefore more bonding sites for the pesticide to adsorb. Drier soils are more likely to adsorb pesticides since water molecules do not preferentially block pesticides from bonding.[23] Adsorption coefficients for particular pesticides in certain soil types are necessary to determine the transport of pesticides through soil.[26] Pesticide adsorption in soil can also affect plant life, since pesticides used to treat one type of pest can destroy desired plants that come into contact with treated soil. In some cases, the adsorption into soils is considered during application of the pesticide and higher concentrations are recommended for use in order to have the same effects. However, the environmental fate of these pesticides used in high concentrations because they are known to be mobile must be kept in mind.[23] Table 7 reports the likelihood of groundwater contamination based on pesticide and soil characteristics as well as the water volume that contributes to transport.[24]

Table 7: Groundwater Contamination Potential as Influenced by Water, Pesticide, and Soil Characteristics[24]

	Risk of Groundwater Contamination	
	Low Risk	**High Risk**
Pesticide Characteristics		
Water solubility	Low	High
Soil adsorption	High	Low
Persistence	Low	High
Soil Characteristics		
Texture	Fine clay	Coarse sand
Organic matter	High	Low
Macropores	Few, small	Many, large
Depth to groundwater	Deep (>100 ft)	Shallow (<20 ft)
Water Volume		
Rain/irrigation	Small volumes at infrequent intervals	Large volumes at frequent intervals

Transport in Soil and Water

There are five main ways in which pesticides can be transported in the environment after application: runoff, leaching, absorption, volatilization, and crop removal. Many of these ways involve water transport but all can contribute to movement into new environment. In many cases, multiple environmental fate processes interact and lead to variable byproduct formation as well as transport.[24]

Runoff

Runoff is the transport of pesticides in water above the earth's surface that does not absorb downwards into soil but instead flows over the surface of the earth due to gravity. Flooding, rainwater, or applied watering can lead to runoff if the water added is greater than the amount that the soil can absorb. As pesticides are dissolved in this water, they are carried along with it. The degree to which pesticides are carried by the water depends on their solubility at that pH and temperature, the amount of vegetation in the area that would potentially hinder the pesticide flow, and other factors.[23]

Leaching

Leaching describes the transport of pesticides downward through the soil into the water table. This movement depends on the chemical properties of the pesticide as well as of the soil type, as discussed before, since strongly adsorbed compounds are less likely to undergo leaching. Water present within soil can also affect leaching. Water may dissolve pesticides and carry them downward or into groundwater supplies, contaminating them. This movement within water is dependent on the compound's solubility in water as well as the water flow patterns. The permeability of the soil itself, or how rapidly it allows movement of water and pesticides through it, also affects leaching. Typically, pesticides leach downward towards water tables. The depth of the water table affects how likely groundwater leaching will be. That is, for a deep water table, the time that a pesticide needs to reach downward to the water table is longer and provides more allowance for possible adsorption into the soil or degradation of the compounds. Again, heavy precipitation or heavy water application can affect leaching since greater amounts of water increase both the ability of pesticides to dissolve in larger quantities of water and also increases the movement of the water itself. Different materials composing the soil layers also affect leaching; for example, limestone dissolves more readily and allows water saturated with pesticides to travel through sinkholes and enter groundwater.[23]

Absorption

This is the transport of pesticides into plants cells. Once in the plants, pesticides may degrade or they may remain within the plant for the length of the plant's life. The toxins may then be transferred to any organism that consumes the pesticide, contributing to bioaccumulation of the pesticide which can have long-term effects through large spans of the food chain.[2]

Volatilization

Volatilization is the transfer of pesticides from a solid or liquid phase into the gaseous phase. This then allows for much more widespread possible movement of the pesticide since gases expand indefinitely in open volumes and may be carried easily by air currents. These transported vapors can then damage other plants, but also affect animal life and humans that inhale them. Pesticides with a high vapor pressure are most likely to volatilize. Ambient temperature, pressure, and humidity also affect the rate at which pesticides will volatilize.[23]

Crop Removal

This is the movement of plants from their original location to other areas for use or sale, which can lead to pesticide transport and contamination of new water supplies or other media. For example, the washing of the plants that were treated with pesticides may remove the pesticides from the plants themselves, as desired, but only transfers the pesticide to water used to clean the pesticides.[23]

Pesticides of Interest

Three pesticides were studied specifically because of their wide use and occurrence in the environment: alachlor, atrazine, and glyphosate.

Alachlor

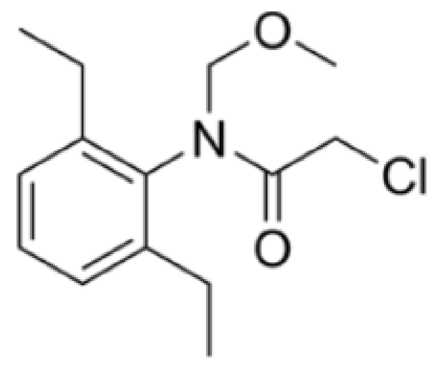

Figure 2: Chemical Structure of Alachlor

Alachlor, 2-chloro-2'-6'-diethyl-N-(methoxymethyl) acetanilide, is an herbicide widely used in the United States and other countries on crops such as corn, soybeans, and peanuts. Figure 2 shows the chemical structure of alachlor. It is an organochloride pesticide, more specifically, a chloroacetanilide. Trade names include "Lasso," "Partner," and "Bronco." It is often found with another member of the cloroacetanilide family, metolachlor, with similar byproducts formed during degradation. Alachlor is highly water soluble and therefore poses a problem for water contamination and transportation through ground and surface water supplies.[27] As of 1992, concentrations significantly greater than the MCL have been detected in at least

15 states by EPA's Pesticide in Ground Water Database studies.[28] According to the USEPA, the best treatment technology available is granulated activated charcoal, which has been shown to remove it down to the MCL.

Usage

Alachlor was first registered for use in 1969. It is primarily used for broadleaf weeds and grasses.[28] It mixes well with other pesticides and is therefore commonly found in conjunction with others such as atrazine, glyphosate, trifluralin, and imazaguin. Alachlor is heavily used in the Midwest of the US, particularly Illinois, Indiana, Iowa, Minnesota, Nebraska, Ohio, and Wisconsin.[28]

The solubility of alachlor in water is 242 mg/L at 25°C.[29]

Legislation

The maximum contaminant level acceptable in drinking water has been set at 0.002 mg/L (2 ppb) by the USEPA. The maximum contaminant level goal is set at zero. It began being regulated under the Safe Drinking Water Act in 1992.[30]

As of 1986, the Department of Agriculture, Trade, and Consumer Protection (DATCP) classified alachlor as a priority pesticide because of its "high use, high leach, and detection." This indicates that not only is it a problematic issue in terms of environmental damage and in magnitude since it is widely used, but it is also difficult to identify contaminated areas.[27]

Alachlor use in the European Union has been banned since 2006 due to known hazards to human health and the environment.[31]

Health Implications

Alachlor has an acute toxicity rating of slightly toxic (category III). The oral LD_{50} is 1350 milligrams per kilogram of body mass, the dermal LD50 is 4982 mg/kg, and the inhalation LD_{50} is greater than 4.67 milligrams per liter of air.[31] According to the EPA, individuals who repeatedly are exposed to concentrations of alachlor higher than the MCL may experience health complications including liver problems, eye problems, kidney problems, or spleen problems. They may also experience anemia. Alachlor has not yet been conclusively evaluated for the EPA based on its carcinogenic potential for humans. It has, however, been categorized as a probable human carcinogen (Group B2) based on animal studies.[32]

European studies have classified alachlor into carcinogenic category 3, which include substances that cause concern for humans due to potentially carcinogenic effects but for which insufficient data is available so no complete conclusions can be made. When byproducts are formed during degradation, it is "extremely unlikely" that an active metabolite of alachlor would lead to cancer but it cannot be completely out ruled.[31] There is similarly no conclusive data for possible endocrine disruption.[31]

Degradation

Degradation of alachlor, like other pesticides, varies in different environmental conditions. Surface water, soil, and groundwater degradation rates vary. Furthermore, degradation during transport conditions complicates the identification of degradation products since new compounds can be introduced from passing environments during transport. Biodegradation and chemical oxidation produce similar byproducts for alachlor, called metabolites. According to the USEPA, at concentrations between 1 and 5 ppb, higher temperatures led to greater degradation rates.[30]

The primary degradation that alachlor undergoes has been shown by many studies to be biodegradation by aerobic microorganisms in soil. Thus the majority of degradation occurs in soil. Since alachlor is largely water soluble, these byproducts may then transfer into groundwater supplies where they can retained for extended periods of time since the half-life in groundwater has been proposed to be between 808 and 1518 days by one study[33] and 320 to 324 by another.[34] Degradation occurs primarily in soil or in surface water supplies, but not once the compounds have reached ground water.[35]

Byproducts

Alachlor forms a large number of byproducts when degraded through different mechanism. A 1995 study by researchers at UMass detected 20 degradation products found in groundwater. The identities of many products were confirmed with standards either purchased or synthesized with great purity, but many, including the two most abundant compounds, were not identified definitively and were classified only as "alachlor related". A structure was proposed for one. In this study, four wells near a Massachusetts corn field were sampled for concentrations of alachlor and related compounds at two intervals, the second 30 months after the first. The same compounds were detected in both studies (with one exception), demonstrating the long half-life and low rate of degradation once reaching ground water. Interestingly, in one of the wells, no alachlor was detected but byproducts that were detected were well over the MCL of 2 ppb demonstrating that byproducts may be a more significant issue than the parent compound in some cases. The concentrations of each byproduct ranged from less than 2 nanograms per liter up to 410 nanograms per liter. In all monitoring wells, the total concentration of byproducts was at least two times the concentration of alachlor detected, indicating that byproducts form in significant concentrations. One byproduct that was reported in other studies but not found by the researchers in Massachusetts were [[(2-6-diethylphenyl)methoxy]-methyl)amino]-2-oxoethane sulfonate (ES).[35]

ES and 2-hydroxy-2,6-diethylacetanilide, 2',6'-diethylaniline (DEA) have been identified by other studies as two of the major alachlor degradation byproducts. A 1992 study focusing on ES studied 11 ground and surface water sources in Indiana and Ohio and found concentrations ranging from 0.6 to 74 ng/L in 9 of these locations, below the MCL for alachlor.[36] No MCLs have been set for alachlor byproducts. ESA, the sulfonic acid analog of ES, was detected along with DEA by a study of 303 near-surface aquifers in the US. The concentration of ESA was significant and greater than that of the parent compound found alongside it. DEA was also detected frequently in this study but at lower concentrations.[37] DEA was found in many other studies and is highly water soluble, which presents a potential risk to ground water.[38]

These and other studies have demonstrated that the concentrations of byproducts can be significant. In terms of the toxicity of the byproducts themselves, each must be evaluated individually to truly determine the potential harmful effects. This has not been fully investigated for all byproducts since many remain unidentified.

In the following research, byproducts for alachlor will not be investigated due to time and detection limitations presented by the wide range of byproducts that may be formed.

Persistence and Toxicity of Byproducts

A structural isomer of ES was shown by the Monsanto Company to have a half lethal dose of over 5000 mg/kg from short-term animal testing. It was reported to be non-mutagenic and to not bioconcentrate or undergo significant metabolic transformations when consumed by animals. As such, ES is not a very toxic byproduct of alachlor.[36] Out of the wells from the UMass study that showed ESA concentrations above 0.10 micrograms per liter, 90% of them retained concentrations above this level during annual subsequent samplings. ESA is therefore persistent, like its analog ES, but also remains relatively non-toxic and not problematic though certainly abundant and persistent.[37]

DEA is water soluble and therefore mobile. However, in general, researchers at UMass comment that the cleavage of the methoxymethyl group of alachlor may translate into detoxification. They point to studies performed by Jacobsen et al that show that in mice, formaldehyde is liberated from the methoxymethyl (MOM) group. If separated, the MOM-free degradation product would provide less potential for formation of formaldehyde and as such have a powerful mutagenic potential.[35] Another study by Tessier showed that two compounds without the MOM group, 2-chloro-2',6'-diethylacetanilide and 2-hydroxy-2',6'-diethylacetanilide were only weakly mutagenic to Salmonella strain TA100. Therefore, although the specific toxicity of DEA has not been tested and a similar situation exits for the numerous other byproducts of alachlor, the toxicity of compounds with a cleaved MOM group can be considered low. Further studies are required to confirm this hypothesis proposed by researchers at UMass.[35,39]

Atrazine

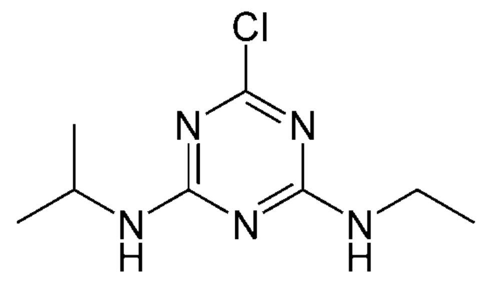

Figure 3: Chemical Structure of Atrazine

Atrazine is an organochloride herbicide, often used in the Midwest. Figure 3 shows the chemical structure. Atrazine is found in the form in of a white crystalline solid. The IUPAC name for atrazine is 2-chloro-4- (ethylamino)-6-(isopropylamino)-s-triazine. The chloro refers to the chlorine attached on the top of ring which identifies atrazine as an organochloride pesticide. The ethylamino and isopropylamino both refer to the two amino sites on the molecule signified by the side chains of nitrogen. The center ring made up of doubled and single bonded nitrogens is also called a triazine ring with the 's' detailing the specific configuration of said ring. The configuration of the inner ring is that of a 1-3-5-trazine ring. That is, the nitrogen appears at the 1, 3 and 5 position in the ring.

Usage

Atrazine is used worldwide as an herbicide. More specifically, atrazine is often used on field corn. In the US, the EPA reports that 75% of field corn acreage grown in the U.S. is treated with atrazine making it the most heavily used herbicide in the US. Typical methods of delivery for this pesticide include many different forms of sprayers, aircraft and tractor delivery. [40]

Legislation

In the United States and the international community, atrazine has been labeled as a dangerous herbicide. The process and time it took different communities to reach this conclusion was very different. In the United States the first step in regulating atrazine use took place in 1974 when the *Safe Drinking Water Act* set the MCL for atrazine at 3 ppb (parts per billion) in drinking water.[41]

In 1991, communities in Wisconsin noticed a large spike in atrazine concentration in their drinking water. This examination led to the *Wisconsin Atrazine Rule,* which demands that no atrazine be used in any areas that contain more than the allowed 3 ppb concentration in the drinking water in order to avoid further increase of concentrations. The rule also only allows the use of atrazine from April 31st to June 31st. The time restriction was implemented in order to greatly reduce the amount of atrazine used on crops and therefore the amount of atrazine that could potentially infiltrate the local watersheds. Shortly following the *Wisconsin Rule,* the federal government made steps to officially recognize atrazine as a harmful contaminate and ban purchase of the pesticide from any persons other than licensed applicators.[40] It wasn't until 1998, however, that the U.S. classified atrazine as a potential human carcinogen[42]. Two years later, the EPA made an announcement that atrazine will be regulated at the highest standards in the agency. In 2002, a startling discovery was made that atrazine, in far lower levels than was set by the EPA, was causing the emasculation of frogs. Because of this discovery, the EPA was sued that year by the National Resources Defense Counsel for not having stricter guidelines on a clearly dangerous chemical. In the US today, the last evaluation of the threat of atrazine was conducted in 2003 although discussion within the EPA are said to have taken place in 2010 and are to continue in 2011. These discussions could potentially add/loosen restrictions on atrazine use based on any up and coming environmental data.[40, 42]

The toxicity of atrazine was realized much more quickly in Europe. In 1991 both Italy and Germany banned use of atrazine. The European Union followed suite in 2004 with the banning of Atrazine for all countries that are members of the union. One can deduce that regulation was delayed as long as it was in the United States due to the incredible demand and dependency of the pesticide in the Midwest.[42]

Health Implications

Analyzed by a research department at Cornell University, atrazine is classified as slightly to moderately toxic.[43] In studies made by the same research group, atrazine was found to cause skin irritation, vomiting, diarrhea, stomach pains, muscle spasms and hypothermia. While there are numerous acute symptoms of atrazine poising, the chronic toxicity is more severe. In one study, rats that were given a steady dosage of twenty milligram of atrazine per kilogram of body mass (mg/kg) for six months showed severe paralysis and respiratory damage. Forty percent of that sample population died within those first six months of exposure. Rats exposed to a lower concentration of five mg/kg showed a strong reduction in growth over six months. Among these symptoms, atrazine was shown to affect many different species in many ways including having reproduction effects, teratogenic effects, mutagenic effects, carcinogenic effects and organ toxicity.[43]

Degradation

While the solubility of atrazine (33 mg/L) is relatively low in water, the half-life of the chemical is very long, with aqueous samples lasting more than 60 days to 100 days. The actual degradation of atrazine is due to chemical hydrolysis followed by degradation by microorganisms in the soil. As is common with most pesticides, degradation occurs faster in non-neutral pH water and noticeably slower in pH neutral water. The addition of humic material has also been noted to rapidly increase the degradation. Figure 4 depict the difference pathways in which atrazine would degrade.[44]

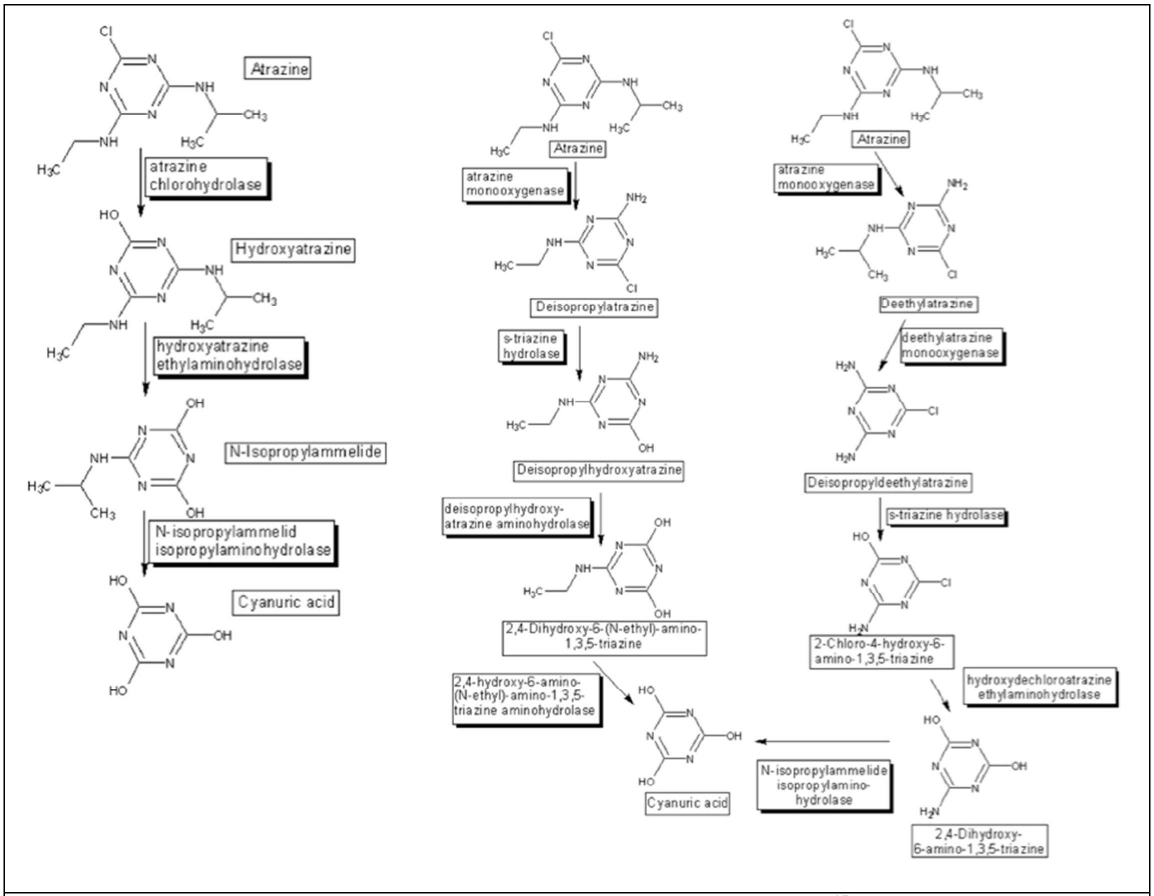

Figure 4: Degradation Pathways of Atrazine[45]

Byproducts

Based on the primary pathway through which Atrazine degrades, there are three main byproducts: hydroxyatrazine, deisopropylatrazine and deethylatrazine. The first byproduct is produced from chemical hydrolysis while the latter two byproducts occur via microbial decomposition in the soil. While there is significant research investigating the toxicity of atrazine, there is very little information regarding byproducts toxicity. Research performed in 2009 accounted for this lack of information and looked into the effect and toxicity of both atrazine and its metabolites deethylatrazine and deisopropylatrazine. This paper suggests that compared to atrazine, the two metabolic byproducts (excluding hydroxyatrazine) show very little potency.[44]

Glyphosate

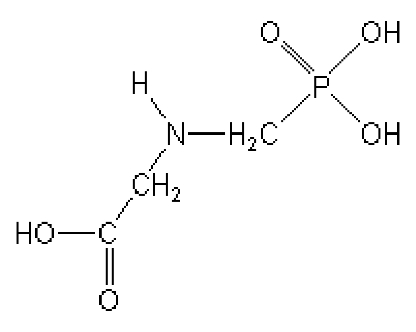

Figure 5: Chemical Structure of Glyphosate

Glyphosate, N-(phosphonomethyl)-glycine, is a broad-spectrum, nonselective herbicide used to control annual and perennial plants. It is often used in both croplands and residential areas. Glyphosate is an organophosphate compound, which are generally highly acutely toxic to humans and wildlife alike. The structure of glyphosate, shown in Figure 5, has several dissociable hydrogen atoms, including the first hydrogen on the phosphate group. The presence of these groups suggest potential for chemically treating glyphosate.[46]

Glyphosate was first discovered to have herbicidal properties by John Franz in 1970, while working for the Monsanto Company. By 1973, the Monsanto Company was marketing its glyphosate-based herbicide, under the trade name Roundup. As glyphosate is highly soluble in water (up to 12 grams per liter at 25 degrees C), it poses a significant threat to water contamination and transport through ground water. [47]

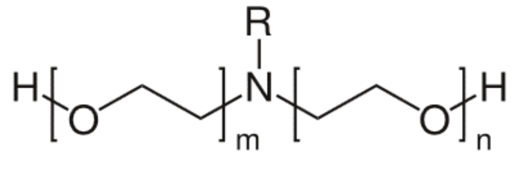

Figure 6: Chemical Structure of POEA

Glyphosate is rated to be less dangerous than other herbicides from the organophosphate family, which is evidenced by its EPA Toxicity Class of III (on a 1 to IV scale, where I is the most dangerous).[46] However, studies suggest that when combined with other ingredients, the Roundup mixture may be considerably more toxic than glyphosate alone. The chemical surfactant used in the commercial formulation, polyoxyethylated tallow amine (POEA), has garnered attention recently, for causing damage and cell death to human cells. The structure of POEA can be seen in Figure 6. Currently, because it is listed as an inert ingredient of glyphosate formulations, POEA is not regulated under environmental laws.[47]

Usage

Glyphosate is one of the most abundantly used pesticides in the United States. In 2007, the EPA estimated that the agricultural market within the US was using up to 185 million pounds of glyphosate per year. Additionally, as of 2005, genetically altered glyphosate resistant crops accounted for approximately 75% of all genetically modified crops.[48] Glyphosate is still legal throughout the world, despite mounting evidence of the toxicity of its formulations.

Health Implications

The LD_{50} of glyphosate in rats is 4,320 mg/kg.[49] The Monsanto Company lists the LD_{50} as 5,600 mg/kg. In 2010, a European Food Safety Authority (EFSA) conference assessed the health effects of glyphosate exposure as well as the health risks of genetically modified food and crops. One finding that came out of this was that in human cells, Roundup is capable of causing total cell death within 24 hours of contact.[47]

Glyphosate acts as an enzyme inhibitor, and when applied to plants, it cuts off the shikimic acid pathway, which does not exist in humans. However, this pathway is rather specific in action–it is the method with which phenylalanine and tryptophan are consumed in plants and microorganisms—and while the shikimic pathway does not exist in humans, glyphosate does prevent the binding of phosphoenol pyruvate from binding to enzymes. Phosphoenol pyruvate is a metabolite that is present in all living organisms, and therefore, glyphosate has the potential to interfere with other metabolic pathways.[48]

In an EPA study, data suggested that glyphosate can cause birth defects in certain test subjects. In this study it was found that the effects of glyphosate on pregnant rats included “diarrhea, decreased weight gain, nasal discharge. . .death of mothers, and kidney and digestive disorders”. [48] In 2009, Andres Carrasco, an Argentinian government scientist, released his finding that exposure to glyphosate herbicides caused malformations in frog and chicken embryos. However, his tests were conducted with considerably lower concentrations of glyphosate than used in agricultural spraying. Malformations in frog and chicken embryos were seen in samples which were injected with 2.03 mg/kg of glyphosate. The maximum limit of residual glyphosate in soy in the European Union is 20 mg/kg, nearly 10 times higher. Carrasco stated that people living in areas dependent on genetically modified soy crops had been reporting problems in 2002, two years after the implementation of genetically modified crops in Argentina. In April 2010, a survey of the population of La Leonesa and other areas of Argentina in which genetically modified crops are harvested revealed that the childhood cancer rate had tripled in the previous ten years; the rate of birth defects also increased four-fold.[51] This information may indicate severe health effects at concentrations much lower than the tests were conducted at.

An epidemiological study of Ontario farming populations showed that exposure to glyphosate through application to crops nearly doubled the chances of late spontaneous miscarriages. Professor Eric-Giles Seralini from Caen University has shown that glyphosate is toxic to human placental cells, killing a large proportion of cells after an 18 hour exposure to concentrations well below agricultural use.[49] Additionally, glyphosate interacts with the active site of the enzyme aromatase, which is responsible for making estrogen. The interaction between glyphosate and aromatase was minimal unless a surfactant was present. After 18 hours of incubation with a Roundup solution, enzyme activity had been inhibited. The inhibition can be directly associated with a decrease in mRNA synthesis, which suggests that Roundup decreases the rate of gene transcription.

Health Implications of POEA

While glyphosate on its own is not acutely toxic, it is likely that the most common formulations of the pesticide, such as Roundup, are more toxic than glyphosate itself. Roundup, one of the most common implementations of glyphosate in pesticide use, includes a surfactant, called polyoxy-ethyleneamine (POEA), which is used to prevent the chemical from forming droplets and rolling off the leaves on which it is sprayed. Ethylated amines are significantly more toxic than glyphosate; they are irritants to the eyes, respiratory tract, and the skin, and can contain dioxane contaminants, which are suspected of being carcinogenic. The Food and Agriculture Organization of the United Nations has set standards of 1 ppm of 1,4-dioxane in formulations, which may be present in POEA surfactants.[52] Additionally, new research shows that commercial Roundup caused liver damage in rats. In this study it was found that glyphosate and its surfactant in Roundup acted in synergy to increase the damage to the liver. It was theorized as early as 1988 that the toxicity of POEA was much greater than that of glyphosate alone. The acute lethal dose of POEA is “less than one third that of roundup and its active ingredient.” The lethal dose of POEA is approximately 1200 mg/kg, compared to the 4,320 mg/kg reported for glyphosate.[7]

In the environment, it has been observed that POEA is more toxic than glyphosate. This is in part due to the fact that POEA is more toxic in alkaline water than in acidic water, and therefore, the relative potency of the chemicals is pH dependent.[53] Data comparing the ecological toxicities of glyphosate, POEA, and Roundup can be seen in Table 8.[52]

Table 8: Estimates of Relative Potency of Toxicological Interaction of Glyphosate and POEA[52]				
Observed LC_{50} values				
Species	Glyphosate	POEA	Roundup	Relative Toxicity
pH 2				
Rainbow Trout	240	0.65	2	369.2
Bluegills	220	1	2.6	220
pH 6.5				
Rainbow Trout	140	7.4	10.8	18.9
Bluegills	140	1.3	6	107.7
pH 7.2				
Rainbow Trout	140	2	11.8	70
Bluegills	140	3	7.1	46.7

Legislation

Glyphosate has garnered significant legal attention throughout the years. In 1996, the Monsanto Company was accused of false advertising in regards to the safety of its product. Again in 2007, the company was convicted of false advertising, having stated that their product left the soil clean after use. The second case began in 2001 with the basis that glyphosate is both dangerous for the environment and toxic to aquatic organisms. Additionally, the United States EPA has caught scientists deliberately falsifying test results for Monsanto studies of glyphosate.[54]

In the United Kingdom, glyphosate is the most frequent cause for complaint recorded by the Health and Safety Executive's Pesticides Incidents Appraisal Panel. Between 1990 and 1995, there were 33 complaints recorded, as well as 34 poisonings involving glyphosate. Within the United States and in other countries, glyphosate has frequent detrimental health effects. In California, it is the most commonly reported cause of illness or injury to workers from pesticides. However, glyphosate is not yet banned in any country.[55]

Degradation

The degradation pathway for glyphosate in the environment can be seen in Figure 7.

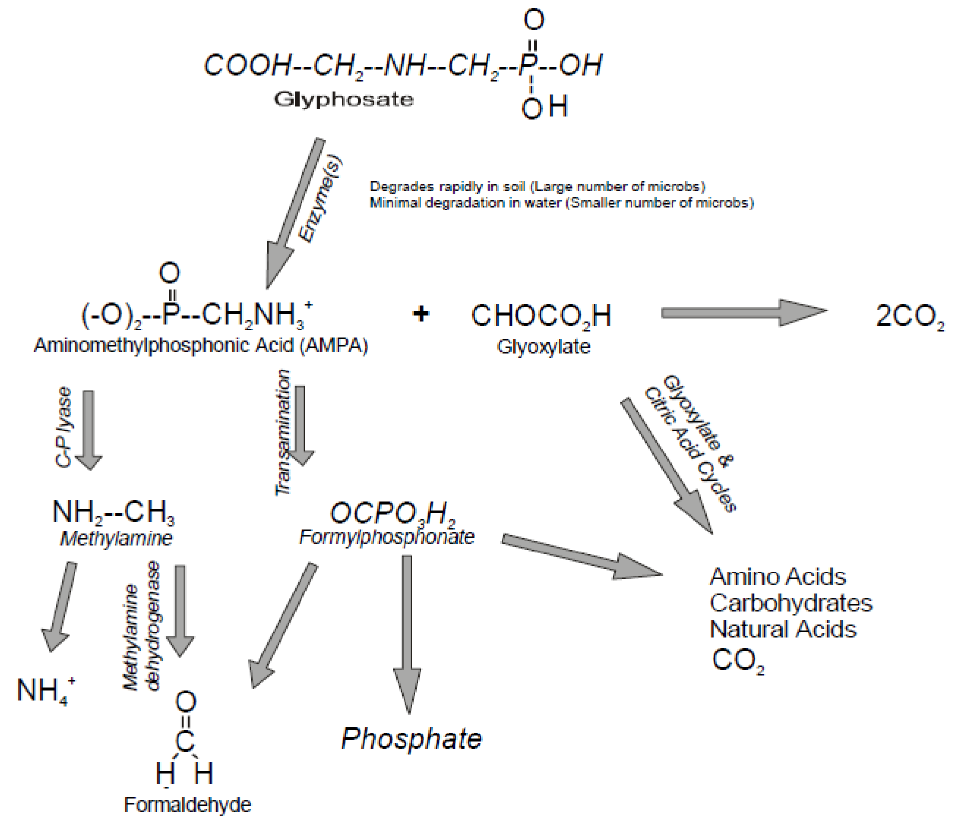

Figure 7: Degradation Pathway for Glyphosate[52]

The primary metabolite in the degradation of glyphosate is aminomethylphosphonic acid (AMPA). The primary metabolite in the degradation of glyphosate is aminomethylphosphonic acid (AMPA). However, this byproduct remains dangerous to humans and the environment. According to case studies, AMPA alters cell cycle checkpoints in sea urchin embryos by interfering with the physiological DNA repair machinery. Such disruption is known to lead to genomic instability and could lead to the possible development of cancers in humans.[52]

There is no pathway for glyphosate to be carried and degraded through air. This is because the vapor pressure for glyphosate is extremely low. Additionally, its low Henry's Law Constant indicates that it will readily bind to soil particles. Glyphosate is persistent in soil, with studies determining the half-life of the herbicide to range from 3 to 130 days. Glyphosate that is bound to soil particles is degraded slowly, with some concentrations potentially remaining unchanged and inactive in the soil for years. Glyphosate particles that have not bonded with the soil particles are rapidly degraded to carbon dioxide through microbial activity.[52] Additionally, when in a soil environment, glyphosate is resistant to chemical degradation, is stable in sunlight, is difficult to leach from the soil, and also has a low tendency to runoff.

Because it is difficult to leach from soil, glyphosate is unlikely to contaminate ground water. However, the chemical is used in water for the control of aquatic weeds, and additionally can be carried into surface water through soil erosion. According to *Pesticides in water: Report of The Working Party on the Incidence of Pesticides in Water,* glyphosate is not typically looked for in water, because it is highly difficult to isolate and is not considered to be of major concern as a water contaminant.[52] Glyphosate is highly soluble in water, with up to 11,600 ppm dissolving at 25 degrees C. Its hydrolysis half-life is greater than 35 days, as glyphosate shows little propensity towards hydrolytic decomposition. Studies suggest that its loss from water is due to sediment adsorption and microbial degradation. Therefore, the degradation of glyphosate in water is slower than that of glyphosate in soil, as there are fewer microorganisms in water to carry out the chemical pathways. However, fish and aquatic life are more sensitive to glyphosate and its formulations than mammals and birds. Higher water temperatures and pH levels have been observed to increase the toxicity of these compounds.[7]

Methods for Pesticide Removal from Water

Pesticides can degrade in the environment through microbial decomposition, UV photolysis, chemical oxidation, or hydrolysis. The following treatment techniques may be employed in wastewater and drinking water treatment facilities to actively reduce pesticide concentrations prior to human consumption or discharge to the environment.

Activated Carbon Adsorption

In modern day water treatment, activated carbon is one of the most common techniques used by both large and small treatment facilities. Activated carbon is very popular throughout industry for various reasons including its low price, ease of use, and the limited machinery required. The mechanism in which activated carbon purifies water is adsorption, where the pesticide or contaminant associates with the porous carbon surface. Activated carbon offers high surface area for adsorption. While only being as large as a grain of sugar, the high porosity of an activated carbon grain yields a very large surface area for adsorption to occur. In fact, studies show that activated carbon can have a surface area of 500 m^2 per gram. While the most common form of activated carbon is a black powered substance made from coal, activated carbon can also be made from wood or coconut shells. The production of activated carbon requires the intense burning of the raw material: coal, wood or coconut shells. Once sufficiently burned, the ash is then activated using compounds such as zinc chloride or calcium chloride. These chemical activate the ash by creating pores inside each individual ash particle.[56]

Types of Activated Carbon

There are two main types of activated carbon. Powered activated carbon (PAC) has a much smaller particle size and in turn has a low initial cost. Manufacturing costs are lower due to the small size. Unfortunately the size is PAC's greatest detriment. Due to the small size, powdered activated carbon is known to clog machinery and other filters throughout a plant. Conversely, granular activated carbon (GAC) has a much larger particle size and a higher initial cost. The advantage of using GAC is the ability to reuse it. GAC particles are large enough that when the activated carbon reaches its breakthrough point and is saturated, the particles can be recovered and the regenerated. Regeneration effectively destroys contaminants and allows the GAC to be reused in the process. [57]

Mechanism

One common misconception regarding activated carbon is the difference between absorption and adsorption. While many techniques utilize absorption, a process in which material is transferred into a solid, activated carbon utilizes adsorption. The contaminant in the water sticks onto the wall of this carbon. This adsorption mechanism is broken into three main parts. The contaminant first adheres to the exterior surface of the activated carbon particle. Due to equilibrium, the process by which a particle will travel from high to low pressure, the contaminant moves along the wall to larger pores located deeper inside the activated carbon particle. The third stage of the mechanism includes the contaminant coming to a final resting place on the inner surface of the grain of activated carbon. This process continues until a no more contaminants can be adsorbed by the activated carbon. The maximum adsorption can vary from substance to substance and is also directly affected by type of activated carbon, temperature, pH and contact time.[58]

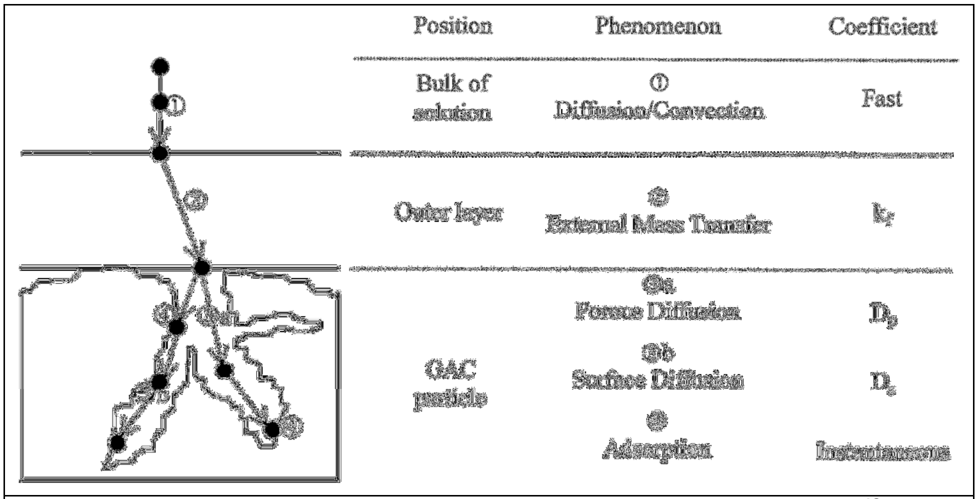

Figure 8: Activated Carbon Adsorption Process and Mechanism[48]

Figure 8 depicts the process by which chemicals travel into the porous piece of carbon. In step one, the particle is in solution. For this figure, it can be assumed that the pressure in the solution is higher than that of the inside of the carbon particle. In step two the particle is drawn into the pores of the activated carbon due to the low pressure inside the pore. Once again this is the particle being brought to equilibrium. In step three the particle is fully inside of the particle but has yet to find the lowest pressure area. Step four involves the particle traveling further inside the pore and coming to rest at a wall, thus reaching equilibrium.[58]

Economics

Traditionally, PAC has been used in smaller facilities where the capital cost drives the demand. Because of the GAC's high capital cost but low difference, the actual particle size also impacts the choice of which form to use. This consideration takes into consideration the types of equipment found downstream of the filtration process. Due to the small particle size, PAC requires more involved filtration systems and can potentially damage precision equipment downstream. GAC is therefore more commonly used.[59]

Modeling Adsorption with Activated Carbon

One common way to model the adsorption of activated carbon is via the Freundlich model. In this model, the following equation is used to predict specific parameters in the adsorption mechanism.

$$\frac{X}{M} = K * C_e^{\frac{1}{n}}$$

Where:

$X = Mass\ of\ Solute\ Adsorbed$
$M = Mass\ of\ Adsorbent$
$K = Experimental\ Constant$
$n = Experimental\ Constant$
$C_e = Equilibrium\ Concentration\ of\ Solute$

Using this equation, a log-log graph such as Figure XX can be formed in order to ascertain the experimental constants. The goal of using this model is to ascertain the experimental constant (K/n). With this constant one can theoretically find the mass of adsorbent needed to dissolve a certain percentage of pesticide. Conversely, this model can accurately calculate a certain amount of pesticide an adsorbent material can extract form a solution. In order to find the constant, one must experimentally mix a mass of activated carbon and measure its resulting adsorption rate. With this rate one can get the resulting amount of pesticide, C_e, left in the solution. With values experimentally found, a log-log graph can be created and the resulting constants can be calculated. Experimental constants are specific to that particular contaminant and form of activated carbon and in turn can be used to theoretically obtain adsorption information. When plotted correct, as shown in Figure 9, the graph should show a linearly increasing line.[60]

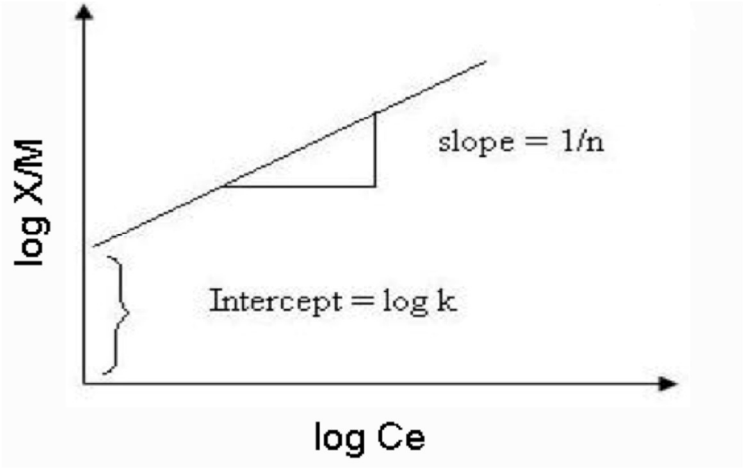

Figure 9: Sample Freundlich Isotherm Curve[60]

Chlorination

Chlorination is a common technique used in water treatment facilities to deactivate undesired microorganisms as well as reduce some pesticide concentrations through chemical oxidation. Chlorine reacts with a number of species, including bacteria, viruses, nitrogen, hydrogen sulfide, cystide, cyanides, phenols, and other organics through disinfection and oxidation.[61] According to the World Health Organization (WHO) in 2004, “chlorine is [the] most widely and easily used [disinfectant], and the most affordable of the drinking water disinfectants.” Similarly, it is an affordable oxidant.[62]

Chlorine can be used in a number of forms to treat drinking water. Concentrations of either pure chlorine or chlorine compounds are added to water supplies and are termed either free or combined chlorine. Free chlorine is either in the form of pure chlorine gas Cl_2 dissolved in water, hypochlorous acid HOCl, or the hypochlorite ion OCl^- which is formed because hypochlorous acid is a weak acid that dissociates based on pH. [61]At pH levels between 6-9, less than 1% of the available chlorine will be in the pure form and will instead have hydrolyzed rapidly in water. This hydrolization is nearly instantaneous at 25°C and takes a few seconds at 0°C. Hydrolization Reactions 1 and 2 are shown here. Hypochlorous acid is a stronger disinfectant than the hypochlorite ion, but the ion is a stronger oxidant. Therefore, pH levels greater than the pKa of 7.6, the hypochlorite ion is dominant and conditions are more favorable for oxidation.[63]

$$Cl_2(g) + H_2O \rightarrow HOCl + H^+ + Cl^- \qquad \text{(Reaction 1)}$$

$$HOCl + H_2O \leftrightarrow OCl^- + H_3O^+ \qquad \text{(Reaction 2)}$$

Chlorine referred to as combined is in the form of chloramines, which form because of reactions with ammonia present in natural water supplies at pH levels below 7. These include monochloramine, dichloramine, and trichloramine, though only the first two are active oxidants. Once trichloramine is formed, the chlorine no longer has the potential to degrade carbon compounds. Chloramines are more stable and are therefore more often used to maintain concentrations within a water distribution system.[64]

Chlorine is most commonly added to water either in the form of pure chlorine gas or in the form of a sodium hypochlorite, or simply hypochlorite, also known as bleach. Calcium hypochlorite, the form of chlorine used in swimming pools, is more concentrated and may also be used.[64]

Mechanism

There are three general pathways that chlorine can react with substrate in water: oxidation, addition, and substitution. Chlorine reacts with organic material through combinations of these pathways depending on the material in the water.[65]

These chlorine compounds that are added to water have the potential to oxidize substrate. However, many competing reactions occur that demand consideration when treating for pesticides. Microorganisms are also deactivated by chlorine, though the underlying mechanism is still due to oxidation of cell walls or other materials that destroys the organism. In order of their oxidation potential, chlorine dioxide, then free chlorine, and then chloroamines are most effective. Ammonia within water bonds with chlorine to form chloramines. Other contaminants within water may also scavenge chlorine. Therefore, concentrations greater than the minimum chlorine demand calculated using standard methods might be added to increase the potential for degradation of the target species. Even in pure water to which concentrations of pesticides are added in a laboratory setting, rate constants may limit the time required for oxidation based on concentrations. Excess chlorine may speed up these reactions[9]

Dosages

The dose of any chlorine-containing compounds that is required to completely oxidize a pollutant can be calculated by experiments comparing applied chlorine concentrations to the residual chlorine concentrations in water samples with some chlorine demand. Methods described in *Standard Methods for the Examination of Water and Wastewater*, published by the American Public Health Association, American Water Works Association, and the Water Environment Federation, detail the determination of the chlorine demand of a pollutant. The demand for specific chlorine forms (total, free, combined, monochloramine or dichloramine) can be calculated depending on the test. Some of these tests include amperometric titration with phenylarsine oxide titrant to determine the remaining chlorine after a reaction period, titration with ferrous ammonium sulfate (FAS), or a colorimetric method comparing the quality of samples to chlorine standards using a spectrophotometer.[65] Studies have also been conducted where molar ratios of the chlorine-containing species up to 100:1 relative to the amount of substrate in water.[66]

Effectiveness

The effectiveness of chlorine at oxidizing the desired pesticide depends on the water quality. If other carbon sources are present in the water, these will exert a demand upon the chlorine and result in competing oxidation and a lower overall pesticide reduction. pH levels and turbidity also impact the distribution of available chlorine (free or combined).[9] If the water contains sulfides, excess chlorine must be used since this is a common reaction, which exerts a demand on the chlorine.

A study conducted in Spain measured the efficiency of various treatment techniques, chlorination among them, and their efficiency at removing 44 different pesticides from water. Chlorination was found to be 30% efficient at removing alachlor and 20% efficient for atrazine. Aluminum sulphate was added as a coagulant at various doses but found to have no impact. A number of the 44 pesticides studied did benefit from greater removal rates, up to 100% removal; an overall average removal of 60% of the studied pesticides was found. However, the chlorine dose applied to these pesticides was the minimum required to satisfy the chlorine demand. Higher concentrations may have contributed to greater removal since impurities in the water may have exerted a chlorine demand, or the contact time may have been insufficient for thorough oxidation.[67]

A study on glyphosate and glycine removal with chlorine used chlorine in molar ratios to substrate ranging from 1:1 to 100:1 to observe products formed. With the 100:1 molar ratio assumed to be comparable to the excess that water treatment facilities would use, glyphosate was totally degraded to end products phosphoric acids, carbon dioxide, methanediol, nitrogen, and nitrate.[66]

Feasibility and Economics

Chlorination is one of the least expensive treatment procedures. As such, it is commonly used in wastewater and drinking water treatment facilities. It is essential that concentrations too great are not consumed by humans and therefore controls and monitoring is necessary, which incur extra costs. However, the treatment procedure itself is easy to implement and run, as well as low in installation and chlorine costs.[64]

Dechlorination

In water treatment facilities, residual chlorine concentrations must be reduced before discharge to public water distribution systems in order to meet acceptable limits. While some chlorine must remain in the water throughout the piping, excess can be dangerous. Many compounds that are formed from chlorine, such as trihalomethanes (THMs), have been proven to be carcinogenic. Dechlorination is achieved through use of sulfur dioxide, solutions of sulfite compounds, or granulated activated carbon (GAC)[61] Use of carbon adsorption is more expensive and would be used when total dechlorination is required.

UV Photolysis

Treatment of waters and wastewaters with UV is another technique used to degrade organic pollutants and inactivate microorganisms to acceptable levels. UV light is a form of electromagnetic radiation in the wavelength range of 100 to 400 nanometers. There are four classes: UV-A between 400 and 315 nm, UV-B from 315 to 280, UC-C from 280 to 200, and vacuum UV from 200 to 100 nm. When used for water treatment, low-pressure mercury lamps (LP-UV) generate UV at a wavelength of 253.7 nm and medium-pressure lamps (MP-UV) generate UV and visible light in the 200-800 nm range. LP-UV lamps can provide up to 0.4 kW while MP-UV lamps can provide up to 30 kW of power. [68]

When used to degrade pollutants rather than targeting microorganisms, UV treatment is used as an advanced oxidation process (AOP) inducing photodegradation of organic and inorganic contaminants in water. The breakdown of pollutants into simpler compounds due to UV exposure is also called photolysis. Since this occurs at a very low rate with UV alone, hydrogen peroxide is often added to provide active hydroxyl radicals to bond to pollutants and oxidize compounds as a simultaneous and parallel reaction to the photolysis. Semiconductor catalysts may also be added to encourage this process through more rapid formation of active hydroxyl radicals or other radicals that attack pesticide substrate; this is then called photocatalysis.[69]

UV treatment provides a number of benefits, including potentially complete oxidation of organics within a few hours. In general, UV has the potential for many compounds to provide more successful removal than other treatment procedures such as biodegradation, or activated carbon that involves a phase transfer of pollutants rather than their actual destruction. Low concentrations may also be treated, in the ppb range.[69] Again, this varies by compound, but through the use of cost-effective catalysts that can be adapted to specific systems, photolysis can be optimized.

Mechanism

Photolysis is the chemical decomposition of compounds when a photon of light is absorbed. This however, does not fully degrade them. This is often a successfully destructive pathway for pesticides in surface water or on plant or soil surfaces that are therefore contacted by sunlight. The absorption of a photon induces molecular excitation and elevation from a ground state to an elevated state. This excited molecule may then undergo a degradation reaction through "direct" photolysis, or contact a ground-state molecule which will then undergo the reaction through "indirect" photolysis. Many photoreactions then involve bond cleavage. Typical photoreactions involve isomerization, dealkylation, carbon oxidation, dehalogenation, ester cleavage, cyclization, or sulfur oxidation.[70]

Oxidation involves the transfer of electrons when two compounds are combined. In the case of UV degradation, UV creates active bonding radicals that oxidize carbon (gain electrons from the carbon) and other organic and inorganic components of pesticides. Oxygen replaces hydrogen or other bonded molecules and, if complete oxidation is achieved, oxygen replaces all potential bonding sites provided by carbon's valence electrons. Hydroxyl radicals are often the oxidizing species. If the pesticide is completely mineralized, the final products are carbon dioxide as well as other fully oxidized compounds containing any other inorganic elements in the base compound. If incomplete degradation occurs, any number of byproducts may be formed and will vary in composition based on the parent compound and water quality overall. [68]

In natural waters with some amount of hardness, carbonate and bicarbonate species in the water react with hydroxyl radicals and UV degradation can be assumed to occur predominantly through photolysis rather than oxidation.[68] Expected byproducts vary with water quality, pH, the base pollutant compound, and the type of UV lamp (low or high pressure).

Hydrogen Peroxide Use

Peroxide use with UV radiation has been used traditionally to provide high concentrations of hydroxyl radicals to attack oxidation sites. For these reactions, UV radiation between 250 to 300 nm successfully generates the radical that propagates the oxidation reactions. The first reaction, Reaction 3, to generate the hydroxyl radical is as follows.[71]

$$H_2O_2 + hv \rightarrow \ 2OH^{\cdot} \qquad \text{(Reaction 3)}$$

Photocatalysis

Many semiconductor catalysts have also been used with UV for photocatalytic treatment, including iron oxides, cadmium and zinc sulfides, and oxides of zinc, tungsten, titanium, chromium, aluminum, and others. One of the most widely used is titanium dioxide, TiO_2 due to its "stability under harsh conditions, commercial availability, different allotropic forms with high photoactivity, [the] possibility of coating it as a thin film on solid support, ease of preparation in the laboratory, etc."[69] These may be used in conjunction with hydrogen peroxide or alone. Catalysts may be in a water suspension at a low concentration or in fixed or fluidized beds through which a flow is passed in order to receive treatment.

Pelizzetti reports that the major reactions occurring during photocatalysis with titanium dioxide are as follows:[72]

$$TiO_2 + hv \rightarrow e^- + h^+ \qquad \text{(Reaction 4)}$$

$$(O_2)ads + e^- \rightarrow (O_2^{\cdot})ads \qquad \text{(Reaction 5)}$$

$$Ti(IV) - OH^- + h^+ \leftrightarrow Ti(IV){-}{}^{\cdot}OH \qquad \text{(Reaction 6)}$$

$$Ti(IV) - OH_2 + h^- \leftrightarrow Ti(IV){-}{}^{\cdot}OH + H^+ \qquad \text{(Reaction 7)}$$

The UV radiation splits the semiconductor oxide into an anion and cation pair, which then initiates redox reactions with substrates in the water. The catalyst then encourages the formation of hydroxyl radicals which are highly reactive and begin oxidation of substrate. The active oxidizing agent in this set of reactions is the ${}^{\cdot}OH$ radical. Hydrogen peroxide may be added to systems where a semiconductor catalyst is being used to encourage the rapid formation of radicals.[72]

Dosage

Typical UV doses are in the range of 500 to 750 mJ/cm^2. The contact time may range from less than 30 minutes to over 24 hours.[68] Various doses and contact times should be investigated since kinetics vary among compounds and with respect to other water quality parameters. Increased energy inputs lead to greater ease in bond breaking.[68] In a glyphosate study that used three different UV lamps at 15 W, a 16% filter over a 40 W, and a unfiltered 40 W, the highest powered lamp (40 W) saw the greatest removal.[73] (Note – this study was performed on glyphosate alone and did not include surfactants and other ingredients in commercial formulations.)

Hydrogen peroxide concentrations are known to have a strong impact on the rate of decomposition. This specific peroxide concentration varies with the compound and can be represented by a range of ratios to the pollutant concentration. For some pollutants, the rate of pollutant photolysis increase asymptotically with the hydrogen peroxide concentration. When the concentration was increased from 5×10^{-3} to 5×10^{-1} M in a UV/H_2O_2 system for a pyridine herbicide, this led to a decomposition rate increase of almost 700%.[74] For the same glyphosate study, the molar ratio of hydrogen peroxide to substrate was optimally between 7.3 and 19.7.[73]

Effectiveness

UV is an important technique for investigation of the degradation of many types of pesticides, and can be particularly useful when applied to stable and persistent pesticides or pesticides that form many problematic byproducts.

Although organochlorides are stable and therefore difficult to degrade, photocatalytic oxidation has been shown to be effective. Depending on a number of operating conditions, the percentage of organochloride removal may exceed 90%. Main intermediates involve carboxylic acids and aldehydes.[68] The organochloride pesticide lindane was degraded 99.9% using a high-pressure UV lamp and a titanium dioxide catalyst.[74] DDT was degraded also with a titanium dioxide catalyst.[73] Pentachlorophenol, a wood preservative, experienced complete oxidation under UV treatment with a titanium dioxide suspension.[68]

Atrazine does not undergo complete mineralization because the triazine ring strongly resists hydroxyl radical attacks.[75]

Feasibility and Economics

UV is currently in use in many water treatment facilities after its growth in popularity in the mid-1980s. It is a successful method for inactivating pathogens and is therefore popular from the microbial standpoint, but it can also be effective at reducing organic concentrations such as pesticides. With increasingly strict chlorine discharge limits and the corresponding concentration reduction costs for chlorine treatment as well as the potential risks that stores of chlorine gas pose, UV is a viable alternative for large scale treatment facilities.

In a large scale system, sets of UV lamps housed in quart sleeves would be suspended in a water chamber that the wastewater would flow through. A particular residence time to obtain a required exposure would be determined based on the typical composition of the water and then applied to this chamber. Low-pressure UV systems would be used for low to medium water flows (up to 10 MGD). The contact time may be as low as 10 to 20 seconds, much lower than the 30 to 60 minutes of chlorine contact time.[75] For high flow systems, medium-pressure lamps would be used.

Ozonation

Ozonation is a popular water treatment technique that was first discovered and actively used in the 1800's. Today, the process is more commonly found in European and Asian water treatment plants as opposed to ones in the United States. It is a powerful oxidation technique in which a form of oxygen is forced through a contaminated liquid sample. With ozonation, O_3 can oxidize irons, sulfurs, and other various metals and contaminants through and oxidation reaction. Byproducts are agents that leave the solution as a gas, along with excess ozone and oxygen gas. After the ozonation process however, there will always be a residual amount of ozone left in the solution. A typical half-life of ozone in water is about 30 minutes. There are various methods to extract the residual ozone out of the solution. These methods include using a simple stir bar and agitating the water as well as putting the solution in a sonication bath. On a much larger scale, treatment plants can still use agitation. The implementation of agitation however, would be to use the water's turbulent flow rather than a physical object mixing.[76]

Mechanism

To begin the process, ozone must first be created from an oxygen source: air. To do this, electric current is run through air, charging the molecules and converting diatomic oxygen (O_2) to ozone (O_3). The ozone is then fed through the water. Because of the instability of ozone, the molecules actively degrade to diatomic oxygen and a free radical (O^{2-}). Free radicals are highly unstable and can only be found in nature for a matter of nanoseconds before they bond to the closest molecule that will accept them. In solution, the oxygen radicals search for the nearest component to bind to, forming a compound that can escape with the ozone. Figure 10 describes the mechanism in terms of the oxygen content (O) and the relevant pesticides or contaminants (R).[77]

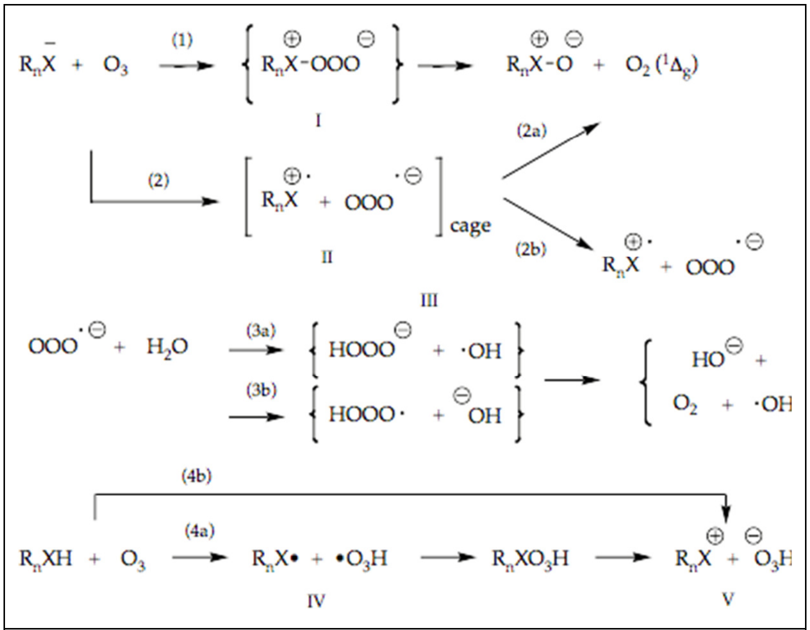

Figure 10: Ozonation Mechanism[77]

Perozone

Hydrogen peroxide may be added during the ozonation process, called perozone. The mechanism is the same as ozonation but the addition of hydrogen peroxide quickens the decomposition of ozone molecules and increases the amount of hydroxyl radicals in the solution. The reason for the increase in reaction while using the perozone process, is directly related to the oxidation potential. With greater oxidation potential, the solution is more reactive and contaminants can be removed more quickly. Below are the reactions behind both ozonation and perozonation reactions; equations 8 and 9 show standard ozonation, while reaction 10 shows that of a perozone reaction. The oxidation potential for the perozone reaction is clearly the highest and in turn is the most efficient. The disadvantage of using the perozone process over a standard ozonation reaction is the actual cost. For obvious reason adding large quantities of hydrogen peroxide into contaminated water can become very expensive very quickly. Therefore for large quantities of water, standard ozonation is the preferable method.[78]

$$OH + e^- \rightarrow OH^- \qquad E^0 \rightarrow +2.8V \qquad \text{(Reaction 8)}$$

$$O_3 + 2H^+ + 2e^- \rightarrow O_2 + H_2O \qquad E^0 \rightarrow +2.07V \qquad \text{(Reaction 9)}$$

$$O_3 + H_2O + 2e^- \rightarrow O_2 + 2OH^- \qquad E^0 \rightarrow +1.24V \qquad \text{(Reaction 10)}$$

Effectiveness

Over the course of many studies, ozonation has been determined have varying results in removing inorganic and organic material as well as pesticides from drinking water. In terms of inorganic and organic material from water, studies have shown that the average removal rate is between 40-60% of the matter. The introduction of a coagulant however, can increase the average removal rate. The reason for this is due to the increase in efficiency of the coagulant. When ozonation is used in conjunction with a coagulant, removal rates increase to an average 65%.

Ozonation has been found to be very effective to reduce concentrations of pesticides in drinking water. Table 9 reports removal rates of some common pesticides treated with ozone. In 50% of the tested pesticides, a sufficient amount of the contaminant was removed from the drinking water. A sufficient amount, as assumed by the study, was a removal rate of 80% or higher.[79]

Table 9: Degradation of Selected Pesticides through Ozonation[79]			
Pesticide	**pH 7,2; 5 °C; O_3/DOC = 1,0**	**pH 7,2; 20 °C; O_3/DOC = 1,0**	**pH 8,3; 20 °C; O_3/DOC = 1,0**
diazinon	86	92	92
dimethoate	97	97	97
parathion-methyl	85	91	91
diuron	91	95	98
linuron	67	81	89
methabenzthiazuron	78	90	94
metobromuron	83	91	94
MCPA	83	87	90
MCPP	91	93	93
chlortoluron; isoproturon; metoxuron; vinclozolin	> 99	> 99	> 99

Feasibility and Economics

Many studies have been conducted over a long period of time to study the economic feasibility of ozone use in water treatment plants. One particular study, conducted by Spartan Environmental Technologies, notes that the benefit of using clean oxygen as opposed to chemicals is offset by the high capital and operational cost of using an ozonation system. This study considered a system lifetime of twenty years in order to evaluate the lifetime cost (capital and expenditure) of an ozone generator. For a large scale plant, liquid oxygen (LOX) would be used to generate ozone. On a smaller, experimental or pilot scale, an air source would be used to produce the oxygen. The study concludes that ozonation in a large scale treatment plan, where LOX would be a necessity, is incredibly expensive. The study also made assumptions regarding typical system life spans and calculated the overall lifetime cost of using an air ozone generator versus using an ozone generator with LOX. The general assumptions included the interest rate over the twenty year life span, capital cost of an ozone generator system as well as the cost of electricity used to produce the ozone. The cost difference between ozone generation systems was calculated. Table 10 reports the net present value (what the system is worth in today's market) of an ozonation system (capital and operational cost) for both ozone generation methods, considering three different LOX pricing values since the price of the liquid oxygen can vary significantly (sometimes over 200 $/ton) depending on the economy and the distributer. For the assumptions used to generate this table, see Appendix B.[80]

Table 10: Price of Using Ozonation[80]			
Net Present Value of System			
LOX Pricing ($/ton)	**Cost of Ozone Generation from Air**	**Cost of Ozone Generation from LOX**	**Difference***
50	$2.15 MM	$1.74 MM	$410 M
100	$2.15 MM	$2.25 MM	($100 M)
150	$2.15 MM	$2.27 MM	($620 M)
*Indicates that value is negative. I.e., LOX more expensive			

Ferrate

Ferrate is an iron compound in which iron is present in its +6 oxidation state (Iron VI). It is represented by the formula FeO_4^{2-}. Ferrate is applied in the form of a solid, which then dissolves into solution in solution to form its ionic components. Ferrate, in an ionic solution, is considered to be unstable.

Mechanism

The mechanisms by which ferrate acts on a contaminant is summarized by Figure 11.

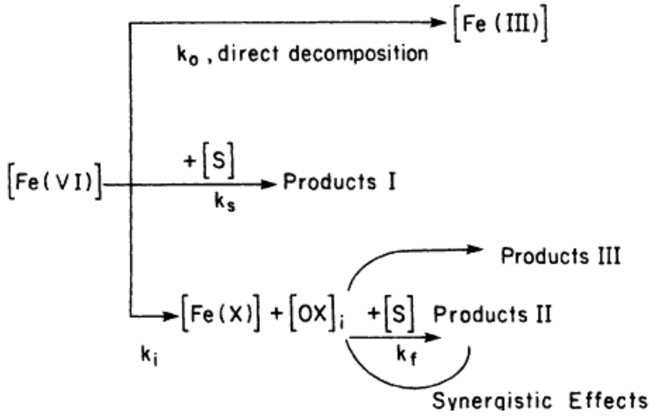

Figure 11: Ferrate Reaction Mechanism[81]

When added to water, ferrate(VI) ions rapidly decompose through Reaction 11:

$$2FeO_4^{-2} + 5H_2O \rightarrow 2Fe_3^{+3} + \frac{3}{2}O_2 + 10OH^- \qquad \text{(Reaction 11)}$$

The ferric ion produced by this decomposition is non-toxic and is a strong oxidant. It is the ferric ion that acts upon the contaminant during the water treatment process. Additionally, the ferric oxide that is produced from ferrate(VI) acts as a coagulant that is effective in removing metals and non-metals.[82] This coagulant can then help agglomerate insoluble contaminants in the water and facilitate separation steps later, such as filtration to remove the insoluble particles. Because ferrate can deliver multiple ways to treat contaminants in water from one dose, i.e. coagulation, oxidation and disinfection, it is regarded as one of the most cost efficient water treatment methods.

Effectiveness

Table 11 shows the oxidation/reduction potential for common oxidants used in wastewater treatment. Ferrate has the greatest oxidative potential of the evaluated disinfectants/oxidants.

Table 11: Oxidative Potentials of Common Disinfectants/Oxidants[81]		
Disinfectant/Oxidant	**Reaction**	**E^0, V**
Chlorine	$Cl_2(g) + 2e \leftrightarrow 2Cl^-$ $ClO^- + H_2O + 2e \leftrightarrow Cl^- + 2OH^-$	1.358 0.841
Hypochlorite	$HClO + H^+ + 2e \leftrightarrow Cl^- + H_2O$	1.482
Chlorine Dioxide	$ClO_2(aq) + e \leftrightarrow ClO_2^-$	0.954
Perchlorate	$ClO_4^- + 8H^+ + 8e \leftrightarrow Cl^- + 4H_2O$	1.389
Ozone	$O_3 + 2H^+ + 2e \leftrightarrow O_2 + H_2O$	2.076
Hydrogen Peroxide	$H_2O_2 + 2H^+ + 2e \leftrightarrow H_2O$	1.776
Dissolved Oxygen	$O_2 + 4H^+ + 4e \leftrightarrow 2H_2O$	1.229
Permanganate	$MnO_4^- + 4H^+ + 3e \leftrightarrow MnO_2 + 2H_2O$ $MnO_4^- + 8H^+ + 5e \leftrightarrow Mn^{2+} + 4H_2O$	1.679 1.507
Ferrate (VI)	$FeO_4^{2-} + 8H^+ + 3e \leftrightarrow Fe^{3+} + 4H_2O^-$	2.20

Preparation of Ferrate

There are three ways to produce ferrate (VI): via dry oxidation, electro-chemically, and via wet oxidation. In dry oxidation, various iron oxide containing minerals are heated or melted under conditions of heavy alkalinity and strong oxygen flow. However, this method of formulating ferrate is considered to be extremely dangerous, since the synthesis process could cause detonation at high temperatures. Either potassium or sodium ferrate(VI) can be prepared by the calcination of a mixture of ferric oxide and potassium peroxide, while the mixture is held between 350-370°C. This yields a product containing FeO_5^{4-}, which is immediately hydrolyzed to form ferrate. An additional dry oxidation preparation method involves mixing galvanizing wastes with ferric oxide in an 800°C furnace.[81]

In the electro-chemical formulation of ferrate, ferrate (VI) salts are formed by using cast iron as an anode. The iron is then dissolved and oxidized to form K_2FeO_4 when potassium hydroxide (KOH) is used as an electrolyte. It has been found that current density through the electrolytic system and the temperature and concentration of the electrolyte have large effects on the production yield of ferrate. The greatest production yield observed was 40%, which was seen at a current density of 3 mA/cm^2 and an electrolyte temperature of 30°C. It was also determined that a 14M solution of NaOH is optimal for the electrochemical synthesis of ferrate (VI) salts. The overall reactions for the electrochemical production of ferrate (VI) salts are as follows[81]:

Anode Reaction: $$Fe + 8OH^- \rightarrow FeO_4^{2-} + 4H_2O + 6e \qquad \text{(Reaction 12)}$$

Cathode Reaction: $$2H_2O \rightarrow H_2 + 2OH^- - 2e \qquad \text{(Reaction 13)}$$

Overall Reactions: $$Fe + 2OH^- + 2H_2O \rightarrow FeO_4^{2-} + 3H_2 \qquad \text{(Reaction 14)}$$

$$FeO_4^{2-} + 2K^+ \rightarrow K_2FeO_4 \qquad \text{(Reaction 15)}$$

Production of ferrate(VI) salts via wet oxidation is done by oxidizing a ferric containing solution to form a ferrate(VI) solution under high alkaline conditions. Wet oxidation to form ferrate(VI) salts has been studied since the 1950's; one of the primary mechanisms for the formation of ferrate via Reactions 16-18[81]:

$$Fe^{3+} + 3OH^- \rightarrow Fe(OH)_3 \qquad \text{(Reaction 16)}$$

$$2Fe(OH)_3 + 3NaClO + 4NaOH \rightarrow 2Na_2FeO_4 + 3NaCl + 5H_2O \qquad \text{(Reaction 17)}$$

$$Na_2FeO_4 + 2KOH \rightarrow K_2FeO_4 + 2NaOH \quad \text{(Reaction 18)}$$

Because the ferrate solution would decompose rapidly, the ferrate must be precipitated, washed and dried to obtain a solid stable product.

In one case study conducted by Waite and Gilbert,[81] allylbenzene, chlorobenzene, benzene, 1-hexene-4-ol, aniline, phenol, and nitrobenzene were exposed to controlled doses of ferrate and monitored. The experiments were conducted at substrate to ferrate molar ratios of 1 to 5 for allylbenzene, 1 to 3.3 for chlorobenzene, benzene, and 1-hexene-4-ol, and 2 to 1 for aniline, phenol, and nitrobenzene. The pH of the solution was varied from 7 to 11 while the temperature was held constant at 20°C. In order to determine whether the ferrate was reacting with the substrates, Waite and Gilbert tracked the absorbance of the ferrate over time, using UV spectroscopy at a wavelength of 505 nanometers. The decomposition curve of ferrate in water is shown below. As shown by Figure 12, ferrate decomposes more rapidly at a lower pH, which shows that ferrate is more reactive, and therefore better at removing impurities, at those levels of pH. [81]

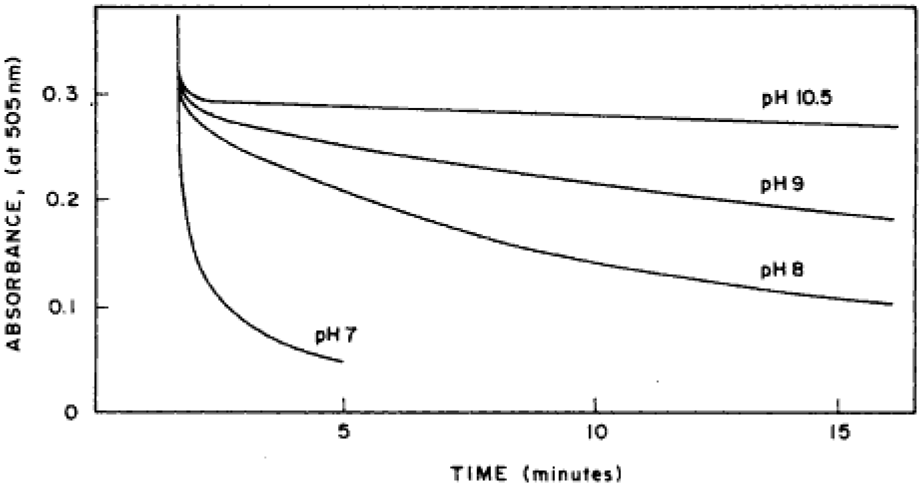

Figure 12: Decomposition of Ferrate over Time[81]

It was observed that allylbenzene, chlorobenzene, nitrobenzene and 1-hexene-4-ol were found to have no effect on the rate of decomposition of ferrate, suggesting that there is a slow selective reaction between a pH of 7 and 11. Phenol and aniline caused an acceleration of the ferrate decomposition, suggesting a direct reaction with ferrate. Lastly, benzene retarded the rate of decomposition, suggesting that there is some protective effect from the benzene. Additionally, the maximum oxidation percentages at pH less than 8 were 18-47%, 23-47%, 85-100%, and 32-55% for benzene, chlorobenzene, allylbenzene and phenol respectively. These rates were achieved at molar ratios of ferrate to organic matter between 3:1 and 15:1. This shows that higher molar ratios of ferrate added to solution will have a more complete reaction with contaminants. Additionally, this shows that acidic pH's are more suited for the use of ferrate as a decontaminant. [81]

Other case studies were carried out to measure ferrate's ability to oxidize other common inorganic pollutants, such as cyanide, ammonia and hydrogen sulfide. It was concluded that ferrate is an appropriate removal method for cyanide. At temperatures between 15-30°C and pH's ranging from 8-12, more than 22% of ammonia was removed when the ratio of ferrate to solvent was greater than one. Under the same conditions, approximately 99.9% of hydrogen sulfate was removed from water.[81]

Methodology

This research aimed to:

1. Research and establish the historical toxicity, health implications, and prevalence in the environment of the three pesticides alachlor, atrazine, and glyphosate.
2. Evaluate the removal efficiency of ozonation, chlorination, activated carbon, UV radiation, and ferrate at removing atrazine, glyphosate, and alachlor from water.

The oxidative potentials of the oxidants studied are listed in Table 12.

Table 12: Oxidative Potentials of Oxidants Studied

Oxidant	Oxidative Potential, E^0 (V)
Hypochlorite	1.48
Ozone	2.07
Ferrate (VI)	2.20
Hydroxyl Radical	2.80

Accordingly, the strongest oxidants with the highest oxidative potentials were hypothesized to be most effective, namely ferrate and UV oxidation when hydrogen peroxide was added and converted into the hydroxyl radical. The removal for time-based reactions (UV photolysis and ozonation) was hypothesized to increase with exposure time and with the addition of hydrogen peroxide to increase the concentration of free radicals. The removal achieved with activated carbon, chlorine, and ferrate was hypothesized to increase with the amount of activated carbon, hypochlorite ion, or ferrate used.

Solution Preparation

Stock solutions were prepared for each chemical as to control the input variables to the experiments. A set value of chemical was added to a likewise predetermined volume of water. The samples were prepared with the intention of being able to retrieve detectable concentrations without approaching the solubility limits. Solutions were mixed at 25°C for 24 hours or until no insoluble particles were observed.

For atrazine, the stock solution was prepared with 19 mg of chemical added to 1 liter of purified water. This corresponds to approximately 57% of atrazine's solubility limit (33 mg/L). This corresponded with an absorbance of approximately 0.32 at a wavelength of 265 micrometers.

For alachlor, samples were prepared with between 147 and 188 mg of chemical added to 1 liter of purified water, which corresponds to between 60 and 70% of the maximum solubility of alachlor (242 mg/L). At these concentrations, the absorbance fell between 0.315 and 0.4 at 265 micrometers.

Glyphosate samples were prepared with 300 mg of chemical in one-half liter of purified water. This solution is equivalent to 5% of glyphosate's maximum recorded solubility (12 g/L). The primary reason for using a considerably lower percentage of glyphosate's solubility was availability of the chemical. The absorbance at this concentration was 0.023 at 255 micrometers.

All water used for this research was purified through reverse osmosis with a Thermo Scientific E-Pure unit.

Formation of Calibration Curves

Calibration curves were constructed so that the absorbance results collected through experimentation could be directly converted into correlated concentration values. In order to create the calibration curves, the standard solutions of known concentrations for each chemical were prepared. Each dilution was measured in a Cary 50 Scan UV-Visible Spectrophotometer at the chemical's wavelength where the chemical absorbed the greatest amount of UV light.

The absorbance values were then plotted against the known concentrations of the standard solutions. R^2 values greater than 0.97 were used to demonstrate linearity in accordance with Beer's Law relating absorbance to concentration.

The calibration curves for the three chemicals are shown in Figures 13-15.

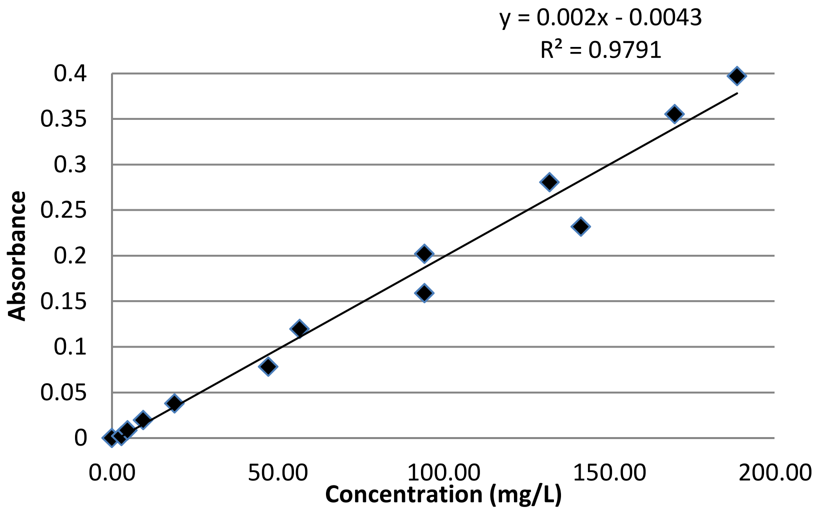

Figure 13: Calibration Curve for Alachlor at 265 nm

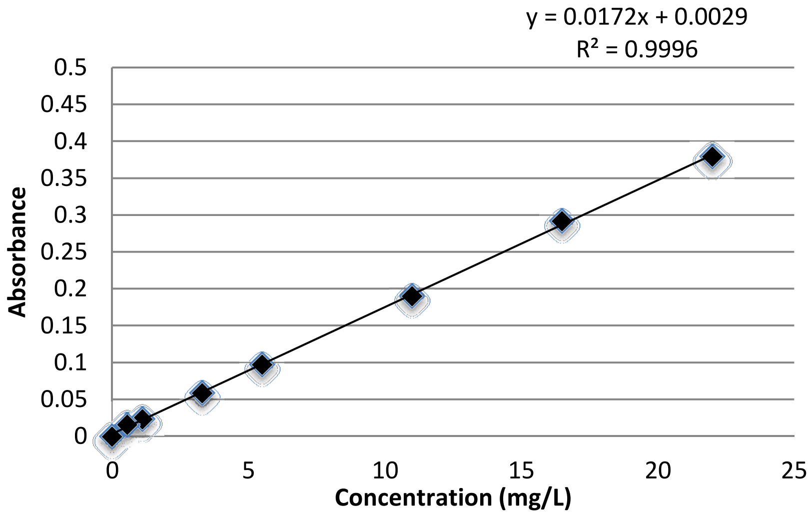

Figure 14: Calibration Curve for Atrazine at 265 nm

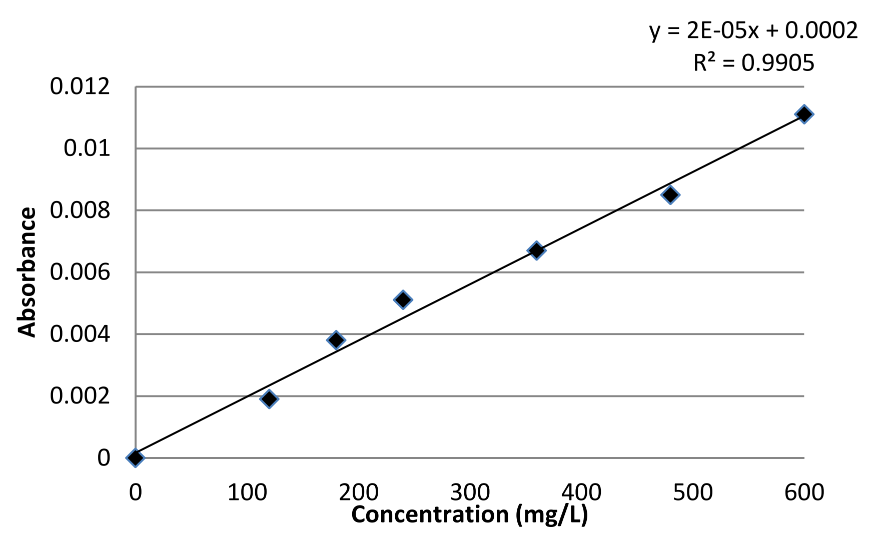

Figure 15: Calibration Curve for Glyphosate at 255 nm

Activated Carbon Adsorption

Figure 16: Activated Carbon

Two types of activated carbon were used to remove the pesticides from water. The two types of activated carbon are powdered and granular activated carbon. As the name suggests, the powdered activated carbon (PAC) came in a powder while the granular activated carbon (GAC) came in slightly larger particles. The same procedure was used for both types of activated carbon to ensure consistency. For each sample, a mass of activated carbon was added to 40-mL glass vial. A volume of the aqueous pesticide solution was then added to the vial. Vials were sealed and put into a rotator to mix for 24 hours. The rotator rotated at a constant speed during this time. For all samples, the 24-hour residence time remained constant while amounts of pesticide and activated carbon varied.

The mass ratio of pesticide to activated carbon was varied in order to find the most effective ratio for removal. Isotherms were plotted in which the equilibrium concentration was graphed versus the mass of pesticide removed divided by the mass of activated carbon used. Depending on resulting data, the mass ratio between pesticide and activated carbon was changed in order to get data points at the appropriate range.

Once the vial was removed from the rotator, it was transferred to an Eppendorf Centrifuge 5804. Due to the physical constraints of the glass vial, the centrifuge was set to spin the vials at 2600 rpm. Depending on the type of activated carbon, the time in which the centrifuge would spin the sample would vary from 15 to 45 minutes. After centrifugation, samples were removed and scanned in the UV spectrophotometer to measure the absorbance level. The level was recorded and the resulting concentration was plotted.

Chlorination

Samples were treated with a calcium hypochlorite in water solution at 10% of the maximum solubility. Molar ratios relative to the pesticide concentration ranging from 1 to 25 were used but were limited to these ratios in order to maintain final absorbance readings within the reliable, linear region of the UV spectrophotometer absorbance readings closer to or below a value of 1 where absorbance was known to correlate very strongly to concentration.

Samples of the pesticide (20-30 mL) were added to 30-mL vials along with small (up to 1 mL) samples of the 10% solubility calcium hypochlorite corresponding to the various molar ratios. Two sets were prepare for each pesticide: at pH 4 and at pH 9, above and below the pKa for hypochlorous acid of 7.6. Samples were rotated and mixed for 24 hours. Final absorbance values were recorded and samples were mixed for an additional 24 hours to ensure equilibrium removal had been reached.

Solutions of calcium hypochlorite in water were measured at both 255 and 265 nm, at pH 4 and pH 9, to observe any absorbance at these wavelengths that would interfere with and compound upon the pesticides' absorbances, making calculations of absorbance to concentration unreliable. Since significant interference was absorbed, calibration curves were created. Table 13 reports the calibration curves and R^2 values for these four curves.

Table 13: $Ca(OCl)_2$ in Water Calibration Curves

	pH 4		pH 9	
	Equation	R^2	Equation	R^2
265 nm	Abs = 0.00001*conc - 0.0009	0.9995	Abs = 0.0006*conc + 0.0031	0.9933
255 nm	Abs = 0.00001*conc + 0.0003	0.9979	Abs = 0.0005*conc - 0.0044	0.9995

To determine the final pesticide concentration, the calibration curve for the correct wavelength and pH was used to calculate the contribution to the absorbance from the unreacted calcium hypochlorite. The reacted amount was assumed to be between 0 and 1 moles since at high molar ratios, the calcium hypochlorite concentration reduction was small but at lower molar ratios it was much more significant. Remaining calcium hypochlorite was then either the initial concentration or one mole less than the initial concentration, noting that one mole of calcium hypochlorite yields two moles of the hypochlorite ion. This calcium hypochlorite contribution was then subtracted from the final absorbance measured after 24 hours to determine the final pesticide concentration and the removal percentage.

Ozonation

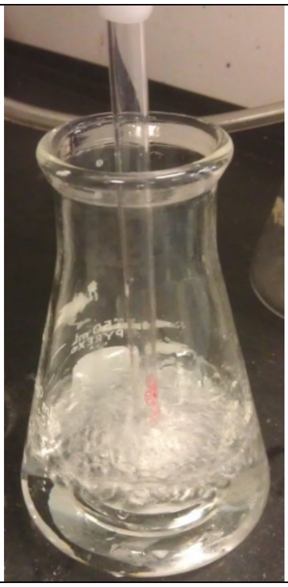

Figure 17: Ozonation Contactor

A tank containing oxygen was connected to an ozone generator and pumped into the generator at a flow rate of 8 SCFH. The flow of oxygen was acted upon by an electrical current to convert diatomic oxygen into ozone. While the actual potential supplied by the unit was unknown, the setting was kept at its maximum setting for the duration of the experiment in order to maximize the amount of ozone produced. The value of the applied potential was unknown due to the limited information reported by the generator. No units were given, only a numerical dial having no without a provided correlation to the actual potential. The ozone gas and resulting free radicals were then fed through a contactor into a flask containing the solution, shown in Figure 17.

An Erlenmeyer flask was used to contain 75 mL of pesticide solution. The ozone contactor was then placed into the flask and ozone generator was started, feeding ozone gas into the solution. The flask remained uncovered to allow the escape of waste gases. The contact time for the ozone ranged from 5 to 90 minutes with absorption readings taken every 5-15 minutes.

After each 5-15 minute interval, aqueous samples were extracted in order to measure the absorbance. Before the sample could be measured however, the excess ozone in the solution needed to be extracted. The half-life of ozone dissolved in water is about 30 minutes.[54] In order to expedite this process, two extraction methods were used. The two methods were an agitator and a sonification bath. The agitator was a simple stir bar in the solution. The stir bar was set to an approximate speed of 350 rpm. The contact time for the agitator was in a ratio of 1:1 with how long the solution was treated with ozone. The second extraction method used was a sonication bath. The bath was filled with purified water just deep enough to cover the portion of the flask filled with the solution. The contact time for the sonication bath was approximately 20 minutes regardless of the contact time with the ozone generator. Because of the static residence time, the sonification bath was the primary means of extraction.

UV Photolysis


Figure 18: UV Treatment Set Up

UV photolysis was used to reduce pesticide concentrations with and without the addition of hydrogen peroxide. A low pressure mercury vapor Pen-Ray 5.5 watt lamp (ACE No. 12132-08) supplying UV light at 254 nm and a medium pressure UV lamp, the Spectronics Corporation Spectroline 36-380 Long Wave Pencil Lamp, supplying UV light at 365 nm were both used. Figure 18 shows a schematic of the lab scale reactor where a glass tube holds an aqueous sample (6 mL) while UV light is applied for a specified period of time. After this exposure time, the sample's absorbance was measured at the specified wavelength to determine the final pesticide concentration as compared to the initial concentration.

The exposure time was first set at 90 minutes to measure the maximum removal when any subsequent UV exposure did not lead to further concentration reduction. Both lamps were used at this stage to determine which wavelength would be most effective for each pesticide. The determinedly more effective lamp was used for each respective pesticide for further tests. For all three pesticides, the low-pressure lamp was more effective.

Trials at smaller time intervals were then conducted to observe the rate of degradation under UV exposure. These were run at 5 minutes, 15 minutes, 30 minutes, and 60 minutes. Further treatment was added as necessary if the removal of the pesticide was less than 90%. For all samples, the absorbance at the pesticide's characteristic wavelength was recorded. Scans of absorbance's from 500-200 nm were run to observe the potential for byproducts of the oxidation reactions that could have contributed to the absorbance and have been misrepresented in concentration-based analyses.

Hydrogen Peroxide Addition

Hydrogen peroxide was then added in molar ratios of 12.5:1, 25:1 50:1 and 100:1 relative to the amount of pesticide. The higher ratios were expected to be more effective based on the literature.

After each ratio was allowed to react for 45-60 minutes, the most effective ratio was then rerun and monitored in shorter time intervals to observe the changes in concentration with respect to time. The absorbance was measured at all time intervals. Scans of the absorbance from 500-200 nm were run when byproducts were anticipated based on the results of UV treatment without hydrogen peroxide. A sample size of 6 mL and the low pressure lamp were used for all runs.

Ferrate Oxidation

Figure 19: **Potassium Ferrate Solid and in Aqueous Pesticide Solution**

Potassium ferrate, shown in Figure 19, was used in various molar ratios relative to the pesticide used. Molar ratios of 1:1, 2:1, 4:1, 8:1 and 16:1 of potassium ferrate to glyphosate in water were used, with respect to the iron species that reacts (1 mole of iron in 1 mole of potassium ferrate). Solid potassium ferrate was added to 40-mL vials with volumes of the pesticide between 20-30 mL. The pH was lowered to 3 with the addition of 1 M HCl since the reaction proceeds further under acidic conditions. The vials were then capped and then rotated and agitated for 24 hours. Solids and suspected dissolved solids were present at this point so the pH was then raised to 8 to minimize the solubility of ferric hydroxide.[86] Various methods of removal were then attempted, including centrifugation at 2600 RPM for up to 2 hours, gravity filtration through 25 mm Whatman glass microfiber filters, and vacuum filtration through 0.22 um Millipore Durapore (PVDF) filters. Final samples were measured in the UV spectrophotometer at glyphosate's characteristic wavelength and also scanned from wavelengths of 500-200 nm to observe any other wavelengths where the solution absorbed.

Results and Analysis

Results are presented by treatment method, for the pesticides studied with each method.

Activated Carbon Adsorption

Alachlor

Granular activated carbon was highly effective at removing alachlor from water. Solutions were treated with varied amounts of activated carbon corresponding to mass ratios from 1 to 50 of activated carbon t alachlor. The maximum removal was 98%, achieved at a 25:1 mass ratio. Removal past this ratio did not significantly increase the percentage of removal. For ratios up to 25:1, Table 14 reports the removal.

Table 14: Alachlor Removal with Granular Activated Carbon (24 Hour Contact Time)

Mass Ratio of GAC : Alachlor	Alachlor Concentration Removal %
1	40
3.5	66
5	60
10	89
15	91
20	94
25	98

The Freundlich empirical model for adsorption was used to represent this data, where constants were found from experimental data to develop a logarithmic curve. Figure 20 compares the Freundlich model isotherm to the experimental data. Here, the x-axis is the equilibrium concentration after 24 hours and the y-axis is the loading, or amount of alachlor adsorbed per unit of activated carbon. The point at which the curve would be expected to reach an asymptote represents the maximum removal, beyond which no greater removal per unit of activated carbon (definition of loading) can be achieved.

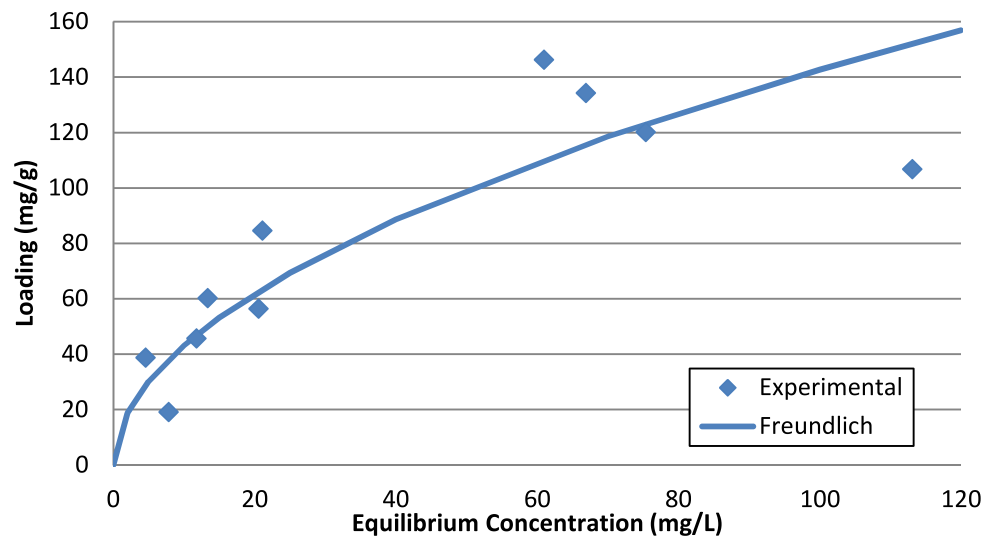

Figure 20: Alachlor GAC Isotherm

Atrazine

Activated carbon was highly effective in removing atrazine from water. Since the concentrations of atrazine in solution were low and limited by the solubility of atrazine, greater masses of activated carbon were required for accurate measurements. Therefore, mass ratios up to approximately 4,000:1 of activated carbon to pesticide were used. Table 15 reports the various ratios and their corresponding removal rates. A 92% removal was achieved with a mass ratio of activated carbon to atrazine of 18:1. Mass ratios greater than this did lead to slightly greater removal, but the much larger amounts of activated carbon required indicate that a lower molar ratio would be more appropriate from a cost analysis. A mass ratio between 18 and 117 should be investigated to determine the minimum mass ratio required to increase removal up to 95%.

Table 15: Atrazine Removal with Granular Activated Carbon (24 Hour Contact Time)

Mass Ratio of GAC : Atrazine	Atrazine Concentration Removal %
3	21
7	53
18	92
117	96
2270	97
3422	97

Figure 21 shows the Freundlich isotherm for the atrazine experimental data. The data points logarithmically ascend until a horizontal asymptote is reached at a loading of approximately 400.

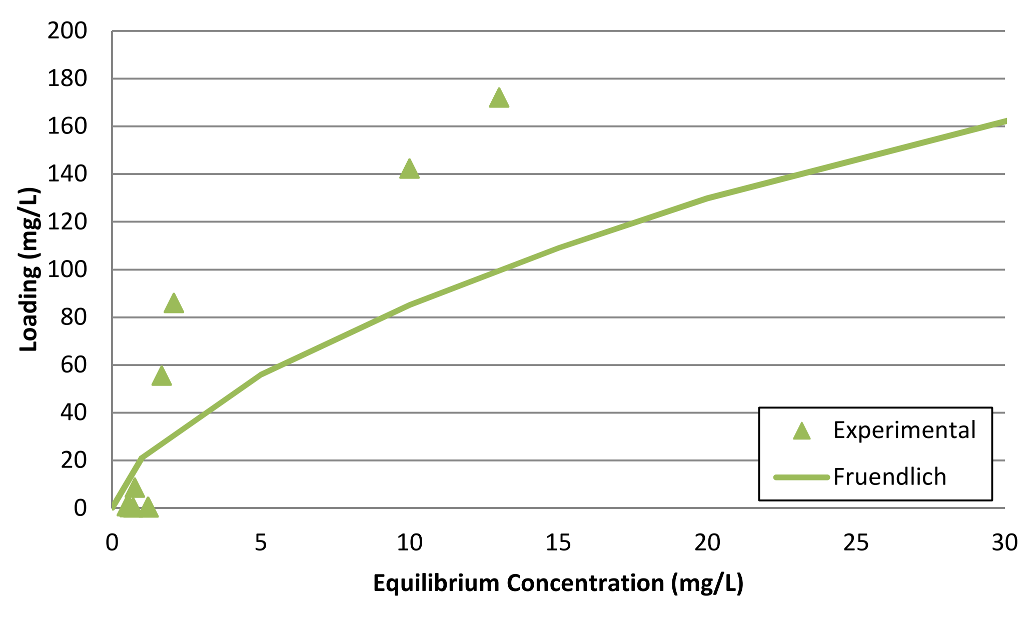

Figure 21: Atrazine GAC Isotherm

Glyphosate

Granular activated carbon displayed only a poor to moderate level of removal of glyphosate from water. Solutions of glyphosate were treated with the activated carbon for 24 hours in mass ratios of activated carbon to glyphosate ranging from 0.1:1 to 25:1. The solutions displayed unpredicted behavior, with ratios above 15:1 displayed drastically decreased adsorption of chemical. The greatest removal was approximately 51% at this 15:1 ratio. Table 16 shows the percent removal of glyphosate achieved at corresponding mass ratios.

Table 16: Glyphosate Removal with Granular Activated Carbon (24 Hour Contact Time)

Mass Ratio of GAC : Glyphosate	Glyphosate Concentration Removal %
0.1	3.7
0.5	4.7
1	3.8
5	15.7
10	45.4
15	50.7

Figure 22 shows the Freundlich isotherm alongside the experimental adsorption data.

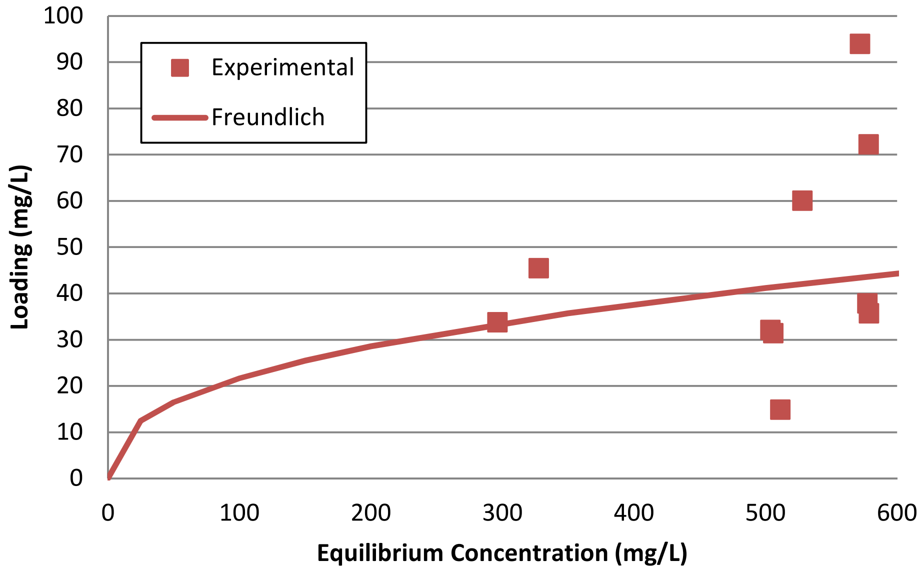

Figure 22: Glyphosate GAC Isotherm

Activated Carbon Adsorption Summary

Figure 23 shows Freundlich isotherms modeling the removal for all three pesticides using granular activated carbon for the 24 hour equilibrium contact period. Points are experimental data and the curves are Freundlich isotherms. The different slopes of the curves indicate that the amount of pesticide that may be removed per unit of activated carbon, or the loading (y-axis), varies largely among the three pesticides. Atrazine requires less activated carbon for its removal than alachlor or glyphosate. The equilibrium concentrations remaining in solution differ largely as well but this is affected by the starting concentrations in solution. Since solutions were prepared based on each pesticides' individual solubility, similarity over the x-axis is not expected. That is, glyphosate was roughly 30 times as concentrated as atrazine.

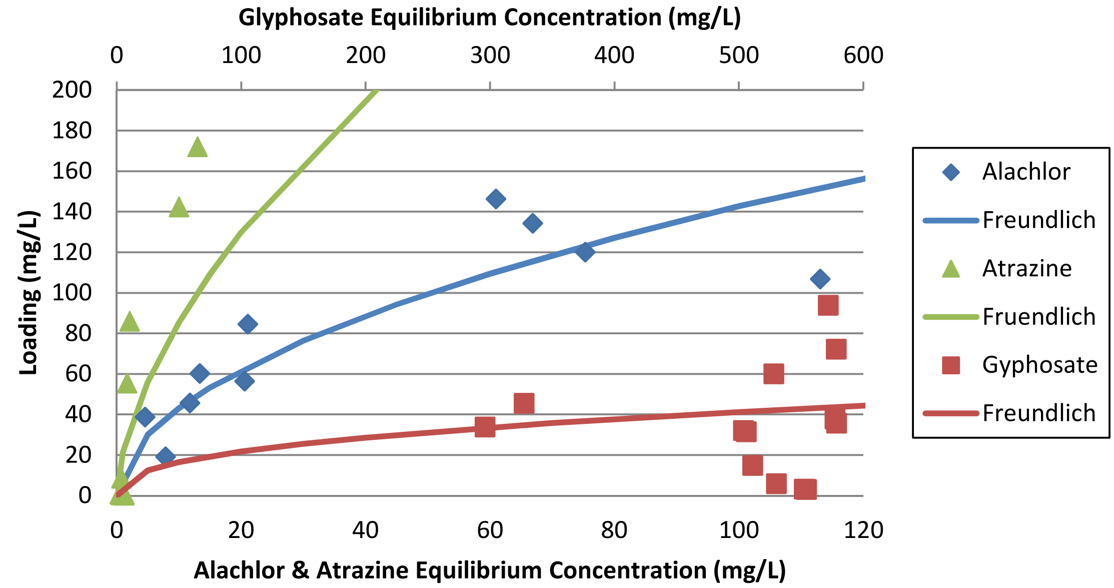

Figure 23: Activated Carbon Isotherms and Freundlich Models

The maximum removals observed were 98% of alachlor at a 25:1 mass ratio of activated carbon to pesticide, 92% of atrazine at a 18:1 ratio, and 51% of glyphosate at a 15:1 ratio. Greater amounts of activated carbon may have removed more glyphosate.

One hypothesized reason as to the limited reduction of glyphosate in solution is the relative size of the molecule in relation to the size of alachlor and atrazine. Alachlor and atrazine are both aromatic organochlorides, whereas glyphosate is a branched organophosphate. Therefore, the overall length of a glyphosate molecule could be longer than that of alachlor or atrazine, inhibiting adsorption. Additionally, the phosphorous atom present in glyphosate is 1.26 angstroms, compared 0.97 angstroms for chlorine, which could further hinder adsorption onto the activated carbon surface.

Chlorination

Alachlor

Treatment of alachlor with a calcium hypochlorite solution prepared at 10% of its solubility was most effective at reducing alachlor concentrations at high molar ratios and an elevated pH. Table 17 shows the concentration reduction percentages of the different molar ratios of calcium hypochlorite to alachlor at pH 9. The highest molar ratio, 25:1, was most effective and reduced concentrations by between 51 and 59%. Higher molar ratios would be expected to further increase removal but were not tested here due to constraints of the measurement technique—higher molar ratios would have led to an absorbance interference from the calcium hypochlorite too great to accurately factor out. This assumption had error associated with it, particularly at low molar ratios when the amount of hypochlorite ion actually consumed in a reaction represented a significant portion of what was added. At greater molar ratios when the amount reacted was lower compared to the amount added, the error associated with the assumption was lower.

Since the calcium hypochlorite in solution contributed to absorbance at a wavelength of 265 nm, this had to be factored out of the final absorption values measured in the spectrophotometer. To simplify analysis, the assumption was made that no moles of the hypochlorite ion were reacted with the pesticide and removed from solution by consumption. Therefore, final absorbance values after the 24 hour contact period were adjusted to remove the UV absorbance interference from the total amount of calcium hypochlorite that was added.

Table 17: Alachlor Concentration Reduction with Calcium Hypochlorite Treatment (pH 9)	
Molar Ratio of OCl^-(ion):Alachlor	**% Concentration Removal (Assuming No OCl^- Reacted)**
1	6
5	12
10	19
15	32
25	59

When samples were scanned over wavelengths from 500-200 nm, the only sharp peak observed was at 265 nm which did not seem to indicate any new, distinct byproducts being formed. However, additional products at 265 nm may have been present. Figure 24 shows scans of solutions of different molar ratios over wavelengths from 500-200 nm, where higher concentrations of calcium hypochlorite led to greater overall absorbances and higher lines. All peaks were centered at 265 nm. The elevation of the various peaks above where an alachlor-only solution would absorb (around 0.3 absorbance units) is due to the contribution of calcium hypochlorite at this wavelength. The background from calcium hypochlorite was removed using the calibration curves developed for either pH 4 or 9.

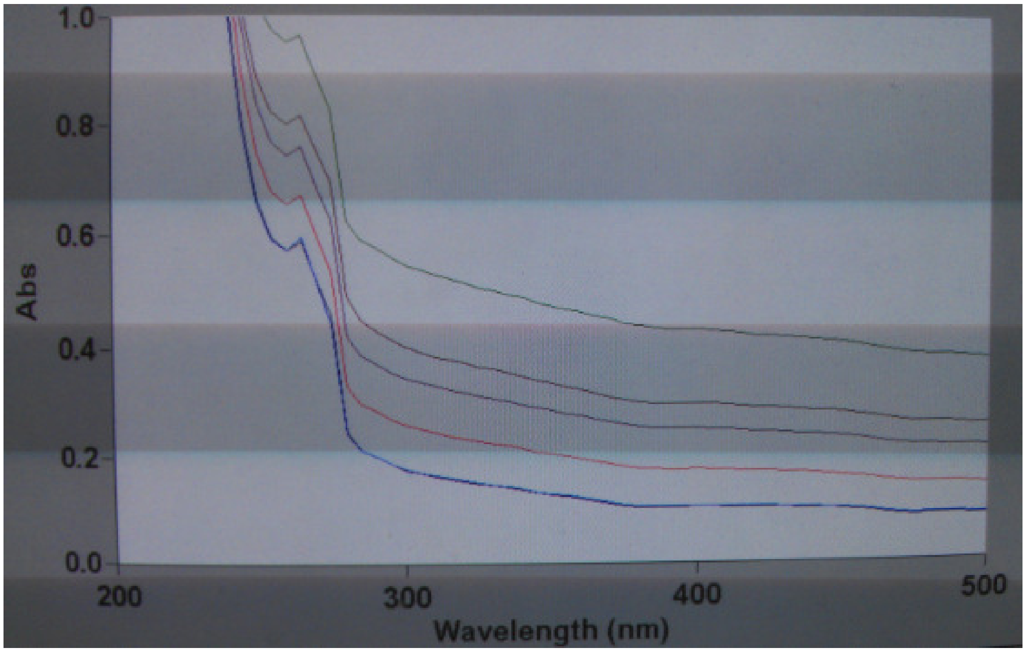

Figure 24: Scans of Calcium Hypochlorite Treated Alachlor Solutions (pH 9)

Contact times beyond 24 hours showed no further decreases in alachlor concentration. At pH 4, the maximum removal at the highest molar ratio, 25:1, was a maximum of 12.5%.

Atrazine

Calcium hypochlorite was similarly more effective at removing atrazine at high molar ratios and an elevated pH of 9. Table 18 reports the concentration reduction percentages for molar ratios from 1:1 up to 25:1 at a pH of 9. The maximum removal at pH 4 was 3%. Scans of the treated solutions after 24 hours showed no new peak locations which would indicate byproducts.

Table 18: Atrazine Concentration Reduction with Calcium Hypochlorite Treatment (pH 9)

Molar Ratio of OCl^-(ion):Alachlor	% Concentration Removal (Assuming No OCl^- Reacted)
1	3
5	9
10	12
15	14
25	11

Chlorination Summary

Chlorine removal was expected to reach a maximum removal at some molar ratio of the hypochlorite ion to the pesticide, and then remain near a constant removal percentage. This maximum removal was observed for atrazine at a molar ratio of 15:1, at which 14% removal was achieved. The slight decrease to 11% removal at a 25:1 molar ratio is attributed uncertainties in measurement techniques. Alachlor removal would be also expected to reach a maximum removal, perhaps at molar ratio greater than 25:1, beyond which the removal would not significantly increase or decrease. These percentages are based on an assumption of 0 moles of hypochlorite being consumed in the reaction and therefore the background from all added calcium hypochlorite being removed from the absorbance measured after treatment. Analysis using this assumption is inaccurate at low molar ratios but becomes less inaccurate at higher molar ratios. Therefore, this assumption of no hypochlorite reacted may be reliable for determination of the maximum removals seen at high molar ratios.

Removal at pH 4 was much lower: 13% of alachlor and 3% of atrazine at the highest molar ratios used (25:1). Since the pKa of hypochlorous acid is 7.6, the pH 4 samples featured the hypochlorous acid as the dominant form of chlorine. The hypochlorite ion, dominant at pH levels greater than 7.6, was present in greater amounts at pH 9 and is concluded to be a stronger oxidant than hypochlorous acid, contributing to greater removal.

Ferrate

The use of potassium ferrate to oxidize glyphosate yielded considerable degradation over the course of a 24 hour contact time. This was determined qualitatively, based upon both the color of the solution formed after the contact time and the amount of precipitate formed. However, soluble ferric byproducts remained in solution despite attempts to filter or otherwise remove these solids. Therefore, due to the high absorbance of these byproducts, quantitative measurements could not be appropriately obtained from these samples to report the quantitative glyphosate removal. Multiple visual cues were instead used to analyze the contaminant oxidation abilities of ferrate.

Firstly, the orange color that solutions developed over time was taken as an indication that reduction of the iron was occurring. Potassium ferrate added to water initially had a purple color. At this point, iron was in its +VI oxidation state. As reactions proceeded, the iron oxidized contaminants and was itself reduced to its +III "ferric" oxidation state. This has a characteristic orange color. This therefore indicated that the iron was reacting. The orange color of solutions can be seen in Figure 25, where samples' molar ratios of ferrate to glyphosate increased from 1:1 on the left-hand side up to 16:1 on the right-hand side.

The depth of the orange color was further indicative of the reaction extent. Excess, unreacted iron in solution is unstable and is known to be reduced to the more stable ferric form, which has a distinct orange color. Therefore, the depth of the orange color in solution was used to infer the extent of reaction—more of an orange color indicated a lower reaction extent since more iron remained unreacted and was converted to ferric iron. Molar ratios leading to greater extents of reaction did not produce as much ferric iron and had less of an orange color. Based on this color analysis, less-orange solutions, or solutions with higher molar ratios, showed more complete reactions with glyphosate.

Solutions in which the reactions proceeded further produced more solid byproducts, which can also be observed in Figure 25 where vials on the right-hand side (greater molar ratios, greater extents) have greater solid content at the bottom. This corresponds to the hypothesis that the extent of the reaction would be expected to increase with molar ratio since higher overall amounts of ferrate in solution provided greater concentrations for the concentration-dependent reaction to occur.

Figure 25: Glyphosate Solutions after Ferrate Treatment, pH 3

These solutions could not be scanned in the UV spectrophotometer since soluble byproducts remained in all solutions and contributed to absorbance values. In attempts to reduce this interference from the byproducts, the experiment was repeated under acidic conditions to further the reaction at all molar ratios. After a 24-hour reaction period, the solution's pH was then raised to basic conditions (pH 8) to minimize the solubility of the ferric compounds. The absorbance's of the solutions were still above the base absorbance of the glyphosate solution after being vacuum filtered and centrifuged at this elevated pH. While the solutions visually appeared quite clear, as seen in Figure 26, it was concluded from the elevated absorbance values was still a significant quantity of ferric component dissolved in the solution that could not be removed sufficiently enough to obtain valid, quantitative data to describe the glyphosate concentration reduction.

Figure 26: Glyphosate Solutions after Ferrate Treatment and Solid Removal, pH 8

Although no valid quantitative data could be obtained from ferrate experiments, it was determined that over the course of a 24 hour exposure time, potassium ferrate did eliminate a considerable percentage of contaminant in solution. A considerable degree of oxidation could be anticipated based on the oxidative potential of ferrate, 2.20. The solubility of byproducts must be evaluated and minimized more thoroughly, if possible, to quantify final concentrations of an isolated pesticide compound.

Ozonation

Alachlor

Ozone treatment of alachlor over time periods from 5 minutes up to 90 minutes did not successfully reduce alachlor and alachlor byproduct concentrations. Instead, the absorbance at 265 nm rose steadily until experiments were stopped at 90 minutes. The dose of ozone was not measurable but may be taken as the maximum amount suppliable by the ozonator used.

As was observed with UV treatment, the peak at 265nm which indicates presence of alachlor decreased rapidly while the total absorbance at 265 nm increased rapidly, and continued to increase with continued exposure. Byproducts which did not show a peak on a scan of wavelengths 500-200 nm are assumed to have contributed to the increase in absorbance. The increase in absorbance with ozone contact time is shown in Figure 27. Exposure was limited to 90 minutes since greater contact time would be impractical for treatment facilities and uneconomical. After 90 minutes, the absorbance had increased by 49%. Again, for comparison, the untreated starting solution of alachlor (shown at 0 minutes of contact time) began with an absorbance value of approximately 0.3.

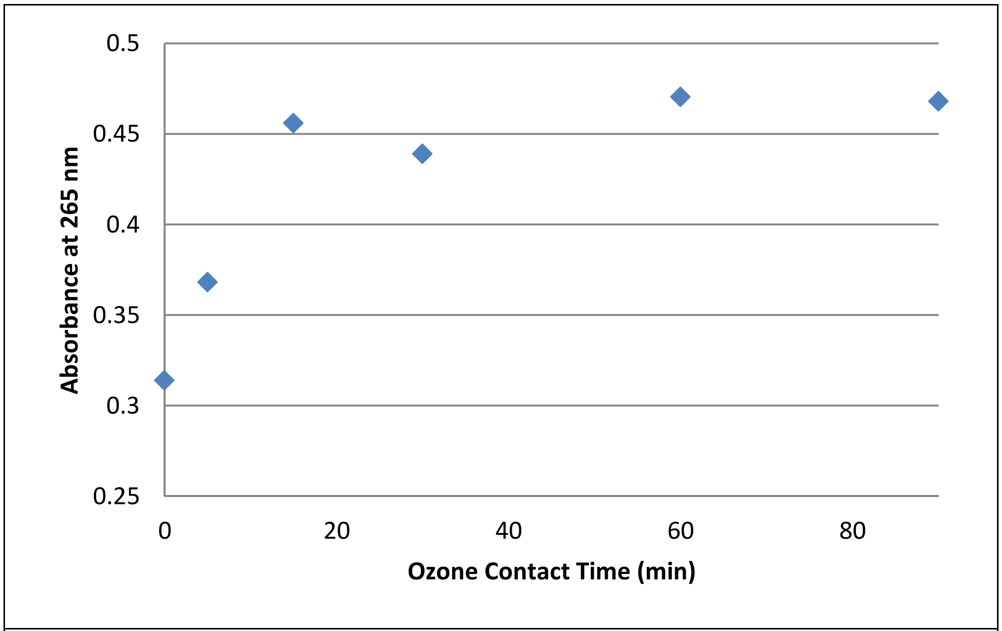

Figure 27: Increase in Alachlor Absorbance at 265 nm with Ozone Treatment

Atrazine

Ozonation did not remove a significant amount of atrazine from water over a 60-minute contact time. Figure 28 shows the reduction in concentration over the ozone exposure time. After 30 minutes of exposure, the removal rate reached a maximum of 17%. This same 17% removal was seen at 60 minutes indicating that no further removal could be achieved. While the ozonation process did remove some atrazine, this would not be a viable option for a treatment facility attempting to remove much more significant percentages from water.

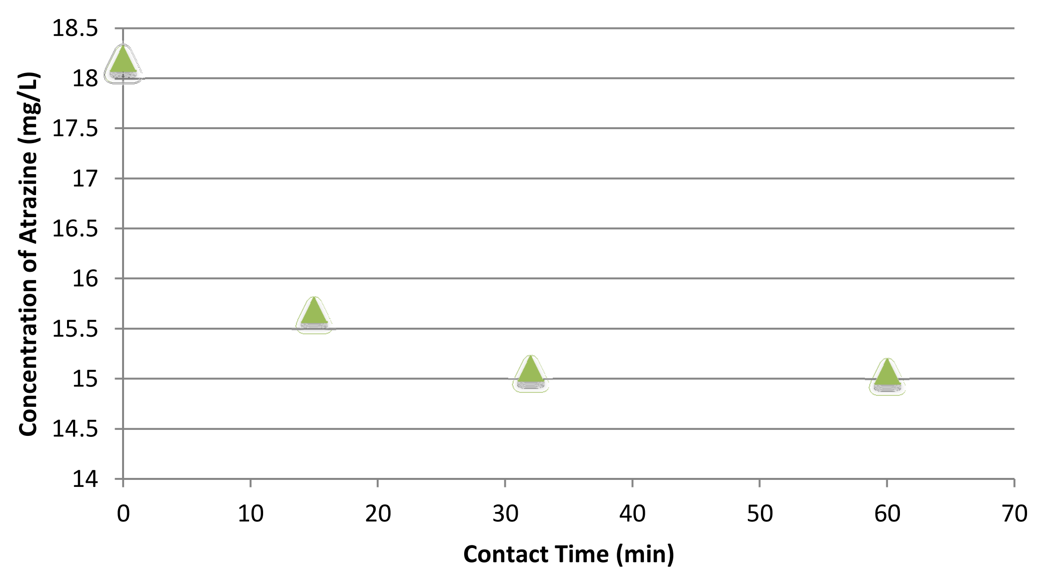

Figure 28: Reduction in Atrazine Concentration with Ozone Treatment

Rate Law Analysis

Atrazine

Analysis of atrazine experimental data did not allow determination of a rate law. Neither zero, first, nor second order rates appropriately modeled this data. When data was manipulated to attempt to determine a rate constant by identifying the most linear plot when axes were adjusted using the inverses or logarithms, no plot was very strongly linear. The data most nearly fit a second order degradation with a rate constant of 0.0002 when the R^2 value for rate determination was 0.65. However, this is not a strong correlation and the second order degradation is only one possibility. A strong possibility is that removal methods used to remove excess ozone from solution could have resulted in skewed data.

Ozonation Summary

Atrazine concentrations were reduced by 17% after 30 minutes of ozone oxidation. This removal was not as significant as other treatment methods, which could be expected since the oxidative potential of ozone is 2.07 whereas the oxidative potential of the hydroxyl radical produced in UV photolysis is 2.80. The byproduct formation and persistence seen during alachlor tests with ozone indicated the need for stronger oxidation.

UV Photolysis

Alachlor

Without Hydrogen Peroxide

Direct UV photolysis of an alachlor solution with a Pen-Ray 5.5 watt low-pressure lamp rapidly degraded the alachlor and formed significant concentrations of byproducts. A scan of the wavelengths 200-500 nm showed no identifiable peak at alachlor's characteristic 265 nm after 5 minutes. This lack of a defined peak at 265 nm indicated the reduction or complete degradation of alachlor. Figure 29 shows a scan after 5 minutes, where no peaks can be identified. The red line in this figure is an untreated solution of alachlor, and the black line is a solution treated for 5 minutes. However, other products were suspected to be present since the total absorbance did not drop to 0 nm.

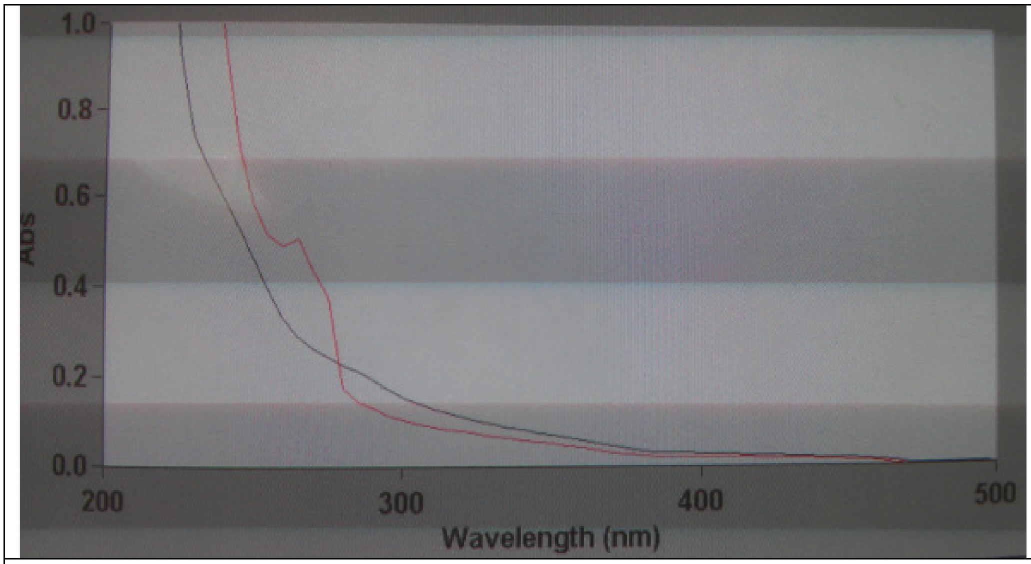

Figure 29: Scan of Alachlor Solution Treated for 5 Minutes with UV light, 500-200 nm

The total absorbance at 265 nm initially increased with time, reached a maximum absorbance at 90 minutes of UV exposure, and then began to decreased. The component which absorbed at 265 nm could not be identified as either alachlor or a byproduct using only the spectrophotometer. It was concluded that a large portion of the alachlor was degraded since no peak was seen on a 200-500 nm scan (Figure 29) but some amount of alachlor may have remained. No other clear peak was visible on the scan which would have been specific to the byproduct(s) and could have helped identify their identities. However, byproducts are believed to be formed, with the assumption that they may have absorbed UV light over a range of wavelengths, lending themselves to no peak at a specific wavelength.

At 90 minutes, the maximum amount of these byproducts had been formed. The absorbance at 265 nm reached 1.7, corresponding to a 5.4x increase from the solution's starting absorbance. Scans of solutions with longer contact times past 90 minutes showed subsequent decreases in the absorbance at 265 nm, but still no formation of a second peak. Byproducts could not be identified but were observed to decrease in concentration with time. Experiments were not extended to attempt to determine the maximum reduction in absorbance beyond 2.5 hours, when the absorbance was still above 1.19 (4x the initial absorbance). Figure 30 shows the increase, maximum, and decrease in the absorbance at 265 nm with continued UV photolysis. This is presented in units of absorbance rather than concentration since unidentifiable byproducts may have potentially contributed to this absorbance at 265 nm. UV photolysis alone was unable to reduce alachlor/alachlor byproduct concentrations significantly in less than 90 minutes.

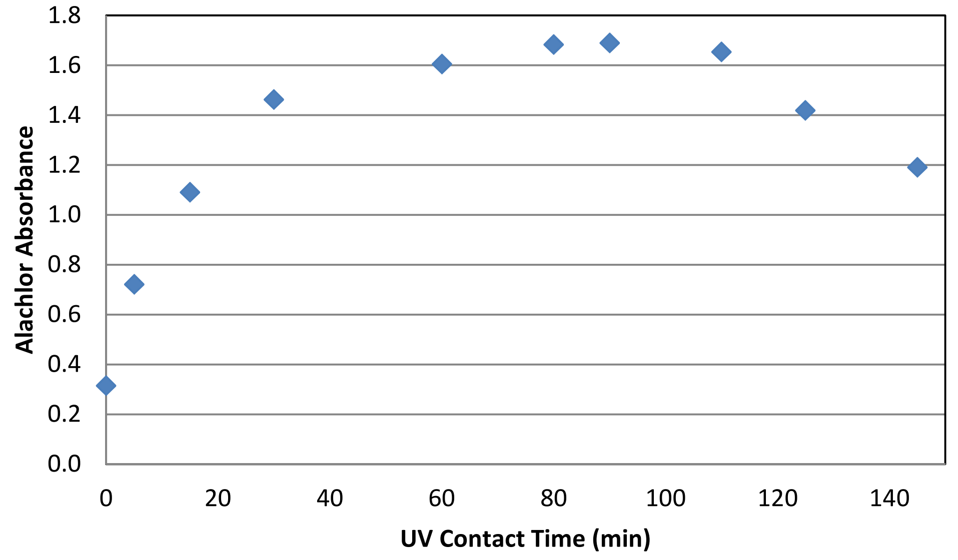

Figure 30: Increase in Alachlor Absorbance with UV Photolysis

With Hydrogen Peroxide

Advanced UV oxidation of alachlor with the addition of hydrogen peroxide showed more rapid and more thorough reduction of the concentration of alachlor and/or alachlor byproducts. Of the 12.5:1, 25:1, 50:1, and 100:1 molar ratios of hydrogen peroxide to alachlor which were employed, the 25:1 molar ratio was most successful at reducing the absorbance at 265 nm after 45 minutes, shown in Table 19. This absorbance was still higher than the starting absorbance of alachlor alone, but longer residence times were expected to reduce the absorbance further. Therefore, this 25:1 molar ratio was used over a longer time period to test the maximum alachlor removal over time. In Table 19, absorbance is again used to report removal since byproducts were again concluded to be present.

Table 19: Evaluation of Various Molar Ratios of Hydrogen Peroxide : Alachlor for Use in UV Photolysis	
Molar Ratio	**Absorbance at 265nm, 45 min**
12.5	0.735
25	0.067
50	0.069
100	0.136

Scans over wavelengths of 500-200 nm for all UV and hydrogen peroxide-treated samples saw a lack of a defined peak at 265 nm that would have indicated a high concentration of alachlor remaining. As was seen without hydrogen peroxide use, no new peak was present that would indicate specific byproducts.

The 25:1 molar ratio of hydrogen peroxide to alachlor removed 88% of the species absorbing at 265 nm after 60 minutes. After 90 minutes, this removal increased to a maximum of 95%. Table 20 reports the percentages of absorbance reduction with increased exposure. Further exposure (up to 105) minutes did not significant increase removal.

Table 20: Reduction in Alachlor Absorbance with UV + H_2O_2 (25:1 Molar Ratio)	
Exposure Time (min)	**Reduction in Absorbance at 265 nm (%)**
5	-312
15	-100
30	49
45	76
60	88
75	93
90	95
105	95

Figure 31 shows the same removal with time as Table 20. Though the identity and number of byproduct(s) could not be determined, analysis using absorbance remains valid through use of Beer's Law linking absorbance to concentration. The calibration curve to quantitatively link absorbance to concentration remains unknown. For comparison, the starting absorbance of the untreated alachlor solution was approximately 0.3 (shown at time zero).

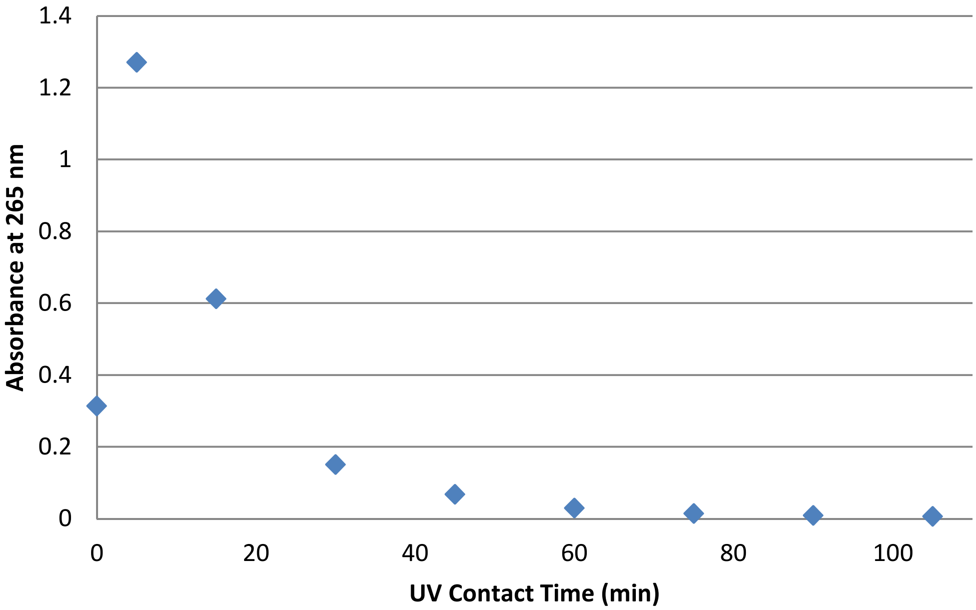

Figure 31: Degradation of Alachlor with UV + H_2O_2 (25:1 Molar Ratio)

Kinetics Analysis

Rate analysis was performed to observe the degradation kinetics for UV treatment using hydrogen peroxide. Since no appreciable degradation was seen without hydrogen peroxide addition, no kinetic analysis was appropriate without hydrogen peroxide addition. For rate law determination, concentration units were used although byproducts were certainly observed. The concentration was calculated as if all absorbance was due to the alachlor base compound though this was known to be inaccurate since no increase in alachlor concentration was possible. Because of this spike in concentration that was attributed to byproduct formation, the rate law for the overall degradation that was observed could be developed in two ways: the rate pertaining to the entire process, including the rapid concentration increase, and that of the rate just concerning the degradation that followed the rapid concentration increase.

Manipulation of the axes to determine the rate order led to a first order rate determination. When the initial spike in concentration was included, the rate constant in this case was determined to be 0.0412 min^{-1}. Without this initial spike in concentration, the rate constant in this case was determined to be 0.0457 min^{-1}. The rates would not vary too greatly by using either analysis method, but here the rate constant was selected so as to include the initial spike in concentration since it seemed to more accurately describe the behavior observed. The rate law was then:

$$-R_{alachlor} = 0.0457 * C_{alachlor} \qquad \text{(Reaction 19)}$$

Figure 32 shows the degradation of alachlor over time (in concentration units) along with the degradation predicted by the rate law that was developed.

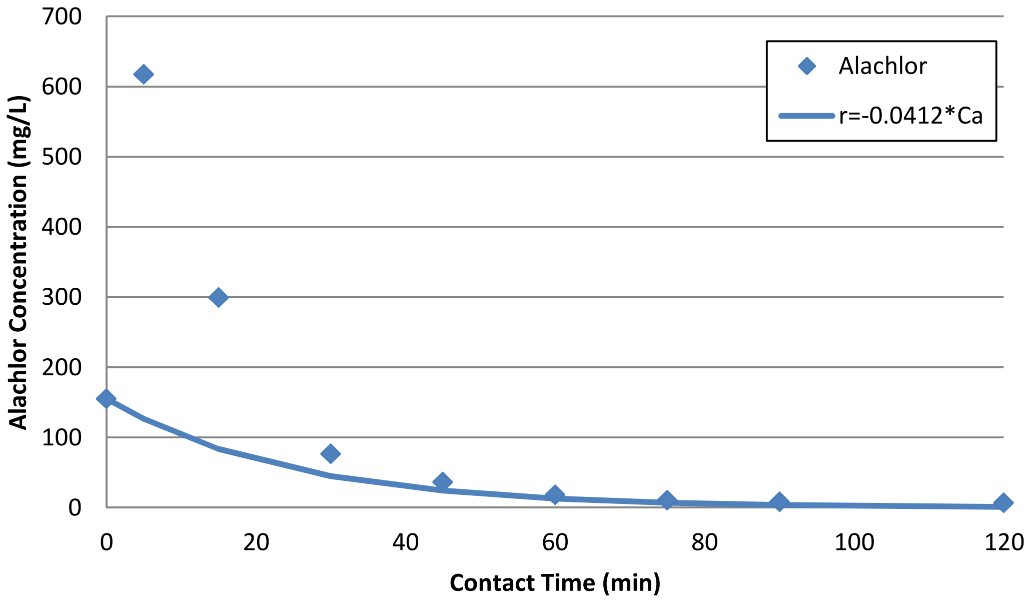

Figure 32: First Order Degradation of Alachlor with UV + H_2O_2 (25:1 Molar Ratio)

Atrazine

Without Hydrogen Peroxide

The low-pressure UV lamp was used to treat atrazine solutions for 90 minutes. The overall removal achieved was 87% in 75 minutes. Removal at 90 minutes was not significantly increased. Figure 33 shows the degradation of atrazine over time.

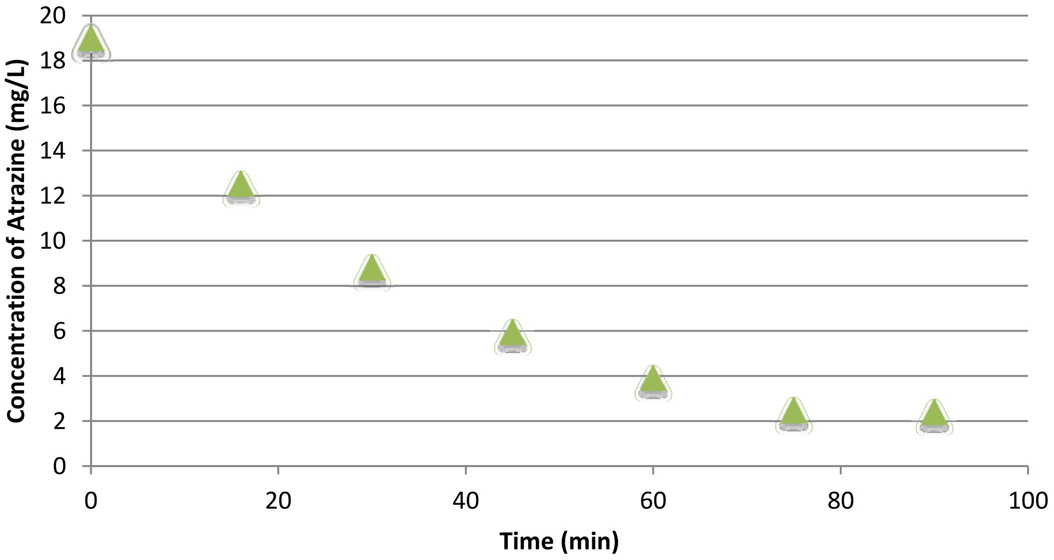

Figure 33: Degradation of Atrazine with UV Photolysis

With Hydrogen Peroxide

Hydrogen peroxide was then added to the atrazine solution at a molar ratio of 100:1. Again, the low solubility of atrazine led to this high ratio since lower ratios would have resulted in nearly negligible amounts of hydrogen peroxide added. A 63% increase in concentration was observed during the first 15 minutes of exposure. The solution was hypothesized to have degraded into atrazine's byproducts due to the hydrogen peroxide addition. Figure 34 shows the atrazine degradation with time. After 120 minutes the removal reached a maximum of 92%.

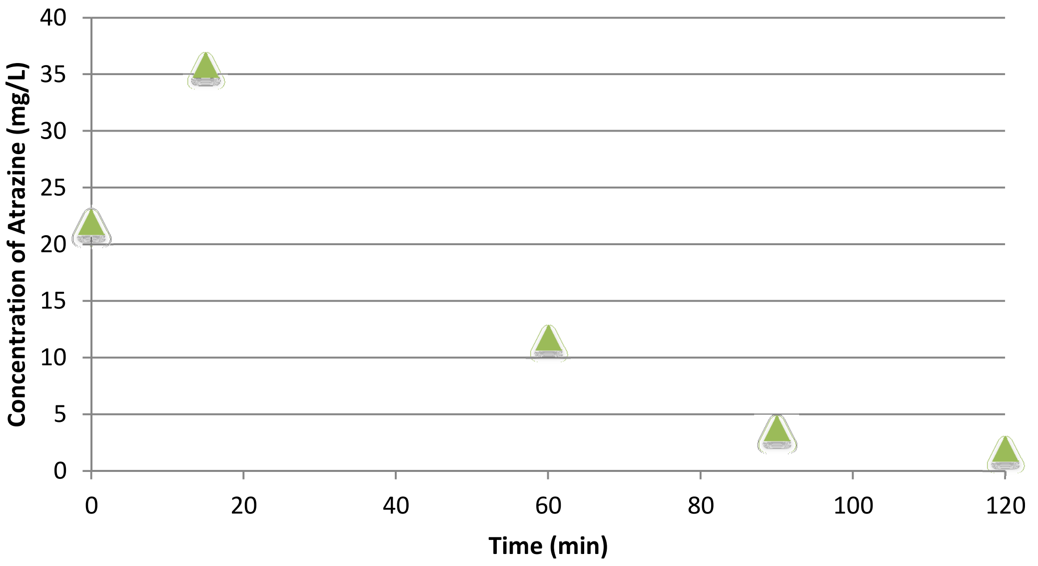

Figure 34: Degradation of Atrazine with UV + H_2O_2 (100:1 Molar Ratio)

Kinetics Analysis

It was determined through graphical analysis that the degradation of atrazine with UV photolysis first order reaction with a rate constant of 0.0245 min^{-1}. The rate law, shown in Figure 35 with experimental data, was then:

$$-R_{atrazine} = 0.0245 * C_{atrazine} \qquad \text{(Reaction 20)}$$

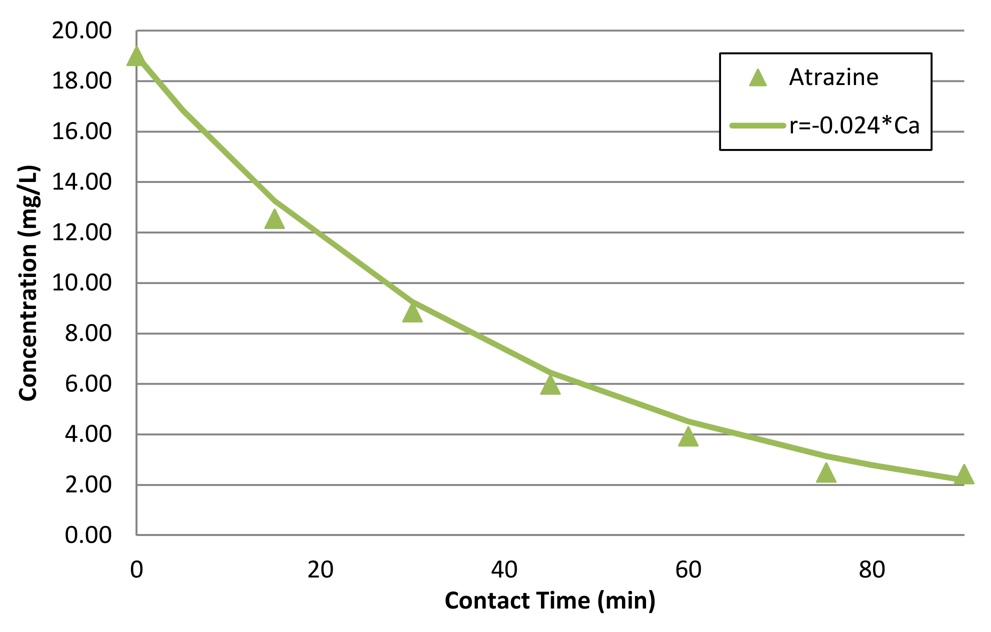

Figure 35: First Order Degradation of Atrazine with UV Photolysis

A first order rate law was also developed for UV photolysis when hydrogen peroxide was used. This analysis included the initial increase in concentration when byproducts were formed. The rate law for this, shown in Figure 36 with experimental data, was:

$$-R_{atrazine} = 0.0232 * C_{atrazine} \qquad \text{(Reaction 21)}$$

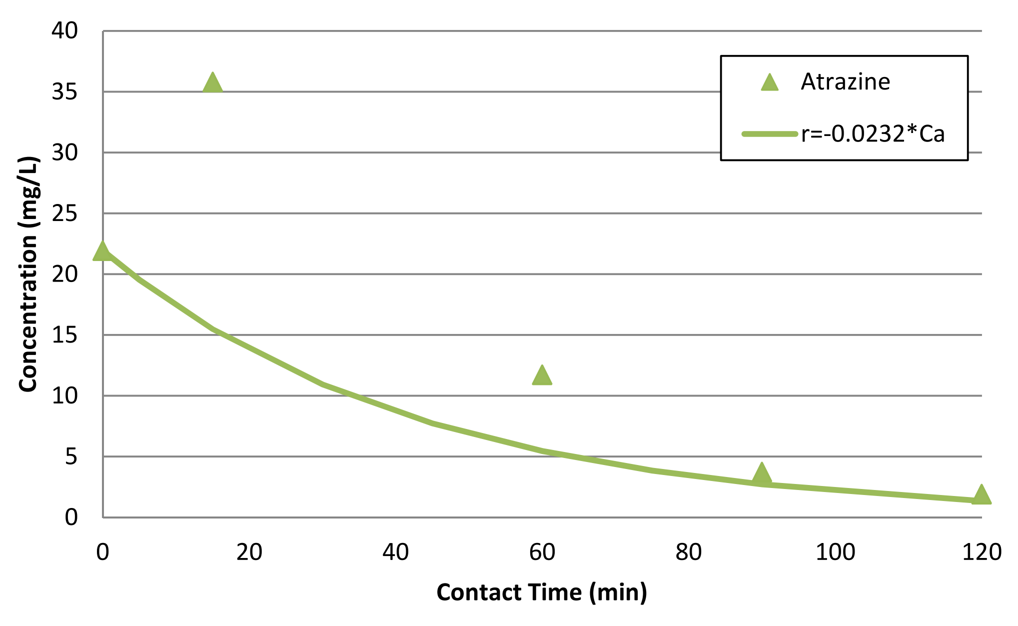

Figure 36: First Order Degradation of Atrazine with UV + H_20_2 (100:1 Molar Ratio)

When the rate constant for degradation with UV with hydrogen peroxide is compared to that of without hydrogen peroxide, it seems that more rapid degradation was achieved when no hydrogen peroxide was added. The rate constant is greater when no hydrogen peroxide was added. This is because the spike in concentration when byproducts were formed was included in the analysis when hydrogen peroxide was added. No spike was seen without hydrogen peroxide, but perhaps at shorter time intervals a similar spike would have been observed. Or, byproducts may have followed a different degradation pathway involving byproducts that did not absorb as much UV light (as measured in the UV spectrophotometer) when a greater concentration of hydroxyl radicals were present. The overall removal when contact times reached 90 minutes became very similar.

Glyphosate

Without Hydrogen Peroxide

Direct UV photolysis of a glyphosate solution under a low-pressure lamp showed significant reactions occurring over a relatively short period of time. However, due to the significant amount of byproducts produced by the degradation of glyphosate, absorbance was seen to rise steadily with time. While the absorbance of alachlor and atrazine ultimately decreased over the course of the UV exposure, the byproducts formed by glyphosate were slow to degrade with the UV exposure. The absorbance level ultimately leveled off after two hours, at which point it could be concluded that the glyphosate had ceased reacting, but did not decrease after this time period. When scanned from 200-500 nm, no visible peaks were observed at 255 nm, which is glyphosate's absorption wavelength. This observation led to the conclusion that after two hours of UV exposure, most of the measurable glyphosate had been reacted out of solution. No other peaks were visible on the scan to denote the byproducts' identities. However, the overall increase in absorbance at 255 nm does lend itself to byproducts, as there were no other components within the solution that could react with the free radicals produced by UV photolysis. Byproducts that formed may not have been measurable by the spectrophotometer.

At the 120 minute point, the absorbance of the solution had reached 0.085, almost seven times greater than its initial absorbance. Figure 37 shows the increase of absorption at 255 nm with respect to UV contact time. The graph is shown in terms of absorbance rather than concentration as the presence of byproducts makes the conversion between absorbance and concentration impossible.

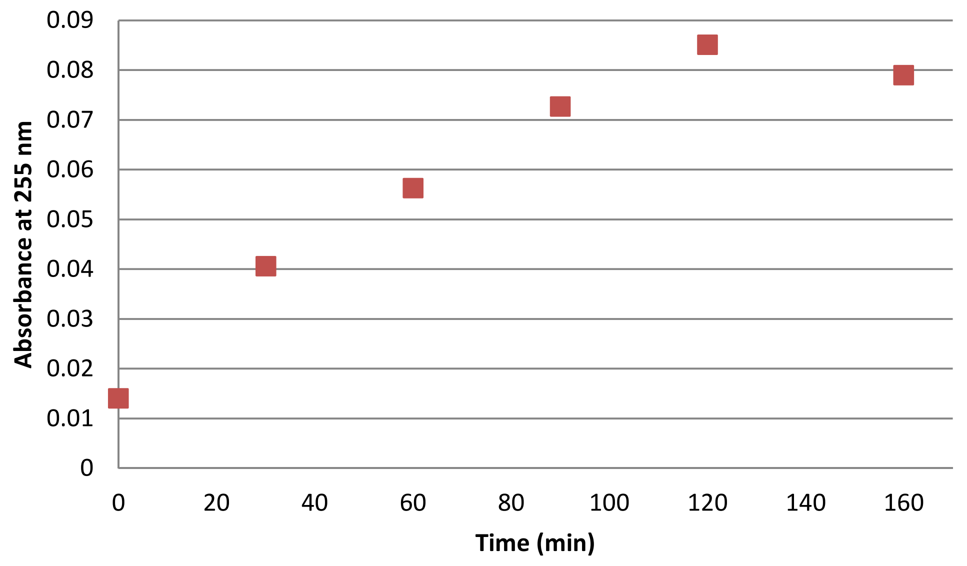

Figure 37: Increase in Glyphosate Absorbance with UV Photolysis

With Hydrogen Peroxide

UV oxidation of glyphosate with the addition of hydrogen peroxide showed considerably better removal of glyphosate and elimination of byproduct as compared to UV oxidation without the hydrogen peroxide. Because alachlor had shown to react best at a 25:1 molar ratio of H_2O_2 to contaminant, this ratio was used for glyphosate as well.

The 25:1 ratio of hydrogen peroxide to glyphosate showed a large increase in absorbance, peaking at 0.193 (starting at 0.023 for untreated glyphosate) after 15 minutes. The absorbance reading gradually decreased from that point until it reached an absorbance of 0.11 after 90 minutes, corresponding to a 52% overall decrease in absorbance. Greater residence times than 90 minutes saw no appreciable changes in absorbance, indicating the maximum removal was at 90 minutes. Table 21 below shows the percent reduction of absorbance with respect to exposure time.

Table 21: Reduction in Glyphosate Absorbance with UV + H_2O_2 (25:1 Molar Ratio)

Exposure Time (min)	Reduction in Absorbance at 265 nm (%)
15	-739
30	-565
45	-404
60	-191
75	-96
90	52

Figure 38 shows the concentration of glyphosate versus time. In accordance with Beer's Law, absorbance can be plotted to represent the concentration.

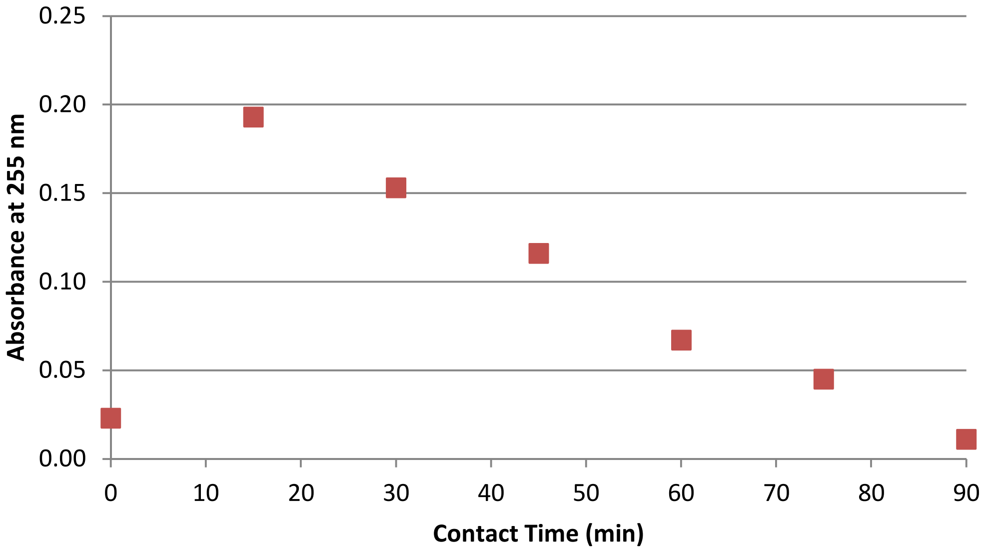

Figure 38: Degradation of Glyphosate with UV + H_2O_2 (25:1 Molar Ratio)

When UV+H_2O_2 treated solutions were scanned over wavelengths 500-200 nm, no definite peak was seen at 255 nm. This would suggest considerable removal of glyphosate from the contaminated water.

As seen previously for UV without hydrogen peroxide, there was no new peak indicating the identity of the byproducts.

Kinetics Analysis

Out of the three chemicals, glyphosate showed the lowest removal percentage when treated with UV radiation. Regardless, rate law analysis was performed and it was determined that the rate governing this degradation was nearer to a zero order reaction than a first order like the other two pesticides' degradation. The rate constant was 132.63 mg L^{-1} min^{-1}. Figure 39 shows the degradation with this zero order reaction, as follows:

$$-R_{glyphosate} = 132.63 \qquad \text{(Reaction 22)}$$

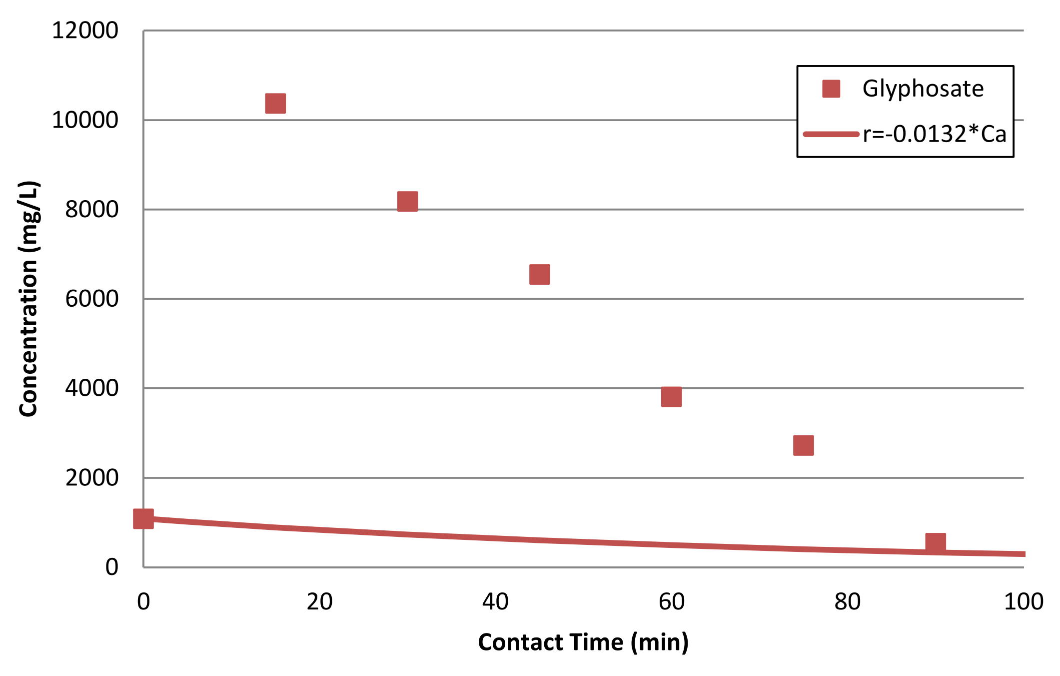

Figure 39: Zero Order Degradation of Glyphosate with UV + H_2O_2 (25:1 Molar Ratio)

UV Photolysis Summary

The degradation of atrazine under UV photolysis followed first order kinetics and was reduced by 87% in 90 minutes. The other two pesticides, however, formed significant amounts of persistent byproducts, shown in Figure 40.

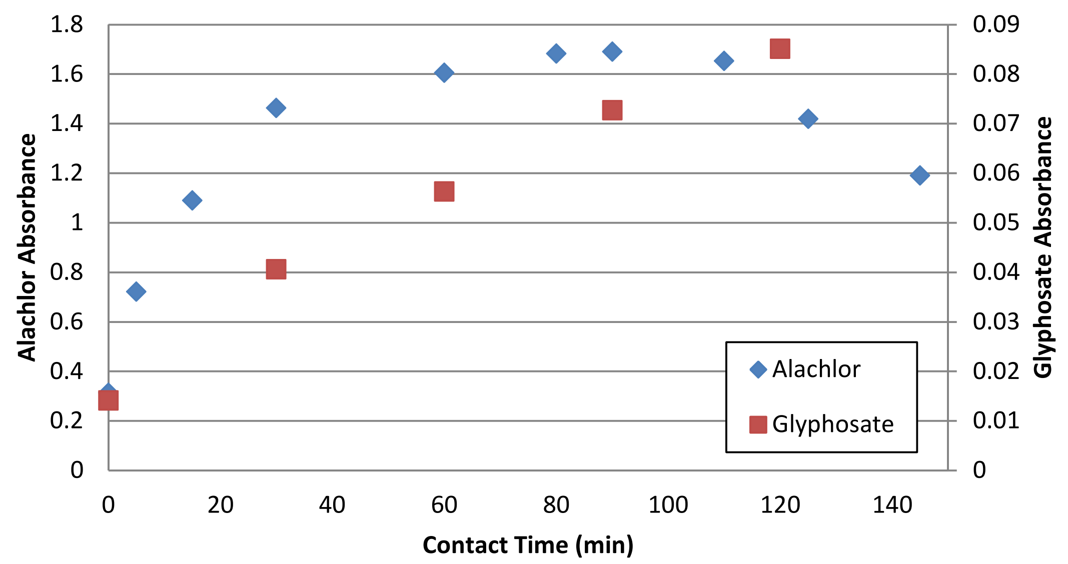

Figure 40: Alachlor and Glyphosate Byproduct Formation with UV photolysis

Stronger oxidation with hydrogen peroxide removed 95% of alachlor, 83% of atrazine, and 50% of glyphosate in 90 minutes. The most effective molar ratios of hydrogen peroxide to pesticide were used. The removal of atrazine slightly decreased with this addition of hydrogen peroxide, but this difference is concluded to be due to errors associated with measurement of concentrations since atrazine concentrations started lower than the other two pesticides. The removal of the other two pesticides increased with the addition of hydrogen peroxide. This is because the hydroxyl radical, the oxidant that is generated from water with UV exposure, is generated in greater concentrations when hydrogen peroxide is added.

Removal of alachlor and atrazine with hydrogen peroxide followed first order kinetics. The experimental data points as well as the curves showing degradation according to the generated rate laws are shown in Figure 41.

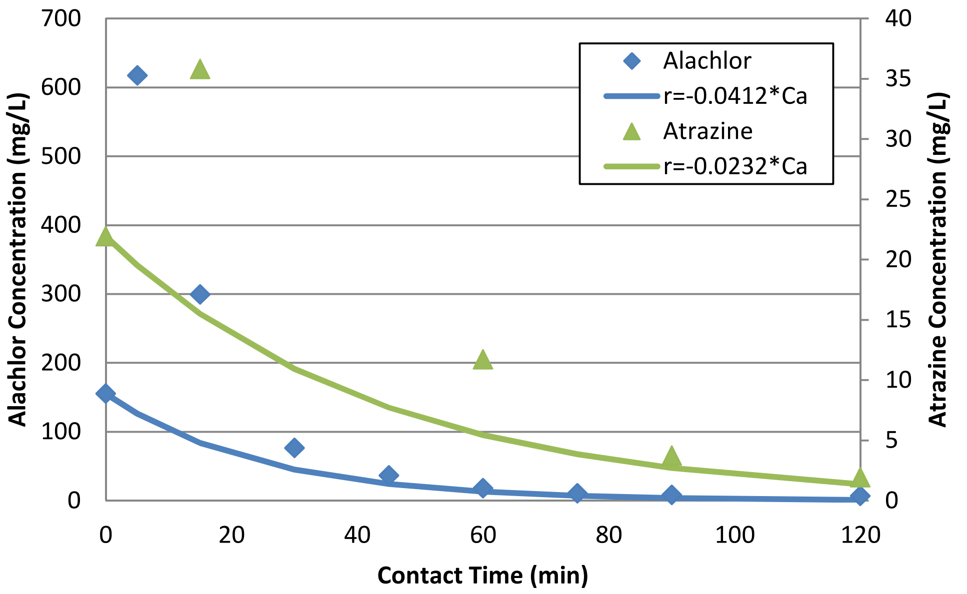

Figure 41: First Order Degradation of Alachlor and Atrazine with UV + H_2O_2

The spike in concentrations between 0 and 20 minutes is due to the rapid formation of byproducts. This spike was included in the development of the rate laws, but the formation of byproducts not modeled by first order degradation. The presence of byproducts is not included at all since the rate is first order with respect to the base pesticide concentration, not the concentration of byproducts. However, since byproducts are then reduced very significantly over the 120-minute period that experiments were conducted for, rate laws can still have practical applications when developed in this way. When initial and final concentrations are the relevant values, first order degradation modeling may still provide useful data.

The degradation of glyphosate seemed to follow zero order kinetics rather than first order. This may be due to the similar structure of glyphosate byproducts to the base glyphosate structure. The reaction may become competitive for hydroxyl radicals once byproducts are present as well. The rate would therefore slow, as observed, and not follow the same overall degradation observed for the other two pesticides in the same time period.

Error Analysis

For much of the analysis performed throughout this research, uncertainty in measurements was a significant consideration. This applied significantly to the use of the UV spectrophotometer, as it was constantly in use within the lab and its zero base line was recalibrated for use with various samples a single day. An assumption of an uncertainty error of ±0.0005, or even ±0.001 absorbance units, measured by the spectrophotometer was made. In order to determine the effect of such an error within further data analysis, the change in concentration resulting from this uncertainty in the UV spectrophotometer was calculated for the three stock solutions.

Atrazine exhibited the smallest amount of error from the UV spectrophotometer, with an uncertainty of 0.0005 and 0.001 resulting in a 0.2 mg/L and 0.23 mg/L change, respectively, in measured concentration respectively. These numbers increased for alachlor to 2.4% and 2.65 mg/L, compared to its initial concentration of 160 mg/L. Glyphosate showed the most noticeable changes, showing a 35 mg/L and 60 mg/L uncertainty with respect to its initial concentration of 600 mg/L.

An additional source of error that was considered during experimentation was the measurement of solids for various mass and molar ratios used throughout research. For solid compounds such as activated carbon and potassium ferrate, there was uncertainty in the readouts of the scales used to mass samples to the thousands decimal. Additionally, the varying size of particles of activated carbon made maintaining precise ratios difficult since the addition of a single granule of activated carbon in some cases pushed the mass ratio slightly beyond what was desired. However, the effect of such an error would be considered to be minimal.

Liquid compounds such as hydrogen peroxide and calcium hypochlorite solution also added sources of error throughout experimentation. The use of micropipettes in conjunction with the use of minute measurements of these liquids meant that any deviation from the desired volume could be significant. For example, a measurement of 10 microliters off by 1 microliter led to a 10% error. In this case, the source of the error would come from the precision and accuracy of a given micropipette or graduated cylinder, as well as human error associated with pipetting and transferring fluids.

The contact time used for time-based treatments (UV photolysis and ozonation) may have deviated slightly from the reported times. This deviation, likely a maximum of 2-3 minutes, would not have led to a very significant alteration in the degradation behavior since most degradation was significant over 30-90 minute time periods. Comparatively, the reaction occurring in a 2-3 minute time period would not have a significant impact. For activated carbon, chlorine, and ferrate samples run for 24 hours, equilibrium concentrations were reached (verified by measurements after 48 hours) so no error associated with prolonged exposure were expected.

Since applied ozone concentrations could not be measured with the equipment and meters in the laboratory, a great deal of uncertainty is associated with ozone treatment. However, experiments were still conducted with the acceptance that quantitative values could not be obtained.

Byproduct formation also contributed to errors in our analysis. Since identities of byproducts could not be determined using the UV spectrophotometer, concentrations of byproducts could not be determined. No calibration curve correlating the absorbance to concentration could be produced. Furthermore, the distinction between base pesticides and byproducts could not always be made since in all cases, no secondary peaks were observed when samples were scanned from 500-200 nm. Byproducts and base compounds were therefore analyzed together. When determining rate laws, concentration units were used, with the acknowledgement that this concentration would be in terms of the base pesticide concentrations—though byproducts were in fact present. This analysis is still valuable for determination of contact time required to reduce concentrations to a significant level.

Conclusions and Recommendations

The most effective treatment methods in terms of maximum possible removal were granular activated carbon and advanced UV oxidation with hydrogen peroxide. The superiority of these two treatment methods was observed for both alachlor and atrazine. Glyphosate was treated only with these two methods and its removal therefore cannot be compared among other treatment methods.

A 25:1 mass ratio of activated carbon to alachlor was most effective at reducing concentrations for a 24-hour contact time—a 98% reduction. An 18:1 mass ratio to atrazine effectively removed 92%. A 15:1 mass ratio to glyphosate effectively removed 51%.

A 25:1 molar ratio of hydrogen peroxide to alachlor was most effective at reducing the absorbance at 265 nm for a 90-minute residence time—a 95% reduction. First order kinetics were used to model this removal. A 100:1 molar ratio of hydrogen peroxide to atrazine reduced the concentration for a 90-minutes residence time by 83%. First order kinetics were again observed. A 25:1 molar ratio of hydrogen peroxide to glyphosate reduced the concentration for a 90-minute residence time by 50%. This removal did not follow first order and was instead concluded to be a competitive reaction for hydroxyl radicals between glyphosate and its byproducts.

Calcium hypochlorite at the highest molar ratio removed only 59% of alachlor and 14% of atrazine at an elevated pH. Significant amounts of byproducts were formed during UV, UV+H_2O_2, and ozone treatment. Only UV+H_2O_2 was successful at reducing concentrations of these byproducts in a reasonable time period (90 minutes). The identities of these byproducts could not be determined, and they did not show a defined peak at any wavelength but were observed to absorb UV light over a range of wavelengths. One recommendation made for future research includes the identification of these byproducts using a mass spectrophotometer and gas chromatograph. These byproducts can be monitored over time to observe degradation kinetics rather than grouping all degradation into first order kinetics with respect to the base pesticide concentration.

The oxidative potentials of hypochlorite and of ozone, respectively, are 1.48 and 2.07. When these potentials are compared to that of the hydroxyl radical, 2.80, the successful removal using UV photolysis with added hydrogen peroxide was to be expected. The hydroxyl radical is the strongest oxidant and was therefore the most successful at oxidizing pesticides.

Since the oxidative potential of ferrate is 2.20, the second highest of treatments used here, a method to determine the quantitative pesticide concentration reduction with ferrate is recommended. This could involve filtration through membranes with tighter pores or different surface chemistries. Another method might involve determination of all reactions occurring, baking of products to determine the amounts of solids, and analysis of the stoichiometry involved to isolate the amount of pesticide remaining in solution as compared to the other solids formed during the reaction. The removal may be determined in that matter.

Other recommendations include either increasing the concentration of ozone applied during ozonation or adding hydrogen peroxide. Both would increase the amount of oxidants in solution and therefore increase removal.

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Appendices

Appendix A: Organic Chemicals' Maximum Contaminant Levels (Complete)[12]

Contaminant	MCLG* (mg/L)	MCL or TT (mg/L)	Potential Health Effects from Long-Term Exposure Above the MCL (unless specified as short-term)	Sources of Contaminant in Drinking Water
Alachlor	zero	0.002	Eye, liver, kidney or spleen problems; anemia; increased risk of cancer	Runoff from herbicide used on row crops
Atrazine	0.003	0.003	Cardiovascular system or reproductive problems	Runoff from herbicide used on row crops
Chlordane	zero	0.002	Liver or nervous system problems; increased risk of cancer	Residue of banned termiticide
2,4-D	0.07	0.07	Kidney, liver, or adrenal gland problems	Runoff from herbicide used on row crops
Dalapon	0.2	0.2	Minor kidney changes	Runoff from herbicide used on rights of way
1,2-Dibromo-3-chloropropane (DBCP)	zero	0.0002	Reproductive difficulties; increased risk of cancer	Runoff/leaching from soil fumigant used on soybeans, cotton, pineapples, and orchards
Dinoseb	0.007	0.007	Reproductive difficulties	Runoff from herbicide used on soybeans and vegetables
Diquat	0.02	0.02	Cataracts	Runoff from herbicide use
Endothall	0.1	0.1	Stomach and intestinal problems	Runoff from herbicide use
Endrin	0.002	0.002	Liver problems	Residue of banned insecticide
Glyphosate	0.7	0.7	Kidney problems; reproductive difficulties	Runoff from herbicide use

Heptachlor	zero	0.0004	Liver damage; increased risk of cancer	Residue of banned termiticide
Heptachlor epoxide	zero	0.0002	Liver damage; increased risk of cancer	Breakdown of heptachlor
Lindane	0.0002	0.0002	Liver or kidney problems	Runoff/leaching from insecticide used on cattle, lumber, gardens
Methoxychlor	0.04	0.04	Reproductive difficulties	Runoff/leaching from insecticide used on fruits, vegetables, alfalfa, livestock
Oxamyl (Vydate)	0.2	0.2	Slight nervous system effects	Runoff/leaching from insecticide used on apples, potatoes, and tomatoes
Picloram	0.5	0.5	Liver problems	Herbicide runoff
Simazine	0.004	0.004	Problems with blood	Herbicide runoff
Toxaphene	zero	0.003	Kidney, liver, or thyroid problems; increased risk of cancer	Runoff/leaching from insecticide used on cotton and cattle
2,4,5-TP (Silvex)	0.05	0.05	Liver problems	Residue of banned herbicide

Appendix B: Price of Using Ozonation[80]

Ozone Economic Assumptions	
Time Frame	20 Years
Power Cost	0.07 $/kWh
Interest Rate	5%
Ozone Production Rate	450 lbs/day (with 100% redundancy)
Ozone Concentration	2%
Power Consumption	10.1 kWh/lb ozone
Capital Cost	$549,000
Ozone Concentration	10%
Power Consumption	4.5 kWh/lb ozone
LOX System Rental	$1,000/month (tank, evaporators, valves and controls)

Appendix C: Activated Carbon Adsorption Data

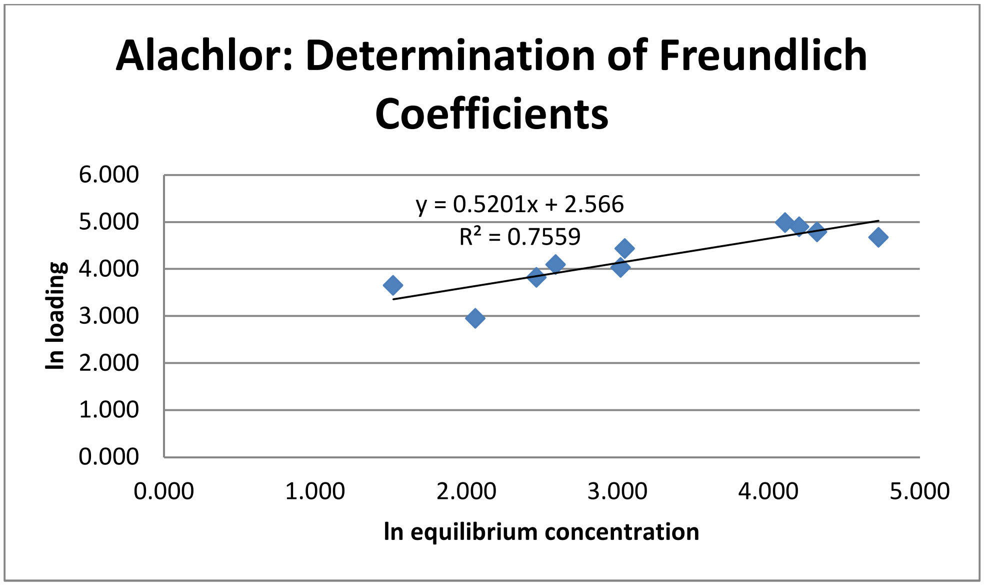
All activated carbon experiments run for 24 hours

Alachlor

Sample Volume (mL)	Mass of Pesticide (mg) in Sample	Mass of GAC (g) in Sample	Mass Ratio (Adsorbate: Adsorbant)	Absorbance	Equilibrium Concentration (mg/L)	**Removal Percentage (%)**
30	5.66	0.0028	0.5	0.3970	195.41	**-4**
30	5.66	0.0057	1.0	0.3405	168.12	**11**
30	5.66	0.0113	2.0	0.2610	129.72	**31**
30	5.66	0.0283	5.0	0.1629	82.33	**56**
30	5.66	0.0566	10.0	0.0041	5.62	**97**
25	4.72	0.0047	1.0	0.3820	188.17	**0**
30	4.00	0.0057	1.4	0.2265	113.05	**40**
20	2.67	0.0099	3.7	0.1309	66.87	**65**
20	2.67	0.0099	3.7	0.1186	60.93	**68**
30	5.66	0.0283	5.0	0.1484	75.32	**60**
20	2.73	0.0273	10.0	0.0361	21.08	**89**
20	2.73	0.0409	15.0	0.0201	13.35	**93**
20	2.67	0.0400	15.0	0.0350	20.55	**89**
20	2.73	0.0545	20.0	0.0168	11.76	**94**
20	2.73	0.0682	25.0	0.0019	4.56	**98**
15	2.00	0.0987	49.3	0.0087	7.84	**96**

Freundlich Isotherm for Alachlor

Loading (mg/g)	ln Loading	1/loading	Equilibrium Concentration (mg/L)	ln Equ. Conc	1/Equ Conc
134.28	4.900	0.007	66.87	4.203	0.015
146.28	4.986	0.007	60.93	4.110	0.016
120.14	4.789	0.008	75.32	4.322	0.013
84.54	4.437	0.012	21.08	3.048	0.047
60.14	4.097	0.017	13.35	2.591	0.075
56.39	4.032	0.018	20.55	3.023	0.049
45.69	3.822	0.022	11.76	2.464	0.085
38.66	3.655	0.026	4.56	1.517	0.219
19.07	2.948	0.052	7.84	2.060	0.128


Alachlor: Determination of Freundlich Coefficients

Slope is 0.5201 and y-intercept is 2.57. The natural log of the y-intercept is 13.01. The Freundlich isotherm for alachlor is therefore:

$$Loading_{alachlor} = Equilibrium\ Concentration^{0.5201} * 13.01$$

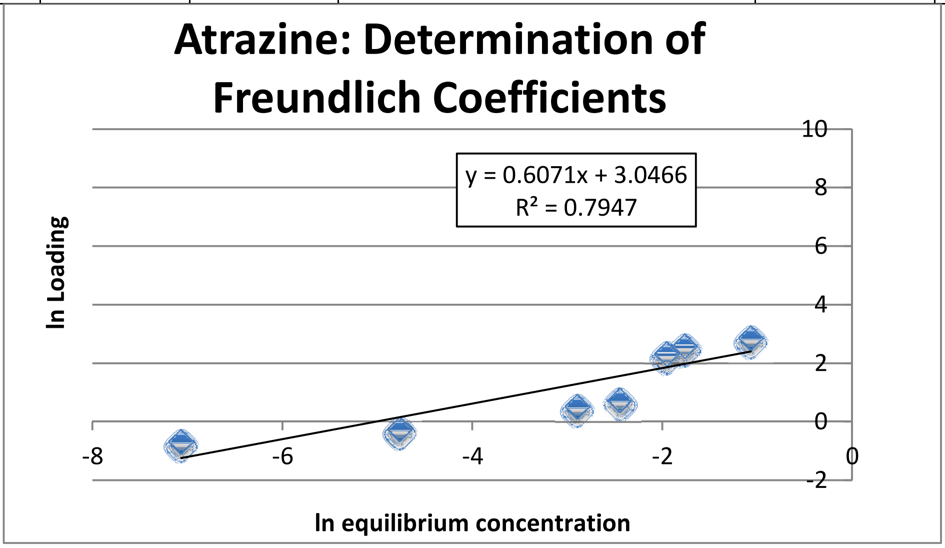
Atrazine

Sample Volume (mL)	Mass of Pesticide (mg) in Sample	Mass of GAC (g) in Sample	Mass Ratio (Adsorbate/Adsorbant)	Absorbance	Equilibrium Concentration (mg/L)	**Removal Percentage (%)**
20	0.010	0.5000	49920	0.0124	0.50	**98**
20	0.033	0.0077	230	0.0325	1.67	**92**
20	0.015	0.0500	3251	0.0170	0.77	**96**
20	0.200	0.0030	15	0.1754	10.00	**53**
20	0.024	0.7518	31169	0.0245	1.21	**95**
20	0.012	0.9990	83264	0.0141	0.60	**97**
20	0.014	1.5058	105938	0.0160	0.71	**97**
20	0.025	2.0021	81057	0.0250	1.24	**94**
40	0.083	0.0100	120	0.0395	2.08	**90**
40	0.520	0.0050	10	0.2270	13.01	**39**
40	0.684	0.0025	4	0.2970	17.09	**21**

Freundlich Isotherm for Atrazine

Loading (mg/g)	ln Loading	1/Loading	Equilibrium Concentration (mg/L)	ln Equ. Conc.	1/Equ. Conc.
0.854	-0.158	1.171	0.501	-0.692	1.997
55.444	4.015	0.018	1.673	0.514	0.598

8.538	2.145	0.117	0.769	-0.263	1.300
142.307	4.958	0.007	10.000	2.303	0.100
0.585	-0.536	1.709	1.206	0.187	0.829
0.440	-0.820	2.270	0.600	-0.511	1.667
0.292	-1.230	3.422	0.711	-0.342	1.407
0.220	-1.515	4.550	1.235	0.211	0.810
86.000	4.454	0.012	2.080	0.732	0.481
172.000	5.147	0.006	13.010	2.566	0.077
344.000	5.841	0.003	17.090	2.838	0.059


Atrazine: Determination of Freundlich Coefficients

Slope is 0.6071 and y-intercept is 3.0466. The natural log of the y-intercept is 21.04. The Freundlich isotherm for alachlor is therefore:

$$Loading_{atrazine} = Equilibrium\ Concentration^{0.6701} * 21.04$$

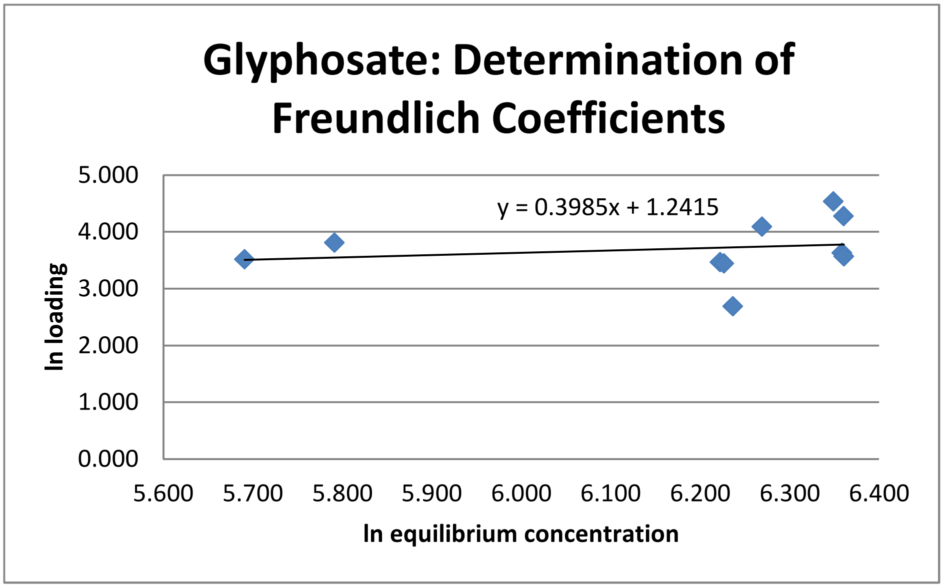
Glyphosate

Sample Volume (mL)	Mass of Pesticide (mg) in Sample	Mass of GAC (g) in Sample	Mass Ratio (Adsorbate/Adsorbant)	Absorbance	Equilibrium Concentration (mg/L)	**Removal Percentage (%)**
40	24	0.0024	0.1	0.0086	195.41	**3.7**
40	24	0.0024	0.1	0.0047	168.12	**2.0**
25	15	0.0030	0.2	0.0201	129.72	**8.7**
25	15	0.0075	0.5	0.0108	82.33	**4.7**
25	15	0.0075	0.5	0.0083	5.62	**3.6**
25	15	0.0150	1.0	0.0087	188.17	**3.8**
25	15	0.0150	1.0	0.0082	113.05	**3.6**
25	15	0.0300	2.0	0.0276	66.87	**12.0**
25	15	0.0750	5.0	0.0369	60.93	**16.0**

25	15	0.0750	5.0	0.0361	75.32	**15.7**
20	12	0.1200	10.0	0.1045	21.08	**45.4**
20	12	0.1200	10.0	0.0341	13.35	**14.8**
20	12	0.1800	15.0	0.1165	20.55	**50.7**
15	9	0.1800	20.0	0.0268	11.76	**11.7**
15	9	0.2250	25.0	0.0182	4.56	**7.9**
15	9	0.2250	25.0	0.0175	7.84	**7.6**

Freundlich Isotherm for Glyphosate

Loading (mg/g)	ln Loading	1/loading	Equilibrium Concentration (mg/L)	ln Equ. Conc.	1/Equ Conc
93.91	4.542	0.011	571.83	6.349	0.002
72.17	4.279	0.014	578.35	6.360	0.002
37.83	3.633	0.026	577.30	6.358	0.002
35.65	3.574	0.028	578.61	6.361	0.002
60.00	4.094	0.017	528.00	6.269	0.002
32.09	3.468	0.031	503.74	6.222	0.002
31.39	3.447	0.032	505.83	6.226	0.002
45.43	3.816	0.022	327.39	5.791	0.003
14.83	2.696	0.067	511.04	6.236	0.002
33.77	3.520	0.030	296.09	5.691	0.003



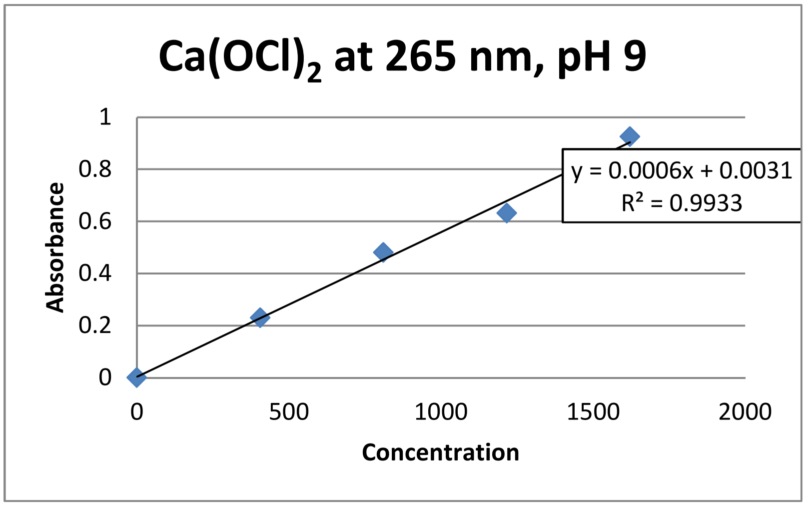
Slope is 0.3986 and y-intercept is 1.2415. The natural log of the y-intercept is 0.215. The Freundlich isotherm for glyphosate is therefore:

$$Loading_{glyphosate} = Equilibrium\ Concentration^{0.3986} * 0.215$$

Appendix D: Chlorination Data

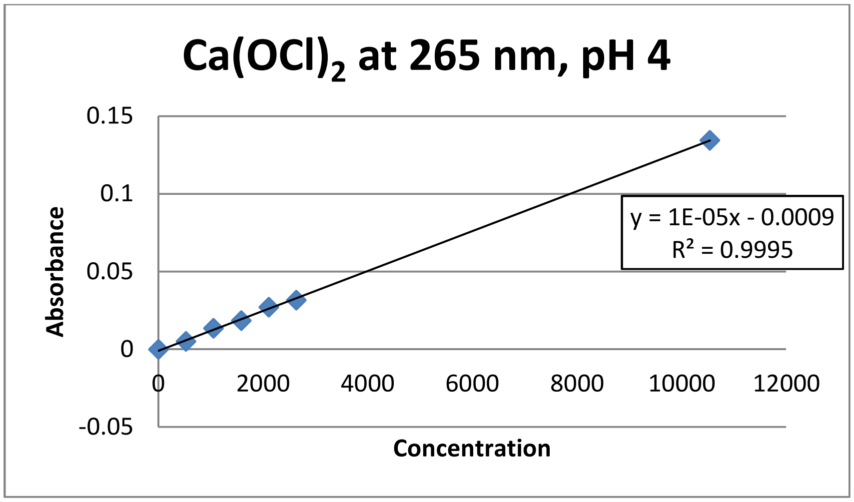
Calcium Hypochlorite Calibration Curve, 265 nm, pH 9

uL Ca(Ocl)2	uL epure	Conc of Ocl- (mg/L)	Absorbance at 265 nm
0	2000	0	0
500	1500	405	0.2303
1000	1000	811	0.48
1500	500	1216	0.631
2000	0	1622	0.925



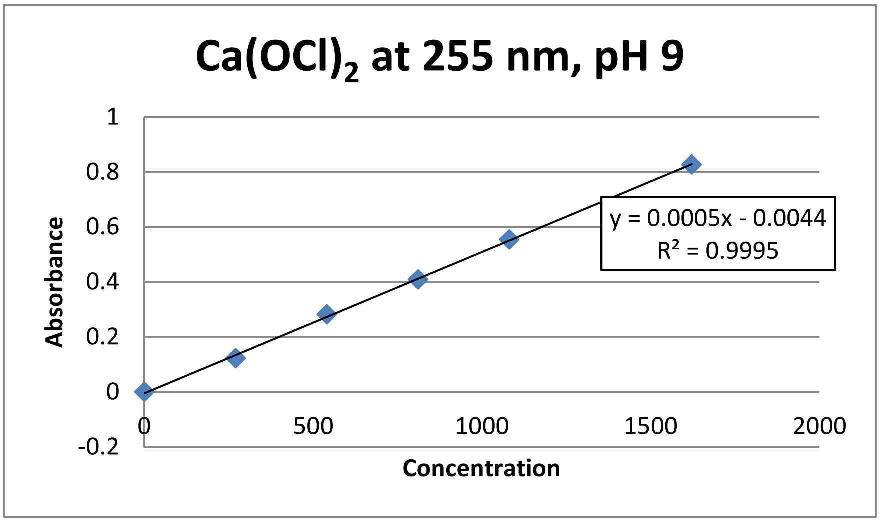
Calcium Hypochlorite Calibration Curve, 265 nm, pH 4

uL Ca(Ocl)2	uL epure	Conc of Ocl- (mg/L)	Absorbance at 265 nm
0	2000	0	0
100	1900	527	0.005
200	1800	1054	0.0135
300	1700	1581	0.0185
400	1600	2108	0.027
500	1500	2636	0.0315
2000	0	10542	0.1345



Calcium Hypochlorite Calibration Curve, 255 nm, pH 9

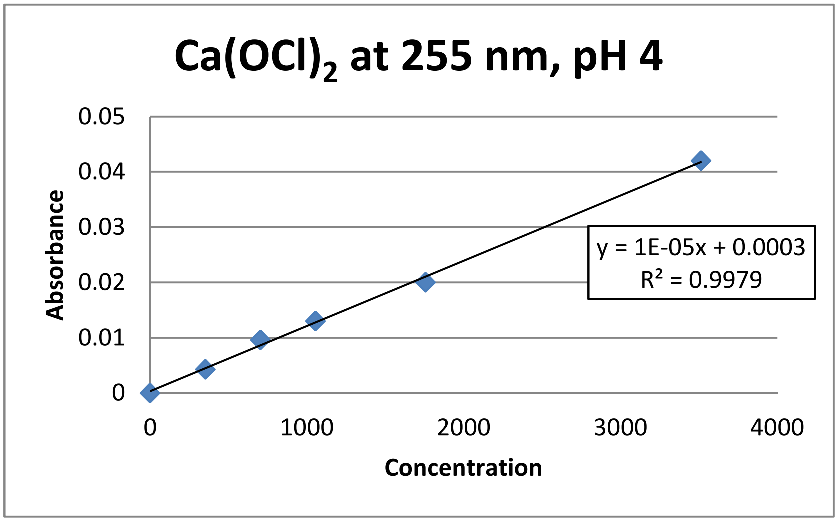
uL Ca(Ocl)2	uL epure	Conc of Ocl- (mg/L)	Absorbance at 255 nm
0	3000	0	0
500	2500	270	0.123
1000	2000	541	0.2815
1500	1500	811	0.409
2000	1000	1081	0.554
3000	0	1622	0.827



Calcium Hypochlorite Calibration Curve, 255 nm, pH 4

uL Ca(Ocl)2	uL epure	Conc of Ocl- (mg/L)	Absorbance at 255 nm
0	3000	0	0
100	2900	351	0.0043
200	2800	703	0.0096
300	2700	1054	0.013

500	2500	1757	0.02
1000	2000	3514	0.042



Alachlor, pH 9

All initial alachlor concentrations 157.25 mg/L

Sample Volumes all 25 mL, corresponding to 0.015 mmoles alachlor

Molar Ratio of Ocl-:Alachlor	mL of Ca(Ocl)2 Added	Moles Ocl- Added	Absorbance (24 hours)	Absorbance Contribution from All Ocl- Added	Final Concentration (mg/L) Removing all OCL-	**% Removal**
1	0.049	0.000007	0.3280	0.0281	148.53	**5.55**
5	0.247	0.000036	0.4075	0.1269	139.19	**11.48**
10	0.494	0.000073	0.5055	0.2483	127.90	**18.66**
15	0.741	0.000109	0.5800	0.3673	106.38	**32.35**
25	1.235	0.000182	0.7260	0.5987	65.14	**58.57**

Alachlor, pH 4

All initial alachlor concentrations 157.25 mg/L

Sample Volumes all 10 mL, corresponding to 0.006 mmoles alachlor

Molar Ratio of Ocl-:Alachlor	mL of Ca(Ocl)2 Added	Moles Ocl- Added	Absorbance (24 hours)	Absorbance Contribution from All Ocl- Added	Final Concentration (mg/L) Removing all OCL-	**% Removal**
1	0.020	0.000003	0.3230	0.0013	159.03	**-1.13**
5	0.099	0.000015	0.3170	0.0112	151.35	**3.75**
10	0.198	0.000029	0.3420	0.0213	158.54	**-0.82**
15	0.296	0.000044	0.3330	0.0313	149.40	**4.99**
25	0.494	0.000073	0.3278	0.0505	137.57	**12.51**

Atrazine, pH 9

All initial alachlor concentrations 19.01 mg/L

Sample Volumes all 25 mL, corresponding to 0.002 mmoles atrazine

Molar Ratio of Ocl- :Alachlor	mL of Ca(Ocl)2 Added	Moles Ocl- Added	Absorbance (24 hours)	Absorbance Contribution from All Ocl- Added	Final Concentration (mg/L) Removing all OCL-	**% Removal**
1	0.007	0.000001	0.3283	0.0069	18.52	**2.58**
5	0.037	0.000006	0.3230	0.0220	17.33	**8.82**
10	0.075	0.000011	0.3300	0.0408	16.65	**12.44**
15	0.112	0.000017	0.3425	0.0596	16.28	**14.35**
25	0.187	0.000028	0.3915	0.0969	16.96	**10.79**

Atrazine, pH 4

All initial atrazine concentrations 19.01 mg/L

Sample Volumes all 15 mL, corresponding to 0.001 mmoles atrazine

Molar Ratio of Ocl- :Alachlor	mL of Ca(Ocl)2 Added	Moles Ocl- Added	Absorbance (24 hours)	Absorbance Contribution from All Ocl- Added	Final Concentration (mg/L) Removing all OCL-	**% Removal**
1	0.004	0.000001	0.3255	0.0010	18.70	**1.63**
5	0.022	0.000003	0.3345	0.0012	19.21	**-1.04**
10	0.045	0.000007	0.3271	0.0015	18.76	**1.32**
15	0.067	0.000010	0.3272	0.0018	18.75	**1.38**
25	0.112	0.000017	0.3225	0.0025	18.44	**3.01**

Appendix E: UV Photolysis/UV + H_2O_2 Data

Alachlor

Run 1 without H_2O_2

Sample sizes were 6 mL

Starting concentration of alachlor solution was 155.32 mg/L corresponding to an initial absorbance of 0.314

Contact Time (min)	Absorbance	Concentration (mg/L)	**% Absorbance Removal**
0	0.314	155.32	**0**
15	1.090	530.16	**-247.13**
30	1.463	710.10	**-365.76**
60	1.605	778.98	**-411.18**
80	1.683	816.61	**-435.99**

Run 1 without H_2O_2

Sample sizes were 7 mL

Starting concentration of alachlor solution was 155.32 mg/L corresponding to an initial absorbance of 0.314

Contact Time (min)	Absorbance	Concentration (mg/L)	**% Absorbance Removal**
0	0.314	155.32	**0**
30	1.518	736.91	**-383.44**
60	1.619	785.70	**-415.61**
90	1.690	819.99	**-438.22**
110	1.653	802.12	**-426.43**
125	1.419	688.85	**-351.75**
145	1.191	578.95	**-279.30**
215	0.927	451.19	**-195.06**

With H_2O_2

For the determination of which molar ratio of H2O2:alachlor to use

Sample sizes 6 mL total, starting concentration again 155.32 mg/L. Run for 45 minutes each.

Molar Ratio	mL of H2O2 Added	Absorbance after 45 min	Concentration (mg/L)	**% Concentration Removal**
12.5	0.005	0.735	358.68	**-139.12**
25	0.010	0.0665	35.76	**76.16**
50	0.019	0.0693	37.12	**75.26**
100	0.039	0.1355	69.09	**53.94**

Using a 25:1 molar ratio of H_2O_2 : alachlor

Contact Time (min)	Absorbance	Concentration (mg/L)	**% Concentration Removal**
0	0.3140	155.32	**0**
5	1.2709	617.55	**-311.70**
15	0.6125	299.51	**-99.67**
30	0.1509	76.53	**48.98**
45	0.0677	36.34	**75.77**
60	0.0300	18.13	**87.91**
75	0.0140	10.40	**93.06**
90	0.0092	8.08	**94.61**
105	0.0065	6.78	**95.48**

Atrazine

Without H_2O_2

Sample size was 5 mL with a starting atrazine concentration of 19 mg/L

Contact Time (min)	Absorbance	Concentration (mg/L)	**% Concentration Removal**
0	0.33	19	**0**
16	0.219	12.544	**34**
30	0.1555	8.84	**53**
45	0.1063	5.97	**69**
60	0.071	3.9168	**79**
75	0.0465	2.488	**87**
90	0.0454	2.4245	**87**

With H_2O_2

A 100:1 molar ratio of H_2O_2:atrazine was used

The initial concentration of alachlor in solution was 21.93 mg/L

The sample size was 6 mL total

Contact Time (min)	Absorbance	Concentration (mg/L)	**% Concentration Removal**
0	0.38	21.93	**0**
15	0.618	35.8	**-63.2**
60	0.205	11.73	**46.5**
90	0.068	3.74	**82.9**
120	0.037	1.93	**91.2**

Glyphosate

Without H_2O_2

A 25:1 molar ratio of H_2O_2:glyphosate was used

The initial concentration of glyphosate in solution was 695 mg/L
The sample size was 6 mL

Contact Time (min)	Absorbance	Concentration (mg/L)	**% Absorbance Removal**
0	0.0141	695	**0**
30	0.0406	2020	**-187.94**
60	0.0563	2805	**-299.29**
90	0.0727	3625	**-415.60**
120	0.0851	4245	**-503.55**

With H_2O_2

A 25:1 molar ratio of H_2O_2:glyphosate was used

The initial concentration of glyphosate in solution was 1086 mg/L
The sample size was 6 mL

Contact Time (min)	Absorbance	Concentration (mg/L)	**% Concentration Removal**
0	0.02	1085.78	**0**
15	0.19	10370.16	**-855**
30	0.15	8185.6	**-654**
45	0.12	6547.18	**-503**
60	0.07	3816.48	**-251**
75	0.05	2724.2	**-151**
90	0.01	539.64	**50**

Appendix F: Ozonation Data

Alachlor

Initial concentration of all alachlor samples was 155.32 mg/L, all sample sizes 75 mL

Contact Time (min)	Absorbance	**% Absorbance Removal**
0	0.314	**0.00**
5	0.3682	**-17.26**
15	0.456	**-45.22**
30	0.4390	**-39.81**
60	0.471	**-49.84**
90	0.4680	**-49.04**

Atrazine

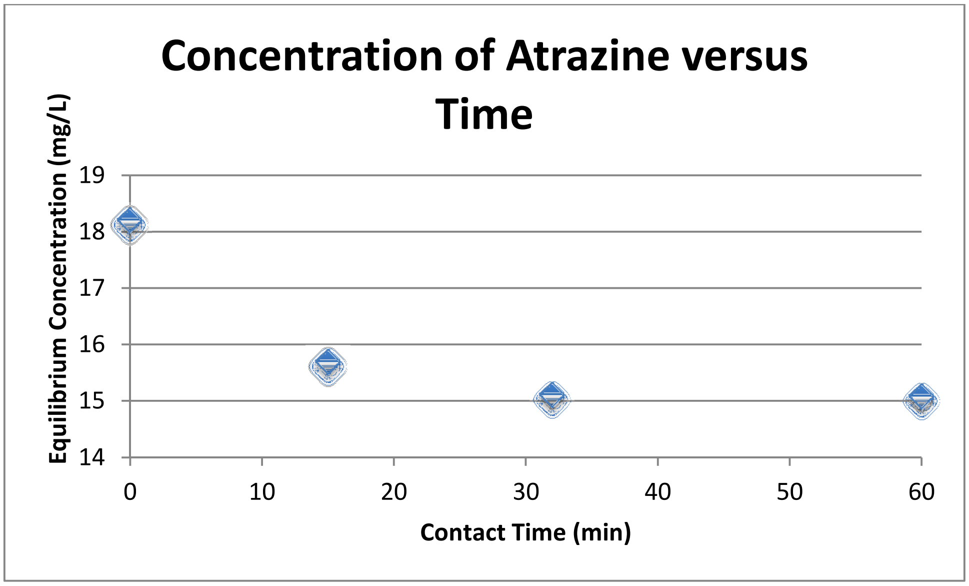
Initial concentration of all atrazine samples was 18.2 mg/L, all sample sizes 75 mL

Contact Time (min)	Absorbance	Final Concentration (mg/L)	**% Concentration Removal**
0	0.05495	18.2	**0.00**
15	0.06373	15.69	**13.79**
32	0.06619	15.11	**16.98**
60	0.06631	15.08	**17.14**

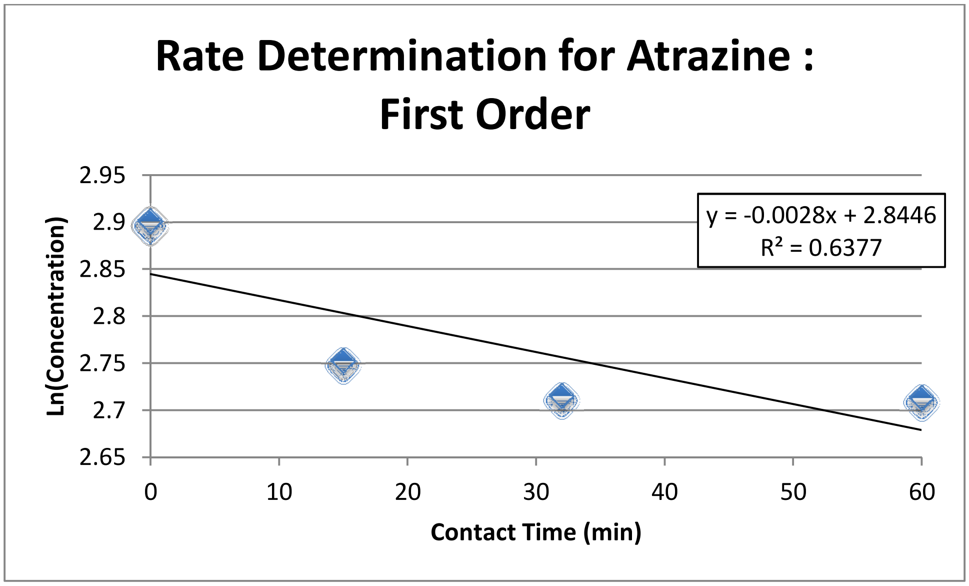
Appendix G: Kinetics Analysis Data

Ozonation Rate

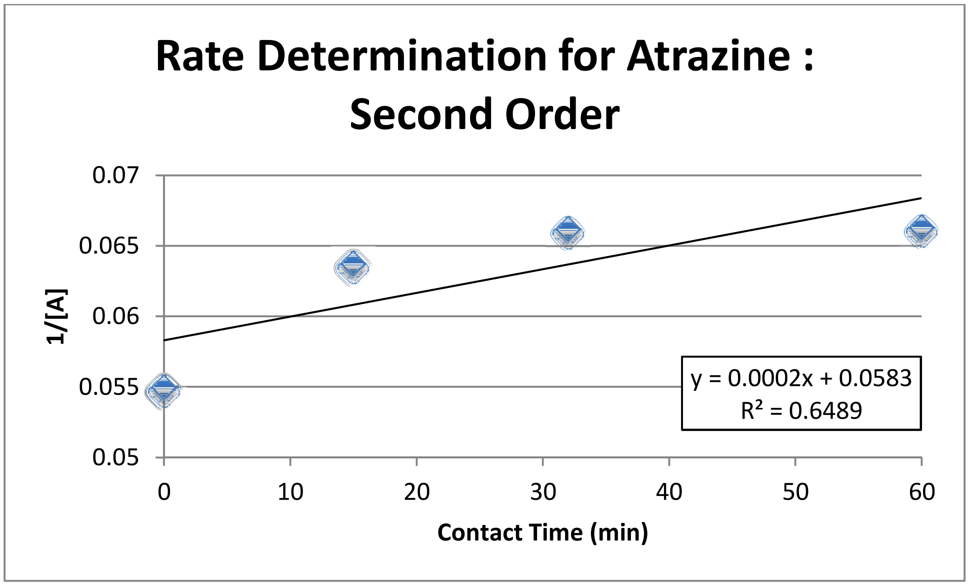
Ozonation of Atrazine



The integrated rate law states that a linear plot showing Ln(Concentration) versus time yields a first order equation. Should a plot of 1/[A] versus time yield a linear graph, the reaction is said to be second order.



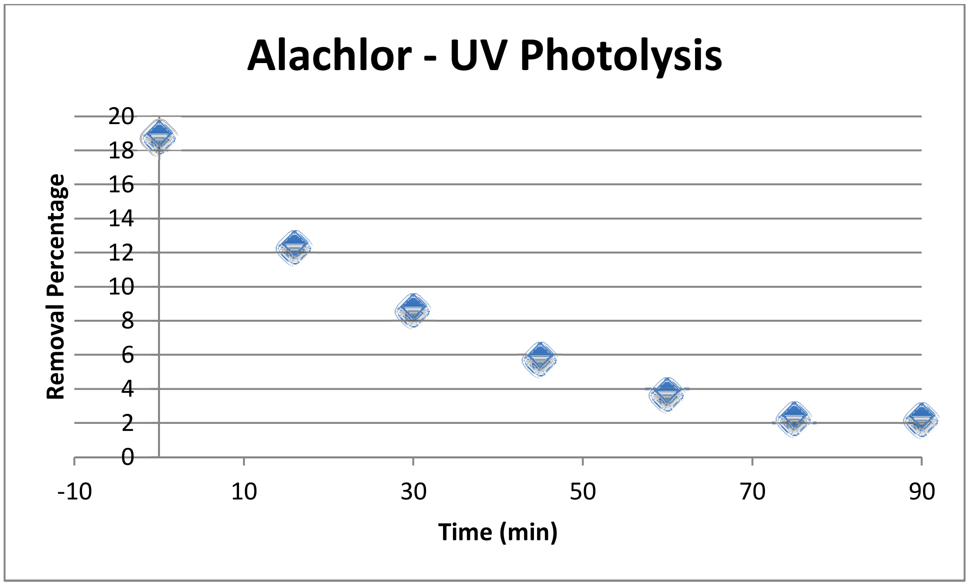
The plot did not result in a strongly linear correlation and therefore the reaction cannot be considered first order.



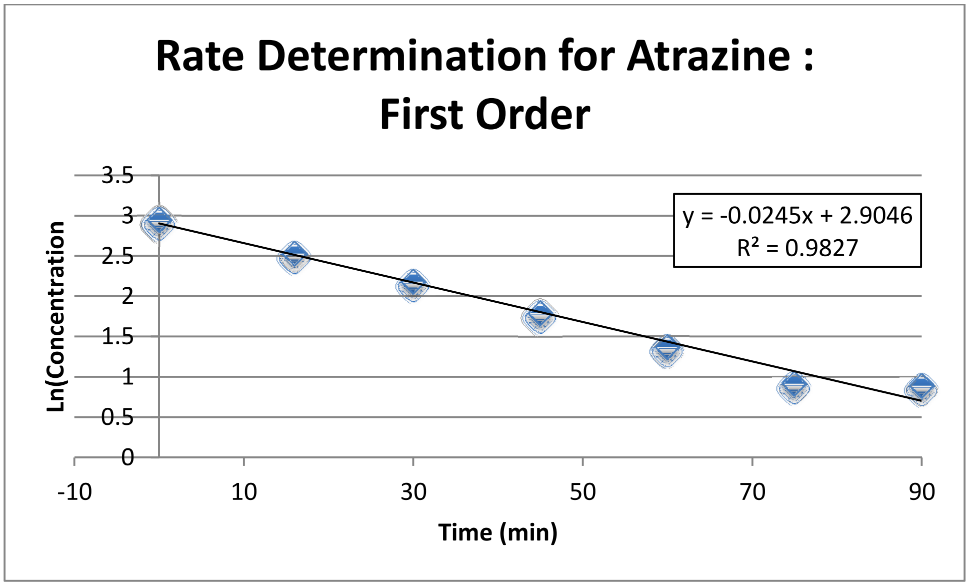
The plot use to determine whether a reaction is a second order reaction did not produce a strongly linear fit either. No rate law can be determined.

UV Photolysis Rates

UV Photolysis of Atrazine (without H_2O_2)

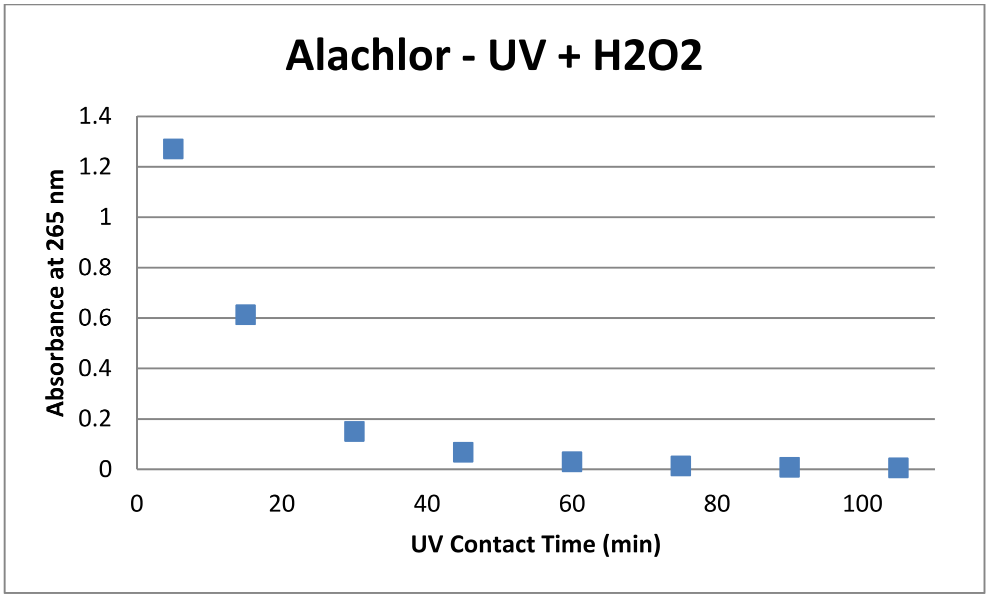


The figure above shows the concentration versus time for UV Photolysis over 90 minutes.

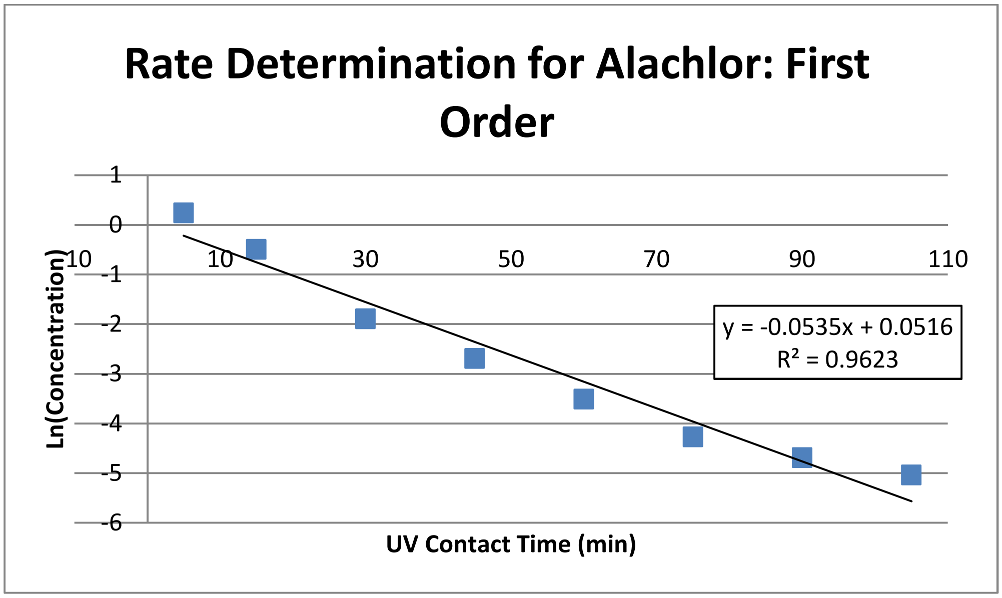


Because the figure above shows a linear plot with an R^2 value of 0.98, it can be determined that this reaction is a first order reaction.

UV Photolysis of Alachlor (with H_2O_2)

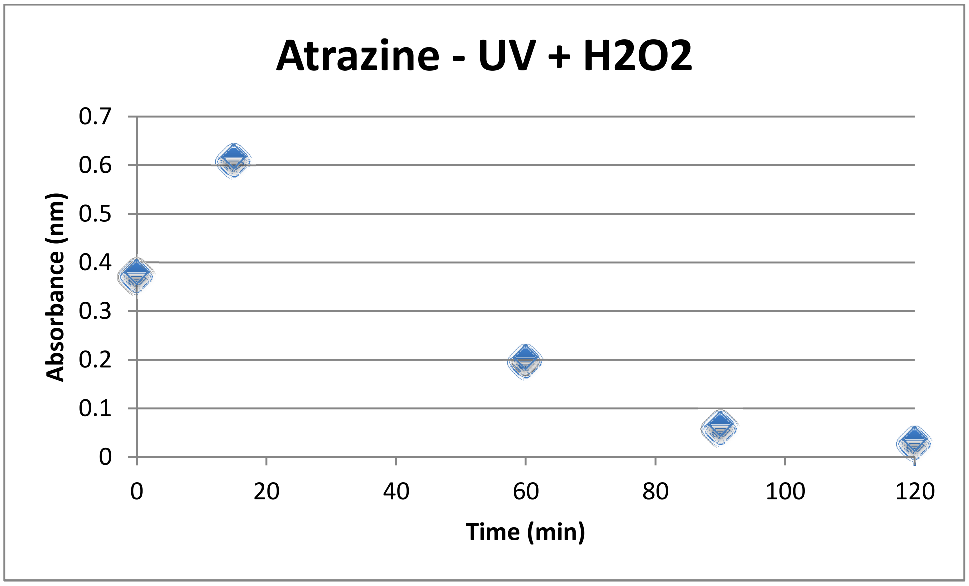


The figure above shows the logarithmic trend or the absorbance of alachlor to the contact time.

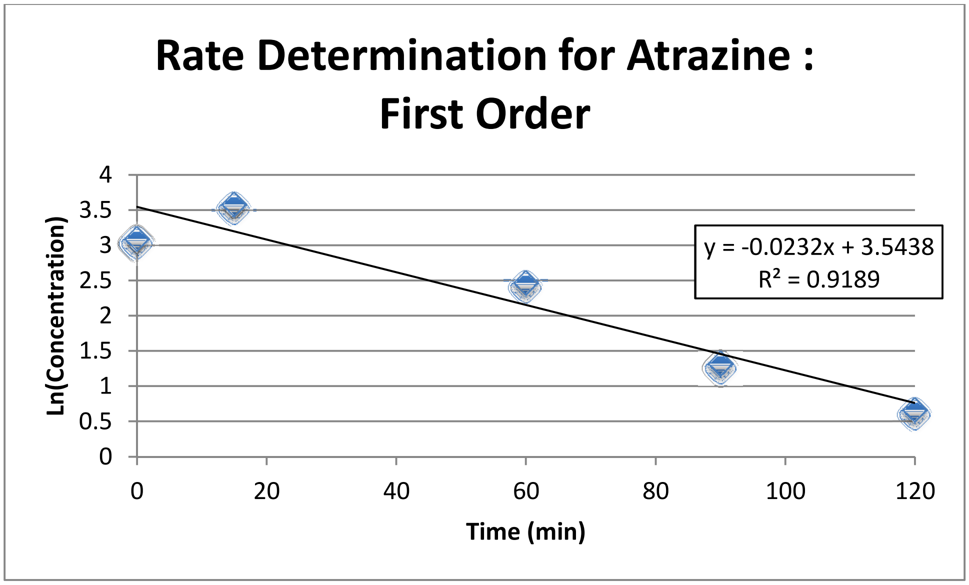


With an R^2 value of 0.96, the reaction was determined to be first order.

UV Photolysis of Atrazine (with H_2O_2)

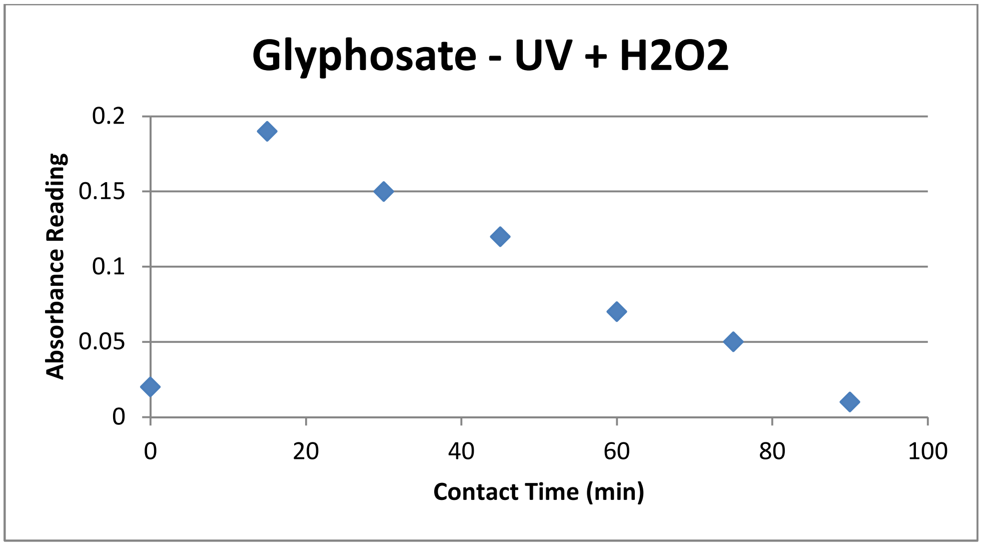


The figure above shows the correlation between the Absorbance and contact time.

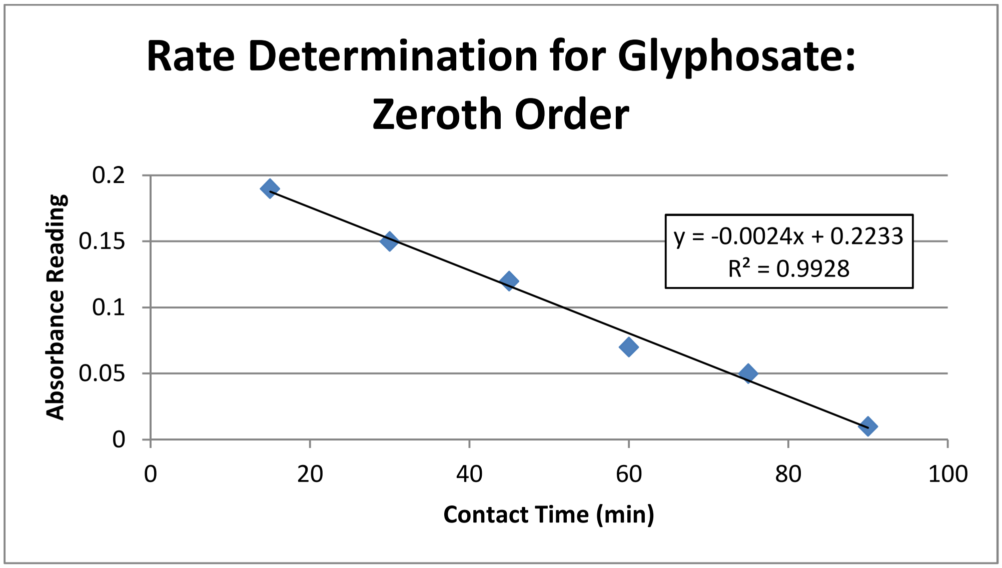


Taking into account the spike at time twenty, the reaction was considered to be first order due to the linearity of the plot.

UV Photolysis of Glyphosate (with H_2O_2)



The above figure shows how after the initial spike of in the absorbance reading at approximately time equals ten minutes, the absorbance decreases in a linear fashion.



When all data points after the initial spike are analyzed, it is clear that the reaction of byproducts and glyphosate follow a typical zeroth order reaction.