CHAPTER 2

BACKGROUNDS AND LITRATURE REVIEW

2.1 Pesticide

U.S. EPA (1996), Federal Insecticide, Fungicide and Rodenticide Act (Federal Environmental Pesticide Control Act) defined "Pesticides" as: "any substance or mixture of substance intended for preventing, destroying, repelling, or mitigating any pest (insect, rodent, nematode, fungus, weed, other forms of terrestrial or aquatic plant or animal life or viruses, bacteria or other microorganisms, except viruses, bacteria or other microorganisms on or in living man or other animals, which the Administrator declares to be a pest)".

While U.S. Food and Drug Administration defined pesticides as any substance or mixture of substance intended for preventing or controlling any pest and included any substance or mixture of substance intended for use as a plant growth regulator, defoliant or desiccant.

2.1.1 Pesticide Classification

Pesticides are divided into eight major groups, which are:

1. Insecticide: use for control insects both in public health and agriculture. Insecticide is divided into four main subgroups, they are:
   - 1.1 Organochlorine insecticides: contain the element chlorine, hydrogen and carbon. Some also contain oxygen and sulfur.
   - 1.2 Organophosphate insecticides: contain the element carbon, hydrogen, oxygen, sulfur and phosphorus such as parathion.
   - 1.3 Carbamates: contain the element carbon, hydrogen, oxygen, and nitrogen and it's effect like a organophosphate pesticide, such as carbaryl, baygon.
   - 1.4 Inorganic insecticides: the oldest insecticides but are still widely used, such as fuel oil, kerosene, cyanide and arsenic.

2. Rodenticide: use for control rodents both in public health and agriculture.
3. Herbicide: only use for control herb in agricultural practice.
4. Larvicide: use for control immature stages of insects, whether larvae or nymphs.
5. Molluscacide: only use for control molluse in agriculture
6. Fungicide: use for control fungi both in public health and agriculture
7. Repellent: use for control biting arthropods such as mosquitoes, black flies, chiggers and other.
8. Fumigants: use for control insect both in public health and pest control operators.

2.1.2 Organophosphate Pesticide

Galo and Lawlyk (1991) reported that organic phosphorus, organophosphate or organophosphorus pesticide shared a common chemical structure, but they differed greatly in the detail of their structure, in their physical and pharmacological properties, and consequently in the uses to which they have been put or for which they have been proposed. The organic phosphorus compound was in which X was the leaving group.

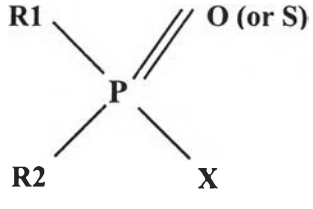

Figure 2.1: Common Structure of Organophosphate Pesticide

All the compounds may be placed in four main categories, depending on the character of the X constituent, as follows:

Categories I X contained a quaternary Nitrogen, this categories was small and Ecothiopate isodide was the example pesticide in this group.

Categories II X = F: Fluorophosphate groups have only a few compounds used or even considered as pesticide. For the example, dimefox and diisoproptyl fluorophosphate or DFP.

Categories III X = CN, OCN, SCN, or Halogen other than F: tabun, parathion were the example pesticide in this group.

Categories IV It may be subdivided into at least eight groups on the basis of their R1 and R2 constituents. Several of these groups differed either quantitatively and qualitatively in toxicity, and in some instance the basis for the difference was known. The eight groups and an example of each were as follows:

1. Dimethoxy compounds: malathion, parathion-methyl, mevinfos, monocrotophos, dicrotophos, dimethoate, fenthion, chlopyrifos-methyl, azinphos-methyl.

2. Diethoxy compounds: parathion, diazinon, phorate, chlopyrifos, azinphos-ethyl.

3. Other dialkoxy compounds: propaphos

4. Diamino compounds: schradan

5. Chlorinated and other substituted dialkoxy compounds: haloxon

6. Trithioalkyl compounds: merphos, *S,S,S*-tributyl phosphorotrithiolate: DEF, merphos.

7. Triphenyl and substituted triphenyl compounds: *o*- and *p*-cresyl saligenin phosphate.

8. Mixed substitute compounds: leptophos, carejin, fonofos, metamidofos

2.1.3 Effect of Organophosphate Pesticides

Sawyer, et al. (1994) stated that organophosphate pesticide effected on insects and toxic to human health. It was potential noncarcinogenic but its effectmay interfere nervous system function. Moreover, it is believied that may result in nerve impulse transmission; or acethylcholinesterase, and enzyme.

There are numerous and diverse effects which related to metabolic and behavioral response in enzyme production, growth, reproduction activity, production of tumors and teratogenic effect, after being exposed to these pesticides.

Britt (2000) stated that the effects of organophosphate compounds can be categorized into two levels which are:

1. Acute Effect of Organophosphate Pesticides

   Sign and symptoms of overexposure to organophosphate compounds would occur fairly rapid and could be noticeable after exposure from five minute to 12 hours. The acute symptoms depended on the degree of inhibition of acetylcholinesterase of each person. The results include salivation-increased, lacrimation, bronchospasm, dyspnea, gastrointestinal effects (vomiting, abdominal cramps, and diarrhea), blurred vision, and incontinence, wheezing, decreased heart rate and finally paralysis. Central nervous system symptoms include giddiness, tension, anxiety, headache, tremors, confusion, coma, and convulsions.

2. Chronic Effects of Organophosphate Pesticides

   Organophosphate induced delayed neuropathy has been associated with exposure to only a small amount of organophosphate compounds, with almost exclusively at near-lethal exposure level. From the study, there was no evident to prove that the organophosphate compounds had the permanent adverse health effects

The Lethal Dose Fifty (LD_{50}) of each organophosphate pesticide depended on its chemical property. LaGrega, et al., 2001, said that the acute oral lethal dose of parathion and malathion for a mouse was 2 mg/kg and 2800 mg/kg. Both of pesticide would be accumulated in the aquatic food chain and conducted to biomagnification.

Galo and Lawryk (1991), the toxic signs and symptoms, which characterized poisoning depended on the inhibition of acetylcholinesterase. This action plused direct cholinergic activity would produces the same end result. The result of toxic could be separated into 4 types as the following:

1. Typical Poisoning

   Sign and symptom were, at least to a very great extent, secondary to cholinesterase inhibition. The usual symptoms included headache, giddiness, nervousness, blurred vision, weakness, nausea, cramps, diarrhea, and discomfort in the chest. Sign included sweating miosis, tearing, salivation, vomiting, cyanosis, uncontrollable muscle twitches following by muscular weakness, convulsion, coma, loss of reflexes and loss of sphincter control.

   In ordinary occupational case, relatively incapacitating symptoms of nausea, cramps, discomfort in the chest, muscular twitching, etc. often followed the initial giddiness, blurred vision, and headache only after a period of 2-8 hours, but the onset of serious symptoms may be more rapid. Treatment of significant illness after excessive exposure to these compounds should not be delayed merely because miosis was absent.

2 Behavioral Effect

   Mild poisoning by organic phosphorus compounds has been associated with lapses of attention or judgment that led to accident.

   Decrements in alertness and memory have been associated with chronic industrial and agricultural exposure to organic phosphorus pesticides. Increased irritability, memory deficits, lethargy, and lack of energy have been associated with multiple or severe acute exposures.

3 Visual Effect

The typical anticholinesterase visual effects of organic phosphorus insecticides constituted a minor aspect of poisoning. Blurred vision may offer an early warning which, if needed, an exposed person could stop exposure and initiate therapeutic management.

The effect may also have special importance for the safety of workers. The inability of the pupils of both eyes to expand in dim light could be a cause of an accident as a worker entered a dimly light area. In addition, this effect greatly interfere judging distances and this, in turn, could interfere with driving vehicles though, cataracts resulting from occupational exposure to organic phosphorus insecticide have been reported.

4. Other Local Effect

Not only some visual effects but also the effects on the respiratory system, skin, muscles and gastrointestinal system which provided the cholinesterase inhibitor involved.

The inhalation of pesticide vapors or aerosols may rapidly lead to illness which was first by respiratory signs and symptoms. In addition, local effect on sweat glands and muscles from dermal absorption in the involved area indicated the peripheral nature of sign which was frequently seen in systemic poisoning.

From NIOSH Method 5600 depicted that ACGIH studied and determined the Permissible Exposure Level (PEL) for each organophosphate pesticide for the industrial and agricultural workers. Table 2.1 showed the physical, chemical property and the exposure permission of organophosphate pesticide which Bang Rieng farmer widely used.

TABLE 2.1: Physical, Chemical Properties and Exposure Permission of Organophosphate Pesticide

Name	CAS No.	Molecular Weight	Vapor Pressure (mm. Hg)	Exposure Limits			LD_{50}	ADI*
				OSHA PEL (mg/m^3)	NIOSH REL (ppm)	ACGIH TLV (mg/m^3) at skin	Mouse : mg/kg	mg/kg.day
Chlorpyrifos $C_9H_{11}Cl_3NO_3PS$	2921-88-2	350.58	$1.84x10^{-5}$	0.2	0.014	0.2	145	0.01
Dicrotophos $C_8H_{16}NO_5P$	141-66-2	237.19	-	0.25	0.026	0.25	16-21	-
Malathion $C_{10}H_{19}O_6PS_2$	121-75-5	330.35	$4x10^{-5}$	10	0.740	10	1,000-1,375	0.02
Methyl-Parathion $C_8H_{10}O_5PS$	298-00-0	263.20	$1.5x10^{-6}$	0.2	0.019	0.02	14-24	0.02
Methamidophos $C_2H_8NO_2PS$	10265-92-6	141.12	$3x10^{-4}$	-	-	-	25-27	0.004
Mevinphos $C_7H_{13}OP$	198-01-1	224.15	$3x10^{-3}$	0.1	0.011	0.1	3.7-6.1	0.0015
Monocrotophos $C_7H_{14}NO_5P$	919-44-8	223.17	$7x10^{-6}$	0.25	0.027	0.25	17-20	0.0006
Parathion $C_{10}H_{14}NO_5PS$	56-38-2	291.26	$3.78x10^{-5}$	0.1	0.004	0.1	3.6-13	0.005

Note: Summarized from ACGIH recommendation cited in NIOSH Manual of Analytical Method (NMAM): 5600.

ADI recommendation from Thailand: Pollution Control Department.

2.1.4 Example of Fate of Organophosphate Pesticide in the Environment

After the farmer spraying organophosphate pesticide solution is sprayed in the air, the structural transformation by hydrolysis, photolysis or biodegradable will process to another form. The examples for methyl parathion and chlorpyrifos fate in the environment are following:

1. Methyl parathion in air undergoes one of two reactions. The first involves hydrolytic degradation where methyl parathion is converted to two compounds of lesser toxicity, nitrophenol and dimethyl phosphorothioic acid. The second reaction is oxidative, and involves the phototransformation of methyl parathion to methyl paraoxon. This reaction is reported to occur rapidly in air when methyl parathion contacts hydroxyl radicals in the presence of ultraviolet light. Although the rate for this reaction is not known, once methyl paraoxon is produced, it is considered resistant to further atmospheric reactions. (California EPA, 1999)

   Methyl parathion has a half-life in aqueous solution of 175 days, and 10 days up to two months in soils. The rate of degradation increases with temperature and with exposure to sunlight. When large concentrations of methyl parathion reach the soil, as in an accidental spill, degradation will occur only after many years. The US-EPA may have detected 4-nitrophenol, a methyl parathion breakdown product, at very low levels in drinking water wells. methyl parathion is unlikely to bioaccumulate. (PAN-UK, 1995)

2. Chlorpyrifos is generally active in the soil, strongly adsorbed by most soils and relatively immobile in the soil. The half-life of chlorpyrifos ranges from 11 to 141 days in a variety of different soil types; it is thus considered to be moderately persistent. Chlorpyrifos is less persistent in soils with higher pH values. Soil microorganisms break down chlorpyrifos and it can hydrolyse at a moderate rate. The main break-down product of chlorpyrifos in the

soil (as well as in plants and animals) is 3, 5, 6-trichloropyridinol (TCP) which is weakly to moderately adsorbed, mobile and very persistent in the soil. TCP is considered to be relatively non-toxic. (California EPA, 2001)

2.2 Risk Assessment

Turnbull (1992) stated the definition of risk assessment that it was the scientific process of assessing the probability of an adverse effect caused by the exposure to a hazardous substance. To develop a comprehensive approach to health hazard evaluation of chemical contaminants in the environment, it is necessary to collect and classify all available and pertinent in the information on the subject. In a majority of cases, there is a paucity of relevant data, and scientific judgement as well as policy decision, which play a important role in establishing acceptable levels of population exposure.

The final output of the risk assessment process should define the values which ideally represents concentrations of chemical compounds in the air or in any other environmental media that would not pose any hazard to the human population.

For the pesticide effect in human's life, U.S.EPA used risk assessment as a tool for evaluating the chemicals prior to registration, and in reevaluating older pesticides already on the market to ensure that they can be used with a reasonable certainty of no harm. There are more than 865 active ingredients registered as pesticides, which are formulated into thousands of pesticide products that are available in the marketplace. About 350 pesticides are used on foods, and to protect our homes and pets.

U.S. EPA (1999) uses the National Research Council's four-step process for human health risk assessment Details of steps are the following:-.

Step One: Hazard Identification (Toxicology)

To identify potential health effects that may occur from different types of pesticide exposure. EPA considers the full spectrum of a pesticide's potential health effects. Generally, for human health risk assessments, many toxicity studies are conducted on animals by pesticide companies in independent laboratories and evaluated for acceptability by EPA scientists. EPA evaluates pesticides for a wide range of adverse effects, from eye and skin irritation to cancer and birth defects in laboratory animals.

Step Two: Dose-Response Assessment

The amount of a substance a person is exposed to is as important as how toxic the chemical might be. Dose-response assessment involves considering the dose levels at which adverse effects were observed in test animals, and using these dose levels to calculate an equal dose in humans.

Step Three: Exposure Assessment

People can be exposed to pesticides in three ways; inhaling pesticides (inhalation exposure), absorbing pesticides through the skin (dermal exposure), and getting pesticides in their mouth or digestive tract (oral exposure).

Depending on the situation, pesticides could enter the body by any one or all of these routes. Typical sources of pesticide exposure include:

- **Food:** Most of the vegetables and fruits have been grown with the use of pesticides. Therefore, pesticide residues may be present inside or on the surfaces of these foods.
- **Home and Personal Use Pesticides:** The usage of pesticides in and around our home to control insects, weeds, mold, mildew, lawn and garden pests and to protect your pets from pests such as fleas.

Pesticides may also be used as insect repellants which are directly applied to the skin or clothing.

- **Pesticides in Drinking Water:** Some pesticides that are applied to farmland or other land structures can make their way in small amounts to the ground water or surface water systems that feed drinking water supplies.
- **Worker Exposure to Pesticides:** Pesticide applicators, vegetable and fruit pickers can be exposed due to the nature of their jobs. To address the unique risks workers face from occupational exposure, EPA evaluates occupational exposure through a separate program. All pesticides registered by EPA have been shown to be safe when used properly.

Step Four: Risk Characterization

Risk characterization is the final step in assessing human health risks from pesticides. It is the process of combining the hazard, dose-response and exposure assessments to describe the overall risk from a pesticide. It explains the assumptions used in assessing exposure as well as the uncertainties that are built into the dose-response assessment. EPA's role is to evaluate both toxicity and exposure and to determine the risk associated with use of the pesticide. Simply put,

RISK = TOXICITY x EXPOSURE

This means that the risk to human health from pesticide exposure depends on both the toxicity of the pesticide and the likelihood of people coming into contact with it. At least some exposure and some toxicity are required to result in a risk.

EPA evaluates studies conducted over different periods of time and that measure specific types of effects. These tests are evaluated to screen for potential health effects in infants, children and adults.

1. **Acute Testing**: Short-term exposure; a single exposure (dose) such as oral, dermal (skin), and inhalation exposure, eye irritation, skin irritation, skin sensitization and neurotoxicity (nerve system damage).
2. **Sub-chronic Testing: Intermediate exposure:** Repeated exposure over a longer period of time (i.e., 30-90 days) such as: oral, dermal (skin), and inhalation exposure and neurotoxicity.
3 **Chronic Toxicity Testing:** Long-term exposure, repeated exposure lasting for most of the test animal's life span. It is intended to determine the effects of a pesticide after prolonged and repeated exposures such as chronic effects (non-cancer) and carcinogenicity (cancer).

U.S.EPA has the specific testing to evaluate the pesticide's effect in human as follow:

- Developmental and Reproductive Testing: To identify effects in the fetus of an exposed pregnant female (birth defects) and how pesticide exposure affects the ability of a test animal to successfully reproduce.
- Mutagenicity Testing: To assess a pesticide's potential to affect the cell's genetic components.
- Hormone Disruption: To measure effects for their potential to disrupt the endocrine system and its hormones that help the development, growth, reproduction, and behavior of humans.

Risk Management

Once EPA complete the risk assessment process for a pesticide, they use this information to determine and make a more informed decision regarding whether to approve a pesticide chemical or use, as proposed, or whether additional protective measures are necessary to limit occupational or non-occupational exposure to a pesticide. For example, EPA may prohibit a pesticide from being used on certain crops, requiring workers to wear personal protective equipment (PPE) such as a

respirator, or chemical resistant gloves, or not allowing workers to enter treated crop fields until a specific period of time has passed.

2.3 Exposure Assessment

Turnbull (1992) said that the exposure assessment had two aspects. Firstly, it was the general evaluation of actual or anticipated exposure concerning the type, magnitude, time and duration. In general, knowledge of exposure from all source is needed in recognising the contributions to total body intake from all exposure route. Additional information relation to special population groups at risk based on unusual individual susceptibility or unusually high levels of exposure in specific segments of the population must also be given consideration. Most important, the relationship between present levels of exposure to ambient contaminants and calculated criteria must be carefully evaluated to determine if a human health hazard exists.

The second aspect involved the characterization of a population, the number of people exposed, a profile of particularly sensitive individuals, and other specific exposure data important for the quantitative estimate of risk for any particular population. This aspect was usually a part of the risk management process when decisions concerned a fixed population.

Berglund and et al. (2001), said that exposure was a contact. People were in contact with, expose to potentially harmful chemical, physical and biological agents in the air, food, water, soil, dust, products etc. Exposure does not result only from the presence of a harmful agent in the environment. There must be contact between the agent and the outer boundary of the human body such as airway, skin and mouth.

U.S.EPA defined and explained that the exposure assessment was the determination of the actual levels of exposure and absorption of toxicant among the population of exposed individuals. The levels of exposure were measured on the frequency and duration of exposure as well as the levels of contaminant in the

exposure media such as soil, water, air, and food. Actual absorption was determined by toxicological studies.

The level exposure to contaminants depended upon the initial concentration at the source of contamination and its rate of distribution and dilution as it travels through air, water, soil, and food. The chemical reactions, which occur in the exposure media, may render the agent more or less toxic than the original compound. This was a very important consideration with regards to human contact and the estimated concentration of a toxicant upon exposure to the environmental media. Environmental fate studies provided information about the fate of chemicals in the environmental media and were used in exposure assessment for characterizing the exposure scenario.

2.3.1 Entry and Fate of Chemicals in Humans

A single chemical can enter the body through all three routes of exposure inhalation, ingestion and skin penetration (dermal exposure). A pesticide which was sprayed can be inhaled during use in the mist or vapor form. It would penetrate through the skin during mixing and application; and be ingested through food if not washed off hands or food before eating.

After a chemical entered the body, it was often absorbed into the bloodstream and can move throughout the body. The amount absorbed and the rate of absorption depended on the chemical and the route of exposure. This movement of the substance through the bloodstream was called distribution. Through distribution, a chemical can come into contact with all parts of the body, not only the original site of entry. In some cases, such contact, distant from the site of entry, can lead to adverse health effects. For example, ingestion of the pesticide paraquart into the stomach can lead to damage to the lungs.

Once a chemical was absorbed into the bloodstream, it can have several different fates. In many cases, it was rapidly removed from the

body through the urine or feces. In other situations, it may be stored in various parts of the body, such as fat or bone, and remained in the individual for many years. A compound may also lead to a toxic effect through interaction with certain organs or tissues in the individual or with other compounds in the body.

Often, a substance, which was absorbed into the body interacted with particular body chemicals and was changed into one or more other chemicals. This process was called metabolism and the products were called metabolites. Metabolism may lead to products, which were easier for the body to excrete and can protect the body from possible adverse effects. In other cases, however, the metabolites may be more toxic than the original chemical which was absorbed. The variety of products resulting from metabolism may have the same possible fates as the original chemical storage, excretion or toxicity.

The severity of the adverse effect from pesticide exposure depended on many factors, which were

1 **Chemical Properties:** The particular properties of the absorbed chemical were quite critical to its fate in the body. Certain chemicals were very resistant to metabolism and readily dissolve in fat so that they tended to be stored. Some chemicals were more rapidly metabolized and excreted and were gone before they can cause adverse effects. The organophosphate pesticides tended to behave this way at low doses.

2 **Individual's Characteristics:** The characteristics of the individual, who was exposed were also very important in the fate of the chemical. Age, sex, genetic background, previous exposures, diet and other factors played important roles in the way that the body interacted with a chemical and in turn the potential for adverse effects. Thus, the characteristics of both the chemical and the exposed individual were important factors determining the fate of the chemical in the body.

3 **The time Course for Exposure:** In the case of a single event exposure, it was the total amount of chemical to which a person wss exposed that determined the severity of the toxic effect, if any. The greater the amount of exposure, the greater the potential for adverse health effects. In some cases, this was due solely to the inherent toxicity of the chemical and, in others, also to the overwhelming of the body's ability to respond. In the latter case, the body may not be able to metabolize the chemical rapidly enough to prevent an increase in concentration to toxic levels. In such a situation, there was a clear threshold above which toxic signs and symptoms aappeared.

In the case of repeated or multiple exposures to a chemical, it wass not only the total amount of exposure, but also the rate or timing of exposure that was quite important. All processes in the body normally proceeded at specific rates so that metabolism, excretion and storage occured during a particular period of time after a chemical was absorbed. For a one occurrence exposure, the time needed for the various processes that breakindown the compound to be completed would determine the length of time that a toxic response, if any, persisted.

However, if there were repeated exposures to the same chemical, the situation was more complicated. If there was enough time between exposures so that all of the chemical from the initial exposure was excreted, and no effects persisted, then each exposure was essentially independent of the previous one and can be treated as a single exposure. However, if the time between exposures was so short that some of the chemical remained from the first exposure, then a buildup of the chemical could occur. Over time this buildup could lead to levels which were toxic.

The total amount of exposure can have different results depending on whether the exposure occurred all at once or repeatedly over time (the time course of exposure). A high dose given once may have a toxic effect while the same total dose given in small amounts over time would not.

Dooley (1996) stated that exposure assessment, in other words, exposure analysis examined the fate of the released hazard and identified the receptors that have become exposed to it. These two components, the hazard and the receptors, must come together for exposure to occur. The released hazard created the risk and the receptors may suffer the consequences.

Exposure analysis also determined the presence of the receptors in state, space, and time, and the mechanism, or pathway by which the hazard received by the receptors.

The pathway usually had two parts firstly, the external pathways, which dealed with how the receptor comes into contact with the hazard. The second was the internal pathway, which examined how the hazard reached the particular part of the receptor that becomed affected. The product of exposure analysis was a description of the kinds and the intensities of exposure received by the different categories of receptors.

1. **Exposure pathways**

   The pathways by which hazards can find their way to receptors were combinations of air, water (ground and surface), soil, rocks and the food chain. These were often referred to as the atmosphere, hydrosphere, pedosphere and biosphere respectively and transferred between the sphere may occur. The concept of pathways was a familiar one in the environmental field, and could be applied equally well in risk analysis providing uncertainty was included.

2. **Transformation of released hazard**

   The variety of possible exposure was great and varied, and included the possibility as a transformation, or a change in the hazard. Transformations may be in state, space or time.

   2.1 Change in state: Changes or transformations occured in the physical properties or characteristics of the released energy,

material, environmental stress, or social force. Most often, the hazard was received by the receptors in a state that was different from the one in which it was released. A hazard may increase or decrease as a result of the change.

2.2 Change in space: A hazard changed in space if it moved from one point. Line or area of the initial released to some other location. The hazard may be changing in space as it moved to other location.

2.3 Change in time: A hazard may be released at one moment yet not reach the receptors for some time. This may happen when a hazard was changing its state or its space, or both.

3. Characteristics of exposure

The concerning was to identify the different groups of receptors that were sensitive to the hazard or the sensitive receptors. The means by which they became exposed and the probability of exposure must be included the following:-

3.1 State of the receptor: to determine the characteristics of the receptor, which were important in estimating the potential consequences.

3.2 Space of the receptor: The receptors were often distributed geographically. So the spatial distribution of the different types and classes of receptors needed to be described because the hazard was received only if the spatial distribution of the released hazard coincided geographically with the receptors

3.3 Time of the receptor: The phrase time of reception had two meaning. Firstly, it may refer to the date and time of day on which the reception occurred. The second way time of reception, which may be used was to refer to the time pattern of the exposure such as its frequency or duration. Time pattern could include a toxic leak received for a short duration and only once.

3.4 Intensity: The level of magnitude of the hazard can be expressed in a number of ways. It had potential for

consequences but the exposure was measured in different ways depending on the nature of the hazard. In describing levels of exposure, the term intensity was used to measure the degree, magnitude or level of exposure to the hazard.

4. **Fate Models: Source of literature**

There were many transformation and pathways from releases to receptors and many models have been developed to estimate the transformation and exposure intensities.

5. **Spatial Models**

The hazard and the receptor must come together in time and space before exposure occurs. To determine the probability of an exposure, we must know where the hazard was and where the receptors were at specific times.

The exposure assessment methods of human exposure would be separated into 2 levels:

1 **Indirect method:** it involved questionnaires and diaries (personal and resident characteristics, time-activity patterns and exposure factors), environmental monitoring, and modeling.

Berglund (2001), stated that questionnaire could be used to obtain information of individual characteristics as well as relevant exposure factors and time activity patterns. Questionnaire could provide information on the existence of exposure sources and other characteristics in a community or an industry, and could be used to categorize exposure.

2 **Direct method:** it involved personal exposure monitoring and biological monitoring. The choice of method and strategy depended on the purpose of the study and the quality of data which was needed to address the answerable questions.

Berglund (2001) stated that objectives of monitoring were to repeat observations, measurements and evaluation of pollutant concentration in the

environmental media such as air, water, foods, soil and dust. To assess compliance with exposure standard, the observation of changes over time in pollutant concentration and the assessment of the degree of current approximate human exposure to certain pollutant should be taken into consideration.

In summary, U.S. EPA (1992) stated that exposure was dependent upon the intensity, frequency, and duration of contact. The intensity of contact was typically expressed in terms of the concentration of contaminant per unit mass or volume in the medium to which humans were exposed. Exposure assessments were sought to characterize the "real-life" situations whereby potentially exposed populations were identified, etc.

This study determined Exposure Concentration and Intake Concentration based on the following theory:-

1. Exposure Concentration

Gratt (1996) stated that since Exposure (E) was the chemical concentration (a function of times), it could be obtained by assuming the constants chemical concentration with time. Exposure concentrations were useful when comparing peak exposure to levels of concern, such as short-term exposure limits. It was typical expressed in units such as g/m^3, mg/m^3, or mg/kg, etc. The equation to determine Exposure is the following:-

$$\mathbf{E = C\ \Delta t}$$

where

C = the average chemical concentration

Δt = the time duration of exposure

2. Intake Concentration

La Grega et al. (2001) said that intake concentration or dose, the amount absorbed by the body depended on the concentration in air, particular size distribution, bioavailability to the pulmonary system and rate of respiration. Other factors to be considered in determining the intake concentrations included life style, frequency, duration of exposure (chronic, sub-chronic or acute) and the body weight of the receptor.

Since human population were continuously in contact with various amount of environmental pollutant in air, water, food, and soil. The population-average exposure over a constant time was of interest. A generalized equation for the intake of a chemical via an exposure pathway, I, in units of mg/kg of body weight day was as follows:-

$$\mathbf{I = (C \times CR \times EF \times ED)/(BW \times AT)}$$

where

I = Intake Concentration (mg/kg·day)

C = Chemical Concentrations at the Exposure point (mg/m^3)

CR = Contact Rate (m^3/day)

EF = Exposure Frequency (days/year)

ED = Exposure Duration (years)

BW = Body Weight (average over exposure period: kg.)

AT = Average Time (days)

2.4 Integrated Pest Management Farmers

Galo and et al, (1991) said in the initiation of Integrated Pest Management Program that most environmentally persistent organochlorine pesticides have been used with decreasing frequency since 1975 as these pesticides were replaced by compounds of more capable of biodegradation to less harmful products. Since integrated pest control management involved the use of these less persistent and

more specific pesticides, organic phosphorus pesticides was the first compounds of the choice for these applications, and were later replaced by carbamate pesticides. However, the rapid biodegradation of carbamate pesticides has resulted in the present utilization of both carbamate and organic phosphorus pesticides for these purposes, with the use of organic chlorine pesticide reserved in the event of special problems.

National Integrated Pest Management Network (2000) defined Integrated Pest Management (IPM) Farmers as the farmers who used the way of farming which was socially acceptable, environmentally responsible and economically viable. IPM farmers promoted the minimum of pesticide use, but controlled the pest by biological method to enhance environmental stewardship and sustainable agricultural systems. The IPM approach aimed to create a system that at once kept the good guys alive in the fields, reduced dependence on potentially harmful pesticides, and sidestep the undesirable effects of the standard chemicals, which farmers have used for decades. At the same time, in order to work, IPM must give growers the tools to continue to farm profitably and produce the abundance of safe food, clothing, jobs and exports which we all depended on.

While U.S.EPA defined IPM that it was an effective and environmentally sensitive approach to pest management, which relied on a combination of common-sense practices. IPM programs used current, comprehensive information on the life cycles of pests and their interaction with the environment. This information, in combination with available pest control methods, was used to manage pest damage by the most economical means, and with the least possible hazard to people, property, and the environment.

Pedigo (1989) defined IPM as a comprehensive approach to pest control which utilized all available control strategies to reduce the status of pests to tolerable levels while maintaining a quality environment. The objective of this system was to reduce the use of pesticides and target them more effectively against pests.

Postal (1987) shared that Integrated Pest Management recognized a field of crops as an ecosystem within which many natural forces affected pests and weeds interaction. It drawn on biological controls such as natural predators of pest,

cultural practices e.g., planting pattern, genetic manipulations e.g., pest-resistant crop varieties, and judicious use of chemicals to stabilize crop production while minimizing hazards to health and the environmental. The operating goal was not to eradicate insects and weeds but to keep them below the level at which damaging economic losses occurred. Under this integrated approach, farmers used chemicals selectively and only when necessary, rather than as the first and primary line of attack.

IPM farmers required knowledge of a pest's life cycle, behavior, and natural enemies. The way cropping patterns and chemical use affected pest and predator population levels, and many other features of the crop ecosystem. Biological methods of pest control, either alone or as part of an IPM design, could provide some of the most elegant and long-lasting solutions to pest problems. In classical biological control, a beneficial organism was introduced into a pest-plagued area and became a permanent part of the agro-ecosystem. The pest and the introduced natural enemy reached a population balance, which kept pest damage below the economic threshold.

For the IPM program in Thailand, Pavena Supsawakul (1998) reported that IPM in Thailand was a program initiated by many governmental organizations. The Department of Agriculture, Ministry of Agriculture and Cooperation was one of the organizations, which supported the IPM program in Thailand. In 1983, Department of Agriculture initiated IPM program to reduce the toxic chemical residue in fruit and vegetable products. Moreover, this program was to control and standardize the process of fruit and vegetable production with the hygienic technology. The harvest of the project's member were from the usage of pesticide as instructed and under controlled of the agricultural technician who used the biological method such as organic substance and use less pesticide. After their cultivation passed the quality testing from the program, the specific logo (Hygienic Vegetable) would be labeled on the package and were allowed to sell in local and export to other countries.

At Tambon Bang Rieng IPM program originated to reduce the pesticide residues in the vegetable including the adverse effect from pesticide exposure in farmer. The IPM method, that farmers promoted, minimized pesticide use, but control the pest by biological method for example

1. Growing plant in the net area

2. Using Nuclear Polyhedrosis Virus (NPV)
3. Using Bacillus Thuringiensis Bacteria
4. Using earthworm as Steinernema (Neoaplectana) Carpocapsae
5. Spraying Margosa juice
6. Spraying the fermentation juice from Sherry Shell
7. Insect entrapment (light or glue), and etc.

2.5 Review of Literature:

Baker (1992) studied the prevention in reducing pesticide-related illness in the American farmers. He found that farmers needed to be educated and assessed for the potential of poisoning injury related to pesticide exposure. The studies also showed a somewhat greater risk of specific cancers in farmers using pesticides. Acute exposures and chronic conditions resulting from years of exposure to pesticides were oftentimes not attributed to pesticides.

California EPA (1999) reported while studying on exposure of methyl marathion as a toxic air contaminant, that concentrations of methyl parathion were in ambient air and in air associated with a specific pesticide from the monitoring studies. The highest 24-hour average concentration of methyl parathion total residue (methyl parathion plus methyl paraoxon) for positive samples was 2.1 ppt. as measured at Maxwell. The mean 24-hour average methyl parathion concentration for positive samples in Colusa County was 0.8 ppt. In contrast, the mean 24-hour concentration of methyl parathion reported for positive samples in Sutter County was less than 0.1 ppt.

Concentrations of methyl parathion in air reported varily between 0.5 to 688.2 ppt. Concentrations ranging from 101.8 to 688.2 ppt. have been reported in 1- to 2-hours air samples as collected immediately following application at field boundaries. These levels decreased to 1.7 to 4.7 ppt. over the six days following application. Air concentrations ranged from 0.5 to 188.5 ppt. in 12- to 24-hours air samples collected at urban and rural sites.

Air concentrations of methyl parathion were also measured during and after an application. Samples were placed approximately 20 yards from a Sutter

County rice field of approximately 80 acres. Eighty pounds of methyl parathion active ingredient were applied to this field. Samples were collected beginning at the onset of application, and then for three days. The highest average value, 47.6 ppt. was contained in the sample collected during and 1.5 hours after application. After 48 hours, methyl parathion concentrations were below 3 ppt.

California EPA (2001) studied on exposure of chlorpyrifos as a toxic air contaminant which currently had a wide range of agricultural and non-agricultural uses for the control of a variety of foliar, soil, and household pests. This document was an assessment of public exposure to ambient concentrations of airborne chlorpyrifos and chlorpyrifos oxon in the community. This evaluation was to estimate individual and public exposure to airborne chlorpyrifos. The observed chlorpyrifos concentrations were used to estimate absorbed doses for children and adults from acute and chronic exposure to chlorpyrifos. Additional studies were reviewed to assess the exposure to individuals from airborne chlorpyrifos present at the treatment site from applications made underneath and around residential structures. Airborne chlorpyrifos from these usages contribute to the ambient airborne chlorpyrifos present in the community.

The estimated absorbed dose of chlorpyrifos from a single-day exposure to off-site concentrations of chlorpyrifos from an agricultural application ranged from 11.7 μg/kg/day for a six-year-old child to 3.01 μg/kg/day for an adult female. Ambient air concentrations, while were measured in urban areas during a peak use season resulted in a daily exposure of 0.27 μg/kg/day for a six-year-old child to 0.07 μg/kg/day for an adult female. Chronic exposure (annual exposure) to chlorpyrifos in ambient air ranged from 0.03 μg/kg/day for the child to 0.01 μg/kg/day for the adult female.

Robson et al. studied "An assessment of Regulatory, Market and Financial Obstacles to Integrated Pest Management in New Jersey". This project was a pilot study to explore the reasons why more farmers did not use Integrated Pest Management for their operations. From the question of "How growers determine when to spray", the first rank of the answer from IPM respondents was that they

would spray when pest population wondered it, while the second rank was that they sprayed when they found the first sign of problem of pest.

In Thailand, there were some of the IPM and organophosphate pesticide studies as follows:-

The study of Wanwimol Pengprasith and et al. (1998), in "The Correlation of Pesticides Using of Farmers Associated with Residual Pesticides in Soil, Water and Farmer's Blood in Phayao Province" which was the basic studies in the pesticide situation of Northern Thai farmer. She said that from the blood screening test by the reactive paper to determine the acetylcholinesterase level, approximately 25.32% of farmers were at risk from exposure with the organophosphate pesticides. In addition, the acetylcholinesterase level in the traditional farmers and IPM farmers were not significant difference ($p<0.05$).

Furthermore, several researches have studied particularly on the organophosphate pesticide in the knowledge, attitude, behavior and exposure of farmers. Kriengsak Pattamarakha and et al. (1998) studied the insecticide knowledge of Songkhla farmers. They found that farmers had a good knowledge of the symptoms of plant injury and what insecticides to use, but had little knowledge on the classification of insecticides, the active ingredients, breakdown in the environment, or residual effects. Most farmers followed the recommended guidelines during application. The farmers would use insecticide mechanical physical control, nature, crude botanical extract and delayed cropping systems as alternative choices to control the problem.

Pavena Supsawatkul (1998) studied the Evaluation of Hygienic Fresh Fruit and Vegetable Production Pilot Project, which was a case study in Tambon Bang Rieng, Amphoe Khuan Nieng. to evaluate farmers' knowledge on production, their attitude and practice about hygienic vegetable in that area this was the first IPM pilot project of Department of Agriculture.

The structured questionnaires were used to collect the answer from 50 farmers who were trained by the project. She reported that farmers had moderate

knowledge on hygienic vegetable in production. Most of farmers (84%) produced hygienic vegetable utilizing pesticide in the amount as allowed by the project, and harvested vegetables after at least one week as instructed in the production principle prepared by the project. The rest (16%) produced hygienic vegetable without pesticide use. It can be concluded that the project was successful in providing farmers' knowledge and practice and producing quality of hygienic vegetables, The result were correspondent with the study of Kriensak. However, it failed in terms that farmers did not get favorable income from the production.

For the pesticide residues problem in Tambon Bang Rieng, Nongrat Klabrod (2000) studied in "Organophophorus Pesticide Residues in Soil of Agricultural Areas, Changwat Songkhla". The objective was to investigate residues of organophosphaste pesticide and the relationship between the soil and the amount of pesticide residues in three types of agricultural areas in Changwat Songkhla.

She reported on the findings of monocrotophos, dimethoate, methyl parathion, malathion and fenthion residues in the vegetable areas at Tambon Bang Rieng. The amounts of organophosphate residues were between 0.28 - 134.41 μg/kg of soil, which were collected at 20 centimeters interval with the total depth of 100 cm. The analysis of the soil properties revealed that no differences between the pH and amount of organic matter was found in different seasons. Regarding the relationship between the properties of the soil and the amount of organophosphate pesticide residues, it was found that there was a relationship between the amount of dimethoate residues and the moisture content and organic matter in the soil. It was also found that there was a relationship between the amount of methyl parathion residues and the pH, moisture content and organic matter in the soil.

Danai Tipmanee (2000) studies on "Trace Analysis organophosphate pesticide Residues in Water by Gas Chromatography". 33 the samples from the water pond and water way that passed through the vegetable area in Tambon Bang Rieng during Oct 1998 – Nov 1999, he reported that there were five oganophosphate pesticide residues in the water source which were methamidophos, monocrotophos, dimethoate, methyl-parathion and malathion. The maximum concentrations of each were 0.55, 1.01, 0.85, 1.11 and 1.24 ng/ml.

Phungphari Chaimuti (2001) studied on "Knowledge, Attitude and Behavior of Farmer on The Pesticide Application: A Case study of Vegetable Grower in Tambon Bang Rieng". She collected the questionnaires from 224 vegetable growers and semi-structured interviews were conduced on 112 farmers.

She reported that farmer had good knowledge, attitude and behavior regarding pesticide application. There was a statistically significant positive correlation at the 0.05 level between

1. Knowledge and Attitude with correlation coefficient = 0.65,
2. Knowledge and Behavior with correlation coefficient = 0.65,
3. Attitude and Behavior toward pesticide application with correlation coefficient = 0.53

Moreoverr, the study showed that farmers in Tambon Bang Rieng had good knowledge in pesticide application. However, their pesticides usage behaviors were the main causes of adverse effect form pesticide exposure in the farmers for exxample, using the high concentration and toxicity pesticide, violating the pesticide instruction and using or not using appropriate personal protective devices. In addition, the farmers didn't concern on the pesticide residues in the soil and water sources and were lack of method to dispose pesticide containers.

Pisan Pongsapitch (2002) studied on "Risk Assessment of Parathion-methyl to Thai People. The aim of this study was to be an example to promote understanding in application of risk assessment principle. He selected the parathion-methyl as a chemical in this model because of its high toxicity and large quantity usage by Thai farmer. The study followed the method and guidelines recommended by the Joint FAO/WHO Food standard Program (CODEX) and the result of parathion –methyl in the total diet studies was 0.013 μg/kg.day in 1995, which continuously decreased to 0.00022 μg/kg.day in 2000.

In summary, risk assessed by calculation method and by total diet studies showed that there was a long-term risk of parathion-methyl in food for Thai people at all levels of ages. The level and the risk from consuming rice were within acceptable level.