

EVALUATION OF THE HEALTH EFFECTS OF PESTICIDE USE AMONG LAGUNA FARMERS

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ABSTRACT

This is a preliminary study that aims to document and compare the health profiles of two groups of Laguna farmers groups of rice farmers which are socioeconomically comparable. The very striking difference between the two groups of Laguna farmers is the history of exposure to pesticides. Morbidity or the occurrence of illness or abnormalities is used to measure health status. Two health surveys were conducted, and the results seem to show that there were significant differences between the two groups in so far as the hematologic, cardiovascular and neurologic health impairments are concerned. It is to be noted that many of the Laguna farmers did not adopt adequate health safeguards. Other factors like smoking and drinking habits of both groups were also explored.

Keywords: Pesticides, organophosphates, cholinesterase, pesticide poisoning, polyneuropathy, anemia, adverse health effects, personal, protective equipment.

ADVERSE HEALTH EFFECTS OF PESTICIDES – A REVIEW OF LITERATURE

Designed to disrupt vital biological mechanisms, pesticides do not only affect target organisms but nontarget species as well, primarily man.

The adverse health effects of pesticides have been widely reported in professional literature (Lotti 1987). We are particularly interested in organophosphorous compounds which are commonly used by the farmers in the study. When inappro-

priately handled these compounds cause acute and chronic toxicity. Acute toxicity is defined as the "total adverse effects produced by a toxicant when administered as a single dose". On the other hand chronic toxicity can be defined as "total adverse effects produced by a toxicant when administered continuously over a long period of time" (Duffus 1980).

Acute Toxicity

Acute organophosphate poisoning is caused by the phosphorylation of cholinesterase, a substance normally present in the blood. Cholinesterase degrades acetylcholine, the chemical neurotransmitter, to assure the smooth firing of nerve impulses across synapses. The inhibition of cholinesterase by phosphorylation causes acetylcholine accumulation at critical sites, giving rise to clinical manifestations (Corbett 1974). Such manifestations range from non-specific signs and symptoms such nausea and vomiting in mild poisoning, the paralysis in severe cases.

Diagnosis of acute organophosphate poisoning is confirmed by measuring the cholinesterase activity in the blood. Cholinesterase depression as a biological index of toxicity is one of such indices to be proven over time (Duffus 1980). Various studies show that cholinesterase depression correlate well with the development of acute signs and symptoms (Roberts undated).

Chronic Toxicity

Although organophosphates are not known to be highly cumulative there is a group of entities that are due to chronic low dose exposure, that does not apparently affect cholinesterase levels and are therefore called nonanticholinesterase effects. These are the following:

1. Organophosphate-induced delayed polyneuropathy (OPIDN). Due to damaged myelin sheath of peripheral nerves, this clinical syndrome manifests numbness, pain and paralysis of the extremities developing days and weeks after exposure. Animal experiments have implicated phosphotriesters, cyanophosphos and leptophos. In such experiments, it has been shown that an enzyme other than cholinesterase was depressed. There appears to be no apparent relationship between acute toxicity and the likelihood of the development of a chronic neuropathic effects (Hayes 1982).

2. Chronic exposure may lead to a decrease in platelet. Thrombocytopenia or a decrease in platelet count may manifest as increased bleeding tendency. Thirteen percent of the rice farmers in Castañeda's study and 18% among pesticide formulators in Maramba's study showed thrombocytopenia (Castañeda 1987).

3. Abnormal electrocardiographic (Aldridge and Johnson 1971; Metcalf and Holmes 1969) and electromyographic findings (Jager 1976) in asymptomatic individuals exposed to chronic low doses of organophosphates.

4. There are reports of changes in the liver function tests, gastrointestinal disturbances and behavioral changes associated with chronic low dose exposure (Levin and Rodnitzky 1976; Lewis *et al.* 1973; Metcalf and Holmes 1969).

5. Hyporeflexia has been considered by some authors to be a highly sensitive indicator of chronic exposure (Roberts 1976; Roberts and Trollo 1979).

6. Anemia has been reported by Maramba in 38% of workers in formulating plants in the Philippines.

7. Other suspected but generally unconfirmed effects include those on the brain, heart and lungs. Hypertension and accelerated atherosclerosis have also been suspected health effects (Hock 1987).

SPECIFIC OBJECTIVES

1. To identify the health impairment among the farmers.
2. To determine the extent of the health problems and the factors that affect them.
3. To compare the study and control groups as to the differences in their health profile.
4. To document the acute toxic effects of organophosphorous compounds.
5. To identify potential health indices for adverse health effects due to chronic exposure.

SAMPLING

The Exposed Group

Fifty-six farmers from Cabuyao, Calauan, Biñan and Calamba comprised the study group. All 56 had a documented exposure of 15 to 25 years to organophosphorous compounds (44%) and chlorpyrifos (33%). Only 30% of the farmers wore long-t-shirts and 23% wore long pants when spraying pesticide. Thirty four percent took a bath after application.

The Unexposed Group

The control group is a group of 40 farmers from barangays Kulapi, Palola Kabatete and Kilib of Lucban, Quezon, who had never used pesticides because pest control was never been a major problem, allowing them to use natural or other cultural pest control measures. Also, their farm animals grazed near their fields.

METHODOLOGY

The study utilizes a cross-sectional survey which includes interview, physical examination, a battery of laboratory tests and exposure history. The strength of this design lies, however, in its ability to detect chronic and subacute effects. Its

weakness lies in its inherent bias for survivors, that is, the population being studied may be a healthy population. Some of the individuals who were ill may have been excluded in that one slice of time that the survey was conducted. The same set of tests were done for both the control and exposed groups. SAS computer software was used for statistical analysis.

The study was multiphasic.

PHASE 1. Exploratory Talks

A team from the Social Sciences Department of IRRI held exploratory talks with the farmers. In Biñan, Calamba and Cabuyao, there were one-on-one encounters with farmers whom we perceived to be decision makers in that area. The strategy in Calauan was different in that discussions were carried out in groups. In like manner, group discussions were held in the four barangays in Lucban, Quezon, namely Kabatete, Kulapi, Kilib and Palola.

PHASE 2. Medical Assessment

The medical assessment was carried out by a medical team consisting of a physician, nurse, x-ray technician and medical technologists. The nurse interviewed the farmers regarding their personal, family and occupational histories, including their drinking and smoking habits. The doctor performed a complete physical examination on all the farmers using a protocol specially designed for the project. Cholinesterase determinations were done by the medical technologist while chest x-ray and electrocardiogram (ECG) were done by the x-ray technician. The readings for the ECG and the x-ray were done by two cardiologists and a radiologists, respectively.

PHASE 3. Lecture on Pesticide Management

All the farmers were given a lecturette on pesticide management and on health safeguards they can quickly adopt to protect themselves.

PHASE 4. Cholinesterase Determination

Baseline cholinesterase was established for the Laguna and Lucban farmers using the Ellman method (whole blood). The average of two measurements taken not less than 3 and not more than 14 days apart at the time when there was no exposure to organophosphates was considered as "baseline". Repeat cholinesterase was done at two periods that coincide with peaks of pesticide application in Laguna.

PHASE 5. Feedback and Referrals

An immediate feedback of the results were given to the farmers and appropriate referrals to specialists were made.

PHASE 6. Documentation of Exposure

Exposure to Organophosphates was documented in terms of type, total number of applications and category of pesticides.

PHASE 7. One-Shot Survey

A one-shot survey was done to determine pesticides management practices, food and feeds taken from the paddy. Knowledge and attitudes concerning pesticide use were also recorded.

PHASE 8. Walkthrough Surveys

A quantitative evaluation of the work environment and the work processes was conducted.

RESULTS

Medical Assessment

Table 1 shows the characteristics of the exposed and unexposed group. Of the 56 farmers in the exposed group, 86% were males while in the unexposed group, 89% were males. Thirty seven per cent of the unexposed group belong to the age group 60 years and above. Seventy seven per cent of the exposed group and 70 per cent of the unexposed groups are smokers. There were more drinkers among the control group (98%) than the exposed group (59%). Forty eight percent of the exposed group users of at least one personal protective gear. Average number of pesticides applications for the past four years in 19 for the study group.

Table 1. Characteristics of the exposed and control groups.

	<i>Exposed (Laguna)</i>	<i>Unexposed (Lucban)</i>
No. of subjects	56	40
Average age (yrs.)	49	56
Age distribution:	%	%
	7	0
	25	0
	18	15
	14	30
	23	18
	13	37
Average income (1990 pesos)	22,522	19,553
Rural Health Unit	4	1
Percentage of smokers	77	70
Percentage of drinkers	59	98

Physical examination revealed that 32% of the exposed group and 45% of the control group had weights appropriate for their height and sex based on the FNRC standards. Average systole and diastole are 120 and 78 for the exposed group, respectively and 129 and 81 for the control group.

Table 2 details the abnormal findings per organ system for both the control and exposed groups. Eye findings in the exposed group were largely due to bilateral pterygium and pale conjunctive. Frequently cited (53.1%) cardiovascular abnormality was high blood pressure while decreased breath sounds (48.7%) was the frequently cited pulmonary abnormality. In comparison, the most frequently cited eye finding in the control group was due to degenerative diseases (48.57%). In the lungs, decreased breath sound (30%) was the most frequently cited abnormality.

Polyneuropathy was noted on physical examination among three farmers in the exposed group, while there was not any indication observed in the control group in spite of the fact that there were more drinkers in this group. Laboratory tests showed normal fasting blood sugar levels for all three farmers with polyneuropathy. Referral to a neurologist one month later showed more severe manifestations of polyneuropathy which was then confirmed by electrodiagnostic studies. Details are shown in Table 3. All were prescribed cyanocobalamine and Vitamin B 1-6-12 for three months and advised to refrain from pesticide application.

Forty one percent of the exposed group had abnormal ECG findings in comparison with 40% among the control group. This is not significantly different. Among those with ECG findings, 44% belonged to the 21-40 age bracket for the exposed group while only 6% for the control group (Table 4). In the 41 to the above 50 age group which are expected to have more cardiac findings, there is no significant difference between the two groups.

There is a significant difference between the exposed and the study group with regard to the hemoglobin levels. Only 5% in the control group had hemoglobin levels lower than normal as compared to 25% in the exposed group. Eighteen per cent in the control group, while only 16% in the exposed group had albumin in their urine, the difference not being statistically significant. Five among the exposed had cholinesterase levels below normal though this is not significantly different from the unexposed.

Table 5 details the abnormal findings read on ECG for both groups of farmers. The authors are well aware of the controversy regarding readings such as "non-specific ST-T changes" which may be read as "normal" by other cardiologists. Two cardiologists were requested to read the ECG and intraobserver variability was not statistically. In both groups the most frequent reading was "sinusbradycardia" which may be expected among manual workers. However, among those exposed, it is to be noted that the second most frequently cited is "nonspecific ST-T wave changes" while in the nonexposed group the second most cited finding is ischemia which may be due to the fact that this is an older age group.

Table 2. Details of abnormal findings per organ system, 56 exposed (Laguna) and 40 unexposed (Lucban) farmers.

ORGAN SYSTEM	LAGUNA			LUCBAN		
	No.	n=abnormal	n=56	No.	n=abnormal	n=40
1. Eye						
a. pterygium	15	30.00	26.79	6	17.14	15.00
b. cataract	3	6.00	5.36	1	2.86	2.50
c. dirty sclerae	3	6.00	5.36	1	2.86	2.50
d. presbyopia	9	18.00	16.07	8	31.43	27.5
e. glaucoma	1	2.00	1.79	0	0.00	0.00
f. icteric sclerae	1	2.00	1.79	0	0.00	0.00
g. pale conjunctivae	13	26.00	23.21	7	20.00	17.50
h. sluggish pupillary reflex	3	6.00	5.36	1	2.85	2.50
i. arcus senilis	1	2.00	1.79	6	17.14	15.00
j. decreased pupillary size	1	2.00	1.79	0	0.00	0.00
k. hemorrhage	0	0.00	0.00	2	5.70	5.00
2. Ear						
a. congestion	1	20.00	1.79	0	0.00	0.00
b. diminished hearing acuity	4	80.00	7.14	0	0.00	0.00
3. Nose						
a. sinusitis	1	100.00	1.79	0	0.00	0.00
4. Throat						
a. tonsillitis	8	61.54	14.29	0	0.00	0.00
b. congestion	3	23.08	5.36	2	100.00	5.00
c. pharyngitis	2	15.38	3.57	0	0.00	0.00
5. CVS						
a. murmur	2	6.25	3.57	0	0.00	0.00
b. high BP	17	53.13	30.36	2	67.00	5.00
c. low BP	13	40.63	23.21	0	0.00	0.00
d. distant heart sound	0	0.00	0.00	1	33.00	2.50
6. Lungs						
a. bilateral harsh breath sound	3	7.69	5.36	0	0.00	0.00
b. wheezing	6	15.38	10.71	3	15.00	7.50
c. rales	2	5.13	3.57	5	25.00	12.50
d. harsh breath sounds	2	5.13	3.57	0	0.00	00.00
e. decreased breath sounds	19	48.72	33.93	7	30.00	15.00
f. decreased chest expansion	6	15.38	10.71	2	10.00	5.00
g. decreased percussion	1	2.56	1.79	0	0.00	0.00
h. basal rhonchi	0	0.00	0.00	2	10.00	5.00
i. apical, bilateral	0	0.00	0.00	1	5.00	2.50
j. dec. b.s.	0	0.00	0.00	1	5.00	2.50

Table 2 (continued)

ORGAN SYSTEM	LAGUNA			LUCBAN		
	No.	Percent <i>n=abnormal</i>	<i>n=56</i>	No.	Percent <i>n=abnormal</i>	<i>n=40</i>
7. GIT						
a. LUQ tenderness	1	20.00	1.79	0	0.00	0.00
b. hepatomegaly	2	40.00	3.57	2	100.00	5.00
c. RUQ tender	2	40.00	3.57	0	0.00	0.00
8. GUT						
a. hypogastric tenderness	1	33.33	1.79	0	0.00	0.00
b. left lumbar tenderness	2	66.67	3.57	0	0.00	0.00
9. Extremities						
a. joint inflammation	1	50.00	1.79	0	0.00	0.00
b. joint pain	1	50.00	1.79	0	0.00	0.00
c. joint tenderness	0	0.00	0.00	5	83.30	12.50
d. + crepitus	0	0.00	0.00	1	16.70	2.50
10. Motor						
a. left gastrocnemius weakness	1	50.00	1.79	0	0.00	0.00
b. weakness of shoulder muscle	1	50.00	1.79	0	0.00	0.00
c. weakness of plantar flexur and dorsiflexur	0	0.00	0.00	1	50.00	2.50
d. weakness of left lower leg	0	0.00	0.00	1	50.00	2.50
11. Reflex						
a. decreased patellar reflex	14	100.00	25.00	5	83.30	12.50
b. negative L reflex	0	0.00	0.00	1	16.70	2.50
12. Sensory						
a. numbness	1	25.00	1.79	0	0.00	0.00
b. sensory deficit	3	75.00	5.36	0	0.00	0.00
13. Skin						
a. papules	7	63.64	12.50	0	0.00	0.00
b. dryness	7	63.64	12.50	4	20.00	10.00
c. erythema	1	9.09	1.79	0	0.00	0.00
d. wounds & scratches in the	1	9.09	1.79	6	30.00	15.00
e. tinea	3	27.27	5.36	10	50.00	25.00
f. eczema	1	9.09	1.79	0	0.00	0.00
g. scars	2	18.18	3.57	0	0.00	0.00

Table 3. Polyneuropathy cases

<i>ITEM</i>	<i>AGE</i>	<i>SIGNS/ SYMPTONS</i>	<i>FBS</i>	<i>ELECTRODIAG- NOSTIC TEST</i>
FARMER A	53	sensory deficit for both upper and lower extremities with associated hyporeflexia and weakness of plantar and dorsiflexors of the ankle.	5.06 (4.18-6.63)	Finding suggestive of sensorimotor polyneuropathy of the lower extremities as evidenced by the delay in the motor stimulation of the posterior tibial nerve and sensory stimulation of both sural nerves.
FARMER B	63	sensory deficit and hyporeflexia for both lower extremities	76 (N:60-100)	Findings suggestive of moderate to severe polyneuropathy. EMG findings showed denervation potentials. Motor stimulation involves both the median nerves and peroneal and tibial nerves. Sensory stimulation involves the sural serves.
FARMER C	49	numbness and paresthesia hyporeflexia and sensory deficit for both lower extremities	66 (N:60-100)	Findings are suggestive of sensorimotor polyneuropathy as evidenced by the delayed sensory nerve conduction velocity of the left common peroneal and the left posterior tibial nerves.

Table 4. Classification by age and sex with abnormal ECG findings among Laguna and Lucban farmers.

<i>AGE</i>	<i>TOTAL</i>		<i>MALE</i>		<i>FEMALE</i>	
	<i>Laguna</i>	<i>Lucban</i>	<i>Laguna</i>	<i>Lucban</i>	<i>Laguna</i>	<i>Lucban</i>
21-30	5 (22)	0	5 (100)	0	0	0
31-40	5 (22)	1 (63)	4 (80)	1 (100)	1 (20)	0
41-50	3 (13)	5 (31)	1 (33)	5 (100)	2 (67)	0
Above 50	10 (43)	10 (63)	9 (90)	1 (10)	1 (10)	1 (10)
Total	23 (41)	16 (40)	15 (94)	15 (94)	4 (17)	1 (6)

Table 5. Details of ECG findings of 56 exposed Laguna farmers and 40 unexposed Lucban farmers.

ORGAN SYSTEM	LAGUNA			LUCBAN		
	No.	Percent <i>n</i> =abnormal <i>n</i> =56		No.	Percent <i>n</i> =abnormal <i>n</i> =40	
Old anteroseptal myocardial infarction	1	3.22	1.79	0	0	0.00
Left ventricular hypertrophy	1	3.22	1.79	1	3.57	2.5
Left ventricular hypertrophy by voltage criterion	0	0	0.00	4	14.28	10.00
Probable right ventricular hypertrophy	1	3.22	1.79	0	0	0.00
LAD	0	0	0.00	2	7.14	5.00
Tendency to LAD	2	6.45	3.57	0	0	0.00
Tendency to LAD	2	6.45	3.57	0	0	0.00
Atrial flutter/fibrillation with controlled ventricular response	1	3.22	1.79	0	0	0.00
ST-T/T wave changes suggestive of inferior wall ischemia	3	9.67	5.36	0	0	0.00
Non-specific ST-T changes	6	19.35	10.71	2	7.14	5.00
Diffuse/inferior/anterolateral wall ischemia	1	3.22	1.79	6	21.43	15.00
Sinus bradycardia	8	25.8	14.29	8	28.57	20.00
Right bundle branch block (RBBB)	1	3.22	1.79	1	3.57	2.50
Widened QRS, wide slurred S at I, V4-6 T wave inversion at I, VI-4 suggestive of RBBB	1	3.22	1.79	0	0	0.00
Presence of U waves	1	3.22	1.79	0	0	0.00
Premature ventricular contractions	2	6.45	3.57	3	10.71	7.50
Sinustachycardia	0	0	0.00	1	3.57	2.50

Table 6 is a summary of the means of the laboratory findings for both unexposed and exposed groups.

The blood urea nitrogen (BUN) and creatinine, both kidney function tests, showed means that were within the normal limits for both the exposed and the unexposed groups.

SGPT is a liver function test and the means for both unexposed and exposed groups were within normal limits. Mean hemoglobin was within normal for both control and exposed groups. The mean white and red blood cell counts were both within normal limits for both unexposed and exposed groups.

Table 6. Cholinesterase and blood chemistry distribution, 56 exposed (Laguna) and 40 unexposed Lucban farmers.

Variable	Lucban	Std. Dev.	Laguna	Std. Dev.	Normal Values
Cholinesterase	4,575.00	888.76	4,978.00	829.60	3,500-4,500
BUN	12.29	3.84	9.07	4.21	6-20 mg/l
Creatinine	1.05	0.18	1.12	0.49	1-2 mg/l
SGPT	19.56	4.39	14.09	3.05	5-30 R-Fruit
Hemoglobin	14.42	1.07	13.73	1.65	13.5-7 m=gms% (M) 12-16 gms% (F)
WBC	7,908.00	1,139.87	7,224.00	1,943.71	5-10,000/cu mm
RBC	4.80	0.32	4.82	0.58	4-6 million/cumm
Platelet	197,750.00	25,164.84	271,607.00	60,265.00	175-350, 000

Cholinesterase Tests

For the exposed group, the average cholinesterase baseline activity was 4978 with the curve fitting the normal. Most of the values clustered in the middle with a few outliers, the lowest value being 2740, while the highest value being 7526. Both were not within normal limits. The average cholinesterase for the control group was 4575 which is significantly different from the exposed group.

Monitoring was done at two periods coinciding with peaks of spraying. In both monitoring periods there was a significant number of farmers who had a depression in their cholinesterase levels in the first monitoring period, 56% and in the second monitoring period, 61%. All farmers that had a depression of 30% or more in the second monitoring period were examined and all had clinical manifestations such as deminished patellar tendon reflexes, nausea, vomiting and sluggish pupillary reflexes. In comparison, 6.2% of the San Leonardo farmers in Castañeda's study had cholinesterase depression.

There was a continuous fall from baseline, first at 3.52% on peak of spraying, dry season and second at 9.9%, peak of spraying, wet season. Individual farmer graphs were plotted and all showed the same downward trend.

Smoking and Drining Factors

Among the exposed group, drinkers have a significantly higher occurrence of bronchopneumonia, significant cough, epigastric pain, vomiting ($\alpha = .01$) and headache, nausea asthmatic attacks and diarrhea ($\alpha = .05$).

Among the exposed groups, the smokers have significantly higher occurrence of diarrhea ($\alpha = .01$). There was no significant difference for the occurrence of other abnormalities in the review of systems.

Use of Personal Protective Equipment

The farmers in the exposed group who used at least one item of personal protective equipment had a lower occurrence of anemia and albuminuria ($\alpha=.05$) x-ray finding ($\alpha=.05$) double vision ($\alpha=.05$) and asthma and dyspnea at $\alpha=.05$.

Intestinal Parasitism versus Low Hemoglobin Level

There was no significant relationship between intestinal parasitism and low hemoglobin level among the exposed group ($\alpha=.01$).

CONCLUSIONS

The available literature showed that there were indeed acute and chronic effects due to organophosphorous compounds. The acute effect was reflected in the cholinesterase levels, while the chronic effects centered on the lungs, cardiovascular, hematologic and neurologic systems.

Acute Toxicity. In the exposed study, there was a depression of the cholinesterase levels at two monitoring periods. This decline during peaks of spraying could be due to the fact that a low number of farmers used appropriate health safeguards.

A comparison of cholinesterase levels for the exposed and control groups showed no significant difference. However, it was noted that there was a higher average among the exposed than the control, and this could be explained by the rebound phenomenon in which after exposure stopped, there was an increase of the cholinesterase levels sometimes as high as twice the normal level.

Chronic Toxicity. The chronic effects on the other hand are less directly affected by the environment concentration and are not directly related to the use of protective gear. Rather the chronic effects depend more on accumulation of pesticides in particular target organs over a period of time.

In this study, the following effects were noted to be significantly higher among the exposed group as compared to the control group- hematologic, neurologic and cardiovascular effects.

In summary the differences are as follows:

Exposed group	Control group	z-test
25% Low hemoglobin	5% Low hemoglobin	*
5% Polyneuropathy	No polyneuropathy	*
54% CVS on p.e.	7% CVS on p.e.	*

To establish an association between adverse health effects and a noxious substance, it is necessary to demonstrate a dose-effect relationship (Roberts and Trollope 1979). In this study, we compared two rice farmer groups whose only striking difference was their exposure to pesticides. One group never used pesticides, while the other group had been cocktail of pesticides, mostly organophosphorous compounds. Thus, the effects noted can be associated to the exposure to pesticides of one group and the absence of such exposure in the control.

In a developing country like the Philippines, it is said that concomitant diseases, intestinal parasitism, smoking and drinking make the farmer more susceptible to the effects of pesticides (Copplesstone 1985).

The study showed that indeed drinking and smoking among the exposed group aggravated the occurrence of pulmonary and gastrointestinal symptoms.

It was noted that the exposed group had not adopted adequate health safeguards against pesticide use. This is a practice seen even among Central Luzon farmers as shown by Castaneda's study (1987). This study showed that the use of at least one piece of protective clothing would protect the farmers from hematologic and pulmonary effects.

A typical farmer who uses pesticides can be characterized as a person who suffers from ups and downs of his cholinesterase levels, and is likely to be anemic, hypertensive and runs the risk of having a polyneuropathy.

A high risk farmer is one who does not use at least one form of personal protective clothing, smokes, drinks and applies pesticides 19 times a year, and does not take a shower after spraying.

It is recommended that further studies on the impact of intervention, such as training and the use of personal protective equipment be studied.

REFERENCE

- Aldridge, W.N. and M.K. Johnson.** 1971. Side effects of organophosphorous compounds: delayed neurotoxicity. *Bulletin of the World Health Organization* 44:259-263.
- Castañeda, C.P.** 1987. A study of occupational pesticide exposure among Filipino farmers in San Leonardo, Nueva Ecija. In proceedings of the Southeast Asia Pesticide Management and Integrated Pest Management Workshop, February 23-27, 1987, Pattaya, Thailand. Teng P.S. and K.L. Heong eds.
- Copplesstone, J.F.** 1985. Pesticide Exposure and Health in Developing Countries. In *Occupational Hazards of Pesticide Use*. Turnbull G.J. Taylors and Francis eds. London and Philadelphia.
- Corbett, J.R.** 1974. *The Biochemical Mode of Action of Pesticides*. London Academic Press.
- Duffus, J.H.** 1980. *Environmental Toxicology*. London: Edward Arnold Publishers, Ltd.
- Hayes, W.J.** 1982. *Pesticides Studied in Man*. Baltimore: Williams and Wilkins.
- Hock, W.K.** 1987. *Pesticide Use: The Need For Proper Protection, Application and Disposal in Pesticides (Minimizing the Risks)*. Ragsdale and R.J. Kuhr eds. Pennsylvania, U.S.A.

- Jager, K.W.** 1976. Organophosphate exposure from industrial usage, electroneuromyography in occupational medical supervision of exposed workers. In: Pesticides-induced delayed neurotoxicity. Proceedings of a Conference, 19-20 February 1976, Washington DC, US Environmental Protection Agency, (Environmental Health Effects Research Series: EPA-600/1-76-025).
- Levin, H.S. and L. Rodnitzky.** 1976. Behavioral effects of organophosphate pesticides in man. *Clinical Toxicology* 9(3): 391-405.
- Lewis, M.F. et al.** 1973. Behavioral changes from chronic exposure to pesticides used in aerial application: effects of Phosdrin on the performance of monkeys and pigeons on variable interval reinforcement schedules. *Aerospace medicine* 44 (3):290-293.
- Lotti, M.** Assessment of human exposure to pesticides in NATO Advanced Study Institute for Toxicology for Pesticides: Experimental, clinical and regulatory perspectives. Costa, L.G. C.L. Galli and S.D. Murphy eds. Springer-Verlag Berlin Heidelberg, Volume H-13, 1987.
- Metcalf, D.R. and J.H. Holmes.** 1969. EEG, psychological and neurological alterations in humans with organophosphorous exposure. *Annals of the New York Academy of Sciences* 16:357-365.
- Roberts, D. V.** (undated) Blood cholinesterase monitoring of workers exposed to organophosphorous pesticides: Theory and Practice. University of Liverpool, Liverpool, U.K.
- . 1976. E.M. G. voltage and motor nerve conduction velocity in organophosphorous pesticide factor workers. *International archives of occupational and environmental health* 36:267-274.
- and I.E. Trollope** . 1979. Nerve conduction velocity and refractory period as parameters of neurotoxicity. *Electroencephalography and clinical neurophysiology* 46:351-354.