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ARTICLE

# Epidemiology of organophosphate pesticide poisoning in Taiwan

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**Introduction.** The nationwide epidemiology of organophosphate pesticide (OP) poisoning has never been reported in detail for Taiwan. **Methods.** This study retrospectively reviewed all human OP exposures reported to Taiwan's Poison Control Centers (PCCs) from July 1985 through December 2006. **Results.** There were 4799 OP exposures. Most OP exposures were acute (98.37%) ingestions (74.50%) of a single OP (80.37%) to attempt suicide (64.72%) in adults (93.25%). Males were the most common gender (64.95%). Most patients (61.97%) received atropine and/or pralidoxime. The mortality rate for all 4799 OP exposures was 12.71%. Exposures to single OPs without co-intoxicants caused 524 deaths; of these, 63.36% were due to dimethyl OPs. **Conclusion.** Dimethyl OPs cause the majority of deaths in Taiwan.

**Keywords** Epidemiology; Organophosphate; Poisoning; Poison Control Centers; Taiwan

## Introduction

Pesticide poisoning is a worldwide problem and a common poisoning in Taiwan (1–37). The most recent published data on the epidemiology of all toxic exposures in Taiwan is from July 1985 through 1993; there were 6872 human pesticide exposures reported to the Network of Taiwan's Poison Control Centers (PCCs) (14). Pesticide exposures were the largest single category (29.3%) of human toxic exposures in Taiwan and included all types of pesticides, including insecticides, rodenticides, herbicides, etc (14). The most common specific type of pesticide exposure was to organophosphate (OP) pesticides (14). Specifically, there were 1875 OP pesticide exposures; this was 26.97% of all types of pesticide exposures (14).

This new study will focus only on OP pesticide exposures because these are the most common specific type of pesticide

poisoning in Taiwan. Carbamate insecticides are not included in this study because they are a lesser common poisoning in Taiwan, because they have different trade names in Taiwan, because they have a different chemical structure than OPs, because they bind reversibly to cholinesterases, and because carbamoylated cholinesterases do not need oximes to decarbamoylate.

A literature search was performed to see if any published study had reported the nationwide epidemiology of organophosphate poisoning in Taiwan. The literature search included the following electronic databases that were searched from their dates of inception through August 2, 2007: MEDLINE, MEDLINE In-Process & Other Non-Indexed Citations, PubMed, Cumulative Index to Nursing and Allied Health Literature (CINAHL), Cochrane Database of Systematic Reviews (DSR), Cochrane Central Register of Controlled Trials (CCTR), American College of Physicians (ACP) Journal Club, Database of Abstracts of Reviews of Effects (DARE), Toxicology Data Network (TOXNET), Toxicology Literature Online (TOXLINE), Web of Science, and International Pharmaceutical Abstracts. Each database search combined the search terms organophosphate, poisoning, and Taiwan. The searches included all languages in each

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**Table 1.** Literature search results combining the search words organophosphate, poisoning, & Taiwan

Databases	Search Periods	Search Results: Reference Numbers
MEDLINE	1950 – August 2, 2007	(38–41)
MEDLINE In-Process & Other Non-Indexed Citations and MEDLINE	1950 – August 2, 2007	(38–41)
PubMed	1950 – August 2, 2007	(38–41)
CINAHL	1982 – August 2, 2007	None
Cochrane DSR	1988 – August 2, 2007	None
CCTR	1998 – August 2, 2007	None
ACP Journal Club	1991 – August 2, 2007	None
DARE	1994 – August 2, 2007	None
TOXNET TOXLINE	1900 – August 2, 2007	(38–65)
Web of Science	1900 – August 2, 2007	(39–40)
International Pharmaceutical Abstracts	1970 – August 2, 2007	None

database. This literature search is summarized in Table 1 (38–65). This literature search found no published nationwide epidemiological study that focused on OPs, one of Taiwan's most common human toxic exposures. Therefore, this retrospective cross-sectional study describes the epidemiology of OP exposures reported to the Network of Taiwan's PCCs, from the founding of its first PCC in July 1985 through December 2006. During that time, Taiwan's population grew from 21 million to 23 million and its number of PCCs grew from one to four. Data from this study could be used to enhance allocation of public health resources to help prevent and treat OP poisoning.

There are two major classification systems for OPs. One is based on the toxicity of the OPs and the other is based on the chemical structure of the OPs. The World Health Organization's WHO Pesticide List is based on oral and dermal LD<sub>50</sub>s in rats (20,66–67). The WHO Pesticide List classifies pesticides as extremely hazardous (Class Ia), highly hazardous (Class Ib), moderately hazardous (Class II) and slightly hazardous (Class III) (20,66). This WHO Pesticide List is a common international system for classifying pesticides (20,22,66–68). The other classification system is based on the chemical structure of the OPs (3,69). Dimethyl OPs possess two methyl groups and diethyl OPs possess two ethyl groups (3,69). These chemical, structural differences are correlated with observed clinical differences (3,69). Diethyl OPs age more slowly with acetylcholinesterase than dimethyl OPs (3,69). Therefore, oximes have more time to reactivate acetylcholinesterase in diethyl OP poisoning because aging is slower between diethyl OPs and acetylcholinesterase (3,69). Another observed clinical correlation related to OP chemical structure was a higher case fatality ratio dimethyl OPs versus diethyl OPs in one study (67). To evaluate

these two OP classification systems, this study classifies OPs based on the WHO Pesticide List, as well as OP chemical structure and correlates mortality with these two classification systems.

## Methods

This study was performed with the support and acknowledgment of the Taiwan Department of Health, the oversight agency in Taiwan. This study was granted exempt status by the Human Subjects Protection Program, the Institutional Review Board of The University of Arizona. Prior to analyzing the data for this study, all patient identifying information was removed to preserve confidentiality.

This cross-sectional study retrospectively reviews all human OP exposures reported to the Network of Taiwan's PCCs from July 1985 through December 2006. Data include names and numbers of specific OPs, patient demographics, sites of callers to PCCs, chronicity of exposures, routes of exposure, reasons for exposures, therapeutic interventions, and medical outcomes. Chronicity of exposure was based on the following definitions from *Casarett and Doull's Toxicology: The Basic Science of Poisons*, "In human exposure situations, the frequency and duration of exposure are usually not as clearly defined as in controlled animal studies, but many of the same terms are used to describe general exposure situations. Thus workplace or environmental exposures may be described as *acute* (occurring from a single incident or episode), *subchronic* (occurring repeatedly over several weeks or months), or *chronic* (occurring repeatedly for many months or years)." (70). Otherwise, data were analyzed and reported using the definitions of the American Association of Poison Control Centers (15).

Descriptive statistical analyses were done with Excel software. Mortality rates were compared using a Chi-square analysis among WHO Pesticide Hazard Classes. Mortality rates were also compared using a Chi-square analysis among chemical types of OPs, e.g., dimethyl and diethyl OPs. Chi-square analysis and p-values were determined with Stata 9.2 (College Station, TX, USA).

## Results

There were 4799 human OP exposures reported to the Network of Taiwan's PCCs from July 1985 through December 2006. The case numbers of organophosphate poisoning each year are shown in Table 2. Of these, 3117 were males (64.95%), 1657 were females (34.53%), and 25 had no gender reported (0.52%).

The mean patient age and standard deviation were 46.28 ± 18.32 years. The minimum patient age was 0.1 years. The maximum patient age was 96 years. There were 101 people less than six years old (2.10%), 181 people six through

**Table 2.** The case numbers of organophosphate poisoning each year

Year	Age (years)				Total per year
	<6	6–19	> 19	Unknown	
1985			3		3
1986	1	5	35		41
1987	4	12	92	1	109
1988	4	11	158	2	175
1989	3	4	152	2	161
1990	3	11	193	5	212
1991	9	12	247	5	273
1992	13	7	189		209
1993	5	7	194	1	207
1994	2	8	157	3	170
1995	8	18	250	5	281
1996	2	17	326	7	352
1997	10	9	325	3	347
1998	3	11	339	1	354
1999	8	7	293		308
2000	4	10	304	5	323
2001	3	7	251	1	262
2002	4	5	272		281
2003	5	8	219	1	233
2004	6	5	196		207
2005	1	1	169		171
2006	3	6	111		120
Total	101	181	4475	42	4799

19 years old (3.77%), 4475 people over 19 years old (93.25%), and 42 people had no age recorded (0.88%).

Hospitals were the most common sites of callers to PCCs, with 4596 of 4799 OP exposures (95.77%) reported by healthcare professionals at hospitals. Family members reported 200 OP exposures (4.17%) from their residences. The site of the caller to the PCCs was not recorded in three OP exposures (0.06%).

The reasons for exposures are detailed in Table 3. Adults had more intentional OP exposures. Children had more unintentional OP exposures. Of the 4799 OP exposures, 4721 (98.37%) were acute, 35 (0.73%) were chronic and sub-chronic, and 43 cases (0.90%) had unknown chronicity. Ingestion was the most common route of exposure (74.50%). All routes of exposure are detailed in Table 4.

Of the 4799 OP exposures, 3857 (80.37%) involved only a single OP (Table 5). The maximum recorded number of coingestants was 9. The top five exposures to a specific, single OP were from mevinphos (18.41%), chlorpyrifos (17.60%), methamidophos (8.04%), dimethoate (5.16%), and fenitrothion (4.90%) (Table 5). The name of a specific OP could not be determined in 573 (14.86%) of 3,857 OP exposures that involved only one OP (Table 5). These 573 cases were coded as an unknown OP because the specific name could not be determined (Table 5). In all 573 cases, the symptoms, signs, and laboratory data were consistent with OP poisoning.

Of the 4799 OP exposures, 2974 patients (61.97%) received atropine and/or pralidoxime. Clinical outcomes of OP exposures in Taiwan are detailed in Table 6. The mortality rate for all 4799 OP exposures was 12.71%. The mortality rates for OP exposures with only one involved OP are detailed in Table 5.

The largest numbers of deaths (n) from exposures that involved only a single, specific OP were from mevinphos (138) [WHO Pesticide Hazard Class: Ia], methamidophos (68) [Class: Ib], dimethoate (33) [Class: II], chlorpyrifos (30) [Class: II], parathion (25) [Class: Ia], and monocrotophos (25) [Class: Ib] (Table 5) (20,66). Of these OP exposures with the largest numbers of deaths (n), four were also among the top five exposures involving only one OP (Table 5).

Of the 524 deaths caused by exposure to a single OP, 320 deaths (61.07%) were due to WHO Pesticide Hazard Class I OPs, 90 deaths (17.17%) were due to WHO Pesticide Hazard Class II OPs, 10 deaths (1.91%) were due to a WHO

**Table 3.** Reasons for OP exposures related to patient age in years

Reasons for OP exposures		Age (years)				Total	Percentage
		<6	6–19	> 19	Unknown		
Unintentional	Accidental	83	56	490	8	637	13.27%
	Environmental	4	5	28		37	0.77%
	Occupational	1	5	746	7	759	15.82%
	Misuse due to ignorance			10		10	0.21%
	Unintentional unknown	1	1	29		31	0.64%
Intentional	Suicide	4	107	2975	20	3106	64.72%
	Malicious	4	2	14		20	0.42%
	Intentional unknown		1	79	3	83	1.73%
Adverse reaction	Food contamination		1			1	0.02%
Unknown	Unknown	4	3	104	4	115	2.40%
Total		101	181	4475	42	4799	100.00%
Percentage		2.10%	3.77%	93.25%	0.88%	100.00%	

**Table 4.** Routes of OP exposures related to patient age in years

Routes of OP exposures	Age (years)				Total
	<6	6–19	> 19	Unknown	
Dermal	10	10	207	1	228
Ingestion	67	141	3337	30	3575
Ingestion +Dermal	8	1	12		21
Ingestion +Inhalation	1		6		7
Ingestion +Inhalation +Dermal			6		6
Ingestion +Inhalation +Ocular+Dermal			1		1
Ingestion +Ocular			4		4
Ingestion +Ocular+Dermal			1		1
Ingestion +Parenteral			1		1
Inhalation	7	19	436	5	467
Inhalation +Dermal	4	7	346	4	361
Inhalation +Dermal+Other			1		1
Inhalation +Dermal+Parenteral			1		1
Inhalation +Ocular			4		4
Inhalation +Ocular+Dermal			5		5
Inhalation +Parenteral			1		1
Ocular			11		11
Ocular+Dermal		1	8		9
Other			3		3
Parenteral			13		13
Unknown	4	2	71	2	79
Total	101	181	4475	42	4799

Pesticide Hazard Class III OP, one death (0.19%) was due to a WHO Pesticide Hazard Class whose active ingredients are obsolete or discontinued, and 103 deaths (19.66%) were due to OPs whose names were unknown and could not be classified within the standard WHO Pesticide Hazard Classes (Table 5) (20,66). This distribution of deaths based on WHO Pesticide Hazard Classes was statistically significant with a  $p$ -value < 0.001.

Of the 524 deaths caused by exposure to a single OP, 332 deaths (63.36%) were due to dimethyl OPs, 65 deaths (12.40%) were due to diethyl OPs, 23 deaths (4.39%) were due to mixed OPs, one death (0.19%) was due to a diphenyl OP, no death (0.00%) was due to a dipropyl OP, and 103 deaths (19.66%) were due to OPs whose names were unknown and whose chemical structure could not be classified (Table 5) (20,66,69,71). This distribution of deaths based on the chemical structures of OPs was statistically significant with a  $p$ -value < 0.001.

Of the 524 deaths caused by exposure to a single OP, 320 deaths (61.07%) were due to WHO Pesticide Hazard Class OPs and 332 deaths (63.36%) were due to dimethyl OPs (Table 5). This raised the question of how many WHO Pesticide Hazard Class I OPs are dimethyl OPs and vice versa. Of the 23 identified WHO Pesticide Hazard Class I OPs, 13 (56.52%) were also dimethyl OPs (Table 5). Of the 27 identified dimethyl OPs, 13 (48.15%) were also WHO Pesticide Hazard Class I OPs (Table 5).

## Discussion

This study used a nationwide PCC database (the Network of Taiwan's PCCs) and found that hospital healthcare providers made most calls to PCCs (95.77%) and most OP exposures were acute (98.37%) ingestions (74.50%) of a single OP (80.37%) to attempt suicide (64.72%) in adults (93.25%). Males were the most common gender (64.95%) in this study. OP exposures caused signs or symptoms of OP intoxication in 87.21% of all those exposed. Minor effects occurred in 40.28%. However, significant morbidity occurred in over a third of all OP exposures, with moderate effects in 21.03% and major effects in 13.19%. The mortality rate was 12.71 % for all 4799 patients in this study.

These findings are similar to the epidemiology of OP poisoning in other Asian countries (14,21,22,25,29,31,33,34, 36,37,39,40). The majority of published OP exposures in Asia involve acute ingestions in adult males intent on self harm (14,21,22,25,29,31,33,34,36,37,39). The mortality rate for this study is similar to the commonly reported OP poisoning mortality rate of 10% to 20% in many Asian and developing countries throughout the world (3,22,30,32).

Although OP poisoning is a significant problem in Taiwan, OPs are less of a problem in other countries of the world (26). Good, et al.'s recent article documented the 20 most common exposures resulting in inquiries to Poison Centers in 11 countries (26). Only one country, Sri Lanka, specifically listed OPs within their top 20 poisoning exposures (26). OPs were not listed in the top 20 poison exposures for those PCCs included from Australia, Germany, Iceland, Ireland, Netherlands, New Zealand, Norway, Philippines, Scotland, and the United States of America (USA) (26). OP poisoning is on the decline in the USA (23). Most OP exposures in the USA are unintentional OP exposures that do not result in mortality or significant morbidity, and do not require care in a healthcare facility (15,23).

Most OP exposure calls to PCCs in the USA are from the general public and are not from healthcare providers in healthcare facilities (15). Only 28.52% of OP exposures reported to PCCs in the USA were from healthcare providers in healthcare facilities (15). In contradistinction, 95.77% of OP exposure calls to Taiwan PCCs were from healthcare providers in hospitals. This empiric observation may be because the majority of OP exposures in Taiwan are suicidal ingestions that require EMS intervention and transportation to the hospital for care, rather than exposure advice over the phone (Table 3). This is in contradistinction to most OP exposures in the USA that are unintentional and do not require care in a healthcare facility (15,23).

In this current Taiwanese study, the top five exposures from a specific, single OP were from mevinphos (18.41%), chlorpyrifos (17.60%), methamidophos (8.04%), dimethoate (5.16%), and fenitrothion (4.90%). This compares to a Sri Lankan study where the top five specific OP exposures were to dimethoate, methamidophos, malathion, monocrotophos, and fenthion (19). Another Sri Lankan study found the top

**Table 5.** Mortality rates for patients who were exposed to only one OP

Chemical type of OP**	OP generic name	Exposures to a single OP (n)	Deaths caused by exposures to a single OP (n)	Mortality rates due to exposures to a single OP (%)	WHO Pesticide Hazard Class*
Diethyl	Chlorpyrifos	679	30	4.42%	II
Diethyl	Parathion	172	25	14.53%	Ia
Diethyl	Phorate	66	3	4.55%	Ia
Diethyl	Ethion	23	3	13.04%	II
Diethyl	Terbufos	23	1	4.35%	Ia
Diethyl	Diazinon	9	0	0.00%	II
Diethyl	Isoxathion	8	2	25.00%	Ib
Diethyl	Triazophos	5	0	0.00%	Ib
Diethyl	Pyrazophos	5	0	0.00%	II
Diethyl	Phosalone	4	0	0.00%	II
Diethyl	Pyridaphenthion	3	0	0.00%	III
Diethyl	Mephospholan	2	1	50.00%	DC
Dimethyl	Mevinphos	710	138	19.44%	Ia
Dimethyl	Methamidophos	310	68	21.94%	Ib
Dimethyl	Dimethoate	199	33	16.58%	II
Dimethyl	Fenitrothion	189	7	3.70%	II
Dimethyl	Monocrotophos	112	25	22.32%	Ib
Dimethyl	Malathion	90	7	7.78%	III
Dimethyl	Demeton-S-methyl	80	11	13.75%	Ib
Dimethyl	Parathion-methyl	78	11	14.10%	Ia
Dimethyl	Fenthion	72	11	15.28%	II
Dimethyl	Acephate	42	3	7.14%	III
Dimethyl	Omethoate	33	6	18.18%	Ib
Dimethyl	Dichlorvos	31	4	12.90%	Ib
Dimethyl	Methidathion	27	4	14.81%	Ib
Dimethyl	Phenthoate	27	3	11.11%	II
Dimethyl	Trichlorfon	14	0	0.00%	II
Dimethyl	Pirimiphos-methyl	14	0	0.00%	III
Dimethyl	Formothion	11	0	0.00%	II
Dimethyl	Phosmet	4	0	0.00%	II
Dimethyl	Azinphos-methyl	3	1	33.33%	Ib
Dimethyl	Oxydemeton methyl	3	0	0.00%	Ib
Dimethyl	Vamidothion	3	0	0.00%	Ib
Dimethyl	Naled	3	0	0.00%	II
Dimethyl	Tolclofos-methyl	2	0	0.00%	DC
Dimethyl	Thiometon	2	0	0.00%	Ib
Dimethyl	Menazon	1	0	0.00%	DC
Dimethyl	Dicrotophos	1	0	0.00%	Ib
Dimethyl	Temephos	1	0	0.00%	NC
diphenyl	Edifenphos	16	1	6.25%	Ib
dipropyl	Iprobenfos	7	0	0.00%	III
dipropyl	IPSP	1	0	0.00%	DC
Mixed: ethyl & 2,4-dichlorophenyl	Prothiophos	17	2	11.76%	
Mixed: ethyl & 4-bromo-2-chlorophenyl	Profenofos	45	1	2.22%	
Mixed: ethyl & 4-methylmercapto-3-methylphenyl	Fenamiphos	3	0	0.00%	Ib

(Continued)

Table 5. (Continued)

Chemical type of OP**	OP generic name	Exposures to a single OP (n)	Deaths caused by exposures to a single OP (n)	Mortality rates due to exposures to a single OP (%)	WHO Pesticide Hazard Class*
Mixed: ethyl & nitrophenyl	EPN	130	20	15.38%	
Mixed: ethyl & phenyl	Fonofos	1	0	0.00%	Ia
Mixed: ethyl & phenyl	Disulfoton	1	0	0.00%	Ia
Mixed: methyl & 2,5-dichloro-4-bromophenyl	Leptophos	2	0	0.00%	DC
NC	Unknown OP	573	103	17.98%	NC
Total		3857	524	13.59%	

**WHO Pesticide Hazard Class\*:**

Extremely hazardous (Class Ia)

Highly hazardous (Class Ib)

Moderately hazardous (Class II)

Slightly hazardous (Class III)

DC: Active ingredients believed to be obsolete or discontinued for use as pesticides.

NC: Not classified as one of the above by the World Health Organization (WHO).

World Health Organization (WHO). WHO Pesticide List. National Poison Control and Information Service 2003. <http://www.wpro.who.int/hse/pages/wholistpertype.html>. Accessed August 12, 2007.**Chemical type of OP\*\*:**United States National Library of Medicine. ChemIDplus Advanced. <http://chem.sis.nlm.nih.gov/chemidplus/>, accessed August 17, 2007.Eyer P. The role of oximes in the management of organophosphorus pesticide poisoning. *Toxicol Rev* 2003;22(3):165–190.

Table 6. Clinical outcome of OP exposure related to patient age in years

Outcome	Age (years)				Total	Percentage
	<6	6–19	> 19	Unknown		
No effect	33	14	149	1	197	4.11%
Minor effect	39	75	1804	15	1933	40.28%
Moderate effect	14	42	943	10	1009	21.03%
Major effect	7	23	594	9	633	13.19%
Death	3	12	588	7	610	12.71%
Unknown	5	15	397		417	8.68%
Total	101	181	4475	42	4799	100.00%
Percentage	2.10%	3.77%	93.25%	0.88%	100.00%	

five specific OP exposures were to monocrotophos, malathion, profenophos, pirimiphos, and dimethoate (35). The two most common OP exposures in a Turkish study were to methamidophos and parathion (8). The top four specific OP exposures in a Chinese study were to parathion, omethoate, dimethoate, and dichlorvos (29).

A majority of deaths caused by exposure to a single OP in this study were caused by WHO Pesticide Hazard Class I OPs (Ia Extremely Hazardous and Ib Highly Hazardous) (Table 5) (20,66). This finding is similar to other Asian OP studies (19,21). For example, the WHO Pesticide Hazard Class I OPs monocrotophos and methamidophos caused the majority of deaths in a Sri Lankan study (20). Likewise, monocrotophos accounted for the majority of OP deaths in an Indian study (22).

In this current study, 320 of 524 deaths (61.07%) were caused by exposure to a single WHO Pesticide Hazard Class

I OP (Table 5) (20,66). This suggests that controlling and limiting access to WHO Pesticide Hazard Class I OPs in Taiwan could significantly cut the number of deaths due to OP exposures. This could be an important public health initiative in Taiwan. This specific approach has been suggested by others (21,30,32,68), and was specifically tested when Sri Lanka banned WHO Class I OPs in 1995 (22). This had the desired result of decreasing the number of deaths due to WHO Class I OPs; however, the number of deaths due to WHO Class II OPs subsequently increased (22). Taiwan should consider controlling and limiting access to WHO Class I OPs.

In this current study, 332 of 524 deaths (63.36%) were caused by exposure to a single dimethyl OP (Table 5) (20,66,69,71). This suggests that controlling and limiting access to dimethyl OPs in Taiwan could significantly cut the number of deaths due to OP exposures. This could be an important public health initiative in Taiwan. This specific

approach, banning dimethyl OPs, has been previously suggested by others, but has not been done yet (67).

Another possible approach to try and cut the number of deaths from OP exposures in Taiwan would be controlling and limiting access to both WHO Pesticide Hazard Class I OPs and dimethyl OPs. Results from using this combined approach have not been reported in the literature. This could be an important public health initiative in Taiwan. Taiwan should consider controlling and limiting access to both WHO Class I OPs and dimethyl OPs.

Eddleston et al. found higher case fatality ratios for the dimethyl OPs dimethoate (23.1%) and fenthion (16.2%) than for the diethyl OP chlorpyrifos (8.0%) (67). This current study confirms Eddleston et al.'s observation (67) by also finding higher case fatality ratios for the dimethyl OPs dimethoate (16.58%) and fenthion (15.28%) than for the diethyl OP chlorpyrifos (4.42%).

WHO Pesticide Hazard Class I OPs are not necessarily dimethyl OPs and dimethyl OPs are not necessarily WHO Pesticide Hazard Class I OPs. About 50% of the 23 identified WHO Pesticide Hazard Class I OPs are dimethyl OPs. Specifically, 13 (56.52%) of the 23 identified WHO Pesticide Hazard Class I OPs in this study were also dimethyl OPs and 13 (48.15%) of the 27 identified dimethyl OPs were also WHO Pesticide Hazard Class I OPs (Table 5).

## Limitations

This study is a retrospective analysis of prospectively collected information in a PCC database and suffers from the weaknesses of all retrospective studies, including reporting bias, recall bias, selection bias, and possible confounders that cannot be controlled for in retrospect. This study uses PCC data and the name of a specific OP could not be determined in all cases (Table 5). The identities of the OPs in this study were based on the history. This study uses PCC data that do not necessarily include every OP poisoning in Taiwan, because reporting to PCCs is voluntary.

## Conclusion

WHO Pesticide Hazard Class I OPs and dimethyl OPs cause the majority of OP poisoning deaths in Taiwan.

## References

1. Bardin PG, Van Eeden SF. Organophosphate poisoning: grading the severity and comparing treatment between atropine and glycopyrrolate. *Crit Care Med* 1990; 18:956–960.
2. Balali-Mood M, Ayati MH, Ali-Akbarian H. Effect of high doses of sodium bicarbonate in acute organophosphorous pesticide poisoning. *Clin Toxicol* 2005; 43:571–574.
3. Buckley NA, Eddleston M, Szinicz L. Oximes for acute organophosphate pesticide poisoning. *Cochrane Database Syst Rev* 2005;CD005085.

4. Karalliedde L, Baker D, Marrs TC. Organophosphate-induced intermediate syndrome: aetiology and relationships with myopathy. *Toxicol Rev* 2006; 25:1–14.
5. Nhachi CF. Organophosphate poisoning and management, an update. *Cent Afr J Med* 2001; 47:134–136.
6. Srivastava A, Peshin SS, Kaleekal T, Gupta SK. An epidemiological study of poisoning cases reported to the National Poisons Information Centre, All India Institute of Medical Sciences, New Delhi. *Hum Exp Toxicol* 2005; 24:279–285.
7. Zhang J, Zhao J. [Progress in the treatment of acute organophosphate poisoning]. *Zhonghua Yu Fang Yi Xue Za Zhi* 1999; 33:248–249.
8. Aygun D. Diagnosis in an acute organophosphate poisoning: report of three interesting cases and review of the literature. *Eur J Emerg Med* 2004; 11:55–58.
9. Eddleston M, Szinicz L, Eyer P, Buckley N. Oximes in acute organophosphorus pesticide poisoning: a systematic review of clinical trials. *QJM* 2002; 95:275–283.
10. Rousseau JM, Ruttimann M, Brinquin L. [Acute neurotoxic organophosphate poisoning: insecticides and chemical weapons]. *Ann Fr Anesth Reanim* 2000; 19:588–598.
11. Horowitz BZ, Giffin S, Thomsen CL. Pesticide-related illness: are poison centers reporting to the state health department? *Vet Hum Toxicol* 2002; 44:49–51.
12. Davanzo F, Settini L, Faraoni L, Maiozzi P, Travaglia A, Marcello I. [Agricultural pesticide-related poisonings in Italy: cases reported to the Poison Control Centre of Milan in 2000–2001]. *Epidemiol Prev* 2004; 28:330–337.
13. Kalkan S, Erdogan A, Aygoren O, Capar S, Tuncok Y. Pesticide poisonings reported to the drug and poison information center in Izmir, Turkey. *Vet Hum Toxicol* 2003; 45:50–52.
14. Yang CC, Wu JF, Ong HC, Hung SC, Kuo YP, Sa CH, Chen SS, Deng JF. Taiwan National Poison Center: epidemiologic data 1985–1993. *J Toxicol Clin Toxicol* 1996; 34:651–663.
15. Lai MW, Klein-Schwartz W, Rodgers GC, Abrams JY, Haber DA, Bronstein AC, Wruk KM. 2005 Annual Report of the American Association of Poison Control Centers' national poisoning and exposure database. *Clin Toxicol* 2006; 44:803–932.
16. Leveridge YR. Pesticide poisoning in Costa Rica during 1996. *Vet Hum Toxicol* 1998; 40:42–44.
17. Hu SC, Wang LM. [Study of patients arriving by ambulance in Taipei City]. *J Formos Med Assoc* 1993; 92 (suppl 1):S25–32.
18. Hu SC, Tsai J, Kao WF, Chern CH, Yen D, Lo HC, Chang CH, Chern TL, Lee CH. [Three years' experience of emergency medical services in Ilan County]. *J Formos Med Assoc* 1995; 94 (suppl 2):S87–93.
19. Karalliedde L, Senanayake N. Acute organophosphorus insecticide poisoning in Sri Lanka. *Forensic Sci Int* 1988; 36:97–100.
20. International Programme on Chemical Safety. The WHO recommended classification of pesticides by hazard and guidelines to classification. Geneva, Switzerland: World Health Organization; 2000–02.
21. Srinivas Rao C, Venkateswarlu V, Surender T, Eddleston M, Buckley NA. Pesticide poisoning in south India: opportunities for prevention and improved medical management. *Trop Med Int Health* 2005; 10:581–588.
22. Roberts DM, Karunaratna A, Buckley NA, Manuweera G, Sheriff MH, Eddleston M. Influence of pesticide regulation on acute poisoning deaths in Sri Lanka. *Bull World Health Organ* 2003; 81:789–798.
23. Blondell JM. Decline in pesticide poisonings in the United States from 1995 to 2004. *Clin Toxicol* 2007; 45:589–592.
24. Leverton K, Cox V, Battershill J, Coggon D. Hospital admission for accidental pesticide poisoning among adults of working age in England, 1998–2003. *Clin Toxicol* 2007; 45:594–597.
25. Aygun D, Doganay Z, Altintop L, Guven H, Onar M, Deniz T, Sunter T. Serum acetylcholinesterase and prognosis of acute organophosphate poisoning. *J Toxicol Clin Toxicol* 2002; 40:903–910.
26. Good AM, Kelly CA, Bateman DN. Differences in treatment advice for common poisons by poisons centres—an international comparison. *Clin Toxicol* 2007; 45:234–239.



27. Ai P, Kaiyuan Z, Xinhua L, Changbin L, Buckley N, Roberts D. Extracorporeal blood purification for organophosphorus pesticide poisoning. (Protocol). Cochrane Database of Systematic Reviews 2006; CD006253.
28. Roberts D, Buckley NA. Alkalinisation for organophosphorus pesticide poisoning. Cochrane Database Syst Rev 2005;CD004897.
29. He F, Xu H, Qin F, Xu L, Huang J, He X. Intermediate myasthenia syndrome following acute organophosphates poisoning—an analysis of 21 cases. Hum Exp Toxicol 1998; 17:40–45.
30. Eddleston M, Phillips MR. Self poisoning with pesticides. BMJ 2004; 328:42–44.
31. Munidasa UA, Gawarammana IB, Kularatne SA, Kumarasiri PV, Goonasekera CD. Survival pattern in patients with acute organophosphate poisoning receiving intensive care. J Toxicol Clin Toxicol 2004; 42:343–347.
32. Buckley NA, Karalliedde L, Dawson A, Senanayake N, Eddleston M. Where is the evidence for treatments used in pesticide poisoning? Is clinical toxicology fiddling while the developing world burns? J Toxicol Clin Toxicol 2004; 42:113–116.
33. Eddleston M. Patterns and problems of deliberate self-poisoning in the developing world. QJM 2000; 93:715–731.
34. Fernando R. The National Poisons Information Centre in Sri Lanka: the first ten years. J Toxicol Clin Toxicol 2002; 40:551–555.
35. Van der Hoek W, Konradsen F, Athukorala K, Wanigadewa T. Pesticide poisoning: a major health problem in Sri Lanka. Soc Sci Med 1998; 46:495–504.
36. Eddleston M, Sheriff MH, Hawton K. Deliberate self harm in Sri Lanka: an overlooked tragedy in the developing world. BMJ 1998; 317:133–135.
37. Litchfield MH. Estimates of acute pesticide poisoning in agricultural workers in less developed countries. Toxicol Rev 2005; 24:271–278.
38. Tsai JR, Sheu CC, Cheng MH, Hung JY, Wang CS, Chong IW, Huang MS, Hwang JJ. Organophosphate poisoning: 10 years of experience in southern Taiwan. Kaohsiung J Med Sci 2007; 23:112–119.
39. Lin CL, Yang CT, Pan KY, Huang CC. Most common intoxication in nephrology ward organophosphate poisoning. Ren Fail 2004; 26:349–354.
40. Hsieh BH, Deng JF, Ger J, Tsai WJ. Acetylcholinesterase inhibition and the extrapyramidal syndrome: a review of the neurotoxicity of organophosphate. Neurotoxicology 2001; 22:423–427.
41. Wu YQ, Wang JD, Chen JS, Chung SC, Hwang SY. Occupational risk of decreased plasma cholinesterase among pesticide production workers in Taiwan. Am J Ind Med 1989; 16:659–666.
42. Lin TJ, Jiang DD, Chan HM, Hung DZ, Li HP. Prognostic factors of organophosphate poisoning between the death and survival groups. Kaohsiung J Med Sci 2007; 23:176–182.
43. Tsai CY, Wu CH, Chan SH, Chang AY. Muscarinic receptor-independent activation of cyclic adenosine monophosphate-dependent protein kinase in rostral ventrolateral medulla underlies the sympathoexcitatory phase of cardiovascular responses during mevinphos intoxication in the rat. Shock 2007; 27:559–564.
44. Chan JY, Chan SH, Dai KY, Cheng HL, Chou JL, Chang AY. Cholinergic-receptor-independent dysfunction of mitochondrial respiratory chain enzymes, reduced mitochondrial transmembrane potential and ATP depletion underlie necrotic cell death induced by the organophosphate poison mevinphos. Neuropharmacology 2006; 51:1109–1119.
45. Tsai MH, Tsai NW, Chen SF, Tsai HH, Lu CH, Huang CR, Chang WN. Organophosphate intoxication-related coital-like involuntary movements: report of a case. Acta Neurol Taiwan 2006; 15:34–37.
46. Lee F, Lin JL. Intermediate syndrome after organophosphate intoxication in patient with end-stage renal disease. Ren Fail 2006; 28:197–200.
47. Li FC, Tseng HP, Chang AY. Neuroprotective role of coenzyme Q10 against dysfunction of mitochondrial respiratory chain at rostral ventrolateral medulla during fatal mevinphos intoxication in the rat. Ann N Y Acad Sci 2005; 1042:195–202.
48. Yen DH, Chan JY, Huang CI, Lee CH, Chan SH, Chang AY. Coenzyme q10 confers cardiovascular protection against acute mevinphos intoxication by ameliorating bioenergetic failure and hypoxia in the rostral ventrolateral medulla of the rat. Shock 2005; 23:353–359.
49. Wu ML, Deng JF, Tsai WJ, Ger J, Wong SS, Li HP. Food poisoning due to methamidophos-contaminated vegetables. J Toxicol Clin Toxicol 2001; 39:333–336.
50. Yen DH, Yen JC, Len WB, Wang LM, Lee CH, Chan SH. Spectral changes in systemic arterial pressure signals during acute mevinphos intoxication in the rat. Shock 2001; 15:35–41.
51. Yen DH, Yien HW, Wang LM, Lee CH, Chan SH. Spectral analysis of systemic arterial pressure and heart rate signals of patients with acute respiratory failure induced by severe organophosphate poisoning. Crit Care Med 2000; 28:2805–2811.
52. Liu WF. Effects of cholinesterase inhibitors on a two-component chained schedule performance in rats. Neurotoxicol Teratol 2000; 22:389–396.
53. Satoh T, Hosokawa M. Organophosphates and their impact on the global environment. Neurotoxicology 2000; 21:223–227.
54. Wang AG, Liu RS, Liu JH, Teng MM, Yen MY. Positron emission tomography scan in cortical visual loss in patients with organophosphate intoxication. Ophthalmology 1999; 106:1287–1291.
55. Lee WC, Yang CC, Deng JF, Wu ML, Ger J, Lin HC, Chang FY, Lee SD. The clinical significance of hyperamylasemia in organophosphate poisoning. J Toxicol Clin Toxicol 1998; 36:673–681.
56. Wang MH, Tseng CD, Bair SY. Q-T interval prolongation and pleomorphic ventricular tachyarrhythmia (“Torsade de pointes”) in organophosphate poisoning: report of a case. Hum Exp Toxicol 1998; 17:587–590.
57. Sheu JJ, Wang JD, Wu YK. Determinants of lethality from suicidal pesticide poisoning in metropolitan HsinChu. Vet Hum Toxicol 1998; 40:332–336.
58. Chuang CC, Wang ST, Yang CC, Deng JF. Clinical experience with pendimethalin (STOMP) poisoning in Taiwan. Vet Hum Toxicol 1998; 40:149–150.
59. Chuang FR, Jang SW, Lin JL, Chern MS, Chen JB, Hsu KT. QTc prolongation indicates a poor prognosis in patients with organophosphate poisoning. Am J Emerg Med 1996; 14:451–453.
60. Hsiao CT, Yang CC, Deng JF, Bullard MJ, Liaw SJ. Acute pancreatitis following organophosphate intoxication. J Toxicol Clin Toxicol 1996; 34:343–347.
61. Fang TC, Chen KW, Wu MH, Sung JM, Huang JJ. Coumaphos intoxications mimic food poisoning. J Toxicol Clin Toxicol 1995; 33:699–703.
62. Jang SW, Lin JL, Chuang FR. Electrocardiographic findings of organophosphate intoxication in emergency department as predictors of prognosis: a retrospective analysis. Changgeng Yi Xue Za Zhi 1995; 18:120–125.
63. Tseng FY, Chen CS. [Thyroid function tests in acute drug intoxication]. J Formos Med Assoc 1992;91 (suppl 1):S68–73.
64. Liu WF. A symptomatological assessment of organophosphate-induced lethality in mice: comparison of atropine and clonidine protection. Toxicol Lett 1991; 56:19–32.
65. Tsao TC, Juang YC, Lan RS, Shieh WB, Lee CH. Respiratory failure of acute organophosphate and carbamate poisoning. Chest 1990; 98:631–636.
66. World Health Organization (WHO). WHO Pesticide List. National Poison Control and Information Service 2003, August 12, 2007, <http://www.wpro.who.int/hse/pages/wholistpertype.html>.
67. Eddleston M, Eyer P, Worek F, Mohamed F, Senarathna L, von Meyer L, Juszczak E, Hittarage A, Azhar S, Dissanayake W, Sheriff MH, Szinicz L, Dawson AH, Buckley NA. Differences between organophosphorus insecticides in human self-poisoning: a prospective cohort study. Lancet 2005; 366:1452–1459.
68. Eddleston M, Karalliedde L, Buckley N, Fernando R, Hutchinson G, Isbister G, Konradsen F, Murray D, Piola JC, Senanayake N, Sheriff R, Singh S, Siwach SB, Smit L. Pesticide poisoning in the developing world—a minimum pesticides list. Lancet 2002; 360:1163–1167.
69. Eyer P. The role of oximes in the management of organophosphorus pesticide poisoning. Toxicol Rev 2003; 22:165–190.
70. Eaton DL, Klaassen CD. Principles of toxicology. In: Klaassen CD, ed. Casarett & Doull's Toxicology: The Basic Science of Poisons. 6th ed. New York: McGraw-Hill; 2001: 14.
71. United States National Library of Medicine, ChemIDplus Advanced, August 17, 2007, <http://chem.sis.nlm.nih.gov/chemidplus/>