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OCCUPATIONAL AND ENVIRONMENTAL PESTICIDE STUDY IN SOUTH FLORIDA

Health Effects Research Laboratory
Office of Research and Development
U.S. Environmental Protection Agency
Research Triangle Park, North Carolina 27711

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OCCUPATIONAL AND ENVIRONMENTAL PESTICIDE EXPOSURE STUDY IN SOUTH FLORIDA

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U.S. ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF RESEARCH AND DEVELOPMENT
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RESEARCH TRIANGLE PARK, N.C. 27711

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4. Page 60 - Appendix A: 3,5,6-TCP (under "Phenols") should be 3,5,6-TC Pyridinol

5. Page 68 - Attached is a new page 68. The 4th line of the abstract was incorrectly left out from the original version.

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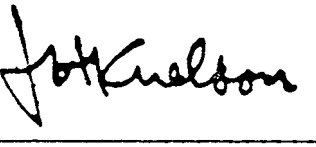
FOREWORD

The many benefits of our modern, developing, industrial society are accompanied by certain hazards. Careful assessment of the relative risk of existing and new man-made environmental hazards is necessary for the establishment of sound regulatory policy. These regulations serve to enhance the quality of our environment in order to promote the public health and welfare and the productive capacity of our Nation's population.

The Health Effects Research Laboratory, Research Triangle Park, conducts a coordinated environmental health research program in toxicology, epidemiology, and clinical studies using human volunteer subjects. These studies address problems in air pollution, non-ionizing radiation, environmental carcinogenesis and the toxicology of pesticides as well as other chemical pollutants. The Laboratory develops and revises air quality criteria documents on pollutants for which national ambient air quality standards exist or are proposed, provides the data for registration of new pesticides or proposed suspension of those already in use, conducts research on hazardous and toxic materials, and is preparing the health basis for non-ionizing radiation standards. Direct support to the regulatory function of the Agency is provided in the form of expert testimony and preparation of affidavits as well as expert advice to the Administrator to assure the adequacy of health care and surveillance of persons having suffered imminent and substantial endangerment of their health.

This investigation was designed to assess the occupational and environmental exposure to pesticides in South Florida, an area where pesticides are widely used. In addition to the conventional approach for measuring exposure to organophosphates and carbamates by means of cholinesterase determination, emphasis is given to the study of metabolites of pesticides in urine, the analysis of pesticide residues in samples of adipose tissue, and the monitoring of air for pesticide pollution. Attention is focused on the diagnostic and epidemiologic potential of certain biologic and environmental indices, such as the urinary metabolite profile and the concentration of pesticides in air, which are important with regard to the occupational health and safety of the worker and the protection of the general population against incidental exposure to pesticides.



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PREFACE

OCCUPATIONAL AND ENVIRONMENTAL PESTICIDE EXPOSURE STUDIES IN SOUTH FLORIDA

Possessing a semi-tropical climate and with a year round growing season Florida is second only to California in pesticide usage. Pesticides are widely used in agriculture, with citrus, fruits and vegetables being the chief agricultural products grown. They are also widely used by public health for vector borne disease control and for the control of nuisance pests; additionally, various types of different pesticides are used by homeowners for interior pest control and horticultural purposes. It is not surprising, therefore, that high priority has been given to human pesticide safety and protection of the environment of Florida.

In the past decade, a community pesticide study in this area amply demonstrated that there are a wide variety of real and potential human health hazards which are due to the widespread use of pesticides. The organophosphates and carbamates are widely used, causing acute systemic poisonings, and illnesses which are due to topical effect of these pesticides on the skin and in the eye. Ethyl parathion and mevinphos (Phosdrin) are the two most toxic pesticides used by agriculture. They have caused numerous incidents of serious pesticide poisonings. Currently these are seen predominately in the agricultural worker, especially those workers occupationally exposed to these two more toxic materials.

Today, over 60% of the pesticides used in agriculture in Florida are applied by aerial application, and systemic poisonings have been identified in two different types of occupational categories. The first category has been the pesticide applicator, and includes people who formulate, mix, load and apply pesticides, and the second occupational category is the "picker" group which is composed of a sizeable occupational work force whose jobs require picking and thinning of crops on a seasonal basis. Poisonings in these two groups are sometimes called "applicator poisoning" and "picker poisoning" respectively. Workers in the former category, are at some time or other exposed to varying degrees of the pesticide concentrate. Pesticide mixers and loaders have been found to be the group with the most exposure and at greatest risk of sustaining systemic poisoning. The clinical picture is a cholinergic illness exhibiting the symptomatology of parasympathetic stimulation. This is the result of cholinesterase inhibition in the central nervous system which is confirmed in the laboratory by the demonstration of significant inhibition of the red blood cell and plasma enzymes. Inhibition of these enzymes usually precedes the appearance of an acute cholinergic illness and this measurement is used as a surveillance tool of the pesticide applicator since the test has shown to be a reliable predictor of cholinergic poisoning. Laboratory tests which are predictive are of major importance to the occupational health and safety of the worker.

In contrast, picker poisoning, which is also occasionally seen in this area, involves workers who pick and thin the crops. The exposure is primarily dermal and is the result of contact with the diluted

pesticide residues on the leaf of the particular crop. The illness which results from this is milder than that which is seen in the pesticide applicator.

Both the applicator and the picker are also prone to dermatitis. This results either because of the irritant properties of the chemical or is more commonly due to the allergic makeup of the individual. These topical lesions are not dose related, and therefore, the magnitude of the pesticide exposure is not a primary factor, the specific allergic potential of the individual being the most important variable. This health effect has not been studied in this project.

Since continued pesticide usage is essential for modern agricultural technology, occupational health is vitally concerned with the protection of the exposed workers and with the development of a variety of strategies which are designed to minimize this exposure. The efficacy of these interventions are measured in terms of reduction in the amount of pesticide exposure and through ongoing studies of cholinesterase surveillance of the workers, especially those workers who are at special risk from working with the more toxic pesticides. Cholinesterase surveillance, the mainstay of present pesticide occupational surveillance, has many limitations and at times is inadequate, non-specific, and unpredictable. Over the last decade, increased interested has been shown in surveillance by the urinary metabolites. Human and animal studies have demonstrated that these urinary metabolites are excellent measures of exposure to the organophosphate and carbamate pesticides. The potential of these metabolites as a surveillance instrument has been further stimulated by the significant developments in analytical methodologies.

Two types of metabolites are identified following exposure to the organophosphate and carbamate insecticides. These are the alkyl phosphates and phenolic metabolites, and both are excreted rapidly in the urine following exposure. With organophosphate exposure, the alkyl phosphate and phenolic metabolites are the major urinary metabolites, and the phenols are the major metabolites of carbamates.

Urinary alkyl phosphates (dimethyl phosphates (DMP), diethyl phosphates (DEP), dimethyl thio phosphates (DMTP) and diethyl thio phosphates (DETP) appear as metabolites after exposure to the inorganic organophosphorus pesticides. The urinary dimethyl phosphate and diethyl phosphate are metabolites of the oxon of the intact pesticides. These metabolites can result from exposure to the oxidation products of the thio organophosphate pesticides, to the oxon, or to organophosphates not containing any sulfur such as DDVP and phosdrin. It is a known fact that oxons are toxic and inhibit the cholinesterase more than the thio organophosphate pesticides.

To summarize therefore, the major alkyl phosphate metabolites which can be identified following human exposure to the organophosphates are the dimethyl thiophosphates (DMTP), diethyl thiophosphates (DETP) and the dimethyl (DMP) and diethyl (DEP) alkyl phosphates.

The phenolic metabolites reflect the nitro and chlorophenol moieties of the insecticides. These two types of urinary metabolites are also usually excreted rapidly in the urine. Being rapidly excreted they should prove to be usable indices of recent exposure.

The other major group of pesticides of epidemiological interest are the organochlorine insecticides. These are stored in fat and persistent in man as well as in the environment. Their concentrations in adipose tissue therefore is another biologic index of exposure, being reflective of long term exposure. The epidemiologic potential is greatest in studies measuring long term exposures.

Today, man is vulnerable to three types of pesticide exposures; acute, chronic, and incidental. Acute exposure is an over exposure in which the illness develops shortly after the over exposure and is reflective of the known toxicity of the offending pesticide. Chronic exposure is the exposure of the pesticide worker, that is usually high and is acquired by working day after day either in the manufacturing, formulation, mixing, handling or application of pesticides. The population at risk is the occupationally exposed. Incidental exposure is the exposure of the general population; it is ubiquitous and man receives small amounts of pesticides through trace amounts of residues found in air, water, food and fiber of his day-to-day living. The magnitude of such exposures can be measured either by determining the residues stored in fat and hair or excreted in the urine of the population at large or can be quantitated by measuring pesticides in the air, water, food and clothing. The latter residues are environmental indices of pesticide exposures.

The aforementioned strategies have been the basis of our occupational and environmental exposure studies in South Florida. The urinary metabolites have been the biologic indices used to measure acute, occupational and incidental exposure of man to organophosphate and carbamate pesticides. The human pesticide residue profile has been the biologic index used to appraise occupational and incidental exposure to the organochlorines in this area. Our environmental assessment of pesticides has focused on concentrations of pesticides in air and these have been the environmental index used to appraise the environmental impact of pesticides in South Florida.

In conclusion, it should be emphasized that all the studies included in this report have been conducted under natural occurring events. Exposures have been measured as they have occurred and nothing artifical has been introduced. Specific dose testing is the more precise way to correlate biologic responses, conclusions being formulated on the basis of observational data acquired in the field.

ABSTRACT

In the past, accurate expressions of human pesticide exposures and environmental pesticide contamination have suffered from interpretative difficulties which have stemmed from the use of biologic indices that were indirect and nonspecific measures of human exposure (cholinesterase inhibition) and environmental indices which lacked precision due to analytical shortcomings. Our studies of the urinary pesticide metabolites have demonstrated that the alkyl phosphates are highly sensitive expressions of recent human exposures to the organophosphate group of pesticides. Urine phenolic metabolites are both sensitive and specific indicators of recent individual organophosphate and carbamate exposures. In addition, the modification of the analytical procedures for pesticides in air and in fat have greatly enhanced the breadth and reliability of estimates of pesticides in the air and have facilitated the recognition of the less polar organophosphate insecticides in human fat.

Because of the sensitivity of our modified methodology, the urinary alkyl phosphates were found to be ideal diagnostic tools of acute pesticide poisonings and epidemiologic measures of subtle and minute differences in organophosphate exposures, a property which lends itself to human monitoring of incidental organophosphate exposure and to epidemiologic studies of the occupationally exposed worker. Because of a lack of specificity for individual organophosphate exposures, however, the alkyl phosphates did not appear to be satisfactory surveillance instruments of the occupationally exposed workers when cholinesterase inhibition or illness was the disease end point of primary concern, and when exposures were to a mixture of pesticides whose toxicities varied considerably. Only in occupational exposures to a single pesticide, particularly to those that have a high toxicity, can these urinary metabolites be used as a surveillance instrument. The usefulness of the alkyl phosphate studies to recognize minute organophosphate exposures permitted the recognition of important and unrealized sources of worker exposure which stemmed from concentrations of pesticides in the air of the work environment and in worker's clothing.

The recognition of bromophos, a fat soluble organophosphate, in the fat of three of the five formulators emphasized the continuing need for human monitoring with newer methodologies to encompass the less polar organophosphates as well as the organochlorine pesticides.

With improvements in air monitoring procedures, the major source of pesticide contamination in air was found to be in areas where pesticides are indiscrimately mixed and loaded in the field and inside the formulating plant. In other areas of South Florida, aerial contamination of pesticides was minimal.

FIGURES

FIGURES - Continued

TABLES

TABLES - Continued

ACKNOWLEDGEMENTS

The investigators acknowledge with gratitude the cooperation and assistance offered us to conduct these studies by Ag-Air Flying Service, Allied Helicopter Service Inc., Tri-State Dusting Service, U.S. Department of Agriculture, Dade County Parks Department, Woodbury Chemical Company, and Jackson Memorial, Hialeah, Glades General and Baptist Hospitals as well as the several volunteers representative of the general population for their willing cooperation and participation to provide blood, urine and adipose samples for appropriate cholinesterase, urinary metabolites and adipose residue studies.

In addition, the investigators at the University of Miami appreciate the advice and guidance provided by Drs. Thomas Scotti and William Durham, and Mr. Jack Thompson and Mr. Frank Wilinski from Research Triangle Park, North Carolina, and Dr. T. M. Shafik and Miss Anita Peoples, both members of our Department.

List of Abbreviations and Symbols

α BHC	alpha benzene hexachloride
β BHC	beta benzene hexachloride
γ BHC	gamma benzene hexachloride
ChE	cholinesterase
DDVP	dichlorvos
Δ pH/hr	delta pH per hour
DEP	diethyl phosphate
DETP	diethyl thiophosphate
DMDTP	dimethyl dithiophosphate
DMP	dimethyl phosphate
DMTP	dimethyl thiophosphate
EC	electron capture detectors
Et. parathion	Ethyl parathion
evap	evaporate
FDA	Food and Drug Administration
FPD	flame photometric detector
GLC	gas liquid chromatography
HCl	hydrochloric acid
HCB	hexachlorobenzene
Hep. Epox.	heptachlor epoxide
m^3	cubic meter
MDA	malathion di-acid
MeCl	methylene chloride
Me. parathion	methyl parathion
mg/kg	milligram per kilogram
ml	milliliter

ml/Osm/l	milliliter per milliosmols per liter
MMA	malathion mono-acid
Na_2SO_4	sodium sulfate
NH_4Cl	ammonium chloride
ND	not detected
ng/m^3	nanogram per cubic meter
PCP	pentachlorophenol
PNP	paranitrophenol
ppb	parts per billion
ppm	parts per million
µg/ml	microgram per milliliter
vs.	vs.

SECTION I

INTRODUCTION

Descriptive epidemiologic studies of South Florida have shown that there are many serious health problems with present pesticide management practices (1,2). Use pattern surveys in the same area have shown that large amounts of pesticides are used (3,4) but what is not known is the true magnitude of the consequences of such exposures in terms of human and environmental effects. The data which have already been collected are generally descriptive and anecdotal and more precise information is needed.

Reliable biological and environmental expressions of this contamination will provide the information which is urgently sought. Insofar as humans are concerned, biologic indices are the preferred instruments for epidemiological appraisal of the situation. For environmental studies, air sampling has reached a high degree of sophistication and is the preferred environmental index of exposure. If these separate indices are both sensitive and specific, health and environmental effects of pesticides can be reliably quantitated.

In the first two years of this project, our studies of acute poisoning cases and occupational exposure studies had suggested that urinary alkyl phosphate and phenolic metabolites were sensitive indicators of worker exposure to certain organophosphates and carbamates (5,6). Significant inverse correlations of cholinesterase levels with alkyl phosphate urinary metabolites were demonstrated under worker exposure to 6-3 ethyl methyl parathion mixtures and mevinphos. DMTP and DETP, and DMP and DEP were the four alkyl phosphate metabolites identified in these exposures. Both quantitative and qualitative information was provided from the alkyl phosphate data. Concentrations of DEP of >0.4 ug/ml were observed in all of the first urines of seven poisonings with ethyl-methyl parathion and these urinary concentrations were in striking contrast to the levels of DEP observed in asymptomatic occupationally exposed workers, where of 99 urines only 1 had concentrations greater than 0.4 ug/ml. The DEP/DETP ratio was also very highly informative because the mean of this ratio for these two metabolites was 4.14 in poisoning cases compared to 0.88 in occupationally exposed workers. This, therefore, was evidence of one biologic index that promised to be a reliable indicator of acute and occupational exposure of the agricultural worker. Being rapidly excreted from the body, the metabolites provide retrospective information on recent exposures especially acute poisoning. Being also sensitive measures of minimal pesticide exposure it was planned to test their reliability in intervention studies designed to limit worker exposure and also to measure the incidental exposure of the general population in the area.

Earlier studies had also demonstrated that a fat soluble organophosphate compound could be detected in human fat in a poisoning case (7).

Finally, earlier monitoring studies had also demonstrated significant differences in air concentrations of pesticides, suggesting that pesticides in air would be a most useful way of expressing environmental air pollution and appeared to be the best environmental index to quantitate the environmental effects of pesticides in this area.

The studies described hereinafter have been designed to expand these earlier findings with the intent of testing the urinary pesticide metabolites as diagnostic and surveillance instruments and exploring their potential as epidemiologic instruments in organophosphate and carbamate exposures. Additionally, adipose residue studies have been conducted to determine occupational and general population exposures and to find out whether any organophosphates were now being stored in fat; air sampling of pesticides has been continued in order to determine environmental exposure to pesticides in this area.

SECTION II

CONCLUSIONS

This year these occupational and environmental pesticide studies in South Florida have sought to measure pesticide exposure by testing the urinary pesticide metabolites as indices of recent exposure and the adipose residue as indices of the more chronically sustained exposures particularly those acquired from organochlorine pesticides. The environmental assessment of pesticides has been based mainly on air sampling of pesticides in different sample sites selected on the basis of being reflective of low, average and high pesticide usage.

The urinary metabolites have been used to measure human exposures in acute poisonings, in the occupationally exposed, and in the general population, in order to test (1) the diagnostic potential of the pesticide metabolites in acute poisoning, (2) the surveillance potential of the metabolites where surveillance has been equated with the assessment of the predictive potential for cholinesterase depression or illness, and (3) the epidemiologic evaluatory potential of the metabolites in exposures to the organophosphate group rather than to a single pesticide.

The diagnostic potential in acute poisoning was amply demonstrated by a series of case studies wherein sequential urines were analyzed during the early phase of the poisoning. If the organophosphate exposure which caused the illness was due to a single pesticide, especially if the pesticide was metabolized to alkyl phosphate and phenolic metabolites, then highly specific diagnostic laboratory data were obtained. If, however, the poisoning was the result of a mixture of organophosphates then the identification of the causal pesticides was impossible since the urinary alkyl phosphate metabolites are group specific (dimethyl or diethyl) rather than pesticide specific.

The initial concentrations of the urinary pesticide metabolites in poisoning were related to (1) the chemical configuration of the pesticide, (2) its metabolism and toxicity (LD_{50}) and (3) the dose and route of absorption. Of these several variables influencing initial urinary concentrations of metabolites, it was found that the dose and toxicity were the most important. The dialkyl phosphates, reflective of the oxon appeared to be more specifically related to cholinergic illness, than did the dithio urinary derivatives. For example, in 32 of 40 urines analyzed from seven poisonings cases due to ethyl parathion, DEP concentrations were higher than DETP.

In the study of these metabolites in the occupationally exposed, the same problems with mixed exposures were encountered. Almost all workers were exposed to a variety of pesticides whose toxicities varied considerably.

The urinary alkyl phosphates while being highly sensitive indicators of organophosphate exposures, lacked specificity for individual organophosphate pesticides, making it impossible to uniformly predict cholinesterase inhibition illness on the basis of a single urinary alkyl phosphate concentration. Thus, with unknown organophosphates, surveillance was impossible. The only exception to this was with workers working with ethyl parathion or mevinphos where cholinesterase depression might be anticipated. Based on findings of these studies, the urinary metabolites could be used as survellance instruments and DEP concentrations greater than 0.9 ug/ml with parathion, or DMP concentrations greater than 1.0 ug/ml in cases of mevinphos exposure, would be regarded as dangerous levels justifying temporary withdrawal of the worker from exposure to these particularly toxic members of the group.

In contrast to specificity, however, sensitivity of these indices was remarkable, often facilitating the identificiation of minute exposures from the environment or from contaminated clothing which was not recognized by the worker himself. It was concluded that the urinary alkyl phosphates were very reliable epidemiologic instruments of organophosphate exposures capable of identifying subtle differences in groups of workers and able to measure small incidental exposure of the general population to these pesticides in the environment.

The adipose pesticide residues showed that (1) fat soluble organophosphate storage was occurring in the formulators, and (2) that there is significant worker exposure from pesticide concentrations to clothing (8) and pesticides in the air of worker's environment. Many of the organochlorine pesticides identified in adipose residues had not been recently formulated but the identification of these in the air of the work environment was the major factor contributing to the continued exposure of these obsolescent pesticides.

In the air sampling studies it was concluded that pesticides were minimal in the suburban and urban areas but higher concentrations were found in the work environment.

SECTION III

RECOMMENDATIONS

Based upon our studies, the following additional steps are recommended:

1. Verification of alleged pesticide poisoning incidence by confirmatory urinary metabolite data.

2. Testing selected applicator surveillance programs by urinary metabolite surveillance when highly toxic pesticides are being formulated or applied.

3. Urgent research on the role of contamination of worker's clothing is needed, making use of the urinary metabolite data to measure this contamination, and residues in the clothing itself, and investigating the potential of treating clothing with fluorocarbon as well as laundering efficacy.

4. Fat soluble organophosphate insecticide studies - it is recommended that environmental human health effects of these less polar insecticides be further investigated.

5. Monitoring programs - The prevalence of incidental exposure of the population at large to organophosphates and carbamates needs expanded research and the sources of these pesticides and their metabolites in food warrant further study.

6. Urinary alkyl phosphate data should always form part of the epidemiologic studies which measure organophosphate exposures. Thus, re-entry studies, new formulation studies, etc. should all be evaluated on the basis of urinary metabolite data.

7. Air monitoring of pesticides should be continued in South Florida especially now that open-air burning of containers is being planned.

SECTION IV

MATERIALS AND METHODS

1. Urinary Pesticide Metabolite Studies

a. Acute poisoning cases - Arrangements were made with physicians and nurses in the Emergency Rooms and Intensive Care Units of Jackson Memorial Hospital, James Archer Smith Hospital, Hialeah Hospital and Belle Glades General Hospital to call us as soon as a pesticide poisoning case was admitted to the hospital. A program was instituted whereby all urines voided during the first two or three days were collected in hexane washed jars, labelled, and frozen at 5° C for later urinary metabolite determinations. 10 cc of blood were collected in a heparinized tube and 10 cc of clotted blood were also collected; the former for red blood cell and plasma cholinesterase determinations and the latter for intact pesticides. Subsequent hospital and field visits were made by project personnel to review and document the clinical findings and to determine the mechanism of the poisoning and the most probable route of absorption. Sequential urines were obtained from eleven cases of systemic poisonings due to organophosphate pesticides. Seven of these poisonings were due to ethyl parathion and all were the result of dermal exposures; four were the result of occupational exposure to 10% ethyl parathion granular formulation and three were the result of occupational exposure to 6% ethyl parathion mixutres. Three cases of mevinphos (Phosdrin) poisoning were similarly investigated but only one is presented in this report, and two cases of chlorpyrifos (Dursban) poisoning due to ingestion of the insecticide and one case of malathion poisoning also due to ingestion are included. All patients were hospitalized shortly after becoming sick so that the initial urine voided almost certainly represented the first voided specimen after developing overt illness.

b. Occupational studies: (a) Pesticide application and loading exposure studies - Two mixer loaders (A and B), one with normal cholinesterase levels (A) and one with significant cholinesterase depression (B), agreed to provide all voided urine specimens over a continuous work period of between 4 and 5 days. These specimens were collected in hexane washed jars, and the date and time of voiding noted. The volume of the urine voided was measured and the specimen frozen to be analyzed at a later date for alkyl phosphate and phenolic metabolites. Prior to analysis, urine osmolality determinations were made using the Fiske osmometer (9) and urinary alkyl phosphates were corrected to a standard osmolality of 800 ml/Osm/l.

(b) Pesticide pickers and foliar residue exposure studies - "Picker poisoning" is not a serious problem in South Florida and in order to explore the potential of protective clothing in the field in the prevention of foliar residue exposure, the Department of Epidemiology and Public Health conducted a field study in El Salvador. This research was an independent project not supported by the U.S. Environmental Protection Agency, however, these activities afforded an opportunity to investigate the sensitivity of the urinary alkyl phosphate metabolites as an epidemio-

logic instrument in these studies. These urinary metabolite data, therefore, were included in these studies in order to throw some light on the overall goals of the E.P.A. supported studies concerned with occupational and environmental pesticide exposure studies.

Twenty seven agricultural laborers picking cotton in El Salvador were asked to participate in a protective clothing project where each would be provided with new sets of long-sleeved shirts, denim pants, cotton socks and heavy leather shoes purchased in a local market. Fourteen workers were given clothing which had been pretreated by spraying with a fluorocarbon solution provided by the 3M Company. Earlier laboratory studies had shown that penetration was reduced when a cloth was treated with a fluorocarbon solution and then sprayed with a 10% xylene dye extract. Thirteen workers were given new but untreated clothing.

The Ministries of Health and Agriculture had requested this field study and participated in the field testing of the protective clothing. The nature of the study was explained to each of the volunteers and signed consent forms obtained.

During the six day study period, urines were collected consecutively at 12 hour intervals in hexane washed bottles. Aliquots were frozen and later analyzed by the Miami Pesticides Laboratory. Osmolality was measured by the Fiske osmometer (9). Urines with osmolar concentrations under 100 ml/Osm/l were excluded under the assumption that they were not valid specimens. All values of alkyl phosphates at or below the limit of detectability were treated as zero in all calculations.

The fields were hot and dry, and temperatures during the six days ranged from 63^{o} F at night to 80^{o} F during the day. The fields had been sprayed from the air with mixtures of ethyl and methyl parathion five days before the study began. Chlordane, toxaphene and methomyl were also in use.

c. General Population Studies: (a) Urinary alkyl phosphates and phenolic metabolite studies - Grab samples of urine were collected in hexane washed jars, each specimen was labelled with the name, age, race, sex and occupation of the participant. These were analyzed for alkyl phosphate and phenolic metabolite data and the concentration of the metabolite corrected to a uniform osmolality of 800 ml/Osm/l.

(b) Orange juice feeding study - In order to determine whether orange juice ingestion was a source of incidental general population exposure to the organophosphates, two volunteers agreed to ingest one quart of orange juice for three days and to provide all urines voided between 9:00 a.m. and 4:00 p.m. Pre-exposure urines were collected over the same time period for two days before ingestion and for two days after ingestion. Pre and post cholinesterase determinations were made in both volunteers. The orange juice was analyzed for alkyl phosphates.

The analytical procedures used for these several studies for the alkyl phosphates was the Shafik et al method (10) which has been modified and is described under "Experimental Procedures" and for the phenolic metabolites the Shafik et al method (11). For the malathion mono and di-acid

metabolites the Shafik and Bradway method (12) was used. The Michel method (13) was used for the cholinesterase determinations.

2. Adipose Pesticide Residue Studies

a. Occupational exposure studies (formulators) - Adipose body burdens of pesticides together with environmental and biological surveys were studied in a group of five formulators working in a formulating plant.

After explaining the nature and purpose of this study to the workers a signed consent form was obtained from each, agreeing to participate in this study and agreeing to provide a single blood, fat and urine sample in order to measure the total body burden of pesticides being sustained in this type of occupation.

The five formulators who wore respirators, rubber gloves, boots, rubber aprons and coveralls which were changed daily, mixed and formulated a wide variety of pesticides from 8:00 a.m. to 12 noon and then from 1:00 p.m. to 5:00 p.m.; they worked 5½ days per week.

J.P., the plant supervisor has worked in the formulating plant for 15 years and A.P., the senior and most experienced formulator has worked in the plant for 18 years. T.W. and J.T. have been employed for three years and E.W. (T.W.'s brother) has worked only four months.

The plant where the men work is a 40 x 200 ft. concrete block building with a high pitched steel corrugated roof. The walls are 8 ft. high and there are 6 - 12 ft. doors; 3 on each wall to provide cross ventilation. Additional ventilation is supplied by 2 - 4 ft. exhaust fans near the peak of the ceiling structure. The offices are located in the north end of the building; and approximately three quarters of the area is occupied by columns of 55 gallon pesticide drums, cardboard and paper packages containing granular and powdered mixtures of different types of pesticides. The only formulation done takes place at the south end of the building. Here, there are two closed-system formulation vats for liquid pesticides with listed capacities of 500 and 165 gallons each. In addition, there is a semi-automatic roller mixer for granular and powdered formulations which mixes 50 cubic feet.

Storage of the formulated products consisting of drums and bags are as described above with center lane thorough fares with side aisles; all materials are placed on pallets.

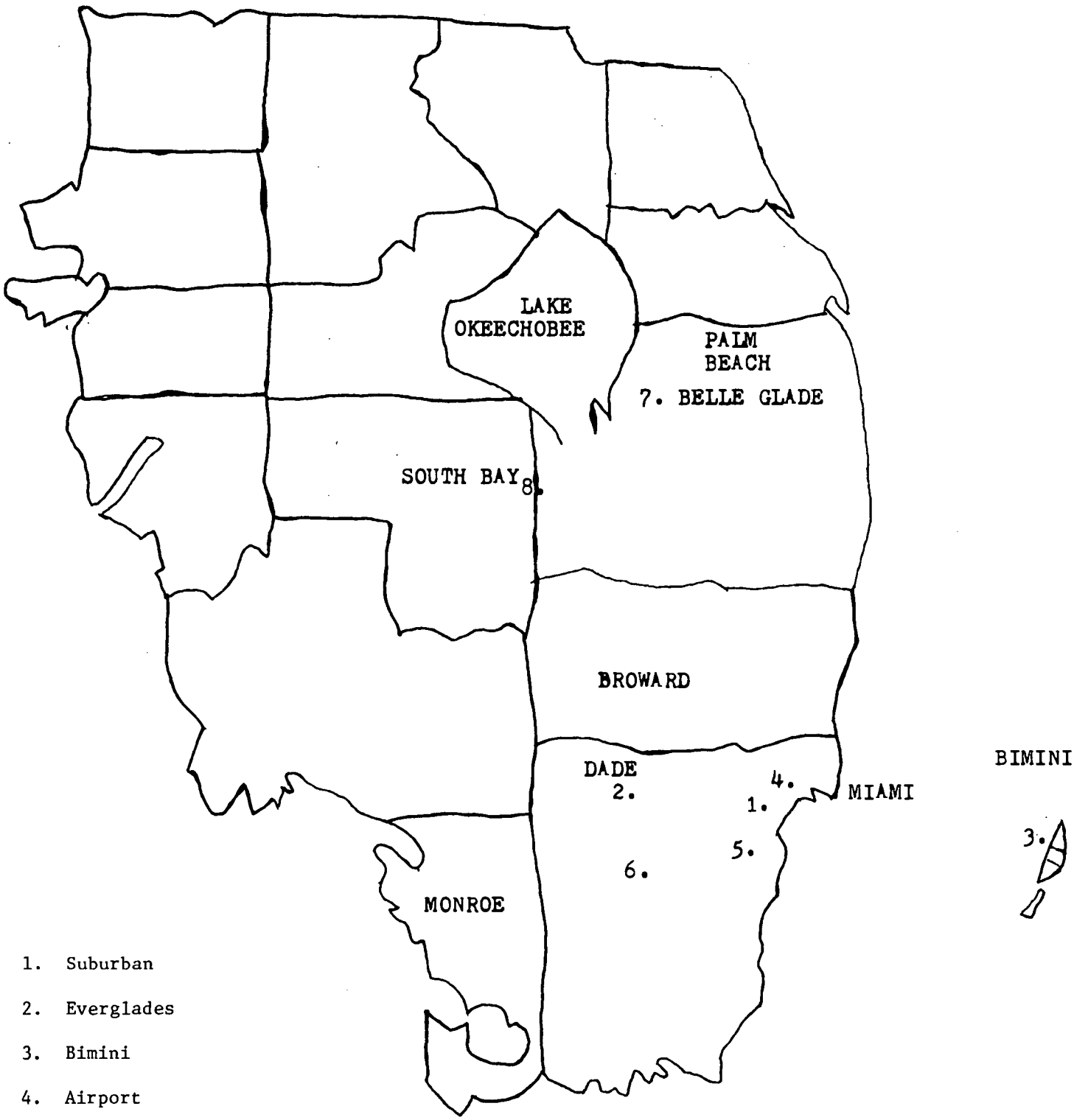
Between 1 and 2 gms of fat were obtained from the gluteal region from each of the volunteers under aseptic conditions and under local anesthetic by a board certified surgeon at the University of Miami Hospitals and Clinics. The specimens were placed in hexane washed jars with aluminum foil screw top caps, frozen and later analyzed by gas chromatography using electron capture and flame photometric detectors for organophosphate pesticides.

b. General population adipose studies - 20 surgical biopsy specimens from a random group of patients coming to surgery at Jackson Memorial Hospital Dade County, Florida were obtained. Between 1 and 2 grams of fat were collected from the surgical site, placed in hexane washed jars, frozen and later analyzed.

The Sherma and Shafik (14) method was modified for organochlorine and organophosphate determinations and is listed under "Experimental Procedures." The urines were analyzed by the Shafik et al. (10) method and cholinesterase determinations were analyzed by the Michel (13) method.

3. Air Monitoring Studies

Forty air monitoring samples for pesticides were analyzed and some of the selected sites are shown in Figure 1. Twenty four hour samples were collected using an M.R.I. air sampler equipped with four impingers charged with 400 ml of ethylene glycol. Each set of impingers was used for 12 hours to collect the 24 hour air sample. The standard F.D.A. (15) procedure has been modified by extracting the ethylene glycol with methylene chloride and chromatographed on a silica gel column. A modification of the Sherma and Shafik method (14) for analysis of pesticides in air was used and modified as described in "Experimental Procedures."



1. Suburban
2. Everglades
3. Bimini
4. Airport
5. Formulating Plant
6. Aerial Applicator
7. Aerial Applicator (H)
8. Storage Shed

Figure I. Selected air sampling sites in south Florida

SECTION V

EXPERIMENTAL PROCEDURES

1. Urinary Alkyl Phosphate Methods Development

The measurement of alkyl phosphates in urine using an amberlite resin was modified to enable the analysis to be performed rapidly, efficiently and accurately.

Urine is diluted with acetone and collected on an Amerlite CG400 resin. This resin is placed in a culture tube containing .05 ml 6 NHCl and 1 ml acetone. After 30 minutes 3 ml of diazopentane is added and the derivatization is complete in one hour. The diazopentane derivaties are extracted with hexane and are concentrated to 1 ml for injection into the gas chromatograph equipped with an FPD detector. An OV-210 column that has been treated with carbowax allows for definition of all alkyl phosphates in one sample.

Urine was spiked with 0.5 ppm, 0.1 ppm and 0.05 ppm alkyl phosphates, diluted with acetone, concentrated on the Amberlite resin and derivatized with diazopentane.

Recovery of alkyl phosphates from urine:

	DMTP	DETP	DMP	DEP
0.05 ppm	64%	82%	80%	90%
0.1 ppm	63%	84%	80%	91%
0.5 ppm	68%	78%	86%	80%

This technique proved useful and practical as 30 urine samples could easily be analyzed per day.

Urinary inorganic phosphates are also derivatized by diazopentane and while they do not interfere with alkyl phosphate peaks they do slow down the analysis by showing up as very large and very late peaks.

At least 10 ml of urine were made alkaline with KOH in order to precipitate inorganic phosphates in the urine. After centrifugation, this urine was made acid with HCl and a complex was formed with the remaining phosphate ions by the addition of ammonium molybdate. This complex was precipitated by safranin and the resultant supernatent was distilled with acetone and poured through resin as usual. These procedures resulted in a large decrease of inorganic phosphates but the results varied with different urines probably because of the amount of phosphate present in the urine due to dietary habits.

A problem arose concerning the reagent, diazopentane or more specifically the nitroso guanidine. A reagent blank had also shown a few interfering peaks but posed interpretive problems with concentrations just above the limits of detectability.

After much correspondence and communication with the guanidine source, we decided to use another derivative, and are presently using 3-benzyl-1-O-tolytriazine. Preliminary results are very promising. There is a possibility of having a "one tube" analysis and no interference with inorganic phosphates.

2. Adipose Pesticide Residue Studies - Partitioning for chlorinated and organophosphate pesticide in adipose tissue

The Sherma and Shafik method (14) for pesticides in air was modified for combined chlorinated and organophosphate pesticide residue studies in adipose tissues. Two hundred milligrams of fat were weighed and extracted three times with 2.5 ml of acetonitrile in a #22 Duall homogenizer. Each extract was transferred to a 45 ml centrifuge tube. The acetonitrile extract was evaporated to a volume of 4 ml to which 25 ml of 2% NA2SO4 solution was added and mixed in a Vortex mixer. This was extracted with one 5 ml and two 2 ml portions of methylene chloride, transfering each extract to the same 13 ml centrifuge tube. The extract was concentrated to dryness with a keeper solution (1% paraffin oil in hexane) and 0.5 ml of hexane was added and then subjected to silica gel column chromatography cleanup.

In the Sherma and Shafik method, the column was washed with 10 ml of hexane and 0.5 ml of the sample was transferred to the column to obtain the first fraction. The second fraction was separated by adding 15 ml of 60% benzene in hexane, and the third fraction was obtained by adding 15 ml of 5% acetonitrile to benzene. The four fraction was extracted with a 15 ml solution of 50% methylene chloride and benzene.

The lower limits of detectability of this modified method were: p,p'-DDT 0.05; p,p'-DDE 0.02; o,p'-DDT 0.04; oxychlordane 0.02; HCB 0.01; BHC 0.02; heptachlor epoxide 0.01; dieldrin 0.019; ronnel 0.06; methyl parathion 0.09; ethyl parathion 0.12; trithion 0.23; ethion 0.12; chlorfenthion 0.05; chlorpyrifos 0.06; diazinon 0.06; and malathion 0.19 ug/ml.

3. Air Monitoring Studies - Modification of the Sherma and Shafik method

The standard F.D.A. procedure (15) for extracting and analyzing pesticides in air involves extraction of ethylene glycol with hexane, fluorosil column chromatography and collection of three fractions. Each fraction contains the pesticides shown in Table 1. The number of pesticides that could be analyzed by the F.D.A. or M.O.G. procedure were limited by the solubility of hexane and those pesticides which could be eluted from the fluorosil column. Table 2 illustrates the new modifications developed by

Thompson and Reed at Research Triangle Park, N.C., and Shafik, Barquet and Morgade at the University of Miami Pesticides Research Laboratory. Here, the ethylene glycol is extracted with methylene glycol, chromatographed on a silica gel column which permits the collection of four fractions. As shown in Table 2, the number of pesticides which can be analyzed greatly increased when this modified was used. The lower limits of detectability of these pesticides are shown in Table 3. The limits ranged from 0.1 - 2 ng/m^3 for chlorinated pesticides; 0.2 - 2.5 ng/m^3 for organophosphate compounds and 3 - 16 ng/m^3 for carbamates.

Table 1. Essential steps of F.D.A. procedure showing the pesticides identified in three fractions

100 ml ethylene glycol = 20 m^3 air/6 hrs

↓ 350 ml 2% Na_2SO_4
↓ 3 x 60 ml hexane

Combined hexane extracts

↓ Wash 2 x with 50 ml H_2O
↓ Pass through 2 inch x 22 mm id Na_2SO_4 column
↓ Evaporate to 3 ml

22 gm florisil column chromatography
4 inch x 22 mm id

Fraction I		Fraction II	Fraction III
200 ml 5% E-PE		200 ml 15% E-PE	200 ml 50% E-PE
5.0 ml/ul; OV-17/QF-1 and SE-30/QF-1; EC		5.0 ml/5 ul; OV-17/QF-1 and SE-30/QF-1; EC	1.0 ml/ 5 ul; SE-30/ QF-1; FPD
α-BHC	p,p'-DDE	Dieldrin	Malathion
β-BHC	o,p'-DDE	Endrin	
γ-BHC	Perthane	Thiodan	
Lindane	p,p'-DDD	Thedion	
Heptachlor	o,p'-DDT	2,4-D esters	
Ronnel	p,p'-DDT	2,4,5-T esters	
Aldrin	Ethion		
Hep. Epox.	Chlordane	1.0 ml/5 ul; SE-30/QF-1; FPD	
o,p'-DDE			
Chlorobenside		Diazinon	
		Ethyl parathion	
		Methyl parathion	

Table 2. Additional pesticides identified in four fractions by the modified method described

100 ml ethylene glycol = 20 m^3 air/6 hrs

↓ 600 ml 2% Na_2SO_4
40 ml $MeCl_2$

$MeCl_2$ extract
Wash 3 times
Evap. to dryness

↓

Silica gel chromatography: 1 g. 20% H_2O

Fraction I		Fraction II		Fraction III		Fraction IV
10 ml Hexane		15 ml 60% benzene in hexane		15 ml 5% CH_2CN in benzene		15 ml 50% $MeCl_3$ in acetone
α BHC	HCB	γ BHC	Trithion	Diazinon	Carbofuran	Diazinon
Aldrin	Heptachlor	β BHC	Et. parathion	Malathion	Matacil	Dimethoate
p,p'-DDE	Chlorbenside	Hep. Epox.	Ethion	Phosdrin	Zectran	Phosphamidon
o,p'-DDT	α Chlordane	Dieldrin	Dursban	DEF	Carbaryl	
p,p'-DDD	γ Chlordane	Endrin	Dichlofenthion	Baygon	Mesurol	
p,p'-DDT	Toxaphene	Methoxychlor	Fenthion	Landrin	Dichlorvos	
		Endosulfan I	Phorate			
		Endosulfan II	Bromophos			
		Ronnel	Dioxathion			
		Me. Parathion				

Table 3. Detectability limits (ng/m^3) for ethylene glycol impinger using silica gel column[a]

α BHC	0.1	Heptachlor	0.1	Chlorpyrifos	0.3
Aldrin	0.2	HCB	0.1	Dichlofention	0.2
p,p'-DDE	0.3	Chlorbenzide	0.7	Phorate	0.1
o,p'-DDT	0.6	Methoxychlor	3.0	Me. Bromophos	0.7
p,p'-DDD	0.8	Endosulfan	0.2	Mevinphos	0.5
p,p'-DDT	0.9	Thiodan II	0.2	DEF	1.4
γ BHC	0.1	Ronnel	0.3	Fenthion	0.9
Δ BHC	0.2	Me. Parathion	0.6	Dioxathion	2.5
Hep. Epox.	0.3	Et. Parathion	0.8	Carbofuran	10.0
Dieldrin	0.3	Trithion	2.0	Carbaryl	16.0
Endrin	0.7	Ethion	1.0	Mesurol	4.0
α Chlordane	0.2	Diazinon	0.2	Propoxur	3.0
γ Chlordane	0.3	Malathion	1.0	Landrin	4.0
β BHC	1.0				

(a) Based on GLC peaks giving 10% full scale deflection (electron capture), or a signal to noise ratio 4:1 (flame photometric) above the background of reagent blanks.

Toxaphene was identified but could not be quantitated because it had 12 peaks in the chromatogram

SECTION VI

RESULTS AND DISCUSSION

A. Results

1. Acute Pesticide Posioning Studies

The objective here is to determine the diagnostic potential of the urinary alkyl phosphates. The major types of urinary metabolites detected in the urine of poisoning cases will vary with the chemical composition of the pesticide. Thus, with ethyl parathion, the diethyl phosphate (DEP) and the diethyl thiophosphate (DETP) and paranitrophenol (PNP) are the major alkyl phosphate and phenolic metabolites identified. With chlorpyrifos (Dursban) the diethyl alkyl phosphates, DEP, DETP and the phenol 3,5,6-trichlorophenol (3,5,6-TCP) are the major metabolites excreted; and with mevinphos (Phosdrin) the dimethyl phosphate (DMP) is the major metabolite observed. In the case of malathion, DMP, DMTP and DMDTP, the alkyl phosphate metabolites of this pesticide are the minor metabolites, and malathion mono-acid and di-acid metabolites are the major metabolites recognized. The thio derivatives are usually reflective of the parent compound and the dialkyl phosphates are reflective of the oxon. Table 4 lists the pesticides and the major metabolites identified in some of the poisoning cases and in occupational workers described in this report.

(a). Poisoning by diethyl organophosphate insecticides - Figure 2 shows the excretion pattern of DEP and DETP in four patients poisoned by dermal exposures to 10% ethyl parathion granular formulations and Figure 3 illustrates the excretions of some of the metabolites in three patients exposed to 6% ethyl parathion mixtures. Metabolite concentrations were greater in the 10% exposure than in the 6% exposures, the initial concentritions of DEP ranged from 8.1 to 0.9 in these seven poisoning cases. Initial DEP concentrations were greater than DETP in five of the seven cases, and in 32 out of 40 urines analyzed in this series suggesting the significant contribution of the oxon to these intoxications. Figures 4 and 5 illustrate the excretion of DEP, DETP and 3,5,6-TCP in two children who were poisoned as a result of ingestion of an unknown amount of chlorpyrifos. It will be noted that the initial urinary concentrations of DEP were 30 - 20 ug/ml respectively and were of a much greater order of magnitude than seen in cholinergic illness due to ethyl parathion; emphasizing the importance of the dose and the LD50 of the organophosphate from which the alkyl phosphate was metabolized rather than the absolute level of the DEP itself. Here too, in these cases, for the most part, DEP concentrations exceeded the DETP concentrations.

The significance of the physical-chemical properties of the pesticide causing the poisoning on the duration of metabolite excretion is exemplified by a dichlofenthion (VC-13) poisoning case reported in our annual

Table 4. Pesticides and their major metabolites identified in some of the poisoning cases and described in this report.

Pesticides	Metabolites	Oral LD_{50} (mg/kg)	Dermal LD_{50} (mg/kg)
A. Diethyl organophosphates - Alkyl Phosphates			
Ethyl parathion	DEP, DETP	3 - 33	21
Chlorpyrifos	DEP, DETP	135	2,000
Dichlofenthion	DEP, DETP	270	6,000
Diazinon	DEP, DETP	100 - 150	900
B. Dimethyl organophosphates - Alkyl Phosphates			
Mevinphos	DMP	7	5 - 33
Methyl parathion	DMP, DMTP	9 - 25	67
Dichlorvos (DDVP)	DMP	56 - 80	75 - 107
Dimethoate	DMP, DMTP, DMDTP	215	
Naled (Dibrom)	DMP	430	1,100
C. Organophosphates - Phenols			
Ethyl, methyl parathion	PNP		
Chlorpyrifos	3,5,6-TCP		
Dichlofenthion	2,4-DCP		
D. Other pesticides - Phenols			
Pentachlorophenol	PCP	125 - 210	
Carbaryl	α naphthol	560	4,000
Lindane and its isomers	2,4,6-TCP, 2,4,5-TCP, 2,3,4,6-TCP, 2,3,4,5-TCP and 2,3,5-TCP	125	
E. Organophosphate - Other Metabolites			
Malathion	MMA and MDA (major metabolites); DMP and DMTP (minor metabolites)	1,000 - 1,375	4,000

LD_{50}s are based on animal studies

SEQUENTIAL URINARY EXCRETION OF DIETHYL ALKYL PHOSPHATES IN FOUR FARM WORKERS HOSPITALIZED DUE TO 10% ETHYL PARATHION GRANULAR DERMAL EXPOSURE

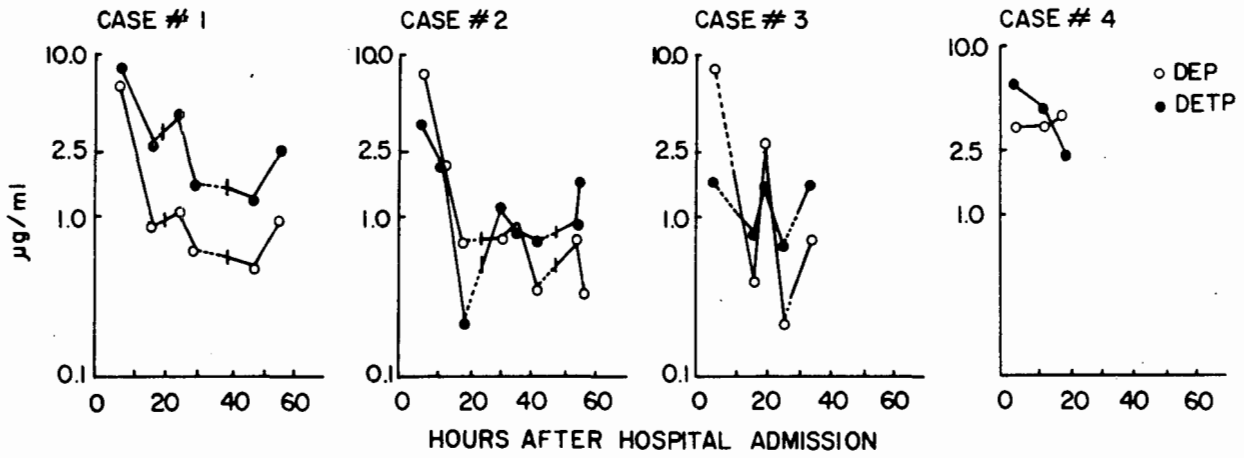


Figure 2

SEQUENTIAL URINARY EXCRETIONS OF DIETHYL ALKYL PHOSPHATES IN THREE CASES OF POISONING BY 6% ETHYL PARATHION MIXTURES DUE TO SPILLAGE OF THE CONCENTRATE

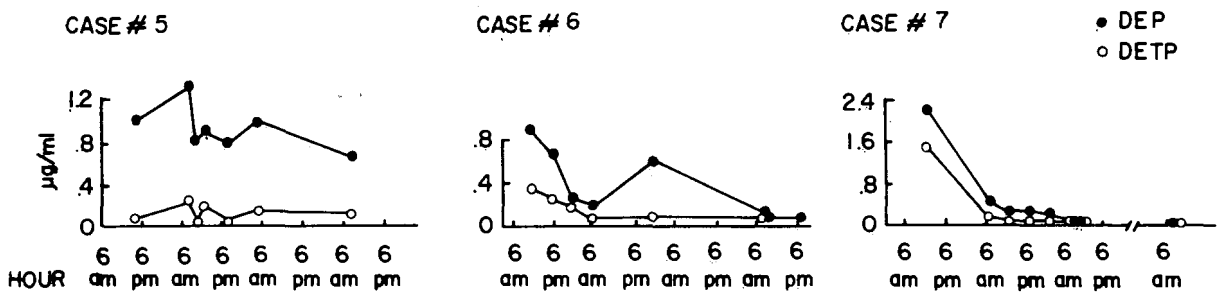


Figure 3

Case #8

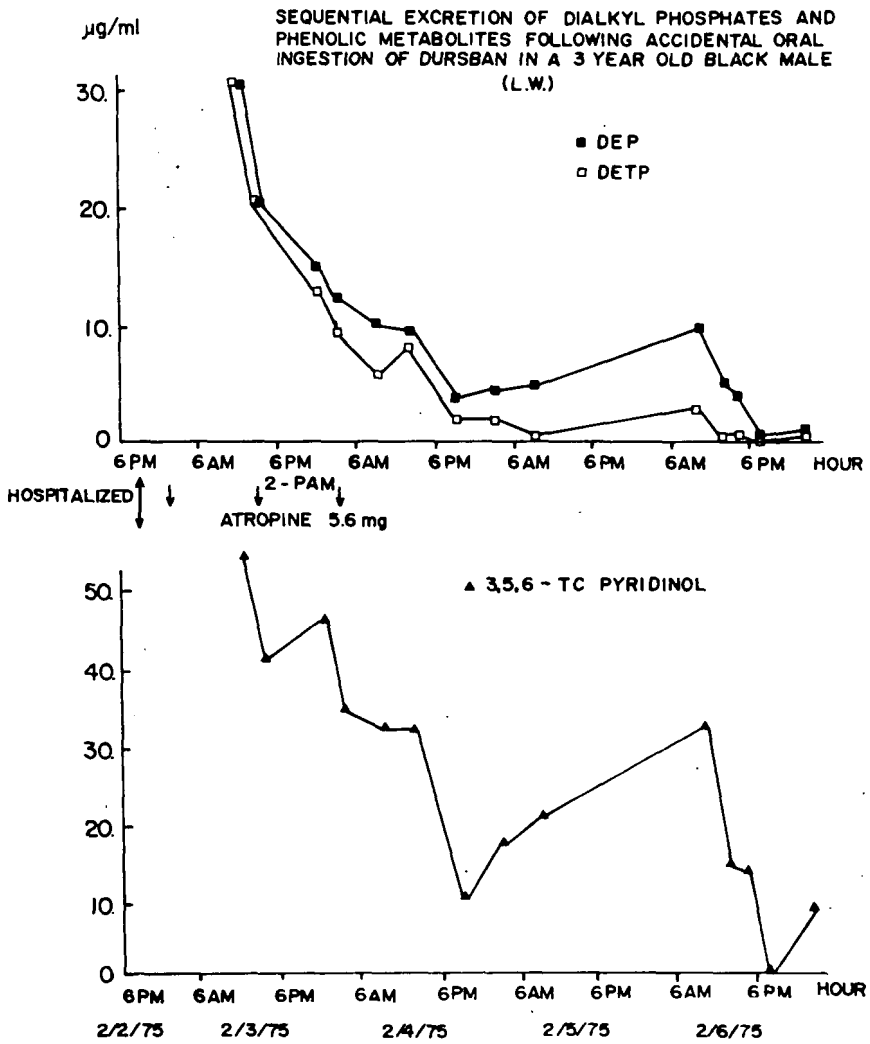


Figure 4.

Case #9

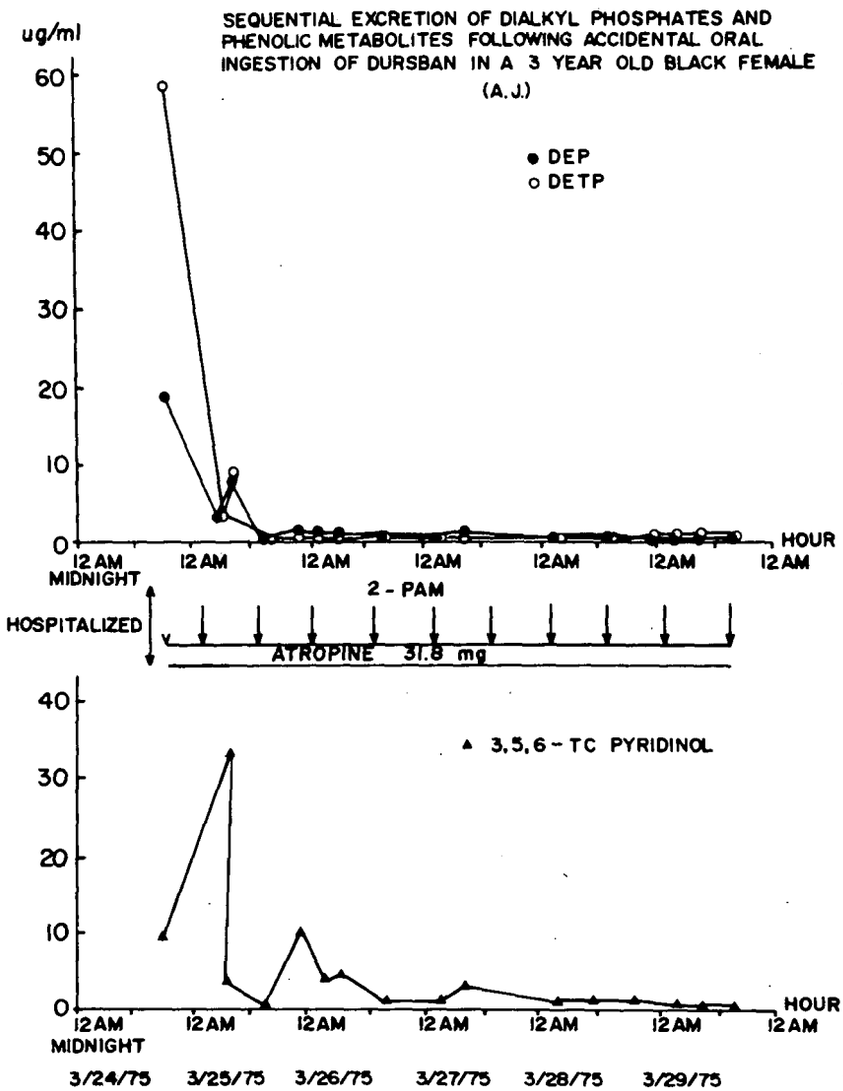


Figure 5

report last year (7). This organophosphate is highly soluble in adipose tissue producing a slow release of the pesticide from the fat into the circulation as serum concentrations shown in Figure 6 exemplify. The excretion pattern of the urinary alkyl phosphates from this pesticide are shown in Figure 7 emphasizing the sensitivity of these indices to the body burden of this pesticide, being still detectable in the urine for as long as 80 days after exposure; this was also the same duration for identifying the pesticide in serum. The DMP alkyl phosphate data shown in Figure 7 was possibly due to contamination of the dichlofenthion with a dimethyl organophosphate.

b. Poisoning by dimethyl organophosphate insecticides - Mevinphos (Phosdrin) is the organophosphate in this group which most frequently causes human poisonings; the major urinary metabolite is DMP. Urinary metabolites were studied in three cases of poisoning by this insecticide of which case number 11, shown in Figure 8 is a typical example. This was a dermal exposure in a mixer-loader and the initial concentration of DMP was 5 ug/ml, rose to 10 ug/ml in 8 hours and then was totally eliminated 8 hours later.

c. Mixed diethyl and dimethyl poisoning - J.T., a 55 y/o w/m was mixing and loading parathion 8E, toxaphene, dimethoate, and mevinphos (Phosdrin). He developed serious cholinesterase illness and was hospitalized shortly thereafter with almost total cholinesterase inhibition. The urine voided shortly after admission showed DMP 5.0 ug/ml; DMTP 0.8 ug/ml; DEP 0.7 ug/ml; DEP 1.5 ug/ml and DETP 3.3 ug/ml. These metabolites illustrate the combined effects of phosdrin and ethyl parathion exposures, demonstrating on the one hand the sensitivity of the index but emphasizing on the other hand the lack of specificity of the index when exposures such as these are mixed exposures.

d. Malathion poisoning - With malathion, the alkyl phosphates DMP, DMTP, DMDTP are the minor metabolites and malathion mono-acid and malathion di-acid being the major metabolites. When a patient (case number 13) ingested an unknown amount of 50% malathion, the initial concentration of DMP was 2.5 ug/ml, DMTP was 87 ug/ml and interpretation of the DMDTP was complicated by interfering peaks. It will be seen from Figure 9 that the urinary excretion of malathion mono-acid and malathion di-acid was very informative.

B. Discussion

Collectively these several poisoning case studies clearly demonstrate the diagnostic potential of the urinary metabolites if the exposure has been to a single pesticide. If additional phenolic metabolite data are available, specific biologic information is obtained, permitting the precise identification of the material causing the cholinesterase illness. With mixed pesticide poisonings, the situation is much more complex

Figure 6. Serum concentrations of dichlofenthion (ppb) and red cell and plasma cholinesterase levels (ΔpH/hr) during recovery following oral ingestion of dichlofenthion (VC-13)

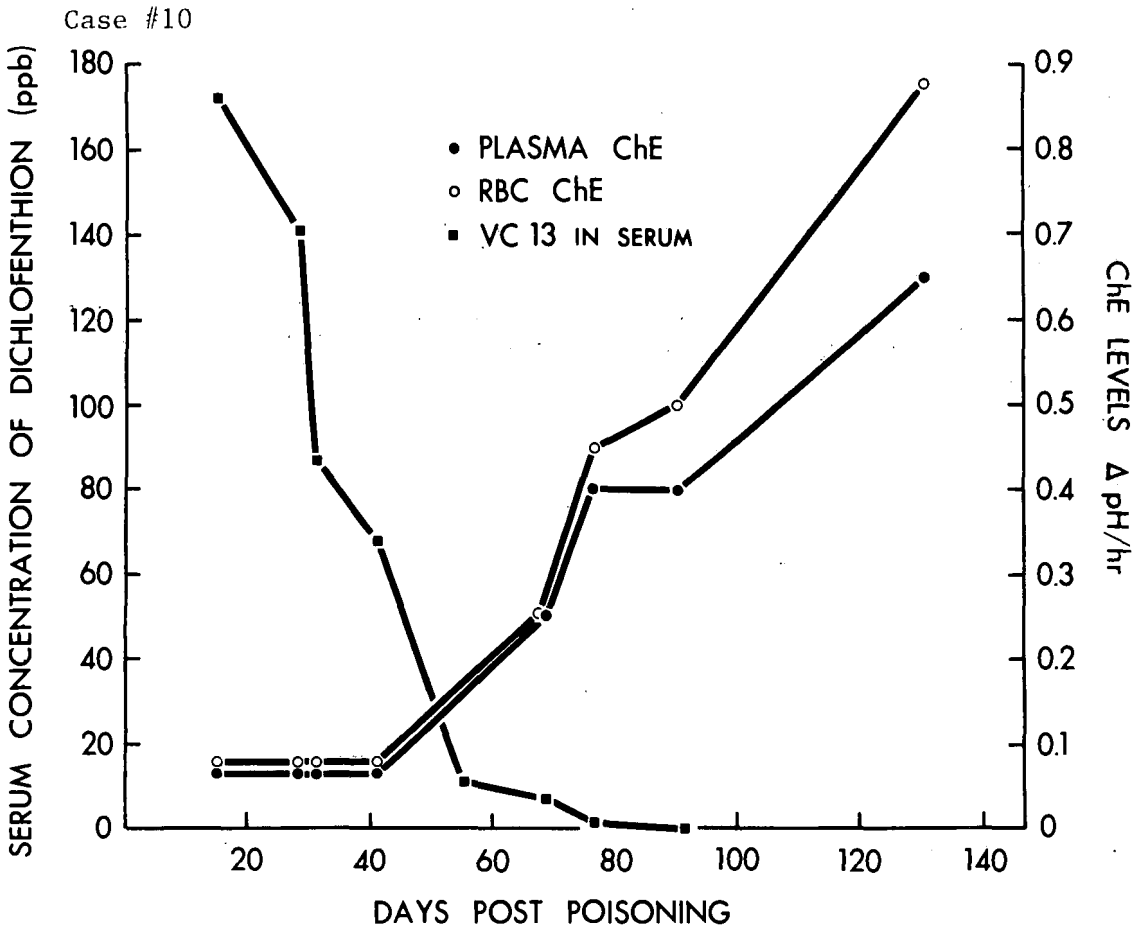
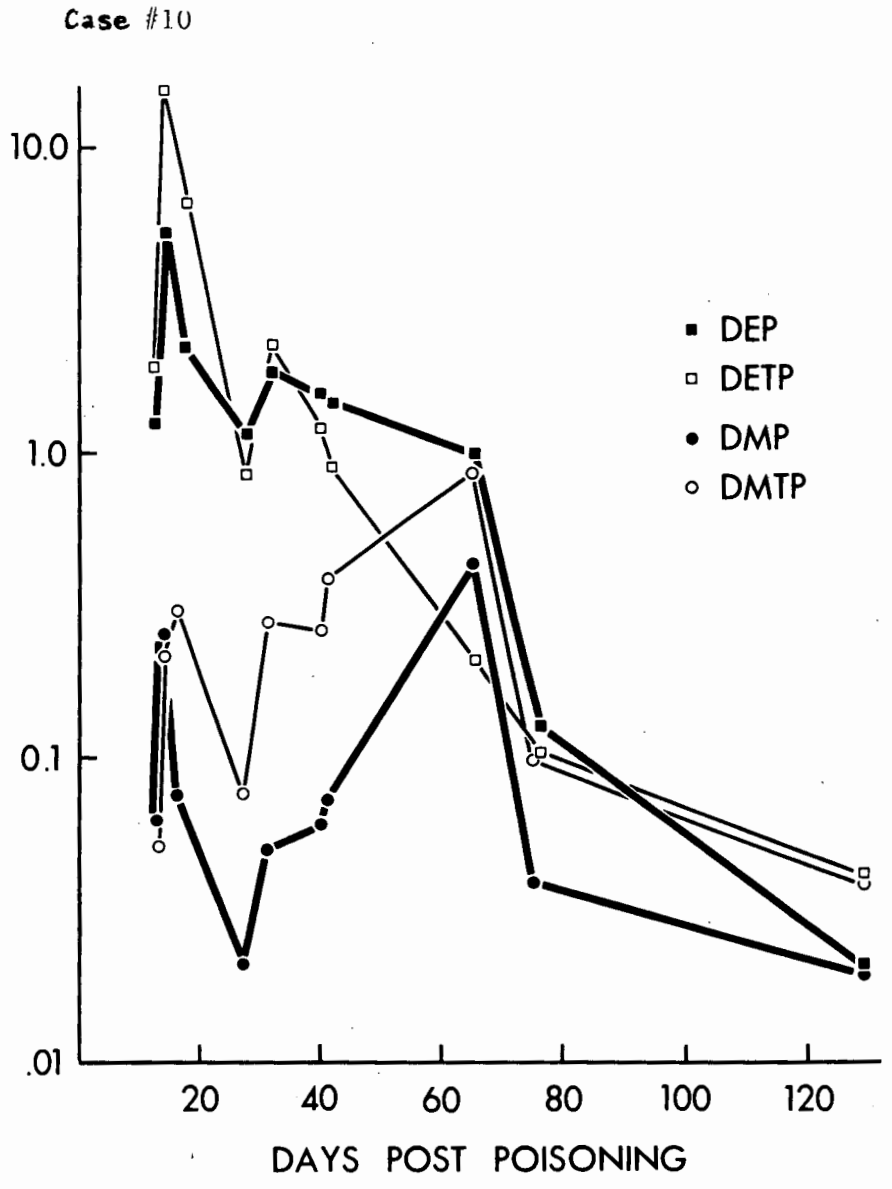


Figure 7. Urinary excretions of alkyl phosphate metabolites of dichlofenthion (VC-13) poisoning. Dade County, Fl. 1974



Case #11

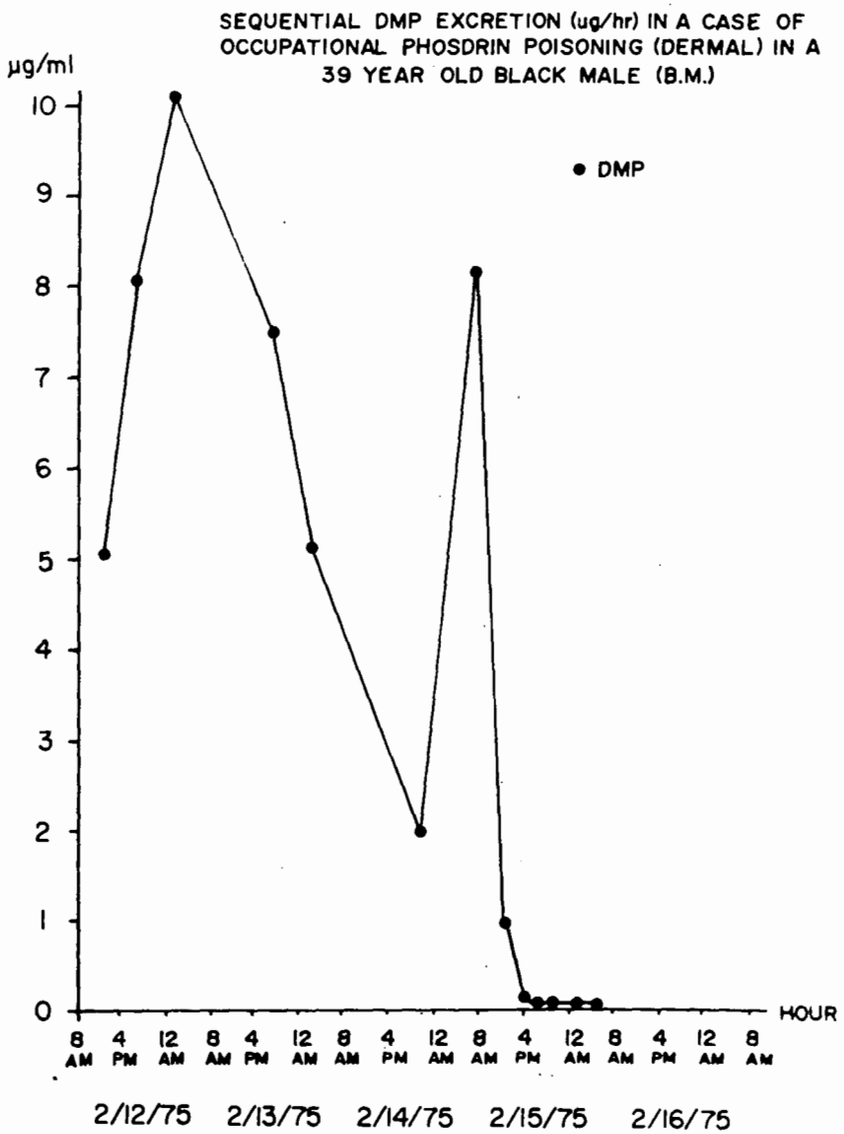


Figure 8

Case #12

Sequential excretion of malathion mono-acid (MMA) & malathion di-acid (MDA) urinary metabolites following oral ingestion of an unknown amount of malathion concentrate

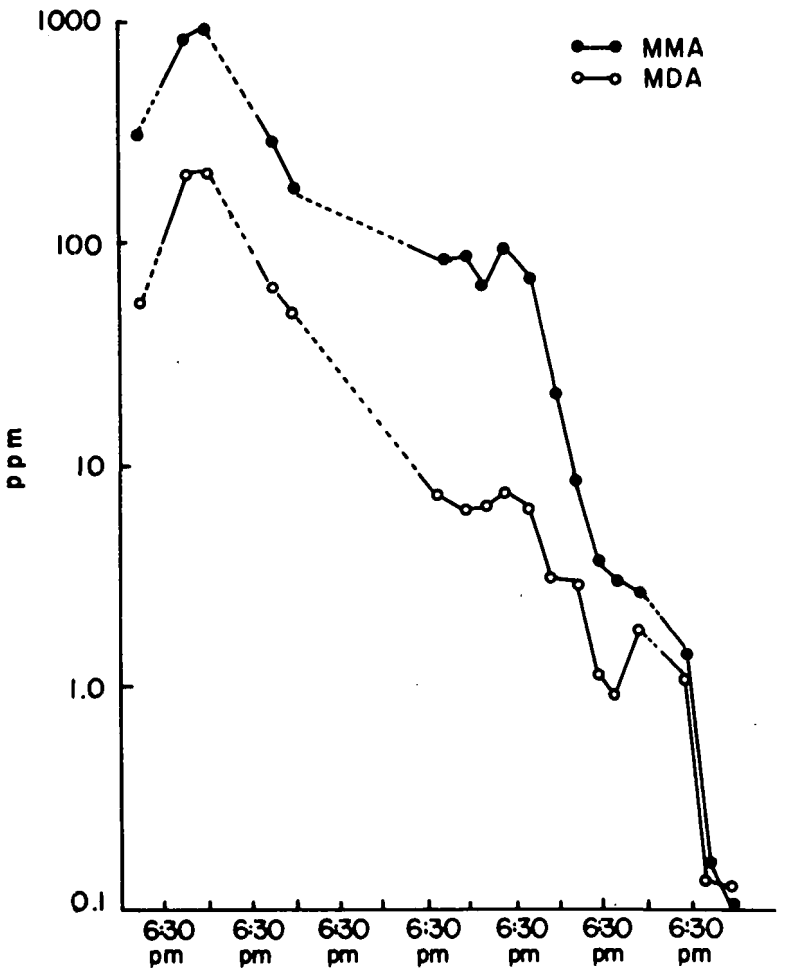


Figure 9

because the urinary alkyl phosphates are group specific (diethyl and dimethyl phosphates) but not pesticide specific. In these circumstances, the identification of the organophosphate group is facilitated but not the individual pesticide. Similarly, from a quantitative point of view there are no absolute concentrations of alkyl phosphates in the urine which can be considered uniformly diagnostic of all organophosphate pesticides because the alkyl phosphates are derived from organophosphates which have varying toxicities. The several variables which influence the concentration of these metabolites in the urine include: (1) the chemical configuration of the pesticide, (2) its metabolism and toxicity, and (3) the dose and rate of exposure; the dose and toxicity of the pesticide are the most important variables.

The ethyl parathion cases and the mevinphos case merit further discussion because these two pesticides are among the most toxic organophosphates being currently used by agriculture and are most often associated with poisoning and/or severe cholinesterase inhibition. In the 7 cases of parathion poisoning the lowest concentration of DEP observed in the initial urine was 0.9 ug/ml with initial concentrations in all these cases ranging from 0.9 to 8.1 ug/ml. These concentrations should be contrasted with DEP concentrations observed in 99 urines from 8 workers primarily exposed to these pesticides and found to have reasonably normal cholinesterase levels. The average DEP concentration was 0.14 ug/ml and only on one occasion was a DEP concentration greater than 0.4 ug/ml identified. Two mevinphos cases were investigated and the initial urine concentration of DMP was 5 ug/ml in both cases; these findings were very similar to those reported by Holmes et al. (16) who found that initial urine concentrations of DMP were 4.7 and 4.0 ug/ml respectively in two cases which they reported.

2. Occupational Exposure Studies with Urinary Alkyl Phosphate Excretions

a. Pesticide loader-mixer study - The objective of these urinary metabolite studies was to measure the surveillance and predictive potential of these biologic indices in the occupational exposed worker concentrating in particular on the pesticide loader and mixer, whose pesticide exposure is to a wide variety of chemicals. The work exposure of two subjects, one of whom had a red blood cell cholinesterase of 0.60 Δ pH/hr and a plasma cholinesterase of 0.50 ΔpH/hr (Worker A) and the other, who had a red blood cell cholinesterase of 0.11 ΔpH/hr and a plasma cholinesterase of 0.10 ΔpH/hr, are shown in Table 5.

It will be noted that Worker A was exposed only to the dimethyl organophosphate pesticides whereas Worker B besides sustaining a mixed exposure to both diethyl and dimethyl organophosphate insecticides was also exposed to pesticides which were far more toxic than those used by Worker A.

Tables 6 and 7 and Figures 10 and 11 describe and illustrate the sequential urinary excretion of pesticide metabolites during the 4 to 5 day work period of mixer loaders A and B. It should be noted that although Worker A was exposed entirely to the DMP organophosphate pesticides, the profile shows that he was sustaining small amounts of DEP exposure whose source was unknown, emphasizing both the sensitivity of the index and also the fact that in pesticide worker situations, unrecognized exposures occur which are not described in the conventional work

Table 5. Pesticide exposure history of two aircraft mixer-loaders, before and during the period of urinary alkyl phosphate surveillance. South Florida, 1975.

	Worker "A"		Worker "B"	
Period	Pesticides mixed	Alkyl phosphate metabolites	Pesticides mixed	Alkyl phosphate metabolites
Day before	Dimethoate (80%)	DMP, DMTP, DMDTP	Dibrom (40%)	DMP
	Dibrom (40%)	DMP	Diazinon	DEP, DETP
	Methomyl	-		
Surveillance Period Day I	None - at home		Dimethoate (80%)	DMP, DMTP, DMDTP
			Methomyl	-
Day II	Methomyl	-	Mevinphos (40%)	DMP
			Dibrom (40%)	DMP
			Parathion (8%)	DEP, DETP
			Toxaphene	-
Day III	Methomyl	-	Dimethoate (80%)	DMP, DMTP, DMDTP
Day IV	Dimethoate (80%)	DMP, DMTP, DMDTP	Mevinphos (40%)	DMP
	Dibrom (40%)	DMP	Diazinon	DEP, DETP
	Toxaphene	-	Dibrom (40%)	DMP
Day V	Methomyl	-	Mevinphos (40%)	DMP
			Dibrom	DMP
			Ethyl parathion (6%)	DEP, DETP
			Methyl parathion (3%)	DMP, DMTP
			Dimethoate	DMP, DMTP, DMDTP
			Methomyl	-
			Toxaphene	-

Table 6. Urinary alkyl phosphate metabolite studies* in a mixer-loader (A) having a normal cholinesterase level and whose pesticide exposure was to dimethoate (Cygon) and dibrom. Belle Glade, Florida. 1975

Date	Hour	DMP ug/ml	DMTP ug/ml	DMDTP ug/ml	DEP ug/ml	DETP ug/ml	$\frac{DEP}{DETP}$ Ratio
3/28/75	6:00 am	0.37	0.33	0.04	0.11	0.10	1.1
	11:45 am	0.19	0.49	0.02	0.07	0.07	1.0
	1:45 pm	0.25	0.10	N.D.	0.09	N.D.	0.0
	6:05 pm	0.19	0.16	N.D.	0.07	N.D.	0.0
	7:30 pm	0.09	0.80	N.D.	N.D.	N.D.	0.0
	10:20 pm	0.10	0.22	N.D.	0.04	0.05	0.8
3/29/75	6:00 am	0.10	0.21	N.D.	0.06	0.04	1.5
	12 noon	0.10	0.21	N.D.	0.06	0.04	1.5
	2:20 pm	0.29	0.17	N.D.	0.10	0.07	1.4
	6:10 pm	0.23	0.20	0.04	0.06	0.06	1.0
	10:00 pm	0.32	0.18	N.D.	0.09	0.06	1.5
3/30/75	6:00 am	0.25	0.15	N.D.	0.09	0.06	1.5
	11:50 am	0.12	0.11	N.D.	0.05	0.03	1.6
	2:20 pm	0.10	0.29	N.D.	0.06	N.D.	0.0
	9:15 pm	0.06	0.32	N.D.	N.D.	0.06	0.0
	11:00 pm	0.13	0.21	N.D.	0.07	0.04	1.7
3/31/75	6:00 am	0.13	0.30	0.04	0.05	0.08	0.6
	11:50 am	0.13	0.19	0.03	0.05	N.D.	0.0
	6:10 pm	0.19	0.17	0.04	0.06	0.06	1.0
	11:00 pm	0.66	1.13	0.17	0.09	0.11	0.8
4/1/75	6:15 am	1.11	0.92	0.19	0.18	0.06	3.0
	12:15 pm	0.69	0.58	0.03	0.12	0.06	2.0
	7:00 pm	0.77	1.06	0.10	0.15	0.13	1.1
	11:00 pm	0.90	0.96	0.11	0.19	0.17	1.1
4/2/75	6:00 am	0.48	0.36	0.12	0.17	0.09	1.8

*corrected for osmolality (800 ml/Osm/l)

N.D. = Not detectable

Limits of detectability for DMP 0.02 ug/ml; DMTP 0.03 ug/ml; DMDTP 0.02 ug/ml; DEP 0.03 ug/ml and DETP 0.02 ug/ml.

Table 7. Urinary metabolite studies in a mixer-loader (B) having an inhibited cholinesterase and whose work exposure was to a variety of pesticides. Belle Glade, Florida. 1975

Date	Hour	DMP ug/ml	DMTP ug/ml	DMDTP ug/ml	DEP ug/ml	DETP ug/ml	PNP ug/ml	$\frac{DEP}{DETP}$ Ratio
4/21/75	3:50 pm	0.21	0.21	N.D.	0.11	0.10	1.16	1.1
	6:45 pm	0.37	0.29	N.D.	0.10	0.15	1.19	0.6
4/22/75	3:00 am	0.24	0.16	N.D.	0.11	0.27	1.09	0.4
	5:30 am	0.23	0.21	N.D.	0.14	0.28	0.98	0.5
	6:20 am	0.41	0.26	N.D.	0.18	0.12	0.98	1.5
	9:35 am	0.14	0.17	N.D.	0.07	0.09	0.58	0.7
	9:05 pm	0.24	0.22	N.D.	0.30	0.31	1.02	1.0
	11:15 pm	0.34	0.20	N.D.	0.40	0.50	1.70	0.8
4/23/75	2:45 am	0.26	0.16	N.D.	0.37	0.14	1.13	2.6
	6:30 am	0.23	0.16	N.D.	0.26	0.19	0.90	1.4
	7:30 am	0.24	0.12	N.D.	0.37	0.13	0.57	2.8
	9:30 am	0.15	0.06	N.D.	0.24	0.06	0.53	4.0
	5:30 pm	0.25	0.07	N.D.	0.41	0.15	0.79	2.7
	9:45 pm	0.26	N.D.	N.D.	0.24	0.12	1.01	2.0
4/24/75	12:00 am	0.22	0.29	N.D.	0.17	0.15	0.22	1.1
	2:00 am	0.24	0.26	N.D.	0.14	0.27	0.34	0.5
	2:15 am	0.31	0.26	N.D.	0.20	0.16	0.47	1.2
	4:55 am	0.22	0.21	N.D.	0.14	0.16	0.43	0.8
	5:30 am	0.30	0.20	N.D.	0.18	0.35	0.52	0.5
	7:30 am	0.42	N.D.	N.D.	0.28	0.15	0.38	1.8
	9:40 am	0.33	0.13	N.D.	0.24	0.10	0.18	2.4
	10:00 pm	0.30	0.20	N.D.	0.21	0.39	0.64	0.5
4/25/75	2:55 am	0.39	0.25	N.D.	0.27	0.12	0.23	2.2
	7:30 am	0.25	0.26	N.D.	0.18	0.12	0.22	1.5
	7:45 am	0.31	0.28	0.03	0.24	0.16	0.29	1.5
	6:05 pm	0.29	0.32	0.03	0.20	0.14	0.23	1.4
	10:30 pm	0.52	0.32	0.03	0.20	0.19	0.19	1.0
	11:30 pm	0.54	0.28	N.D.	0.15	0.12	0.06	1.2

N.D. = Not detectable Limits of detectability are: DMP 0.02, DMTP 0.03, DMDTP 0.02, DEP 0.02, DETP 0.02 and PNP 0.01.

SEQUENTIAL EXCRETION OF URINARY ALKYL PHOSPHATES IN A MIXER LOADER (T.J.) WHOSE RED BLOOD CELL CHOLINESTERASE WAS 0.60 Δ pH/HR. AND PLASMA CHOLESTERASE 0.55 Δ pH/HR. DURING A PESTICIDE EXPOSURE TO DIMETHOATE (CYGON) AND DIBROM

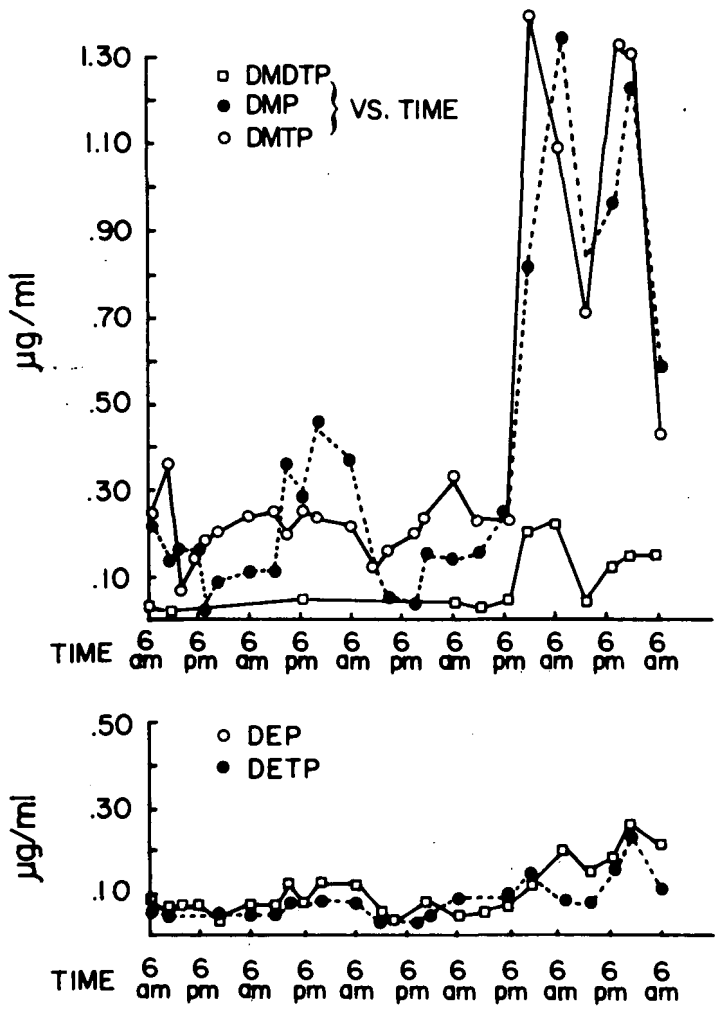


Figure 10

SEQUENTIAL EXCRETION OF URINARY ALKYL PHOSPHATES & PARANITROPHENOL IN A MIXER-LOADER (J.L.) WHOSE RED BLOOD CELL CHOLINESTERASE WAS 0.11 ΔpH/HR. & PLASMA CHOLINESTERASE 0.10 ΔpH/HR. DURING A MIXED EXPOSURE TO PESTICIDES

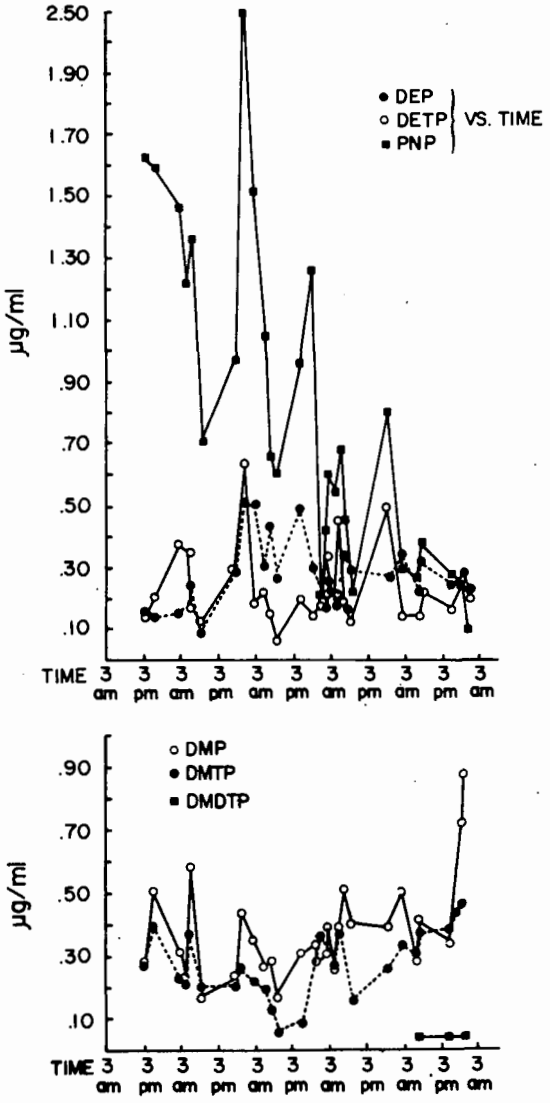


Figure 11

history study. Table 8 summarizes the mean and ranges of the alkyl phosphate metabolite excretions from these two mixer-loaders; there is very little difference in the average urinary excretion of the DMP in either worker although the oxon-parent compound ratio was twice as high in Worker B than in Worker A. There is however, a marked difference in the diethyl metabolite concentrations, Worker B having 1½ times more mean concentrations of DEP metabolites in his urine than what was found in Worker A. As will be seen from Figure 11, DEP excretions in Worker B correlated in time with the paranitrophenol (PNP) excretion indicating that the majority of the DEP was coming from the ethyl parathion exposures suggesting that it was this exposure to this toxic pesticide which was probably the major reason for the cholinesterase differences in these two workers.

Because of the low cholinesterase finding observed in Worker B and because several workers at this site were found to have severe cholinesterase inhibition, a 24 hour air sample was collected at this work site one month after the period of surveillance. The concentrations of pesticides found in air (ng/m) were as follows:

Diazinon	354
Malathion	24
Ethyl parathion	158
Methyl parathion	131
Mevinphos	4,650
γ BHC	1

Interfering peaks prevented the correct interpretation of possible exposures of chlorpyrifos, α BHC, heptachlor, aldrin, dieldrin, p,p' - DDT, o,p' - DDT, endrin, α chlordane and γ chlordane, and toxaphene.

In addition, both mevinphos and methyl and ethyl parathion were identified in a hexane wash extract of a T-shirt worn by one of the mixer-loaders.

There were thus significant and unknown pesticide exposures occurring at the work site where Worker B was employed, a finding which emphasizes the limited interpretability which can be placed on the conventional pesticide worker studies where exposure is entirely limited to pesticide use information of the subjects under study. In this instance, the urinary metabolite information and residue analyses showed that additional exposures were occurring from pesticides in the air of the worker environment and in the worker's clothing. For comparison purposes, the mean and ranges for the urinary alkyl phosphate excretions shown in Table 8 should be compared with the mean and ranges of these metabolites in 8 pilots and loaders also sustaining a mixed pesticide exposure which included ethyl parathion but with normal cholinesterase levels (Table 7).

Table 8. Comparisons of mean and ranges, and ratios of urinary alkyl phosphate metabolites* (ug/ml) in two mixer loaders found to have normal (A) and inhibited (B) cholinesterase levels.

Subjects & ChE levels/	No. study days	No. specimens analyzed	DMP $\bar{x}$ and ranges	DMTP $\bar{x}$ and ranges	DMDTP $\bar{x}$ and ranges	Ratio $\frac{DMP}{DMTP + DMDTP}$	DEP $\bar{x}$ and ranges	DETP $\bar{x}$ and ranges	Ratio $\frac{DEP}{DETP}$
A RBC ChE 0.6 Pl. ChE 0.55	5	25	0.3 (.06 - .09)	0.4 (.11 - .96)	0.4 (ND** - .19)	0.7	.08 (ND - 0.19)	.08 (ND - 0.17)	1.0
B RBC ChE 0.10 Pl. ChE 0.11	4	28	0.3 (.14 - .54)	0.2 (ND - .32)	0.004 (ND - .03)	1.5	.20 (.1 - .41)	.18 (.06 - .50)	1.17

*corrected for osmolality of 800 ml/Osm/l
**Not detectable
/ΔpH/hr - Michel method

B. Discussion

Surveillance of the pesticide worker exposed to organophosphate and carbamate insecticides is designed to detect and prevent serious cholinesterase inhibition and cholinergic illness. These are two human health end points of special concern. In South Florida, past experience has suggested that these effects are most likely to occur with workers working with the more toxic pesticides such as parathion or mevinphos (17, 4, 3). Sequential urinary metabolite studies which were conducted in the second year of this project (6) showed that applicators working with relatively non-toxic organophosphates such as chlorpyrifos, although exhibiting significant urinary excretions of the alkyl phosphates of this pesticide did not demonstrate simultaneous cholinesterase depression. Thus, the the surveillance potential of this metabolite is of primary importance only in situations where the worker is working with the more highly toxic organophosphates. The findings presented in this study show that prediction is impossible when the worker is exposed to a mixture of pesticides, particularly when the mixture includes both highly toxic and relatively nontoxic organophosphate pesticides.

Since today almost all agricultural practices call for the use of a variety of organophosphate and carbamate insecticides, one must conclude on the basis of these studies that although the urinary alkyl phosphate metabolites are highly sensitive indicators of exposure to the organophosphates they do not hold much promise for being reliable non-specific predictors of cholinesterase depression in mixed exposure situations where the pesticides involved differ greatly with regard to inherent toxicities. This broad generalization of the potential of the urinary alkyl phosphates as a surveillance instrument should be tempered by one clarification. If in the future, occupational programs based upon metabolite excretions are pursued, then standards might be set which would have to be based on the worse theoretically possible situations, namely exposures to ethyl parathion and mevinphos. One can envisage a situation where future occupational surveillance programs would be required only when workers were working with the more toxic pesticides. Under the circumstances, standards for safe and unsafe levels of urinary alkyl phosphates could be set which would be based on the metabolite concentrations observed with known exposures to parathion and mevinphos. This biologic index would then become a highly efficient surveillance instrument because safe and unsafe levels would be based on single pesticide exposure to pesticides whose toxicity was known.

A. Results

b. Urinary alkyl phosphate studies in workers exposed to foliar residues - The objective of these studies was to test the evaluatory potential of the urinary metabolites, imposing an intervention procedure designed to produce subtle differences in exposure to foliar residues in two groups of workers and to determine whether the urinary metabolites were sensitive enough to detect these differences. Mean differences in urinary alkyl phosphates between workers wearing fluorocarbon treated

clothing and workers wearing untreated clothing are shown in Table 9. The differences are the mean values of the untreated minus the treated for twenty-four 12 hour urines collected over a six day study period. For all four metabolites these differences were significant at the $p < .01$ level. These showed that the workers wearing treated clothing excreted significantly less amounts of urinary alkyl phosphates than workers wearing untreated clothing. The significance of these differences was the same whether the urinary metabolites were corrected or uncorrected for osmolality. Tables 10a and 10b, and figures 12a and 12b also exhibit striking temporal parallelism between excretion values of the two groups further emphasizing the unique sensitivity of these indices.

B. Discussion

The above tables and figures demonstrate that the mean alkyl phosphate levels for each 12 hour urine period are higher in the untreated group than in the treated group. This is true for 11 of 12 periods for DMP and DEP, and 12 of 12 periods for DMTP and DETP. In addition, the reliability of the urinary alkyl phosphate as an epidemiologic evaluatory tool is amply demonstrated by these data, the sensitivity of these indices readily permitting the identification of the different organophosphate exposures of the two groups.

A. Results

3. Urinary alkyl phosphate studies in the general population

The objectives of these studies was to measure the profiles of incidental exposure of a small sample of the general population of Dade County to organophosphate and carbamate insecticides. Thus, Table 11 presents the mean, ranges and frequency of identification of the urinary alkyl phosphate and phenolic metabolites in this Dade County general population survey. The individual results are shown in Appendix A.

In an orange juice feeding study in which two volunteers participated the orange juice had a DEP concentration of 0.01 ug/ml. Pre and post cholinesterase determinations and urinary alkyl phosphates pre, during and after drinking orange juice are shown in Table 12.

B. Discussion

Table 11 shows that there are minimal incidental exposure of the general population in this area to trace amounts of organophosphates and carbamates. Based upon a small sample size of 37 individuals between 36% and 56% of the population had trace amounts of organophosphates in their urine and since 11 of these had trace amounts of 3,5,6-TCP this would suggest that chlorpryifos was one major source of this incidental organophosphate exposure. There was evidence of minimal incidental exposure in 100% of the study sample to pentachlorophenol (PCP) and 27% of the sample demonstrated small environmental exposure to chlorpyrifos (Dursban).

Table 9. Unadjusted and adjusted (for osmolar corrected) mean differences in urinary alkyl phosphate levels between agricultural workers wearing untreated and treated fluorocarbon clothing. 1975.

	DMP (ug/ml)	DEP (ug/ml)	DMTP (ug/ml)	DETP (ug/ml)
A. Unadjusted				
mean difference (over 12 hr time periods)	.06	.05	.08	.04
standard error	.015	.009	.007	.013
"p"	<.01	<.01	<.01	<.01
B. Adjusted				
mean difference (over 12 hr time periods)	.06	.05	.06	.04
standard error	.016	.009	.006	.012
"p"	<.01	<.01	<.01	<.01

Table 10a. Comparison of the adjusted osmolar mean of twelve hour urinary alkyl phosphates (ug/ml) in workers wearing untreated clothing and workers wearing fluorocarbon treated clothing over a six day period

		DMP (ug/ml)					DEP (ug/ml)		
Day	12 hour urine period	No. subjects	$\bar{x}$ Untreated	No. subjects	$\bar{x}$ Treated	Δ Untreated-Treated	$\bar{x}$ Untreated	$\bar{x}$ Treated	Δ Untreated-Treated
1	1	11	.05	9	.08	-0.03	.11	.12	-0.01
	2	11	.09	11	.07	.02	.16	.13	.03
2	3	11	.11	10	.05	.06	.15	.09	.06
	4	12	.17	10	.11	.06	.19	.13	.06
3	5	12	.38	11	.22	.16	.31	.25	.06
	6	12	.28	9	.15	.13	.27	.20	.07
4	7	11	.26	11	.21	.05	.27	.21	.06
	8	11	.19	12	.19	.00	.22	.21	.01
5	9	11	.22	13	.13	.09	.21	.16	.05
	10	10	.20	11	.15	.05	.25	.19	.06
6	11	11	.23	11	.11	.12	.25	.13	.12
	12	10	.20	11	.19	.01	.24	.20	.04
				mean difference=		.06			.05
				standard error=		.016			.009

$\bar{x}$ = mean
Δ = difference

Table 10b. Comparison of the adjusted osmolar mean of twelve hour urinary alkyl phosphates (ug/ml) in workers wearing untreated clothing and workers wearing fluorocarbon treated clothing over a six day period

		DMTP (ug/ml)					DETP (ug/ml)		
Day	12 hour urine period	No. subjects	$\bar{x}$ Untreated	No. subjects	$\bar{x}$ Treated	Δ Untreated-Treated	$\bar{x}$ Untreated	$\bar{x}$ Treated	Δ Untreated-Treated
1	1	11	.07	9	.03	.04	.08	.02	.06
	2	11	.07	11	.04	.03	.05	.05	.00
2	3	11	.11	10	.06	.05	.09	.08	.01
	4	12	.11	10	.03	.08	.08	.04	.04
3	5	12	.17	11	.11	.06	.13	.15	.02
	6	12	.14	9	.05	.09	.13	.06	.07
4	7	11	.24	11	.16	.08	.14	.09	.05
	8	11	.16	12	.13	.07	.10	.07	.03
5	9	11	.21	13	.11	.10	.10	.08	.02
	10	10	.16	11	.15	.04	.12	.09	.03
6	11	11	.15	11	.06	.09	.19	.03	.16
	12	10	.09	11	.05	.04	.12	.04	.08
				mean difference=		.06			.04
				standard error=		.006			.012

$\bar{x}$ = mean
Δ = difference

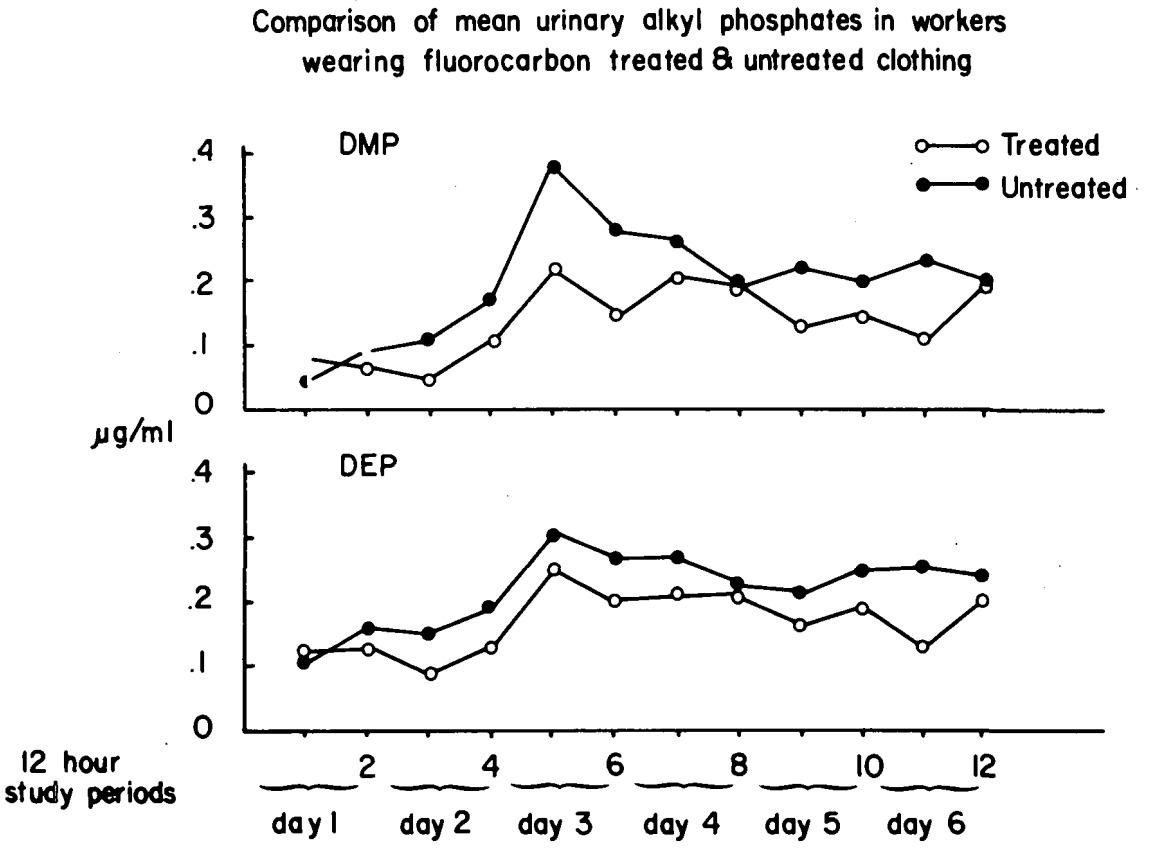

Comparison of mean urinary alkyl phosphates in workers wearing fluorocarbon treated & untreated clothing
DMP
Treated
Untreated
.4
.3
.2
.1
0
µg/ml
DEP
.4
.3
.2
.1
0
12 hour study periods
2
4
6
8
10
12
day 1
day 2
day 3
day 4
day 5
day 6

Figure 12a

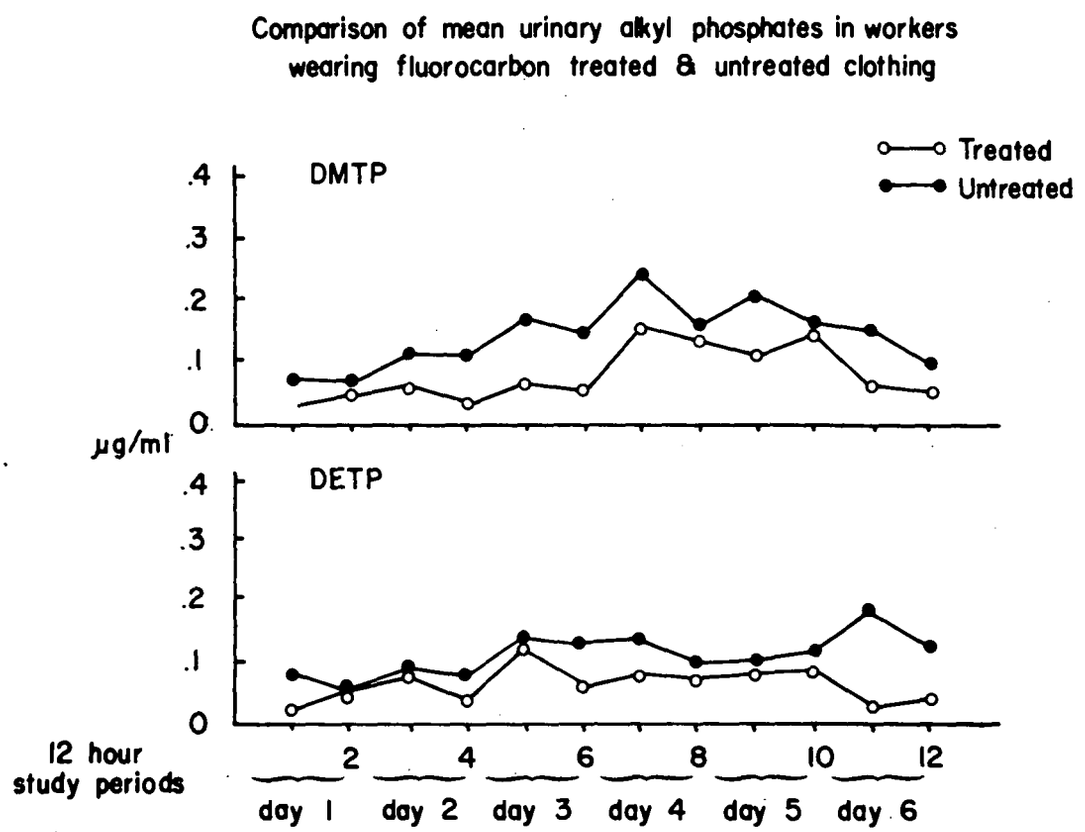

Comparison of mean urinary alkyl phosphates in workers
wearing fluorocarbon treated & untreated clothing
Treated
Untreated
DMTP
DETP
.4
.3
.2
.1
0
µg/ml
12 hour
study periods
2
4
6
8
10
12
day 1
day 2
day 3
day 4
day 5
day 6

Figure 12b

Table 11. Mean and ranges and frequency of identification of alkyl phosphates and selected phenols in the general population of Dade County, Florida, 1975

Metabolites	No. of subjects	Mean	Ranges	% Detected at or above limit of detectability
A. Alkyl Phosphates				
DMP	36	.008	N.D. - .053	56%
DEP	36	.012	N.D. - .11	58%
DMTP	33	.027	N.D. - .22	45%
DETP	33	.021	N.D. - .3	36%
B. Phenols				
3,5,6-TCP	37	.003	N.D. - .032	27%
PCP	37	.009	.001 - .023	100%
2,4,5-TCP	37	.004	N.D. - .10	14%

Table 12. Cholinesterase and urinary alkyl phosphate findings before, during, and after orange juice ingestion

Study Phases	Date	Sex	RBC ChE	Pl. ChE	Vol. Voided	ALKYL PHOSPHATES (ppm) DMP	DMTP	DEP	DETP
Pre-Orange Juice	12/15/76	F			250	N.D.	N.D.	N.D.	N.D.
"	"	M			750	N.D	N.D.	N.D.	N.D.
"	12/16/75	F	0.70	0.68	250	N.D	N.D.	N.D.	N.D.
"	"	M	0.88	0.70	780	N.D.	N.D.	N.D.	N.D.
Orange Juice	12/17/76	F			1,165	N.D.	N.D.	N.D.	N.D.
	"	M			750	N.D.	N.D.	0.35	N.D.
" "	12/18/76	F			870	N.D.	N.D.	N.D.	N.D.
	"	M			1,550	N.D.	N.D.	0.05	N.D.
	12/19/76	F	0.65	0.90	1,200	N.D.	N.D.	N.D.	N.D.
" "	"	M	0.80	0.80	1,230	N.D.	N.D.	N.D.	N.D.
Post-Orange Juice	12/20/76	F			340	N.D.	N.D.	N.D.	N.D.
	"	M				N.D.	N.D.	N.D.	N.D.
" "	12/21/76	F			310	N.D.	N.D.	N.D.	N.D.
	"	M			600	N.D.	N.D.	N.D.	N.D.

N.D. = Not detectable

The orange juice study did not support the hypothesis of incidental organophosphate exposure from this dietary source.

4. Adipose pesticide residue studies in the occupationally exposed

A. Results

The objectives of these studies was (1) to determine whether any fat soluble organophosphates had become part of the adipose pesticide residue spectrum of the occupationally exposed worker, (2) to compare simultaneous adipose and serum exposure of organochlorine pesticicdes and (3) to measure urinary alkyl phosphate and phenolic metabolites in pesticide formulations trying to relate these to worker histories and air concentrations in the work environment. Table 13 presents the simultaneous adipose and serum concentrations of pesticides in five formulators. Bromophos, a fat soluble halogenated organophosphate insecticide was identified in three of the samples analyzed. This is the first time that a fat soluble organophosphate has been identified in persons occupationally exposed to pesticides.

Although with only 5 subjects it is scarcely valid to attach significance to the correlation of serum and fat levels, a bi-variate Pearson correlation (18) was performed on adipose serum values. The following correlations were demonstrated.

COMPOUND	SERUM - FAT CORRELATION COEFFICIENT (p)
α BHC	0.8856
β BHC	0.9841
γ BHC	0.7176
Heptachlor Epoxide	0.9895
Dieldrin	0.9037
p,p'-DDT	0.5373
p,p'-DDE	0.9906
Oxychlordane	0.9770
HCB	0.9789

Correlations of .9 or more were obtained from β BHC, heptachlor epoxide, dieldrin, p,p'-DDE, oxychlordane and HCB when compared to serum levels.

Table 13. SIMULATANEOUS ADIPOSE AND SERUM CONCENTRATIONS OF PESTICIDES IN FIVE FORMULATORS. 1975.

Subject, Initials and years of work Experience	Pesticides												
	α-BHC	β-BHC	γ-BHC	Δ-BHC	Heptachlor epoxide	Dieldrin	p,p'-DDT	o,p'-DDT	p,p-DDE	Total DDT	Oxy-chlordane	HCB	Bromophos
A.P. b/m 18 years													
fat (ppm)	0.345	1.19	0.268	0.035	2.44	0.542	3.35	0.145	21.6	27.5	2.45	0.612	0.03
serum (ppb)	1.3	5.2	0.97		11.8	3.7	7.1	N.D.	70.1	85.2	6.1	5.3	N.D.
J.P. w/m 15 years													
fat (ppm)	0.331	0.89	0.315	0.100	2.91	0.680	2.56	0.162	11.8	17.3	1.15	0.232	0.02
serum (ppb)	1.7	4.5	1.9		12.6	4.6	6.2	N.D.	37.6	48.1	3.5	2.3	N.D.
T.W. b/m 3 years													
fat (ppm)	0.482	0.65	0.436	0.166	3.89	0.724	1.05	0.131	4.78	13.0	0.49	0.141	0.04
serum (ppb)	2.9	3.1	1.6		19.3	4.5	6.2	N.D.	15.4	23.4	1.9	1.9	N.D.
J.T. b/m 3 years													
fat (ppm)	0.283	0.648	0.168	0.038	1.33	0.407	2.23	N.D.	11.06	14.6	0.23	0.108	N.D.
serum (ppb)	1.1	2.7	1.0		5.4	N.D.	5.8	N.D.	38.3	48.5	N.D.	1.0	N.D.
E.W. b/m 4 months													
fat (ppm)	0.160	0.15	0.142	0.036	0.39	0.148	1.36	0.2	14.54	17.7	0.14	0.05	N.D.
serum (ppb)	1.0	N.D.	1.0		2.6	N.D.	N.D.	N.D.	53.4	59.5	N.D.	N.D.	N.D.

Table 14 presents cholinesterase and urinary alkyl phosphate concentrations in these five formulators. The pesticides which were being formulated during the week that these specimens were collected were: chlorpyrifos, DDVP, carbaryl, sulphur, BHC, toxaphene, chlordane, dithane and M-45. Two 24 hour air samples were collected inside the formulating plant. The first was collected five months before the fat biopsy was obtained and the other one month after the collection of the fat biopsy. The concentrations of the pesticides in air are shown below:

	Sample #14 (ng/m^3) June 11-12, 1975	Sample #29 (ng/m^3) December 12-13, 1975
Chlorpyrifos	168	65
Diazinon	470	168
Malathion	170	9
α BHC	3,977	2,301
Heptachlor	7,315	25,243
Aldrin	655	N.D.
γ BHC	4,791	3,779
Dieldrin	88	24
p,p'-DDT	73	N.D.
o,p'-DDT	43	N.D.
Endrin	43	N.D.
Ethyl parathion	86	17
Methyl parathion	182	Interference
Methyl bromophos	4,130	1,715
Mevinphos	8	Interference
α chlordane	432	597
γ chlordane	2,328	3,068
Phorate	N.D.	1
HCB	N.D.	387
Ronnel	N.D.	2

B. Discussion

When one looks at the pesticides being handled by these formulators, and the air concentrations of pesticides detected inside the plant and tries to relate these to the epidemiologic information provided by adipose and urine metabolite studies, it is obvious that these two biologic indices reflect recent exposures, expressed by the urine data and long term exposures reflected by the adipose data. As may be expected, the organochlorine residues are significantly higher than those seen in the general population, and although many of the pesticides identified in the adipose residues have not been formulated recently, their presence in the fat almost certainly reflects the persistent exposure to these chemicals through concentrations in air. Total DDT levels in fat correlated well with person years of employment in the plant but this association was not so constant with the other organochlorine pesticides detected. The recognition of bromophos (an organophosphate of low toxicity) in the

Table 14. SIMULTANEOUS CHOLINESTERASE AND URINARY ALKYL PHOSPHATE AND PHENOLIC METABOLITES OF PESTICIDES IN FIVE FORMULATORS. DADE COUNTY, FLORIDA. 1975

Initials and years of work experience	RBC ChE* (ΔpH/hr)	Pl. ChE* (ΔpH/hr)	Alkyl phosphates (ug/ml)				Phenol (ug/ml)≠					
			DMP	DMTP	DEP	DETP	PCP	2,3,5-TCP	4-bromo-2,5 DCP	2,4,6-TCP	2,3,4,6, TTCP	2,3,5,6, TTCP
A.P. b/m 18 years	0.89	0.35	0.07	0.13	0.06	N.D.	0.016	0.24	0.32	0.04	0.02	0.002
J.P. w/m 15 years	0.70	0.41	0.01	0.04	0.08	N.D.	0.010	0.11	0.20	0.04	0.01	N.D.
T.W. b/m 3 years	0.70	0.27	N.D.	N.D.	0.05	N.D.	0.006	0.12	0.07	0.02	0.01	N.D.
J.T. b/m 3 years	0.57	0.55	0.08	N.D.	0.04	N.D.	0.032	0.14	0.09	0.04	0.01	0.002
E.W. b/m 4 months	0.80	0.50	0.04	N.D.	0.08	N.D.	0.012	0.18	0.07	0.02	0.004	N.D.

≠We were not able to calculate 2,4,5-TCP and 2,3,5-TCP because they had the same retention time in three different columns (4% SE30-6% QF-1), (1.5% OV17-1.95% QF-1), (5% OV-210).

*Michel method

N.D. = Not detectable

fat of these workers merely adds the added dimension of persistence to some of the newer and less polar organophosphates now coming on the market and emphasizes the need for additional evaluation of this property and the consequences of these properties for man and his environment.

With regard to the urinary metabolite findings of PCP concentrations, these were of an order of magnitude similar to that found in the general population, suggesting that this exposure was incidental rather than occupational. The urinary alkyl phosphate findings as well as the red blood cell cholinesterase levels were generally reassuring suggesting that the occupational health and safety practices in this plant were sufficient to prevent significant human exposures to the organophosphate insecticides. 3,5,6-trichloropyridinol data was indicative of occupational chlorpyrifos exposures since these concentrations were greater than those found in the general population.

5. Pesticide residue studies in the general population

A. Results

The objectives of these studies were (1) to determine whether any of the less polar organophosphates formed any part of the adipose pesticide residue spectrum of the general population, and (2) to compare the prevalence of organochlorine pesticides in the 1975 sample with the levels observed in the earlier surveys. The individual results are shown in Appendix B.

No organophosphate pesticide residues were detected in this small sample of the general population. The arithmetic mean and standard deviation and ranges of eight organochlorine pesticides identified in the adipose tissue are shown in Table 15. These levels are compared with the levels found in 1970 (Table 15). Oxychlordane and HCB were not identified in earlier surveys.

Figures 13a and 13b illustrate the frequency distribution of Total DDT, oxychlordane, HCB, BHC, heptachlor epoxide and dieldrin residues in this small sample.

6. Pesticide Air Sampling Studies

A. Results

The objectives of these studies were to continue monitoring air concentrations of pesticides in the several sites described earlier. Emphasis was placed on (1) the Everglades, (2) Miami suburbs, (3) downtown Miami (Miami International Airport) and (4) a community health center located one mile north of a formulating plant (South Dade Community Health Center). The individual concentrations of pesticides in air in the urban and suburban samples collected and analyzed are shown in Table 16. Work site studies are shown in Table 17; these are air concentrations of pesticides at two crop dusting sites and two samples collected inside a formulating plant. The data collected on all 40 samples are shown in Appendix C.

Table 15. COMPARISON OF ADIPOSE ORGANOCHLORINE PESTICIDE RESIDUES (PPM) IN DADE COUNTY GENERAL POPULATION (ADULTS). 1975 AND 1970.

Pesticides	Year	Number	Mean	Standard Deviation	Ranges
p,p'-DDT	1975	20	0.9	0.8	0.11 - 3.1
	1970	122	1.6	1.6	0.01 - 13.0
p,p'-DDE	1975	20	5.8	3.7	1.00 - 11.5
	1970	122	5.6	4.0	0.05 - 21.0
Total DDT	1975	20	7.5	4.7	1.43 - 14.3
	1970	122	7.8	5.7	0.01 - 28.6
Dieldrin	1975	20	0.2	0.2	0.02 - 0.7
	1970	122	0.3	0.3	.005 - 2.2
Oxychlordane	1975	20	0.1	0.1	0.05 - 0.3
HCB	1975	20	0.1	0.1	0.02 - 0.4
β BHC	1975	20	0.2	0.2	0.08 - 0.6
	1970	122	0.3	0.2	N.D. - 1.4
Heptachlor Epoxide	1975	20	0.1	0.02	0.03 - 0.1
	1970	122	0.1	0.05	N.D. - 2.7

Figure 13a. Frequency distribution of organochlorine residues in adipose tissue of the Dade County general population. 1975.

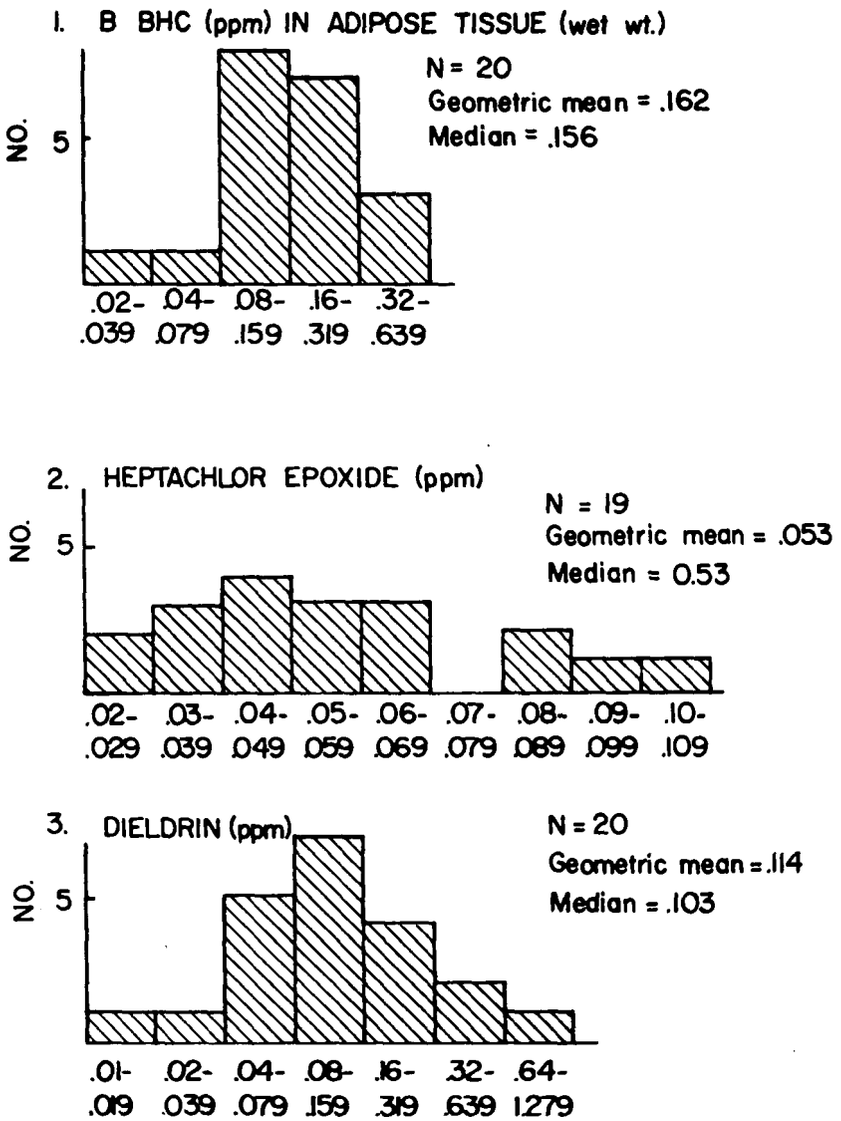


Figure 13b. Frequency distribution of organochlorine residues in adipose tissue of the Dade County general population. 1975.

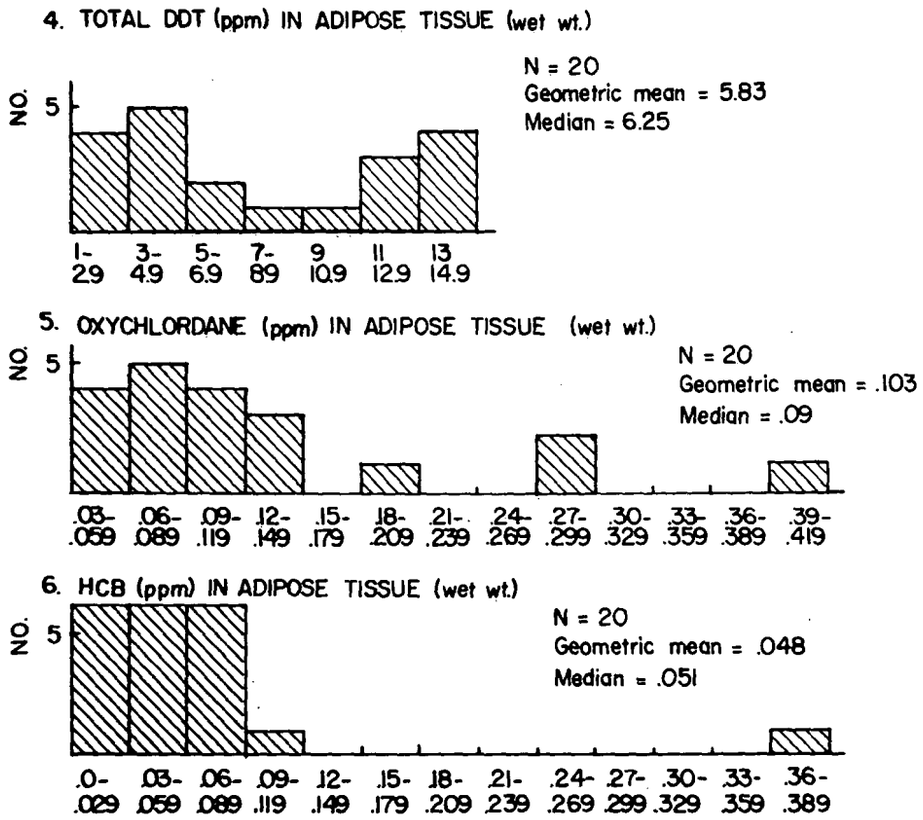


Table 16. Medians and ranges of air concentrations (ng/m^3) of pesticides observed in locations with repeated air sampling surveys. South Florida 1975-76.

Pesticide and Their Limits of Detectability		Suburban Site (17 samples) Med.	Suburban Site (17 samples) Ranges	Airport Site (8 samples) Med.	Airport Site (8 samples) Ranges	So. Dade Comm. Hlth Ctr (9 samples) Med.	So. Dade Comm. Hlth Ctr (9 samples) Ranges	Everglades (2 samples) Ranges
Dichlofenthion	(0.2)	N.D.*	N.D. - 1.4	N.D.		N.D.		N.D.
Chlorpyrifos	(0.3)	1.0	0.3 - 11.5	.75	N.D. - 22.3	0.8	N.D. - 1.6	N.D.
Diazinon	(0.2)	1.5	N.D. - 17.6	1.1	N.D. - 5.9	0.8	N.D. - 5.4	N.D. - 3.2
Malathion	(1.0)	N.D.	N.D. - 9.2	N.D.	N.D. - 6	N.D.	N.D. - 7.2	N.D.
α BHC	(0.1)	0.3	0.1 - 1.5	0.3	0.1 - 0.9	0.3	N.D. - 2.2	0.1 - 0.5
Heptachlor	(0.1)	8.7	0.34 - 8.7	0.75	0.35 - 3.64	1.0	0.64 - 3.9	0.22 - 0.7
Aldrin	(0.2)	N.D.	N.D. - 0.47	N.D.		N.D.		N.D.
γ BHC	(0.1)	0.3	N.D. - 1.3	0.16	N.D. - 0.6	0.4	N.D. - 1.0	N.D. - 0.1
Dieldrin	(0.3)	0.3	N.D. - 1.0	0.18	N.D. - 0.3	0.3	N.D. - 0.7	N.D.
Mevinphos	(0.5)	N.D.		N.D.	N.D. - 3.0	N.D.		N.D.
α Chlordane	(0.2)	0.6	0.3 - 3.0	0.41	0.16 - 0.9	0.3	0.15 - 1.2	N.D. - 0.2
γ Chlordane	(0.3)	1.0	0.41 - 12.7	0.8	0.3 - 2.0	0.9	0.12 - 2.2	N.D. - 0.7

N.D. = Not detectable

All the following pesticides were not detected in any of the samplings: p,p'-DDT, o,p'-DDT, endrin, ethyl parathion, methyl parathion, methyl bromophos, phorate, HCB, Δ BHC, Endosulfan I, Ronnel, Toxaphene, ethion.

Table 17. Air concentrations (ng/m^3) of pesticides and their ranges at four occupational work sites in South Florida, 1975.

Pesticides	South Dade Crop Dusting 3/20-21/75	Belle Glade Crop Dusting 5/19-20/75	Inside Formulation Plant 6/11-12/75	Inside Formulation Plant 12/12-13/75	Ranges of Four Work Sites
Chlorpyrifos	0.20	I	168	65	0.2 - 168
Diazinon	3.23	354	470	168	3.2 - 470
Malathion	N.D.	24	170	9	N.D.- 170
γBHC	1.5	I	3,977	2,301	1.5 - 3,977
Heptachlor	1.1	I	7,315	25,243	1.1 - 25,243
Aldrin	N.D.	I	655	N.D.	N.D.- 655
γBHC	3.5	1.0	4,791	3,779	1.0 - 4,791
Dieldrin	1.0	I	88	24	1.0 - 88
p,p'-DDT	N.D.	I	73	N.D.	N.D.- 73
o,p'-DDT	N.D.	I	43	N.D.	N.D.- 43
Endrin	3.3	I	19	N.D.	N.D.- 19
Ethion	1.1	N.D.	N.D.	N.D.	N.D.- 1.1
Ethyl Parathion	7.6	158	86	17	7.6 - 158
Methyl Parathion	4.5	131	182	I	4.5 - 182
Methyl Bromophos	N.D.	N.D.	4,130	1,715	N.D.- 4,130
Mevinphos	N.D.	4,650	9	I	N.D.- 4,650
Phorate	N.D.	N.D.	N.D.	1	N.D.- 1
HCB	N.D.	N.D.	N.D.	387	N.D.- 387
γChlordane	1.6	I	432	597	1.6 - 597
γChlordane	2.7	I	2,328	3,068	2.7 - 3,068
ΔBHC	N.D.	N.D.	N.D.	1,141	N.D.- 1,141
Endosulfan	6.3	N.D.	N.D.	N.D.	N.D.- 6.3
Ronnel	N.D.	N.D.	N.D.	2	N.D.- 2
Toxaphene	N.D.	Present	N.D.	N.D.	Present
Sample (m^3)	41.5	50	52	47.4	

N.D. = Not detectable
I = Interfering peaks caused by Toxaphene

B. Discussion

The major source of air contamination in South Florida is from areas where pesticides are indiscriminately mixed and loaded in the fields and inside pesticide formulating plants. General concentrations of pesticides in the ambient air from agriculture and horticulture use and in an area in close proximity of the formulating plant, were minimal.

Recently it has been proposed and approved by the Environmental Protection Council of Dade County to resort to open-air burning of pesticide containers. Previously, containers were disposed by destruction and burial. This procedure has been thought to threaten the water table resources of South Florida and because of a sizeable buildup of used containers in this area, the alternative solution of open-air burning has been approved for a period of six months. In the light of the air monitoring data presented in this report and in the light of this decision to resort to open-air burning, it seems to be imperative that the effect of this new method of air dispersal in this area should be monitored for the future.

Finally, DDT has disappeared, except for inside the formulating plant. Malathion is detectable only during the mosquito control programs, and dichlofenthion, commensurate with no further production of this product, is no longer found in the suburban air sampling site.

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APPENDICES

A. General Population Raw Data

B. Adipose Pesticide Residue Raw Data

C. Air Monitoring Raw Data

Appendix A. Urinary metabolite concentrations in grab sample urines from 40 members of the general population in South Florida, 1975

Age & Sex	Occupation	ALKYL PHOSPHATE (ug/ml)				PHENOLS (ug/ml)		
		DMP	DMTP	DEP	DETP	3,5,6-TCP	PCP	2,4,5-TCP
32/f	Cashier	0.053	0.22	0.11	0.022	0.006	0.016	N.D.
69/f	Housewife	0.010	0.040	0.008	0.021	N.D.	0.002	N.D.
23/m	Lab Technician	0.040	0.075	0.043	0.037	N.D.	0.004	N.D.
23/f	Secretary	0.29	0.024	0.021	0.037	N.D.	0.032	N.D.
74/f	Housewife	0.011	0.021	0.012	0.021	0.006	0.004	N.D.
28/f	Housewife	0.010	0.018	0.021	N.D.	N.D.	0.020	N.D.
4/m	Adolescent	0.008	N.D.	0.021	N.D.	N.D.	0.016	N.D.
35/m	Hairdresser	0.023	0.034	0.010	N.D.	N.D.	0.022	N.D.
46/m	Draftsman	N.D.	N.D.	N.D.	N.D.	N.D.	0.016	N.D.
10/m	Student	N.D.	N.D.	N.D.	N.D.	N.D.	0.004	N.D.
42/f	Seamstress	0.003	0.012	0.015	0.030	0.008	0.006	0.1
39/m	Asst. Professor	0.003	N.D.	0.08	N.D.	0.008	0.010	N.D.
49/f	Counselor	0.004	0.015	0.010	N.D.	0.024	0.010	N.D.
36/m	Manager	0.005	N.D.	0.007	N.D.	0.006	0.012	N.D.
32/f	Secretary	N.D.	N.D.	N.D.	N.D.	N.D.	0.010	N.D.
24/f	Lab Technician	0.010	0.027	0.019	0.042	N.D.	0.008	N.D.
40/m	Lab Supervisor	N.D.	0.035	N.D.	N.D.	N.D.	0.002	0.014
21/m	Mail Clerk	N.D.	N.D.	0.006	0.033	0.032	0.006	N.D.
55/m	Mail Supervisor	N.D.	N.D.	N.D.	0.030	N.D.	0.004	N.D.
51/m	College Professor	N.D.	N.D.	N.D.	N.D.	N.D.	0.006	N.D.
33/m	Lab Technician	N.D.	N.D.	N.D.	N.D.	N.D.	0.001	N.D.
26/m	Airplane Mech.	0.009	N.D.	0.011	N.D.	N.D.	0.012	0.004
21/f	Secretary	0.007	0.026	0.006	0.029	N.D.	0.010	N.D.
29/m	Lab Techician	0.010	0.027	0.007	0.019	0.006	0.006	N.D.
19/f	Secretary	N.D.	N.D.	N.D.	N.D.	N.D.	0.006	N.D.
48/m	Unemployed Lab.	0.012	N.D.	0.013	N.D.	N.D.	0.006	N.D.
16/m	Unemployed	0.024	N.D.	0.016	N.D.	N.D.	0.020	0.004
21/m	Lab Technician	0.017	N.D.	0.005	N.D.	0.008	0.006	N.D.
50/f	Reg. Nurse	N.D.	N.D.	0.028	N.D.	N.D.	0.002	N.D.
36/m	Reg. Nurse	N.D.	N.D.	N.D.	N.D.	N.D.	0.002	N.D.
45/m	Plasterer	0.007	N.D.	0.027	N.D.	0.002	0.006	N.D.
23/f	Packing Clerk	N.D.	N.D.	N.D.	N.D.	N.D.	0.023	N.D.
/f	Housewife	N.D.	N.D.	N.D.	N.D.	N.D.	0.002	N.D.
38/f	Chemist	N.D.	0.08	0.04	0.04	0.008	0.016	N.D.
49/f	Chemist	N.D.	0.04	N.D.	N.D.	N.D.	0.015	0.04
53/m	Physician	0.03	0.04	0.04	0.073	N.D.	0.008	N.D.
50/m	Investigator	N.D.	0.16	N.D.	0.05	Not Done	Not done	Not done
72/m	Retired	0.03	0.08	0.04	0.3	N.D.	0.009	N.D.
4½/m	Adolescent	0.03	0.04	0.04	0.04	Not Done	Not done	Not done
51/f	Housewife	N.D.	0.04	N.D.	0.04	Not Done	Not done	Not done

N.D.= Not detected

Appendix B. Individual levels of organochlorine and organophosphorus pesticide residues in adipose tissue from 20 patients coming to surgery in a local hospital. Dade County, 1975

Name	Age, race and sex	p,p'-DDT ppm	p,p'-DDE ppm	o,p'-DDT ppm	Total DDT ppm	Oxychlor-dane ppm	HCB ppm	B-BHC ppm	Hep. Epox.	Dieldrin ppm	OP ppm
M.R.	19 y/o wm	0.11	1.04	N.D.	1.27	0.072	0.07	0.03	0.04	0.08	N.D.
M.L.	38 y/o wf	3.05	9.94	0.15	14.25	0.136	0.11	0.23	0.05	0.07	N.D.
F.T.	33 y/o bf	1.90	9.39	N.D.	12.35	0.050	0.06	0.10	0.03	0.20	N.D.
J.O.	48 y/o bf	0.82	8.59	Inter.	10.39	0.205	0.05	0.35	0.05	0.10	N.D.
O.M.	23 y/o bm	0.45	11.20	0.24	13.16	0.065	0.02	0.04	0.07	0.04	N.D.
A.D.	34 y/o wm	0.53	2.81	0.11	3.77	0.103	0.04	0.15	0.07	0.27	N.D.
E.N.	50 y/o wm	0.53	3.70	N.D.	4.65	0.390	0.02	0.08	Inter.	0.05	N.D.
W.P.	65 y/o bm	1.02	6.41	0.18	8.34	0.281	0.06	0.30	0.08	0.47	N.D.
P.M.	64 y/o wm	0.64	4.72	0.12	6.00	0.280	0.09	0.17	0.10	0.08	N.D.
C.H.	70 y/o wm	0.35	2.12	0.97	2.80	0.138	0.08	0.10	0.11	0.11	N.D.
C.H.	67 y/o wf	1.12	8.92	0.09	11.13	0.122	0.08	0.62	0.03	0.06	N.D.
A.G.	52 y/o wf	0.47	2.57	0.06	3.38	0.092	0.02	0.14	0.06	0.02	N.D.
X.A.	39 y/o wf	0.72	3.46	0.26	4.82	0.080	0.06	0.11	0.08	0.67	N.D.
G.C.	51 y/o bf	0.20	1.00	0.11	1.43	0.087	0.03	0.16	0.04	0.06	N.D.
R.E.	77 y/o bf	0.74	10.15	0.64	12.69	0.058	0.07	0.15	0.04	0.11	N.D.
J.H.	47 y/o wf	2.43	9.33	0.22	13.03	0.084	0.06	0.25	0.04	0.37	N.D.
C.M.	35 y/o bf	1.56	4.28	0.18	6.50	0.103	0.03	0.21	0.05	0.17	N.D.
P.S.	43 y/o wf	0.32	1.02	0.12	1.57	0.037	0.03	0.11	0.04	0.08	N.D.
B.B.	22 y/o wf	0.40	3.31	0.33	4.40	0.046	0.05	0.36	0.27	0.05	N.D.
C.E.	32 y/o bf	1.13	11.46	0.33	14.22	0.096	0.02	0.02	0.49	0.07	N.D.

N.D. - Not detectable

Limits of detectability are: p,p'-DDT 0.09, p,p'-DDE 0.034, o,p'-DDT 0.081, oxychlordane 0.018, HCB 0.008, B BHC 0.015, heptachlor epoxide 0.011 and dieldrin 0.017.

Inter. - Interference, unable to calculate

Appendix C-1. Concentrations of pesticides (ng/m^3) in air samples collected from several sites in South Florida 1975 - 1976

Pesticide	Air #1 ng/m^3	Air #2 ng/m^3	Air #3 ng/m^3	Air #4 ng/m^3	Air #5 ng/m^3	Air #6 ng/m^3	Air #7 ng/m^3	Air #8 ng/m^3	Air #9 ng/m^3	Air #10 ng/m^3
Dichlofenthion	N.D.	0.22	N.D.	N.D.	N.D.	0.3	N.D.	N.D.	1.4	N.D.
Chlorpyrifos	N.D.	3.10	0.72	0.20	0.3	0.5	N.D.	1.6	3.6	1.0
Diazinon	3.2	2.9	3.3	3.23	1.1	1.9	1.5	3.0	5.2	2.2
Malathion	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	7.2	N.D.	N.D.
α BHC	0.10	0.24	0.16	1.5	0.6	0.3	0.4	0.7	0.7	0.4
Heptachlor	0.22	1.3	0.42	1.1	1.2	1.2	0.9	0.8	2.0	8.7
Aldrin	N.D.	0.47	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
γ BHC	0.1	0.26	0.1	3.5	0.6	0.4	0.3	0.5	1.3	0.2
Dieldrin	N.D.	0.49	0.16	1.0	0.2	0.3	N.D.	0.4	0.9	0.4
Endrin	N.D.	N.D.	N.D.	3.3	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
Ethion	N.D.	N.D.	N.D.	1.1	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
Ethyl Parathion	N.D.	N.D.	N.D.	7.6	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
Methyl Parathion	N.D.	N.D.	N.D.	4.5	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
α Chlordane	N.D.	0.36	0.16	1.6	0.3	0.7	0.5	1.1	1.6	3.0
γ Chlordane	N.D.	1.2	0.49	2.7	0.5	1.0	0.7	0.9	2.8	12.7
Endosulfan I	N.D.	N.D.	N.D.	6.3	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
m^3	37.2	50.9	51.7	41.5	53	54	36	38	50	50
Location	Everglades	Suburban	Airport	Tri-State	SDCHC	Suburban	Airport	SDCHC	Suburban	Suburban-Ft. Lauderdale
Wind-Start	E-NE	Calm	W	Calm	NE	SE	S-SE	E-SE	E-SE	SE
Finish	E	E	E	E	Calm	S-SE	S	N	E-SE	E
Date Collected	2/11-12	2/11/12	2/12-14	3/20-21	3/21-22	4/9-10	4/9-10	5/8-9	5/8-9	5/10-11/75

ND-Not detectable

All the following pesticides were not detectable: β BHC, HCB, Carbofenthion, p,p'-DDT, o,p'-DDT, mevinphos, methyl bromophos, chlorbenside, phorate, heptachlor epoxide, methoxychlor, ronnel, Thiodan I and II.

Appendix C-2. Concentrations of pesticides (ng/m^3) in air samples collected from several sites in South Florida 1975-1976

Pesticide	Air #11 ng/m^3	Air #12 ng/m^3	Air #13 ng/m^3	Air #14 ng/m^3	Air #15 ng/m^3	Air #16 ng/m^3	Air #17 ng/m^3	Air #18 ng/m^3	Air #19 ng/m^3	Air #20 ng/m^3
Dichlofenthion	N.D	N.D.	0.4	N.D.	N.D.	N.D.	0.3	N.D.	0.2	N.D.
Chlorpyrifos	Interf.	0.5	3.0	168	1.6	0.8	11.5	1.6	1.0	N.D.
Diazinon	354	2.5	6.2	470	2.9	N.D.	N.D.	N.D.	N.D.	N.D.
Malathion	24	6.0	N.D.	170	N.D.	2.9	9.2	N.D.	1.5	N.D.
α BHC	Interf.	0.1	0.2	3,977	0.3	0.3	0.5	0.5	0.9	0.5
Heptachlor	Interf.	0.6	1.3	7,315	1.0	1.0	1.1	1.5	1.0	0.7
Aldrin	Interf.	N.D.	N.D.	655	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
Lindane	1.0	0.2	0.2	4,791	0.4	N.D.	N.D.	N.D.	N.D.	N.D.
Dieldrin	Interf.	0.2	0.7	88	0.3	N.D.	N.D.	N.D.	N.D.	N.D.
p,p'- DDT	Interf.	N.D.	N.D.	73	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
o,p'-DDT	Interf.	N.D.	N.D.	43	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
Endrin	Interf.	N.D.	N.D.	19	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
Ethyl parathion	158	N.D.	N.D.	86	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
Methyl parathion	131	N.D.	N.D.	182	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
Methyl bromophos	N.D.	N.D.	N.D.	4,130	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
Phosdrin	4,650	3.0	N.D.	9	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
α Chlordane	Interf.	0.4	0.6	432	0.3	0.7	0.8	0.7	0.7	0.2
γ Chlordane	Interf.	1.3	2.1	2,328	1.4	2.2	2.6	2.0	1.7	0.7
Toxaphene	Interf.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
m^3	50	51	47	52	40	36	51	51	51	51
Location	Belle Glade Airport	Miami Airport	Suburban	Formulating Plant	SDCHC	SDCHC	Suburban	Airport	Suburban	Everglades
Wind Direction										
Start:	Calm	SE	E-SE	Indoors	E-SE	Calm	S-SE	W-SW	E-SE	S-SE
Finish:	E	SE	E-SE		E	S-SE	E-SE	SE	SE	SW
Date Collected	5/19-20/75	6/10-11	6/10-11	6/11-12	6/11-12	7/7-8	7/7-8	7/22-23	7/15-16	7/23-24

N.D.-Not detectable
Interf.-Interference

All the following pesticides were not detected: β-BHC, HCB, Trithion, Ethion, Ronnel, Phorate, Fenthion, Chlorobenside, Methoxychlor, Thiodan I, Thiodan II and Heptachlor Epoxide.

Appendix C-3. Concentrations of pesticides (ng/m^3) in air samples collected from several sites in South Florida 1975-1976

Pesticide	Air #21 ng/m^3	Air #22 ng/m^3	Air #23 ng/m^3	Air #24 ng/m^3	Air #25 ng/m^3	Air #26 ng/m^3	Air #27 ng/m^3	Air #28 ng/m^3	Air #29 ng/m^3	Air #30 ng/m^3
Dichlofenthion	N.D.	0.3	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
Chlorpyrifos	0.7	0.7	0.6	1.5	1.5	0.6	0.5	0.6	65	0.3
Diazinon	3.8	3.6	17.6	5.4	4.2	0.6	0.8	0.6	168	0.8
Malathion	1.1	6.1	1.3	N.D.	N.D.	1.0	N.D.	N.D.	9	N.D.
α BHC	1.0	0.3	1.5	1.3	2.2	0.9	1.0	0.4	2,301	N.D.
Heptachlor	0.9	0.6	2.1	2.2	1.0	0.5	2.1	0.8	25,243	3.9
γ BHC	1.2	0.7	0.4	0.8	1.0	0.4	0.7	0.3	3,779	0.1
Ethyl parathion	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	17	N.D.
Methyl parathion	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	Interf.	N.D.
Bromophos	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	1,715	N.D.
Mevinphos	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	Interf.	N.D.
Phorate	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	1	N.D.
HCB	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	387	N.D.
α chlordane	1.1	0.6	1.8	1.2	0.9	0.2	0.3	0.3	597	0.2
γ chlordane	0.5	0.7	2.0	1.3	0.8	0.6	0.7	0.8	3,068	0.9
Dieldrin	1.0	0.6	0.5	0.7	0.7	0.2	0.2	0.2	24	0.2
Δ BHC	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	1,141	N.D.
Ronnel	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	2	N.D.
m^3	48	52	52	37	38	52.7	37.5	50.3	47.4	38.6
Location	Suburban	Suburban	Suburban	SDCHC	SDCHC	Airport	SDCHC	Suburban	Form. Plt.	SDCHC
Wind Direction										
Start:	S-SE	E-SE	E	E	E	SE	E-SE	No Data	Indoors	E
Finish:	S-SE	Calm	NE	S	NE	E	Variable			E-NE
Date Collected	8/18-19	9/21-22	10/11-12	9/22-23	10/11-12	10/16-17	11/25-26	12/9-10	12/12-13	12/12-13

N.D.-Not detectable
Interf.-Interference

All the following pesticides were not detected: β BHC, HCB, Trithion, Ethion, Fenthion, Chlorobenside, Methoxychlor, Thiodan I, Thiodan II, Toxaphene, Heptachlor Epoxide, Endosulfan I, and Endosulfan II

Appendix C-4. Concentration of pesticides (ng/m^3) in air samples collected from several sites in South Florida 1975-1976

Pesticide	Air #31	Air #32	Air #33	Air #34	Air #35	Air #36	Air #37	Air #38	Air #39	Air #40
Dichlofenthion	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
Chlorpyrifos	22.3	4.5	1.35	0.77	0.51	0.80	1.00	N.D.	1.40	N.D.
Diazinon	5.9	1.5	0.52	0.63	N.D.	N.D.	N.D.	N.D.	N.D.	N.D.
α BHC	0.3	0.3	0.20	0.30	0.10	0.10	0.20	0.13	0.21	0.17
γ BHC	0.6	0.6	0.34	0.33	0.15	0.12	0.20	0.16	0.22	0.15
α Chlordane	0.9	1.0	0.35	0.41	0.39	0.30	0.38	0.16	0.42	0.15
γ Chlordane	0.9	1.0	0.98	1.06	0.41	0.30	0.43	0.19	0.50	0.19
Heptachlor	1.7	1.4	1.99	3.64	0.34	0.35	0.66	0.64	0.72	0.67
Dieldrin	0.3	0.5	0.22	N.D.	0.26	N.D.	0.28	0.30	N.D.	0.27
m^3	52.5	46.3	48	49	53	52	52	36	49	37
Place	Airport	Suburban	Suburban	Airport	Suburban	Airport	Suburban	So. Dade Comm. Hlth	Suburban	So.Dade Comm.
Wind - Start	NW	NW	N	E	NE-E	E-NE	E-NE	E-SE	SE	E-SE
Finish	N-rain	N	NE	E	E	E	E	SE	E	E
Date Collected	1/15-16/76	1/15-16	2/10-11	2/10-11	3/2-3	3/2-3	3/5-6	3/5-6	3/15-16	3/16-17

N.D. = Not detectable

All the following pesticides were not detectable: Dichlofenthion, malathion, aldrin, lindane, p,p'-DDT, o,p'-DDT, aldrin, ethyl parathion, methyl parathion, Δ BHC, HCB, ronnel, trithion, ethion, phorate, bromophos, fenthion, methoxychlor, heptachlor epoxide, Thiodan I and Thiodan II.

GLOSSARY

Alkyl phosphates	degradation products; urinary metabolites
BHC	1,2,3,4,5,6 hexachlorocyclohexane (mixtures of isomers)
Bromophos	O-(4-Bromo-2,5-dichlorophenyl) O,O-dimethyl phosphorothioate
Carbaryl	1-naphthyl N-methylcarbamate
Chlorpyrifos (Dursban)	O,O-diethyl O-(3,5,6 trichloro-2-pryidyl) phosphorothioate
cholinesterase	An esterase present in all body tissues which hydrolyzes acetylcholine into choline and acetic acid
DEF	s,s,s-tributyl phosphorothioate
Diazinon	O,O-Diethyl O-(2-isopropyl-6-methyl-4-pyrimidinyl) phosphorothioate
Dichlofenthion (VC-13)	O,O-Diethyl-O-(2,4-dichlorophenyl) phosphorothioate
Dichlorvos (DDVP)	2,2-Dichlorovinyl dimethyl phosphate
Dimethoate (Cygon)	O,O-Dimethyl-S-(N-methylcarbamoylmethyl) phosphorothioate
Ethyl parathion	O,O-Diethyl O-p-nitrophenyl phosphorothioate
HCB	Hexachlorobenzene (fungicide)
Heptachlor	1,4,5,6,7,8,8-Heptachloro-3a,4,7,7a-tetrahydro-4,7-methanoindene
Heptachlor Epoxide	1,4,5,6,7,8,8-Heptachloro-6,7-epoxy-3a, 4,7,7a-tetrahydro-4,7-methanoindene
Lindane	Gamma isomer of benzene hexachloride (1,2,3,4,5,6-hexachlorocyclohexane) of 99+% purity
LD50	in toxicity studies it is the dosage required to kill 50% of the test animals when given a single dosage by mouth or skin contact. The dose is expressed by the weight of the chemical per unit of body weight.
Malathion	O,O-Dimethyl S-[1,2-bis(ethoxy-carbonyl) ethyl] phosphorodithioate

Methyl parathion	0,0-Dimethyl o-p-nitrophenyl phosphorothioate
Mevinphos (Phosdrin)	0,0-Dimethyl 1-carbomethoxy-1 propen-2-yl phosphate
M.R.I.	Air sampling unit built by Midwest Research Institute and used in air monitoring studies by E.P.A.
Naled (Dibrom)	1,2-Dibromo-2,2-dichloro-ethyl dimethyl phosphate
Not Detected	Below the limits of detectability in the gas chromatograph
PCP	2,3,4,5,6 Pentachlorophenol
PNP	paranitrophenol - a urinary metabolite of parathion, EPN, methyl parathion and chlorthion
p,p'-DDT	1,1-Bis-(p-chlorophenyl)-2,2,2-trychloroethane
p,p'-DDE	2,2-Bis-(p-chlorophenyl) 1,1-dichloroethylene
o,p'-DDT	1-(O-chlorophenyl)-1-(p-chlorophenyl) 2,2,2-trichloroethane
o,p'-DDE	1-(o-chlorophenyl)-1-(p-chlorophenyl)-2,2-dichloroethylene
2,4-DCP	2,4-Dichlorophenol
2,4-D esters	2,4-dichlorophenoxy acetic acid
2,4,5-T esters	2,4,5-trichlorophenoxy acetic acid
2,3,4,6-TTCP	2,3,4,6 tetrachlorophenol

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16. ABSTRACT

Studies of the urinary pesticide metabolites demonstrated that alkyl phosphates were highly sensitive indices of recent exposures to DMP and DEP organophosphate pesticides. In contrast, urinary phenolic metabolites when available provided sensitive Urinary metabolites furthered laboratory confirmation of poisonings and facilitated the recognition of subtle differences in organophosphate exposures, properties which lend themselves to use in human monitoring and epidemiologic studies of the occupationally exposed. Urinary alkyl phosphate excretions permitted the recognition of worker protection from residues in the field acquired by wearing fluorocarbon treated clothing. Being group specific rather than pesticide specific, urinary alkyl phosphates were unsatisfactory surveillance instruments when cholinesterase depression or illness was the disease end point of concern and when pesticide exposures were mixed. Important sources of pesticide exposure in a formulating plant included high concentrations in the air and contaminated workers' clothing. Bromophos, a fat soluble organophosphate, was identified in fat for the first time in three formulators, and modification of the analytical laboratory procedures for pesticides in air and in fat increased the number of pesticides identified in air and the recognition of the less polar organophosphates in fat.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
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