

WORKER EXPOSURE TO PESTICIDES IN FLORIDA CITRUS OPERATIONS¹

G. A. WOJECK AND H. N. NIGG
University of Florida, IFAS,
Agricultural Research and Education Center,
P. O. Box 1088, Lake Alfred, FL 33850

Abstract. Studies on the assessment of worker exposure to pesticides identify the sources of pesticide contamination to the worker and indicate any preventive measure necessary to assure a safe work place for agricultural laborers. Methods currently employed in monitoring worker exposure to pesticides were reviewed. Studies on exposure to lead arsenate, ethion, and paraquat used in Florida citrus have shown that the hands receive the highest exposure levels. Wearing vinyl gloves would eliminate up to 75% of the workers' total dermal exposure.

Registration of new pesticides and re-registration of older products often are dependent upon minimizing the degree of exposure sustained by the pesticide applicator. Only a limited base of knowledge is now available on worker exposure to pesticides. Unless an agricultural laborer is wearing adequate protective clothing, he is subject to potential exposure from handling pesticides while mixing and loading concentrated formulations, pesticide drift during application, accidental spills, leaking equipment, cleaning clogged spray nozzles, repairing contaminated equipment (2, 5, 11, 12, 17, 19, 21, 22, 23, 24), wearing contaminated clothing (16), smoking, eating, or drinking in the field (1, 20) and from pesticide residues on recently treated crops (15, 16). The primary route of pesticide exposure has been shown to be dermal, although respiratory exposure increased when dusts were applied (2, 5, 11, 12, 21).

Attempts have been made to protect workers from pesticide exposure by legislative action. The Environmental Protection Agency has proposed guidelines for registering pesticides that include an evaluation of hazards to man (8). Reentry period restrictions were established to protect workers from pesticide residues on treated crops (7). Monitoring worker exposure to pesticides provides information on which to base these regulations.

The purpose of this paper is to briefly review methods currently employed in assessing levels of potential dermal and respiratory exposure of workers to pesticides and to discuss examples of pesticide exposure monitoring studies conducted in Florida citrus.

Review of Methods

Potential exposure is defined as the maximum possible exposure of workers to pesticides, whether from inhalation, direct contact of the skin with pesticides, or indirect exposure from contact of the skin with contaminated clothing. Procedures for estimating potential dermal and respiratory exposure to pesticides were described by Durham and Wolfe (3). Dermal exposure pads made of α -cellulose or 16-ply surgical gauze backed with filter paper were attached to the forearms, shoulders, thighs, shins, and chest. Finley, et al. (10) used patches cut from clothing worn by workers during the exposure period to assess dermal exposure. Exposure to the hands was estimated from pesticide residues

impinged on areas cut from the palms and backs of clean cotton gloves worn by each worker during the monitoring period. Dermal exposure of the hands was also determined from analysis of pesticide residues rinsed from the hands with water or other solvents. Respirators fitted with 16-ply gauze backed with filter pads were used to assess respiratory exposure, under the assumption that the respirator filters were totally efficient in trapping respirable pesticides. An improperly fitted mask would reduce efficiency and thereby, increase experimental error. Air samples collected near the breathing area of workers have also been used to assess respiratory exposure (3).

The dermal exposure pads, respirators, gloves, and air samplers were worn for a carefully timed period while workers mixed, loaded, or applied pesticides. At the end of the monitoring period, all samples were returned to the laboratory for analysis of pesticide residues.

Durham and Wolfe (4) indicated that only 0.1 to 2.8% of the potential dermal exposure to parathion was actually absorbed. All particles or droplets less than 7 μ in diameter were taken into the respiratory tract. Respiratory exposure was arbitrarily assumed to be 10 times more toxic than dermal exposure (4). Since many pesticides or their metabolites are excreted in the urine, urinary levels of pesticides may be used to estimate absorption of pesticides. Biochemical effects of organophosphorus and carbamate pesticides can be measured by changes in blood cholinesterase (ChE) activity (6).

Examples of Monitoring Studies in Florida Citrus

Lead arsenate. Two suppliers, who mixed the concentrated pesticides and filled the spray tank, and 3 applicators, operating tractor-drawn air blast sprayers, were monitored for arsenic exposure on 9 days during the 5-week spray season (22). The average levels of dermal exposure to arsenic for applicators and suppliers were 69 mg/hr and 109 mg/hr, respectively (Table 1). The 5 men were divided into 2 crews. Although, Crew I applied roughly the same amount of lead arsenate in a 0.1% spray mixture as Crew II applied in a 0.3% spray mixture, the average dermal exposure of arsenic to workers in Crew I was 40% lower than the average exposure of workers in Crew II. Estimated respiratory exposure accounted for less than 1% of the total

Table 1. Exposure of workers to lead arsenate, ethion, and paraquat (22, 23, 24).

| | Suppliers | Applicators |
|-----------------------------------|-----------|-------------|
| Arsenic | | |
| Mean dermal exposure (mg/hr) | 109 | 69 |
| Mean respiratory exposure (mg/hr) | 0.005 | 0.005 |
| Mean urinary excretion (ppb As) | 231 | 86 |
| Ethion | | |
| Mean dermal exposure (mg/hr) | 1799 | 1972 |
| Mean respiratory exposure (mg/hr) | 0.005 | 0.004 |
| Urinary metabolites | | |
| Mean ppb DETP | 266 | 133 |
| Mean ppb DEDTP | 350 | 183 |
| ChE (% change) | -10.4 | +2.5 |
| Paraquat | | |
| Mean dermal exposure (mg/hr) | | 22 |
| Mean respiratory exposure (mg/hr) | | 0.004 |

¹Florida Agricultural Experiment Stations Journal Series No. 2789.

arsenic exposure. The mean dermal exposure for all workers in a 10-hr day was 850 mg, a value much lower than the short-term no-effect level for arsenic (100 mg/kg body weight or 7,000 mg total arsenic exposure for a 70 kg man) established by the Environmental Protection Agency (9). Exposure was significantly higher for the hands and legs than for other areas of the body (Fig. 1). Accumulated total body exposure to arsenic was correlated with the urinary arsenic concentration ($R = 0.73$). Although no worker exhibited symptoms of clinical arsenic poisoning, both suppliers in the study exceeded the 200 ppb level of urinary arsenic which is considered suggestive of arsenic poisoning (14). The mean applicator urinary arsenic levels were well below the threshold limit for arsenic excretion.

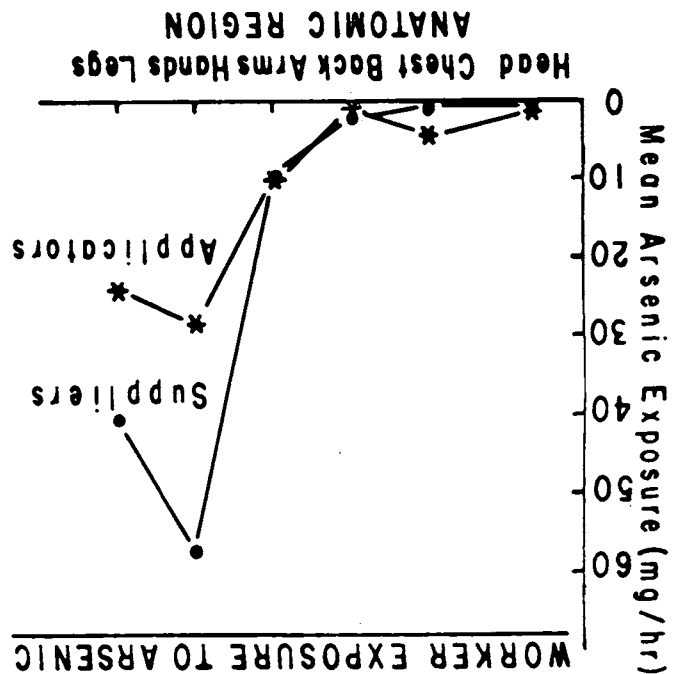


Fig. 1. Worker exposure to arsenic (mg/hr) by anatomical region (22).

Ethion. Seventeen men were monitored for exposure to ethion applied with airblast spray equipment (23). The mean dermal exposure to ethion was 1799 mg/hr for suppliers and 1972 mg/hr for applicators (Table 1). Respiratory exposures to ethion were 0.005 mg/hr and 0.004 mg/hr for suppliers and applicators, respectively. Exposure to the hands was significantly higher than for other anatomical regions. Suppliers received a significantly greater percentage of dermal exposure to the hands than did the applicators. The percentage of total dermal dose of ethion for the head and neck, chest and arms was significantly higher for applicators (Fig. 2).

The only metabolites of ethion found in the urine were DETP (diethyl thiophosphate) and DEDTP (diethyl dithiophosphate). The mean concentrations of these dialkyl phosphates excreted by suppliers were 266 ppb DETP and 350 ppb DEDTP (Table 1). Applicators excreted significantly lower concentrations. A significant correlation was found between the group mean ppb DETP, DEDTP levels and ethion exposure to the hands on a given day. The positive correlation between the concentration of dialkyl phosphates excreted by suppliers was higher than the correlation of applicators, suggesting greater absorption by the hands in comparison to other anatomical regions. Greater decreases in CHE activity also indicate greater absorption of ethion by suppliers (Table 1). There were no

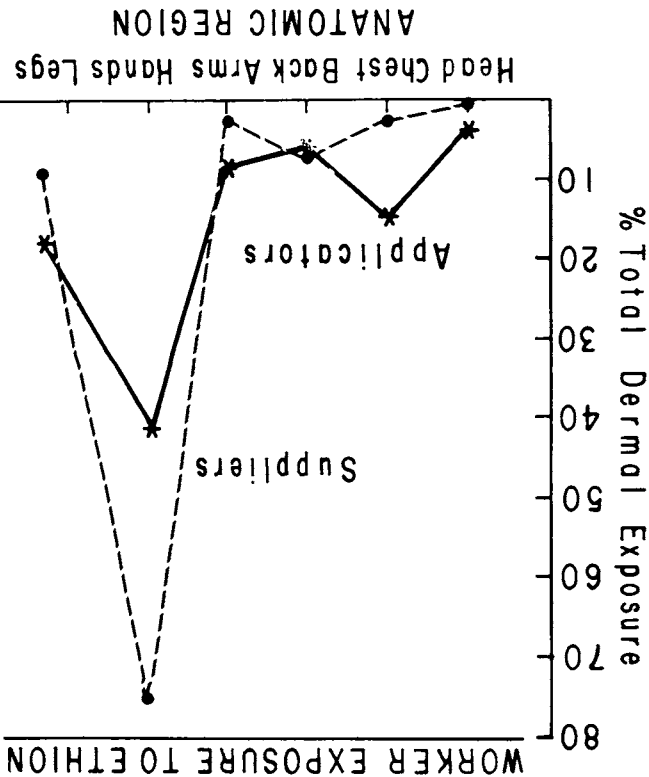


Fig. 2. Worker exposure to ethion (% of the total dermal exposure) by anatomical region (23).

overall significant differences between dermal exposure to suppliers and applicators.

Paraquat. Paraquat was applied by 5 men using tractor-mounted low-boom spray equipment (24). The mean dermal exposure to paraquat was 22 mg/hr (Table 1). The highest exposures were to the hands and legs (Fig. 3).

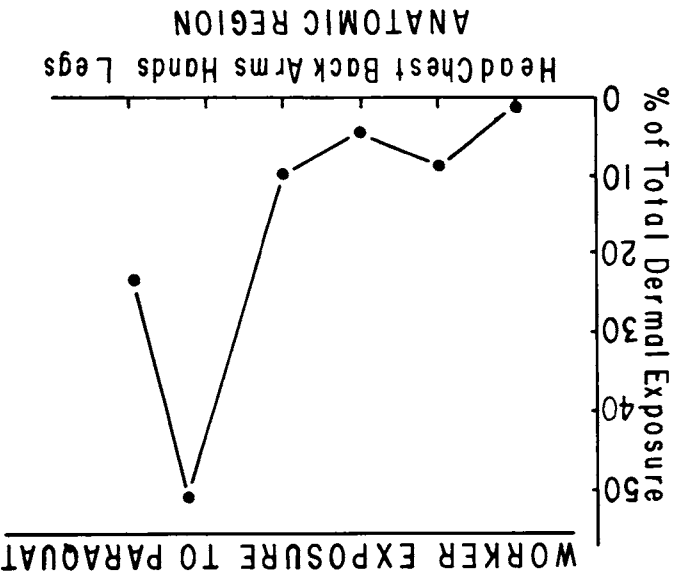


Fig. 3. Worker exposure to paraquat (% of total dermal exposure) by anatomical region (24).

Conclusions

Workers applying ethion received the highest exposure levels. Application of lead arsenate resulted in the second highest exposure levels. Application of paraquat with a low boom sprayer resulted in the lowest exposure. Although,

the exposure levels of all 3 compounds were below the levels expected to result in acute hazard to workers, preventive practices such as wearing elbow-length vinyl gloves while mixing and loading concentrated formulations could reduce dermal exposure by 50 to 75% (22, 23). Other precautionary measures include keeping spray equipment in good condition, providing shower facilities and requiring workers to use them, encouraging workers to wear frequently laundered heavy cotton clothing, teaching the workers to have a healthy respect for all pesticides, and instructing workers what to do in case of accidental spills. The use of a closed system for handling concentrated formulations and loading spray machines would also eliminate a major source of pesticide contamination (13). Although, impermeable clothing provides excellent protection if properly used, covering skin already contaminated with pesticides may lead to greater absorption (18). The use of impermeable clothing may also increase absorption by workers applying fumigants.

Literature Cited

1. Armstrong, J. F., H. R. Wolfe, S. W. Comer, and D. C. Staiff. 1973. Oral exposure of workers to parathion through contamination of food items. *Bul. Environ. Contam. Toxicol.* 10:321-327.
2. Batchelor, G. S. and K. C. Walker. 1954. Health hazards involved in use of parathion in fruit orchards of North Central Washington. *Arch. Indust. Hyg. Occup. Med.* 10:522-529.
3. Durham, W. F., and H. R. Wolfe. 1962. Measurement of the exposure of workers to pesticides. *Bul. Wld. Health Org.* 29:75-91.
4. Durham, W. F. and H. R. Wolfe. 1963. An additional note regarding measurement of exposure of workers to pesticides. *Bul. Wld. Health Org.* 29:279-281.
5. ———, ———, and J. W. Elliot. 1972. Absorption and excretion of parathion by spraymen. *Arch. Environ. Health* 24:381-387.
6. Ellman, G. L., K. D. Courtney, V. Andres, and R. M. Featherstone. 1961. A new and rapid colorimetric determination of acetyl cholinesterase activity. *Biochem. Pharmacol.* 7:88-95.
7. *Federal Register*. 1974. Worker protection standards for agricultural workers. 39:16888.
8. *Federal Register*. 1978. Proposed guidelines for registering pesticides in the U.S. 43:37336-37403.

9. *Federal Register*. 1978. Standard for exposure to inorganic arsenic. 43:48375.
10. Finley, E. L., J. B. Graves, F. C. Hewitt, H. F. Morris, C. W. Harmon, F. A. Iddings, P. E. Schilling, and K. L. Koonce. 1979. Reduction of methyl parathion residues on clothing by delayed field re-entry and laundering. *Bul. Environ. Contam. Toxicol.* 22:590-597.
11. Griffiths, J. T., C. R. Stearns, and W. L. Thompson. 1951. Parathion hazards encountered spraying citrus in Florida. *J. Econ. Entomol.* 44:160-163.
12. Griffiths, J. T., J. W. Williams, C. R. Sterns, and W. L. Thompson. 1951. Health status of parathion when used on citrus in 1951. *Proc. Fla. State Hort. Soc.* 64:79-82.
13. Knaak, J. B., T. Jackson, A. J. Fredrickson, L. Rivera, K. T. Maddy, and N. B. Akesson. 1980. Safety effectiveness of closed-transfer, mixing-loading, and application equipment in preventing exposure to pesticides. *Arch. Environ. Contam. Toxicol.* 9:231-245.
14. Kyle, R. A. 1970. Inorganic arsenic intoxication. In *Laboratory Diagnosis of Diseases Caused by Toxic Agents*. Warren H. Green, Inc., St. Louis. pp. 367-370.
15. Milby, T. H., F. Ottoboni, and H. W. Mitchell. 1964. Parathion residue poisoning among orchard workers. *J. Amer. Med. Assoc.* 189:97-102.
16. Quinby, G. E. and A. B. Lemmon. 1958. Parathion residues as a cause of poisoning in crop workers. *J. Amer. Med. Assoc.* 166:740-746.
17. Williams, J. W. and J. T. Griffiths. 1951. Parathion poisoning in Florida citrus spray operations. *J. Fla. Med. Assoc.* 37:707-709.
18. Wolfe, H. R. 1973. Workers should be protected from pesticide exposure. *Weeds Trees and Turf* 12:1-4.
19. ———, J. F. Armstrong, D. C. Staiff, and S. W. Comer. 1972. Exposure of spraymen to pesticides. *Arch. Environ. Health* 25:29-31.
20. ———, ———, and ———. 1975. Potential exposure of workers to parathion through contamination of cigarettes. *Bul. Environ. Contam. Toxicol.* 13:369-376.
21. ———, W. F. Durham, and J. F. Armstrong. 1967. Exposure of workers to pesticides. *Arch. Environ. Health* 14:622-633.
22. Wojcik, G. A., H. N. Nigg, R. S. Braman, J. H. Stamper, and R. L. Rouseff. 1980. Worker exposure to arsenic on grapefruit in Florida. *Fund. Appl. Toxicol.* (Submitted).
23. ———, ———, J. H. Stamper, and D. E. Bradway. 1980. Worker exposure to ethion in Florida citrus. *Arch. Environ. Contam. Toxicol.* (in press).
24. ———, J. Price, and H. N. Nigg. 1980. Worker exposure to paraquat and diquat. (Manuscript).

Proc. Fla. State Hort. Soc. 93:62-64. 1980.

USE OF MILD STRAINS OF CITRUS TRISTEZA VIRUS (CTV) TO REESTABLISH COMMERCIAL PRODUCTION OF 'PERA' SWEET ORANGE IN SAO PAULO, BRAZIL

G. W. MÜLLER

*Virus Department, Instituto Agronomico
13.100 Campinas, SP., Brazil*

Additional index words. sour orange, tolerant rootstocks, stem pitting.

Abstract. 'Pera' sweet orange (*Citrus sinensis* (L.) Osb.) is susceptible to CTV-induced decline when budded on sour orange (*C. aurantium* L.) and is also highly susceptible to CTV-induced stem pitting. Many 'Pera' groves were severely affected by stem pitting even though propagated on the decline-resistant Rangpur lime (*C. reticulata* var. *austera* hybrid), and by the late 1950's, planting of this important variety was discouraged in the State of Sao Paulo, Brazil. Subsequently, we found that trees experimentally infected with mild strains were protected against the severe, naturally-occurring strains. The stem-pitting effects of tristeza are now being satisfactorily controlled under commercial conditions

by the use of mild strains. Approximately 10,000,000 trees of mild strain-protected 'Pera' sweet orange, the most important variety for Sao Paulo, are in nurseries, young groves, and producing orchards. Good protection also has been observed against the very severe Capao Bonito strain of CTV which occurs in the south of Sao Paulo. The protection of other citrus types with mild CTV strains, as well as the reutilization of the sour orange as a rootstock, is under investigation.

'Pera' sweet orange (*Citrus sinensis* (L.) Osbeck), a late variety that presently represents 45% of the commercial plantings of the State of Sao Paulo, Brazil (6), always has been one of its most important round oranges (16). The fruit holds well on the tree without loss of quality, and also has good storing and shipping qualities.

Along with the other citrus budded on sour orange (*C. aurantium* L.), 'Pera' was nearly wiped out when the tristeza virus was introduced into Brazil. The observation that scions budded on sweet orange, Rangpur lime (*C.*

Proc. Fla. State Hort. Soc. 93: 1980.