AMERICAN JOURNAL OF INDUSTRIAL MEDICINE 39:643–649 (2001)

# Exposure to Endosulfan in Farmers: Two Case Studies

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**Background** *Endosulfan is not a restricted use organochlorine insecticide and is currently under re-registration review. In 1993, one confirmed case and one possible case of endosulfan poisoning in agricultural workers occurred in two southeastern states.*
**Methods** *Two cases of suspected endosulfan poisoning were investigated utilizing record reviews, blood samples, a site visit, and clothing analysis.*
**Results** *Case 1 was fatal; Case 2 resulted in permanent neurological impairment. Additionally, Case 1 mixed and applied two less toxic pesticides, acephate and maleic hydrazide to tobacco plants. Both farm owners had ample opportunity for endosulfan exposure while mixing concentrated endosulfan with water and applying the solution to tobacco with boom sprayers pulled by tractors.*
**Conclusions** *Estimates of the absorbed dose of endosulfan were not available because methods to determine actual personal exposure that would be found in fat or tissue samples were not used. Health and safety issues associated with endosulfan require a closer examination. A cooperative multi-disciplinary approach to providing timely accurate education is needed to prevent pesticide poisonings.* Am. J. Ind. Med. 39:643–649, 2001. *© 2001 Wiley-Liss, Inc.*

***KEY WORDS: endosulfan; pesticide; poisoning; agriculture; protective equipment; public health***

## INTRODUCTION

Endosulfan is not a restricted use organochlorine insecticide and is under reregistration review in the United States (US), (S. Milan [USEPA], personal communication, September 2000). It is no longer manufactured in the US [USDHHS, 1993] and is under regulatory control in several countries [IOCU, 1986]. Currently endosulfan is used in the US on cotton, tomatoes, potatoes, and apples, all of which except potatoes are grown in the South central and Southeastern regions. Non-food crop/site usage includes nurseries and greenhouses [USEPA, 2000]. Like other organochlorines, endosulfan is a neurotoxin that often results in generalized convulsions and respiratory paralysis after exposure to high levels [Ely et al., 1967; Alecksandrowicz, 1979; Blanco-Coronado et al., 1992; Singh et al., 1992; Chugh, 1998]. Reigart and Roberts [1999] reported, ''early manifestations of poisoning by some organochlorine pesticides are often sensory disturbances: hyperesthesia and paresthesia of the face and extremities. Headache, dizziness, nausea, vomiting, incoordination, tremor, and mental confusion are also reported.'' Equally effective insecticides which are less toxic to humans are available. Recent university field trials conducted in the burley and dark tobacco growing areas show that imidacloprid, a flowable, systemic insecticide, is available and can provide insect control equal to or better than several applications of acephate or endosulfan [Townsend, 1992].

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Grant Sponsor: National Institute for Occupational Safety and Health, Centers for Disease Control and Prevention; Cooperative agreement: U60/CCU412957-02.

Accepted 9 February 2001

In 1993, one confirmed case (Case 1) and one possible case (Case 2) of endosulfan pesticide poisoning occurred in two southeastern states. Case 1 was fatal; Case 2 resulted in permanent neurological impairment. Additionally, in Case 1, the farmer mixed and applied two less toxic pesticides: acephate and maleic hydrazide to tobacco plants. Both farm owners had ample opportunity for endosulfan exposure while mixing concentrated endosulfan with water and applying the solution. A nurse from the Community Partners for Healthy Farming surveillance project[1] was informed of Case 1. Subsequent investigation, including a site visit and inquiries to an endosulfan distributor revealed Case 2.

## CASE 1

In September 1993, a 37-year-old male Caucasian farmer died after exposure to endosulfan. On the day of death, he began mixing maleic hydrazide (a herbicide) and acephate (an insecticide) at 8:30 AM and began spraying the tank mix on tobacco at 9:00 AM. The application consisted of using a boom sprayer pulled by a tractor with an unenclosed cab. No respirator or gloves were worn while mixing or spraying the product. Ambient temperature was 62–82°F with winds 0–7 miles per hour. Shortly before noon, the farmer and co-worker exhausted their stock of the less toxic insecticide (acephate) and replaced it with a neighbor's endosulfan. While pouring concentrated endosulfan into a 55-gallon drum, the farmer spilled some on his bare hand and wiped it on his pants without washing his hands.

At noon both the farmer and co-worker felt ill, causing the co-worker to leave the area temporarily while the farmer continued work despite coughing. After completing the first field application at 3:00 PM, the farmer, followed by the co-worker, started driving the tractor home and for unknown reasons swerved off the road without collision. At 3:30 PM, the victim reported nausea, vomiting, headache, and cough to a family member but continued spraying tobacco with tank mixed endosulfan and maleic hydrazide in another field until 4:30 PM, making the total exposure time to endosulfan approximately 4 h. He then drove the co-worker home, returned to his home, removed his damp clothing and showered. At 6:05 PM he was found unresponsive by a family member and transported by emergency medical services (EMS) to the nearest hospital. EMS administered epinephrine, atropine, and sodium bicarbonate prior to transport. Oral intubation was not successful. The emergency department diagnosis was cardiac arrest and the farmer was pronounced dead by the coroner at 8:02 PM. Cause of death was listed as prolonged exposure to the insecticide (endosulfan). An autopsy was not performed.

The farmer in this case was a self-employed carpenter and life-long farmer who produced 22,000 lbs of tobacco in 1993. He was certified to use restricted pesticides in 1990, but it was reported that he did not routinely wear personal protective equipment while handling pesticides. He had a history of cigarette and alcohol use as well as chronic respiratory problems. He used albuterol when needed for respiratory problems but had not used it the day of the incident.

A toxicological chemist analyzed blood samples for pesticides, drugs, and alcohol. Analysis by gas chromatography revealed 0.84 mg/L total endosulfan (endosulfan I: 0.63 mg/L:endosulfan II: 0.21 mg/L). Samples were analyzed as follows: ''Endosulfan I, endosulfan II, and endosulfan sulfate were detected by dual capillary column gas chromatography with electron capture detection. The columns used were a 30 m SE30 and a 30 m SE54. Quantitation was on the SE54 column'' [KHSL, 1993]. The analytical method employed did not detect maleic hydrazide. The report noted, ''given the relatively high published median lethal dose for the small sample volumes available in this case, and the detection of the endosulfan compounds, this compound was not vigorously pursued'' [KHSL,1993]. Further, the analysis method did not detect acephate. In addition, ''the major metabolite methamidophos was examined by gas chromatography on a DB5 meter capillary column with flame photometric detection. The screen for methamidophos was negative based on comparison to a standard. The detection limit could only be estimated at 1 mg/L. Blood alcohol was 0.054 (0–0.10 gm/100 ml). Volatiles were negative. Chlorpheniramine was present at $<0.05$ and diphenhydramine was present at $<0.05$. Acetaminophen and salicylates were not detected'' [KHSL, 1993]. The DB5 is a type of meter capillary column used for gas chromatographic analysis. Nicotine levels were comparable to a typical tobacco user.

Symptoms of exposure to acephate, an organophosphate insecticide used by this farmer, can include headache, nausea, and dizziness. Acute exposure to acephate results in muscle twitching, weakness, tremor, and vomiting. Exposure to extremely high levels results in unconsciousness, incontinence, convulsions, and respiratory depression. Additionally, a third and relatively non-toxic [USEPA, 1994] herbicide, maleic hydrazide, was also used by this same farmer.

### Laboratory Analysis of Clothing

Ten days following the incident, the clothing worn by the victim was sealed in a plastic bag and sent to the National Institute for Occupational Safety and Health

[1] Community Partners for Healthy Farming was a national program funded by the Centers for Disease Control and Prevention's (CDC) National Institute for Occupational Safety and Health (NIOSH). This program brought together local public health nurses who focused on agriculture, rural communities, state health departments, and researchers to conduct surveillance and intervention research related to agriculture-related illnesses, injuries, and hazards that occurred among farmers and their family members. NIOSH supported 16 such state-based projects to assist in reducing the work-related risks for agricultural populations.

**TABLE I.** Results of Clothing Analysis

| | Area of shirt samples ($cm^2$) | Quantity in shirt samples (mg) | Area of pant samples ($cm^2$) | Quantity in pants samples (mg) | Total in pants & shirt samples (mg) | Analytical limit of detection (ug/sample) |
|---|---|---|---|---|---|---|
| Endosulfan I | 1057 | 0.05 | 320 | 1.07 | 1.11 | 0.0001 |
| Endosulfan II | 1057 | 0.01 | 320 | 0.58 | 0.59 | 0.0001 |
| Acephate | 1057 | BDL[a] | 320 | 0.08 | 0.08 | 0.0033 |
| Maleic hydrazide | 1020 | BDL[a] | 328 | 4.10 | 4.10 | 0.400 |

[a]Analyte, if present was below detection limit (BDL). BDL is the lowers level of analyte that can be detected by gas chromatograph.

(NIOSH) for analysis. The laboratory results presented in Table I indicate the clothing worn by the farmer contained measurable levels of endosulfan. These results do not indicate the actual amount of pesticide deposited on the clothing because the samples were not collected in a way that would reduce/eliminate the possibility of compound degradation or volatilization. It is unclear what fraction of the chemicals found on the clothing was from concentrate or diluted spray mix. The quantity found on the clothing confirm the potential for exposure but does not represent actual dermal exposure.

## CASE 2

In August 1993, a 43-year-old tobacco farmer mixed and sprayed endosulfan on tobacco for 5 h and had repeated hand and possibly oral contact with the concentrate and tank mix. He mixed 5–6 quarts of endosulfan concentrate with 300 gal of pond water without washing or wiping his hands. He did not use gloves or a respirator. He pulled a boom sprayer behind an unenclosed tractor while significant wind drift repeatedly caused the tank mix to blow back on him. Ambient temperature was 70–75°F. His clothes appeared soaked and he neither showered nor changed clothes during the exposure period. A broken conduit in the spraying apparatus required welding for 15 min in mid-morning allowing another dermal exposure opportunity. He reportedly drank two canned soft drinks and smoked an unknown number of cigarettes while spraying the tobacco. No other pesticides were used on the day of the incident.

While driving from the tobacco field, the tobacco farmer was observed to wave to other workers, then suddenly slump forward and display jerking motions on the tractor seat while the tractor was still in motion. His tractor then swerved into a ditch and overturned on its side. His legs were pinned under the steering wheel from which he was extricated when medical personnel arrived about 20 min later. No direct physical signs of head or other significant trauma were noted at that time or during hospitalization. His generalized convulsions ceased briefly and he regained consciousness during the interim, but the convulsions recurred and continued as status epilepticus over the next 5–6 h despite aggressive treatment with multiple intravenous seizure control medications.

Hypoxia, acidosis, fever of 101° and status epilepticus were noted on the initial hospital evaluation. His past medical history had been unremarkable. One sibling had a history of a seizure disorder. The physical examination was normal except for tachycardia. Pesticide exposure was initially suspected of precipitating the convulsions. A lumbar puncture revealed bloody cerebrospinal fluid, but a CAT scan showed no definite hemorrhage. Discharge diagnosis from the initial hospital stay was status epilepticus related to either subarachnoid hemorrhage or organochlorine pesticide exposure. He was transferred to a tertiary medical center 3 days post event.

A subsequent 11 week hospitalization was characterized by signs of rhabdomyolysis, gradually resolving epileptiform activity on electroencephalogram (EEG) without observed seizures, and severe encephalopathy with persistent diffuse slowing on EEG. A cervical puncture and two magnetic resonance imaging (MRI) scans were the basis for ruling out subarachnoid and cerebral hemorrhage. Blood samples were collected 3 days post-exposure and were not analyzed until 2 weeks later for endosulfan (due to a lab error); no endosulfan was detected in a residual aliquot. Details about storage and handling of the residual aliquot during this 2 week interval could not be verified with the outside reference laboratory, raising concerns about the validity of the negative endosulfan result. A fat biopsy obtained 4 weeks post exposure detected no endosulfan. A persistent low-grade fever eventually resolved at the tertiary medical center and was attributed to probable drug fever. Final diagnoses at the tertiary care hospital included endosulfan toxicity as the presumptive cause for the severe status epilepticus and toxic encephalopathy. Five years post event he remained profoundly impaired neurologically and under full custodial care.

## Chemical Review

Endosulfan is used in the US on field crops including tobacco, upland cotton, corn, soybeans, wheat, fall potatoes, sunflowers; fruits and vegetables including tomatoes, lettuce, apples, grapes, cherries, peaches, pears, pecans, hazelnuts, and oranges [USEPA, 2000]. Formulations of endosulfan include dust, emulsifiable concentrates, wettable powders, granules and ultra-low volume concentrate (ULV). The oral lethal dose ($LD_{50}$) values for endosulfan in male and female rats are 160 and 22.7 mg/kg, respectively; dermal values are greater than 500 mg/kg [Naegely, 1998].

Handling endosulfan liquid concentrates or applying diluted spray mix (which varies by type of crop) may place farm workers at risk of exposure via three routes: (1) inhalation of spray droplets or vapors; (2) dermal absorption from the mist, splash, or contaminated surfaces; and (3) ingestion via eating, drinking or smoking. Agricultural exposure probably occurs through combined routes during various tasks (e.g., mixing, loading, applying pesticides in both concentrated and diluted forms) [USDHEW, 1977]. Dermal and gastrointestinal absorption and subsequent toxicity are probably enhanced by their organic carrier solvents. Xylene is a common solvent, as in the two cases described in this paper [Maier-Bode, 1968; Gosselin et al., 1984]. Skin and mucous membrane absorption of organic compounds increases with both temperature and moisture content of the skin, and therefore is enhanced in warm, humid climates. Exposure studies have shown hands to be the area of greatest exposure to pesticides [Lavy et al., 1983]. Even though endosulfan is more toxic than xylene, when in direct contact with skin, xylene causes defatting, dryness, and dermatitis. Depending on the concentration when inhaled, acute exposure to xylene causes respiratory irritation, systemic vasodilation, disturbed vision, cardiac stress, confusion and coma [Sandmeyer, 1981].

In published reports of two fatal incidents, endosulfan's interaction with alcohol may have been a contributing factor. In one case, a man consumed endosulfan and dimethoate in xylene and alcohol. In another report, authors concluded that a fatality was due to synergistic effects of alcohol and endosulfan [Demeter et al., 1977].

Animal and human data on endosulfan indicate that ''excretion seems to be principally biliary, although nearly all organochlorines yield measurable urinary metabolites. Unfortunately, many of the unmetabolized pesticides are efficiently reabsorbed by the intestine substantially retarding fecal excretion'' [Reigart and Roberts, 1999]. The most prominent signs of acute poisoning in both humans and animals are hyperactivity, tremors, decreased respiration, dyspnea, salivation, and tonic-clonic convulsions. [Terziev et al., 1974]. Endosulfan's prominent neurotoxicity, central nervous system absorption and metabolism are of particular concern. Mechanisms of endosulfan neurotoxicity are not well established, but neurotransmitter levels appear to play a role.

All previously reported human deaths from endosulfan in the United States have involved ingestion. While many of those reported involved high doses and/or suicide, Terziev et al. [1974] reported one lethal ingestion of only ''drops'' of endosulfan. Symptoms described include nausea, vomiting, headache, and dizziness within 2–3 h of ingestion. In a 1974 study, Terziev et al. concluded ''autopsy findings from casualties after acute intoxication, have shown only non-specific changes such as edema of the brain.''

## DISCUSSION

Endosulfan toxicity is strongly implicated in the Case 1 fatality and the suggested etiology for sudden onset of status epilepticus with resulting severe neurological impairment in Case 2. The Agency for Toxic Substances and Disease Registry (ATSDR) describes neurotoxicity as the primary effect observed in humans following occupational exposure to endosulfan [USDHHS, 1993]. Endosulfan neurotoxicity manifesting as generalized convulsions is repeatedly described in humans [Chugh et al., 1998]. The absence of confirmatory elevated endosulfan biological samples was a significant limitation in establishing the etiology in Case 2. Estimates of absorbed endosulfan dose were not available in Case 1 because methods to determine actual personal exposure were not used. Fat or tissue samples may contain more stored organochlorines than blood [Reigart and Roberts, 1999]. Therefore, the blood levels reported in Case 1 may not be representative of the total body burden. Additive or synergiestic effects of acephate and endosulfan in cases 1 and 2 could not be ruled out although USEPA sources did not indicate any known synergiestic effects [Brassard and Blondell, 2000; USEPA, 2000]. A cholinesterase test was not completed in Case 1. Acephate was not detected at a 1 mg/L level.

In Case 1, the results of the clothing analysis strongly suggest the potential for dermal absorption existed; the post-mortem quantification of endosulfan blood levels were in close agreement with the ranges reported elsewhere [Blanco-Coronado et al., 1992]. Case 1 exhibited blood levels greater than one but lower than some other fatal cases reported in literature. Furthermore, his blood level 0.84 mg/L total endosulfan was higher than five very serious non-fatal hospitalized cases [0.29–0.67 mg/L], four of whom required mechanical ventilation [Blanco-Coronado et al., 1992]. Prior to hospital admission, two had tonic-clonic convulsions and three were asymptomatic. All were treated by gastric lavage and developed tonic-clonic convulsions 1–4 h post ingestion. In another fatal case the reported blood levels were 0.075 mg/L several hours after ingestion which was complicated by the presence of alcohol [Demeter et al, 1977] (see Table II for a comparison of

**TABLE II.** Comparison of Case Studies

| | Source | Endosulfan blood level (mg/L) |
|---|---|---|
| Fatal | Demeter et al., 1977 | 0.075 |
| | Index case | 0.84 |
| | Blanco-Coronado et al., 1992 | 2.85 |
| Non-fatal requiring mechanical ventilators | Blanco-Coronado et al., 1992 | 0.29–0.67 |
| Seizure activity reported with exposure | Ely et al., 1967 | Blood levels associated with seizure activity were not indicated in literature |
| | Alecksandrowicz, 1979 | |
| | Singh et al., 1992 | |
| | Blanco-Coronado et al., 1992 | |
| | Chugh et al., 1998 | |

endosulfan blood levels). Eighteen additional incidents of occupational endosulfan exposure associated with convulsions have been documented in recent literature [Chugh et al., 1998].

## Prevention Strategies\ Recommendations

A cooperative multi-disciplinary approach to providing timely accurate education is needed to prevent pesticide poisonings. Prevention includes replacing use of pesticides with integrated pesticide management (IPM), use of less toxic pesticides, and assurance of the proper handling of pesticides by farmers and farm workers. IPM approaches should include manufacturers, distributors, agricultural extension service, public health professionals, farm employers, and individuals handling pesticides. All involved need to understand and promote compliance with the worker protection standard (WPS) and other EPA regulations (such as pesticide applicator/handler certification programs) designed to protect pesticide workers. Imidacloprid, the active ingredient in a less toxic and equally effective substitution for endosulfan is available [Naegely, 1998]. Cost of the product may be a barrier to substitution at the average cost of US$ 40 per acre for aphids in field crops compared to US$ 6 per acre for the product described in Case 1 with endosulfan as the active ingredient.

All states require commercial applicators to be re-certified, generally every 3 to 5 years, to maintain their certification. It should be emphasized that even pesticides, like endosulfan, that do not require certification for purchase can be lethal or cause serious illness or permanent impairment.

Distributors should encourage the appropriate use of pesticides including the purchase of less toxic pesticides when effectiveness is comparable; promote the sale of personal protective equipment (PPE) and decontamination kits when pesticides are sold and encourage reading of material safety data sheets (MSDS). More importantly, distributors should reiterate precautions listed on the EPA approved label because it is specific to the individual product. (MSDS are not approved by the EPA).

Agricultural extension services should provide periodic instruction to pesticide users in the appropriate storage, handling, and use of pesticides beyond that offered in pesticide certification classes. Emphasis should be placed on recommending less toxic pesticides. Users of pesticides should be instructed in the proper use of PPE, importance of following the directions on the product label and the signs and symptoms of pesticide poisoning. In Case 1, the farmer had attended a certification course but did not use proper PPE or follow directions on the product label. He continued to work although he was not feeling well, indicating that he may not have made the connection between his symptoms and endosulfan poisoning.

Public health professionals should understand the hazards associated with the pesticide use as well as diagnosis and treatment of these types of poisonings. Proceedings from the USEPA [1998] workshop indicate health care providers lack sufficient training in diagnosing and treating cases of pesticide exposure. Information should be disseminated to agricultural workers through the public health systems, hospitals, health fairs, and clinical care settings. Health care providers should take occupational histories to identify pesticide exposures. Because endosulfan has been known to react synergistically with other chemical compounds and substances, healthcare workers should perform cholinesterase tests for organophosphates and other pesticides. Access to respirator fit-testing should be facilitated and enrollment in pesticide training classes encouraged. Because many workers in general industry also farm or garden, on a part-time basis, occupational health nurses and wellness programs should educate employees about pesticide safety.

Farm employers should ensure that each employee is properly trained in handling pesticides, accurate records are maintained, and re-entry intervals adhered to. Decontamination supplies should be readily available or copious amounts of soap and water.

Only trained adults should handle pesticides. However, for agricultural youth to understand the importance of avoiding pesticide exposure, pesticide health risk education should be provided to them through agricultural classes and health curriculums in school. Prior to eating, drinking or smoking, pesticide users should wash their hands thoroughly with soap and water. Care should be taken to avoid respiratory, dermal, and clothing contact; exposure

from tractor-drawn boom sprayers and wind drift should be avoided. Because endosulfan is a Toxicity Class 1 Poison, the protective equipment requirements for mixing, loading and applying are: long sleeve shirt, pants and head covering; chemical resistant apron when handling and mixing the concentrate; goggles/face shield when applying; full/half face NIOSH-approved respirator; and gloves made of rubber/neoprene [Naegely, 1998].

Prevention of pesticide poisoning as well as an individual's susceptibility to poisoning depends upon many factors: species; age; nutritional status; gender; current state of health; the presence of multiple chemicals, including other pesticides, alcohol, and nicotine; and general individual susceptibility [Demeter et al., 1977]. Assessment of applicator exposure, as well as on-going surveillance, may provide researchers with a better approach to preventive measures. Although these two cases of dermal and respiratory exposure to endosulfan appear unique in the literature, thorough occupational histories by clinicians may reveal that endosulfan and other pesticide poisonings are more common.

Health and safety issues associated with endosulfan require a closer examination. The work practices described in these reports were not in accordance with manufacturers' recommendations and may not be typical of those used by small private farmers. However, there is reason to suspect that compliance with safe work practices and PPE use remains inconsistent in agricultural settings. Fifty-four percent of corn growers in New York reported ''nearly always'' wearing chemical-resistant gloves while 29.6% wore them ''occasionally depending on the pesticide'' [Partridge et al., 1994]. Assessments of endosulfan applicator exposure and dissemination of such findings may improve compliance and prevent future incidents like those described here.

## ACKNOWLEDGMENTS

The authors thank the following for their contributions to this manuscript: Ellyn Moon, Amy Scheerer, Karen McClanahan, and Donna Reed at the Kentucky Injury Prevention and Research Center; Gary Palmer, University of Kentucky Department of Agronomy; Ana Maria Osorio, United States Environmental Protection Agency; Ursula Myers, University of Cincinnati, College of Nursing and Health; and Louise N. Mehler, California Environmental Protection Agency; Kentucky Poison Control Center; Virginia Poison Control Center; and the Kentucky Department for Public Health.

## REFERENCES

Aleksandrowicz DR. 1979. Endosulfan poisoning and chronic brain syndrome. Arch Toxicol 43:65–68.

Blanco-Coronado JL, Repetto M, Ginestal RJ, Vicente JR, Yelamos F, Lardelli A. 1992. Acute intoxication by endosulfan. J Toxicol Clin Toxicol 30:575–583.

Brassard D, Blondell J. 2000. US Environmental Protection Agency (EPA).

Chugh SN, Dhawan R, Agrawal N, Mahajan SK. 1998. Endosulfan poisoning in Northern India: A report of 18 cases. Int J Clin Pharmacol Ther 36(9):474–477.

Demeter J, Heyndrickx A, Timperman J, Lefevere M, De Beer J. 1977. Toxicological analysis in a case of endosulfan suicide. Bull Environ Contam Toxicol 18:110–114.

Ely TS, Macfarlane JW, Galen WP, Hine CH. 1967. Convulsions in thiodan workers, a preliminary report. J Occup Med 9:35–37.

Gosselin RE, Smith RP, Hodge HC. 1984. Clinical toxicology of commercial products, 5th edn. Baltimore: Williams and Wilkins. p 286.

International Organization of Consumers Unions (IOCU). 1986. The pesticides handbook profiles for action, 2nd edn. Penang, Malaysia: IOCU. p 2–3.

Kentucky Health Services Laboratory (KHSL) 1993. Report for case #93TM1437. Frankfort, KY: CHR Toxicology Laboratory.

Lavy TL, Mattice JD, Flynn RR. 1983. Field studies monitoring worker exposure to pesticides. In: Seymour KG, editor. Pesticide formulations and application systems. Philadelphia: American Society for Testing and Material. p 60–74.

Maier-Bode H. 1968. Properties, effect, residues and analytics of the insecticide endosulfan. Residue Rev 22:1–44.

Naegely S, editor. 1998. Farm chemicals handbook, Vol. 79. Willoughby: Meister Publishing Company. C158–159.

Partridge MS, Coffman C, Smith WG, Rutz DA. 1994. Pest and pesticide use assessment and personal protective equipment use for field corn production systems in New York State for 1994. Ithaca, NY: Cornell University. p 68.

Reigart RJ, Roberts JR. 1999. Recognition and management of pesticide poisonings, 5th edn. (EPA-735-R-98-003). Washington, DC: US Environmental Protection Agency. p 57–59.

Sandmeyer EE. 1981. Aromatic hydrocarbons. In: Clayton ED, Clayton FE, editors. Patty's industrial hygiene and toxicology, 3rd edn, Vol. 2B. New York: John Wiley & Sons Inc. p 3291–3300.

Singh N, Singh CP, Kumar H, Brar GK. 1992. Endosulfan poisoning: A study of 22 cases. J Assoc Physicians India 40:87–88.

Terziev G, Dimitrova N, Rusev F. 1974. Forensic medical and forensic chemical study of acute lethal poisonings with thiodan. Folia Med 16:325–329.

Townsend L. 1992. Insecticide recommendations for tobacco fields and beds for 1992 (ENT-15). Lexington: University of Kentucky, Department of Entomology.

US Department of Health & Human Services (USDHHS). 1993. Toxicological profile for endosulfan. (TP-91/16). Atlanta, GA: Agency for Toxic Substances and Disease Registry. p 15–119.

US Department of Health Education and Welfare (USDHEW). 1977. Occupational Diseases, A Guide to Their Recognition (Rev. ed.) [DEHW (NIOSH) Publication No. 77-181]. Washington, DC: US Government Printing Office. p 11–21.

US Environmental Protection Agency (USEPA). 1994. Reregistration eligibility decision (RED), maleic hydrazide (EPA 738-R-94-010). Washington, DC: Office of Pesticide Programs. p 208.

US Environmental Protection Agency (USEPA). 1998. Pesticides and national strategies for health care providers. Workshop proceedings (EPA 735-R-98-001). Arlington: author. p 27.

US Environmental Protection Agency (USEPA). 2000. Donaldson D, and Nako S. Analysts. Office of Pesticides Programs, Quantitative Usage Assessment (QUA). p 6.