

Chronic Neurological Effects of Pesticides

Summary of Selected Studies

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Some Notes on the Table

This table is a selective summary of studies of long term effects on the nervous system in adults with potential occupational and environmental exposure to pesticides. Most are from articles published in English in peer-reviewed journals. The studies are listed in chronological order by author – the most recent first.

The source of pesticide exposure could be their occupation as farmers, agricultural workers, sprayers, exterminators, formulators, or other jobs. The exposure could be from household, lawn/garden, pet, or other pesticide use. Or it could be from potential pesticide exposures from living on a farm, in an agricultural spray area, near a pesticide factory, or other environmental exposures.

How The Studies Are Done

Epidemiology is the study of diseases and their causes in human populations. It compares groups of people with an exposure or a disease to those without it.

In the studies in this table, groups of people with Parkinson's disease* or other neurological disease, or with pesticide exposure are the "*cases*". Groups of people without Parkinson's or other neurological diseases, or without pesticide exposure are the "*controls*".

The aim is to find out if the people with neurological disease (the cases) are more likely to have exposure to pesticides than the people without neurological disease (the controls). Or to find out if the people with pesticide exposure (the cases) are more likely to have neurological disease than those with pesticide exposure (the controls).

How Study Results are Reported

Study results are reported as risk ratios. These ratios indicate whether the people with neurological disease were *more* likely to have been exposed to pesticides (at increased risk), *equally* likely to have had pesticide exposure (no difference in risk), or *less* likely to have had pesticide exposure (decreased risk) than the people without neurological disease.

Or whether the people with pesticide exposure were *more* likely to have neurological disease (at increased risk), *equally* likely to have neurological disease (no difference in risk), or *less* likely to have neurological disease (decreased risk) than the people without pesticide exposure.

For example: In a study of Parkinson's disease, the cases would be people with Parkinson's, and the controls people without it. There are three possible outcomes. The people with Parkinson's could be more likely, equally likely, or less likely to have pesticide exposure.

1. More likely: If the ratio is greater than 1 (> 1), this means that the people with Parkinson's were more likely to have pesticide exposure – that pesticide exposure *increases* the risk of Parkinson's. The size of the ratio indicates how much the risk is increased. The larger the number the greater the risk. A ratio of 1.4 means a 40% increase in risk. A ratio of 2.0 means a doubling of the risk, or a 200% increase. At least a doubling of the risk is considered more important than ratios less than 2.

2. Equally likely: If the ratio is equal to one ($= 1$) this means that there was no difference in pesticide exposure found in the people with or without Parkinson's – pesticides did *not* increase the risk of Parkinson's in the study.

3. Less likely: If the ratio is less than one (< 1), this means that people with Parkinson's were less likely to have pesticide exposure than people without it, or the risk was *decreased*. The smaller the number the lower the risk. A ratio of 0.80 means that people with Parkinson's are 20% less likely to have been exposed to pesticides. A ratio of 0.40, that they are 60% less likely.

When studying humans, it is impossible to determine every factor that might influence the results of a study. It might have occurred anyway, by chance. It is possible that any increase in risk was not from pesticides, but something else. This could be something the researcher didn't think of, or didn't even ask

about. Or it could be from pesticide exposure in combination with other unknown or unstudied factors. Therefore, finding an increase in risk does not mean that pesticides “cause” Parkinson’s.

This is why it is common to report increase in risk by stating that “pesticide exposure increases the risk of Parkinson’s”, or “pesticide exposure is a risk factor for Parkinson’s”, and not that pesticides “cause” Parkinson’s.

Are the Study Results “Significant”?

There are methods to determine how strong the link or associations between neurological diseases and pesticides are, or if they occurred by chance. They are called tests of statistical significance. The statistical part is usually left out, and the results reported as “significant” or “not significant”. The two most common tests are the “p” value, and confidence intervals.

1. “p” value: This tests whether the findings could have occurred by chance 5% of the time or less. The 5% is converted to a fraction and written as 0.05. For example, you will see the results as “ $p = 0.05$ ” (read as p equals point 0 5), or “ $p < 0.05$ ” (read as p less than point 0 5), or “ $p \leq 0.05$ ” (read as p less than or equal to point 0 5).

If the “p” value is less than or equal to 0.05, the findings are considered to be statistically significant; that is, they are unlikely to have occurred by chance. The smaller the “p” value the more significant the findings. For example” $p \leq 0.01$ ” (read as p less than or equal to point 0 1) means that it could have occurred by chance 1% of the time or less.

2. Confidence intervals: Another widely used test is called the confidence interval. It shows how close the risk ratio found in the study is to the “true” or expected value. The chosen level is usually 95%. This means that 95% of the time the study results will lie within the calculated interval. Another way of saying this is that 5% of the time they will not.

Because it is an interval, there are two numbers, with the lower number written first. If the lower number of the confidence interval is less than or equal to one (≤ 1), then the increase in risk is “not significant” or “non-significant”. If the lower number of the interval is greater than one (> 1) then the increase in risk is considered “significant”.

If the number of cases is small, the confidence interval can be very wide. When there is a very wide interval between the lowest and the highest number, the less confidence you have in the findings. It usually means that the number of cases found were very small.

The larger the number of people in the study (the sample size), the narrower the confidence interval, and the more significant the findings.

Commonly Used Ratios

FR	Fecundability Ratio	SMR	Standardized Mortality Ratio
OR	Odds Ratio	SHR	Standardized Hospital Ratio
PMR	Proportionate Mortality Ratio	SMbR	Standardized Morbidity Ratio
PCMR	Proportionate Cancer Mortality Ratio	SIR	Standardized Incidence Ratio
PR	Prevalence Ratio	SPR	Standard Proportional Ratio
RR	Relative Risk (or Rate Ratio)	SRR	Standardized Rate Ratio

* Parkinson’s disease is used as an example, but the discussion refers to all of the neurological diseases listed in the table.

Parkinson's Disease

Location	Source of Exposure	Findings (95% CI)	Ref.
US Case-control	Age of PD ¹ onset sibling pairs Pesticide exposure	No differences seen	2002 Maher
Taiwan Case-control	Young onset PD ¹ (before age 40) Well water drinking	Increased risk	2002 Tsai
Italy Case-control	Parkinson's disease Farming as an occupation Well water use	OR 7.7 (1.4-44) p = 0.02 2.0 (1.1-3.6) p = 0.03	2002 Zorzon
Brazil Case report	Parkinsonian syndrome Glyphosate spray accident 54 y man	Follow-up Syndrome developed 1 month later.	2001 Barbosa
India Case-control	Parkinson's disease Well water more than 10 yrs	OR Increased risk - signif.	2001 Behari
US Wash. State Case-control	Parkinson's disease - orchard workers Highest tertile yrs pest. exposure Middle tertile yrs pest. exposure Specific pesticide use Farming Well water use	Prevalence Ratio 2.0 (1.0 to 4.2) 1.9 (0.9 to 4.0) trend not sig. No increased risk No increased risk No increased risk	2001 Engel
Israel Rural south	Parkinson's disease Pesticide exposure	Strong predictor	2001 Herishanu
US Michigan	Parkinson's disease Agricultural occupation	OR 1.74 (0.85- 3.60)	2001 Kirkey
Meta-analysis 55 studies	Parkinson's disease Rural residence. Well water use Farming Pesticide exposure	OR All Studies OR U.S. Only 1.56 (1.18-2.07) 2.17 (1.54-3.06) 1.26 (0.97-1.64) 1.44(0.92-2.24) 1.42 (1.05-1.91) 1.72(1.20-2.46) 1.85(1.31-2.60) 2.16(1.95-2.39)	2001 Priyadarshi
Meta-analysis 19 studies	Parkinson's disease Pesticide exposure - all studies Pesticide expo. all studies U.S. only Duration exposure to pesticides Type of pesticide	OR 1.94 (1.49-2.53) 2.15 (1.14-4.05) No sig. dose-response found No specifics found	2000 Priyadarshi
US California	Parkinson's disease mortality Counties using agric. pesticides Insecticide use by county ²	Mortality Increased Increased - dose-response	2000 Ritz
Denmark	Parkinson's disease Agricult / horticult.- men & women Agricult / horticult.- men only Farmers	Standardized Hospitalization Ratio 1.32 (1.11-1.6) 1.34 (1.1-1.6) 1.3 (1.03-1.6)	2000 Tuchsen
India Mumbai	Parkinsonism syndrome 5 cases of OP ³ poisoning	4 patients recovered completely 1 had repeated episodes w re-exposure	1999 Bhatt

Sweden Southern	Parkinson's disease Agricultural work Pesticide exposure	OR Increased risk Increased risk	1999 Fall
Finland	Parkinson's disease Farming Drinking well water Pesticide/herbicide exposure	OR No association No association No association	1999 Kuopio
Australia Rural	Parkinson's disease Rural residence Herbicide / pesticide exposure	OR 1.8 $p < 0.001$ No significant difference	1999 McCann
US New England Case-control	Parkinson's disease Well-water use Pesticide / herbicide exposure	OR Inverse association (lower risk) No association	1999 Taylor
China Hong Kong	Parkinson's disease Number years exposed pesticides	Increased risk $p = 0.10$	1998 Chan
US Case report	Parkinsonian syndrome Crop duster pilot poisoned OP ³	Possibly pesticide induced	1998 Davis
US Detroit Case-control	Parkinson's disease Occupational exposure herbicides Occupational exposure insecticides Farming as occupation	OR 4.10 (1.37- 12.24) 3.6 (1.8-7.2) 2.8 (1.03- 7.6)	1998 Gorell
Israel Kkibbutzim	Pre-Parkinsonism syndrome Field crop exposure, esp. cotton Landscape work.	Increase risk $p = 0.0007$ Weaker association	1998 Herishanu
US Multiethnic rural community	Parkinson's disease Rural living Farming Drinking unfiltered water	OR Increased risk in blacks Decreased risk in Hispanics Increased risk entire cohort	1998 Marder
Italy	Parkinson's disease Well water use Pesticide / herbicide exposure	OR 2.8 (1.5-5.3) 1.15 (0.6-2.46)	1998 Smargiassi
China Taiwan	Parkinson's disease Paraquat / other herb/pesticide expos. Hrb/pest. other than paraquat expos.	OR Increased risk - dose response Decreased risk	1997 Liou
Italy Sicily	Parkinson's disease Lived most of life as farmer	OR 0.6 (0.3-1.3)	1996 Rocca
Germany	Parkinson's disease Pesticide use Wood preservative exposure Rural factors	OR Increases risk - signif. Increased risk - signif. No association	1996 Seidler
US Miami	Postmortem brain samples Dieldrin detection	PD¹ Alzheimers Controls 30 % 14.3 % 0% $p = .03$	1994 Fleming
Canada British Columbia.	Parkinson's disease - fruit farmers Pesticide handling or direct contact Type/class of pesticide	OR Increased risk No specific associations	1994 Hertzman

Canada British Columbia. Okanaga Valley	Parkinson's disease Working in orchards Occupational exposure to pesticides Paraquat, azinophosmethyl, ferbam Contact with paraquat	OR 3.69 sig. 2.03, 2.3 > 1 Sig. assoc. with postural tremors	1994 Kelly
Italy case report	Extrapyramidal symptoms Occ. expos. maneb fung. 37 y ♂	2 yr latency between exposure and onset	1994 Meco
Spain Caceres	Parkinson's disease Pesticide exposure Agricultural work	OR Increased - borderline signif. No association	1994 Morano
US Washington State	Young onset PD ¹ (< age 50) Insecticide exposure Herbicide exposure Residenc fumigated house Rural residence ⁴	OR 5.75 p < 0.001 3.22 p = 0.033 5.25 p = 0.046 2.72 p = 0.027	1993 Butterfield
US Rural Kansas	Parkinson's disease Pesticide use	Significant predictor of risk	1993 Hubble
Canada Calgary	Parkinson's disease Occupational herbicide use	Significant predictor of risk	1992 Hubble
Canada Calgary	Parkinson's disease Occupational herbicide use	OR Increased risk	1993 Semchuk
Canada Calgary	Parkinson's disease Rural living Farm living Well water use	OR No increase No increase No increase	1991 Semchuk
Spain Madrid	Parkinson's disease Well water exposure ≥30 years Past exposure to pesticides	OR Increased risk p < 0.02 No association	1992 Jimenez
Italy Case report	Severe Parkinson's 72 yr old farmer acute onset Post 10% Diquat spill on hands ⁵	Follow-up 4 months later CT scan, MRI abnormalities ⁶ Persistent signs / symptoms ⁷	1992 Sechi
Italy Ferrara	Parkinson's disease Early onset cases urban areas Early onset cases rural areas All cases 1967-1987 All cases in agricultural workers	Incidence / yr / 100,000 3.1 6.32 10.01 20.6	1991 Granieri
US Case-control	PD ¹ young (< 40) & old (> 60) onset Well water use Herbicide / pesticide exposure Residential history of rural living	OR No associations No associations No associations	1991 Stern
US Washington State	Parkinson's disease Farm job Occup. or home pesticide exposure	OR 3.1 (0.3-35) No clear trends	1991 Wechsler

US Kansas Case-control	Parkinson's disease Rural living Drinking well water Farming Herbicide exposure	OR Increased risk signif. Increased risk signif. No increased risk No increased risk	1991 Wong
Canada British Columbia	Parkinson's disease Working in orchards Contact with paraquat	OR 3.69 (1.34-10.27) Positive association p = 0.01	1990 Hertzman
US Kansas	Parkinson's disease Rural living Drinking well water Farming Herbicide / pesticide exposure	OR Increased risk signif. Increased risk signif. No increased risk No increased risk	1990 Koller
Canada Quebec	Parkinson's disease Residence in rural areas Occupational exposure Mn, Fe, Al ⁸ Exposure more than 30 yrs Pesticide exposure Farm work Well water use	OR 0.31 p = 0.05 2.28 p = 0.07 13.64 p ≤ 0.05 No association No association No association	1990 Zayed
Hong Kong China	Parkinson's disease Long time residence rural areas Farming occupation Herbicide / pesticide use Habitual consump. raw vegetables	OR Increased risk signif. Increased risk signif. Increased risk signif. Increased risk signif.	1989 Ho
China	PD ¹ onset before 47 yr (vs 54 or older) Rural residence Drinking well water	OR Increased risk p ≤ 0.01 Increased risk p ≤ 0.01	1987 Tanner
Italy Case reports	Parkinson's disease 41 yr old farmer 38 yr pest. manif. worker	Author claims 1 st reported cases PD ¹ directly related to pesticide use	1986 Bocchetta
Canada	Parkinson's disease early onset Raised in rural areas	OR Increased p = 0.015	1986 Rajput

Other Chronic Neurological Effects

Location	Source of Exposure	Findings (95% CI)	Ref.
England	Sheep dipping farmers OP ³ exposure Group with normal NCV ⁹ Group with abnormal NCV ⁹ Motor and sensory NCV ⁹ Neuropsychological tests	Neuropathy Symptoms 7 % 52 % Sensory deficits, mainly small fibers Increased anxiety, depression	2002 Jamal
Sri Lanka	Regular sprayers OP ³ pesticides Sensory nerves Motor nerves	NCV⁹ Decreased farmers p = 0.01 Decreased fisherman p = 0.04	2002 Peiris-John

Netherlands	Farmer sprayers OP ³ Sensory nerve conduction velocity Motor nerve conduction velocity Sensory nerve conduction velocity Motor nerve conduction velocity	Between growing season Reduced sig p=.04 Reduced sig p=.04 During growing season Reduced farmers p <.01 Reduced fishers p=.04	2002 Peiris-John
Costa Rica	Banana workers ¹⁰ Psychomotor and visuomotor skills Language skills, affect Digit-Symbol test Neuropsychiatric symptoms Expos. che inhib. ¹¹ prior 3 months	Poisoned Group Poorer performance Poorer performance Poorer performance - sig diff. Marked increase Poorest performance	2002 Wesseling
France 1977-1978	Vineyard workers exposed pesticides ¹² Neuropsychiatric testing.	OR Poorer performance ≥ 2	2001 Baldi
Michigan - Dow	2,4-D Manufacturing workers ALS ¹³ deaths	OR 3.45 (1.1-11.1)	2001 Burns
England	Chronic OP ³ expos. sheep farmers Vibration thresholds	Findings Higher in concentrate handlers ¹⁴	2001 Pilkington
US	Hispanic children Agricultural vs nonagricultural	Cognitive tests Lower performance	2001 Rohlman
Costa Rica 1950-97	DDT exposure - malaria workers Neurobehav. tests overall perform. Verbal attn, visuomot. spd, seqnce Increase risk attributable yrs expos. Increase neuropsych/psychiat. symp	Findings 20 % decrease (mean) Differed most between groups 5 motor, sens., cognit. tests sig. diff 3.98 (1.02-15.6)	2001 VanWend
Belgium Maastricht	Aging study mild cognitive dysfunction Farmers and gardeners	Increased risk	2000 Bosma
India	Pesticide exposed workers ¹⁵ Memory, learning, vigilance	Neurobehavioral Tests Poorer performance	2000 Srivastava
US	Exterminators - total population Postural sway Pegboard turning Nerve conduction velocity Vibrotactile sensitivity Smell, vision No sig. differences Visual/motor skill Chlorpyrifos poisoned workers Postural sway, pegboard 2 of 6 neurobehavioral tests 5 of 5 mood scale tests	Findings Poorer performance - sig. diff. Poorer performance - sig. diff No sig. differences No sig. differences No sig. differences No sig. differences No sig. differences No sig. differences Poorer performance - sig. diff. Poorer performance - sig. diff Poorer performance - sig. diff	2000 Steenland
Poland	Women greenhouse wrkrs OP ³ expos. Reaction times Motor steadiness Tension, depression, fatigue	WHO NCTB²⁰ Increased Reduced More prevalent	1999 Bazylewicz
US Record review	Multiple System Atrophy Environmental toxin exposure ¹⁶	11/100 cases	1999 Hanna

NY State	Male pesticide applicators OPs ³ Vibration sensitivity Electrophysiological abnormalities Acute or subacute symptoms	Findings Increased ¹⁷ One case None found	1999 Horowitz
Germany Case rpt	Severe acute pyrethroid poisoning Persistent chronic effects ¹⁸	Sequelae Clinical picture 2 years later	1999 Muller-M
Germany 15 ♀ /controls	Long term occup. exposure to WPC ¹⁹ Paired-associate learning Benton Reading/naming speed.	Association PCP³² blood levels Poorer performance - signif.. Poorer performance - signif.. Poorer performance - signif..	1999 Peper
US Tennessee	Cortical blindness Intentional carbofuran ingestion	Case report	1998 Baban
US Florida	Structural fumigation workers ³⁶ Pattern Memory Olfactory testing Santa Ana Dexterity dom. hand	Performance Worse sulfuryl fluororide grp sig. Worse all fumigant exposed sig Worse all fumigant exposed sig	1998 Calvert
US	Hispanic farmworkers low OP ³ expos. Sural nerve latency and amplitude Ulnar nerve conduction velocity Ulnar neuromuscular junct. function	NCV⁹ No signif. differences No signif. differences No signif. differences	1998 Engel
Chile	Men chronic methyl bromide expos. Vibration sensory thresholds Dynamometry Nothingham - neg.auto-perception	Findings Increased 2.4 to 2.85 sec Reduced 51.4 to 47.2 kg Increased 11.2 to 13.6.	1997 Acuna
Ecuador	Farmer members of cooperative Visual-spatial	WHO NCTB²⁰ Most sensitive	1997 Cole
France 1973-1994	Methyl bromide poisoning cases Review and follow-up	Findings 2 developed peripheral neuropathy ²¹	1997 DeHaro
US	Male fruit farmers heavy expos. OPs ³ Reaction time, dominant hand Other tests	Neurobehavioral Tests Slower - signif. No signif. differences	1997 Fiedler
South Africa 1993	Fruit farm/sprayers chronic OP ³ expos. Pursuit-Aiming Santa Ana subtest Vibration sense	WHO NCTB²⁰ Small associations Small associations No loss found	1997 London
Germany 1993	“Chronic” pyrethroid illness ²² Causal link acute sym. 6 of 23 cases	Nervous System Effects All reversible.	1996 Altenkirch
UK	OP ³ exposed sheep dippers 2 point discrimin. dorsum of hand 2 point discrimin. dorsum of foot Mean calf circumference	Symptoms No symptoms Controls 22 mm 13 mm 8 mm 34 mm 10 mm 11 mm 35.0 cm 36.3 cm 38.6 cm	1996 Beach
England	OP ³ exposure Reported symptoms / chronic effects	No evidence of any association	1996 Stephens
California Case-control	Cholinesterase-inhibited subjects ²³ Serial digits	Findings Better performance sig.	1995 Ames

US	Chlordane exposure - nonoccupational Balance Reaction time Digit smbol, Trail-making Verbal recall Mood-state Long-term memory	OR Poorer performance signif. Poorer performance signif Poorer performance signif Poorer performance Poorer performance No difference No difference	1995 Kilburn
England	OP ³ exposed sheep farmers Sustained attention Speed of information processing Short-term memory and learning Psychiatric disorders	OR Poorer performance signif. Poorer performance signif. No difference Greater vulnerability	1995 Stephens
New York	Farmer pesticide applicators Long term exposures	Vibration sensitivity threshold Increased dom. hand p<.001 Increased non-dom. hand p<.04	1995 Stokes
Nicaragua	Males methamidophos poisoning ²⁴ Vibrotactile thresholds	Findings > 25 % abnormal thresholds	1994 McConn
India	Agric. sprayers exposed fenthion ¹⁶ Benton Memory Lexand Passalong ERPs ²⁵ - P3 amplitude	Psychometric Tests Poorer performance signif. Poorer performance signif. Poorer performance signif. Poorer performance signif. No significant diffeence	1994 Misra
US Case report	Methyl bromide acute poisoning Concentration, learning , memory	Sequelae Impaired	1994 Reidy
Netherlands	Flower farmers pesticide expos. 20 yrs Median motor Median sensory Sural sensory Peroneal motor Refractory period sural and peroneal	NCV⁹ -1.1 m/s decrease -1.4 m/s decrease -0.9 m/s -1.2 m/s -1.3 m/s decrease Increased	1994 Ruijten
California 1982-90	Severe OP ³ poisoning group Sustained visual attention Mood scales Vibrotactile sensitivity finger, toe Che ²⁶ inhibition group (not hosp) Hospitalization OP poisoning	Findings Poorer performance signif. Poorer performance signif. Poorer performance Poorer performance	1994 Steenland
US Case report 3 young adults	Occup. exposure to ethylene oxide Subacute polyneuropathy	Bilateral foot-drop and denervation potentials on electromyography ²⁷	1993 Finelli
US Washington	Apple orchard applicators OPs ⁹ Neuropsychiatric tests	Pre-season vs Post-season No differences	1992 Daniell
Netherlands	Pesticide exposed bulb growers Neurological exam Visual evoked potentials Electroencephalogram	OR No sig. difference No sig. difference More fast (beta) activity	1992 DeWeerd

US Case report	Chronic ethylene oxide exposure 29 y female records review	Author conclusion: Cognitive dysfunction of psychiatric etiology.	1992 Dretchen see Crystal
Netherlands	Flower growers exposed pesticides EEG findings	Changes mean spectral frequencies	1992 Jonkman
US	Ethylene oxide expos. hospital wrkrs Nerve Conduction Velocity EEG Neuropsychiatric test scores	Findings No sig. differences No sig. differences No sig. differences	1991 Estrin
US Case report	Heavy exposure to dicofol 12 yr male poisoned	Sequelae Cognitive/emotional probs. 18 mos	1991 Lessenger
US 60 cases/ctls	Multiple system atrophy Pesticide exposure	Increase risk signif.	1991 Nee
Nicaragua 1986-88 agricultural workers	Hospitalization OP ³ poisoning 2 yrs after hosp. Motor steadiness and dexterity	WHO NCTB²⁰ Sig. worse 5/6 subtests ²⁸ Sig. worse than controls	1991 Rosenstock
Bulgaria	Pre/post season OP ³ agric. spraying Evoked potentials Median nerve conduction velocity Peroneal nerve conduction velocity	Post Season Findings Increased amplitude - signif. Decreased Decreased	1990 Datsov
US Case reports	Soil fumigation workers Methyl bromide poisoning	Sequelae Neuropsych. symp. several wks	1990 Herzstein
Netherlands	Flower growers pesticide use > 10 yrs Nerve conduction velocity EEG beta-activity Attention, perceptual coding	Findings Small differences - signif. Small differences - signif. Small differences - signif.	1990 VerBerk
California Case report	Optic atrophy (blindness) Methyl bromide expos. 32 yr male	Sequelae No improvement 1 yr later	1988 Chavez
US Case report	ETO ²⁹ occupational exposure 29 yr old woman for 10 yrs	Sequelae Cognitive dysfunction disputed	1988 Crystal see Dretchen
India	Agric. sprayers exposed fenthion ³⁰ Conduction velocity Median latency Peroneal latency F min.H reflex. latency	Peroneal Nerve Slowing p 0.05 Increased p < 0.1 Increased p < 0.05 Increased p < 0.01	1988 Misra
US	Former OP ³ poisoned Memory, abstraction, mood Halstead-Reitan Battery scores MMPI ³¹ PRAPFI ³² Audiometry, vision, EEGs	Findings No significant difference Consistent w. cerebral damage/dysfxn Increased distress Increased complaints of disability No significant differences	1988 Savage
Bhopal India	MIC ³³ victims ³⁴ severe/moderate expos. Learning, motor speed, precision Motor speed and precision tests	Psychometry Results Impaired p < 0.01 Disability score, r = 0.68 sig.	1987 Misra

Germany 1980-1984	Men/women occup. expos. PCP ³⁵ Ulnar, median nerves Peroneal, sural nerves	NCV⁹ Within normal range Within normal range	1987 Triebig
California	Structural and soil fumigators ³⁵ Behavioral tests battery Finger sensitivity 1 cognitive performance test.	Methyl Bromide Group Poorer performance 23 of 27 Poorer performance signif. Poorer performance signif.	1986 Anger
Japan 4 case reports	Multiple neuropathy Ethylene-oxide (ETO) poisoning ³⁶ Chronic ETO occupational exposure	Sensory disturbance lower limbs Gait disturbance in all affected	1986 Fukushima
Yugoslavia Case report	Occupational ethylene oxide exposure 19 yr male heavy exposure	Findings Polyneuropathy - lower limbs	1984 Kovac
US Arkansas	2,4-D, 2,4,5-T manuf. workers One or more slowed NCV Sural nerve below first percentile Sural NCV ⁹ 1.3 SD ³⁷ below cntls Median motor nerve	Nerve Conduction Velocity 46% exposed vs 5% controls 48% exposed vs 10% controls Mean slowing ~ 5.2 meters m/s ³⁸ Mean slowing ~1.9 m/s	1984 Singer
Japan Case reports	Occupational ethylene oxide exposure 2 sterilizer workers	Findings Polyneuropathy ³⁹	1983 Kuzuhara
California farm workers	OPs ³ reentry poisoning 19 workers Recovery time from acute poisoning Normal cholinesterase ²⁹ Eye symptoms	Follow-up 2 to 3 months Persistent 4 months later Persistent 4 months later	1983 Whorton
US	Dieldrin occupational exposure Psychological. tests Psychomotor tests	Findings Poorer performance 5 / 58 Poorer performance 47 /58 p ≤ .05 ⁴⁰	1981 Sandifer
Germany	Agricultural pesticide handlers Exposed 3 years or more	Peroneal NCV⁹ Decreased	1976 Roder
France Case report	Methyl bromide leakage fire exting. Severe poisoning w myoclonus	Follow-up Lived 5 yrs in a stuporous state ⁴¹	1975 Goulon
US Case reports	Agricultural pilots Chronic OP ³ pesticide poisoning	Psychiatric sequelae	1964 Dille

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Footnotes

1. Parkinson's disease.
2. Based on 1982 pesticide use reporting data. At that time not all pesticide use was required to be reported .
3. Organophosphate insecticides.
4. At the time of diagnosis.
5. 10 minutes later developed erythema, hyperkeratosis, conjunctival injection, diffuse hyperemia and tearing. These irritative symptoms cleared up in 4 days.
6. CT: 2 small areas hypodensity left periventricular area near homolateral ventricular wall. MRI: small multiple, bilateral symmetric areas high signal intensity caudate nuclei, putamen, cerebral white matter near ventricular wall.
7. Dysphonia, bradykinesia, and rigidity developed over the next 5 days, stabilized after 1 week and persisted until the time of examination
8. Mn - manganese; Fe - iron; Al - aluminum.
9. Nerve conduction velocity.
10. 81 workers treated for mild poisoning (not requiring hospitalization) from cholinesterase inhibiting pesticides (organophosphates and N-methyl carbamates) compared to 130 who did not seek treatment for pesticide symptoms.
11. Cholinesterase inhibiting insecticides. This includes organophosphates and N-methyl carbamates.
12. 528 directly through mixing/spraying (mean exposure duration: 22 years), 173 indirectly through contact with treated plants, 216 were never exposed.
13. Amyotrophic lateral sclerosis (Lou Gehrig's disease). All 3 case worked in manufacture /formulation of 2,4-D (1947-49, 1950-51, 1968-86), for varying durations of time (1.3, 1.8, and 12.5 years).
14. No association with cumulative exposure.
15. Quinalphos manufacturing workers.
16. One case had occupational exposure to high concentrations of malathion, diazinon, and formaldehyde.
17. In four with peripheral neuropathy.
18. Cerebro-organic disorders; sensorimotor- polyneuropathy; vegetative nervous disorders ; cellular, humoral immune defects
19. Wood-preserving chemicals containing solvents, pentachlorophenol (PCP), and γ -hexachlorocyclohexane (lindane), and other neurotoxicants.
20. World Health Organization's Neurobehavioral Core Test Battery. These information-processing tests were designed for use in subjects with minimal education.
21. In one patient, symptoms improved within five months. In the other, paresthesia still present two years later, associated with visual after-effects.
22. Exposure from carpets, moth killers, pesticide sprays and wood preservatives.
23. History of reduction of RBC cholinesterase activity less than or equal to 70% of baseline ; or reduction of plasma cholinesterase less than or equal to 60% of baseline. Reductions were present without symptoms of frank poisoning.
24. Study done 10 to 34 months after hospitalization. Stratified as: 1) never poisoned; 2) poisoned with organophosphates other than methamidophos and 3) poisoned with methamidophos, a known peripheral neurotoxin.
25. Event related potentials.
26. Acetylcholinesterase. Inhibition of this enzyme occurs in organophosphate poisoning.
27. Gradual and complete return of strength in the lower extremities occurred 4 to 7 months after removal from exposure.
28. Verbal/visual attention and memory, visuo-motor speed, sequencing, problem solving.
29. Ethylene oxide: TWA (time weighted average) 2.4 ppm , 4.2ppm around sterilizer. OSHA stand. 1.0 ppm.
30. No clinical evidence of peripheral neuropathy or muscle weakness.

31. Minnesota Multiphasic Personality Inventory.
32. Patient's and Relative's Assessment of Patient Functioning Inventories .
33. Methylisocyanate, toxic chemical released in explosion at factory manufacturing carbaryl (Sevin) in Dec.1984.
34. 15 severely, 14 moderately, and 23 mildly affected ; mean age 38.2 (15-65);30 males. Neurological examination was normal.
35. Exposure to the fumigants methyl bromide and sulfuryl fluoride (Vikane).
36. Overexposure problems included the reverse flow of an exhaust fan, a choked air conditioner filter and the lack of protective masks in the sterilizing facility.
37. Standard deviation.
38. Sural slowing significantly correlated with duration of employment.
39. Confirmed by sural nerve biopsies.
40. Author notes most did not correlate with dieldrin blood levels, and were within the normal range.
41. Autopsy of brain: necrosis inferior colliculi, gliosis in the upper brain stem reticular formation, moderate changes in the dentate and pontine nuclei.