GREGG M. RADER SR., MD, PLLC
CLINICAL EVIDENCE REVIEW
MEDICAL-LEGAL SERVICES

EVIDENCE RESOURCE

TCE exposure, Parkinson's disease, and the medical record

A concise guide to the current evidence and the documentation needed for an individualized medical-causation assessment. Updated August 2026.

BOTTOM LINE

Human studies support an association between TCE-contaminated environments or occupational TCE exposure and Parkinson's disease (PD). Animal studies provide biologic plausibility. The evidence does *not* establish that TCE caused every individual case, and an exposure history alone is not a diagnosis or a complete causation opinion.

What the research shows

Human evidence	Finding	Important limitation
Camp Lejeune cohort (2023)	Among 340,489 service members, Camp Lejeune veterans had higher PD odds: OR 1.70 (95% CI 1.39-2.07).	Water contained TCE plus other volatile organic compounds; the study cannot isolate TCE as the sole causal agent.
Discordant-twin study (2012)	Ever TCE exposure was associated with PD: OR 6.1 (95% CI 1.2-33).	Only 99 twin pairs; few exposed subjects and a very wide confidence interval make the estimate imprecise.
Pooled occupational analyses (2017)	Pooled estimate for any TCE exposure: OR 1.8 (95% CI 0.9-3.7).	Compatible with increased risk, but confidence interval included 1.0; exposure reconstruction remains difficult.

How TCE could contribute biologically

- **Mitochondrial injury:** chronic TCE exposure has impaired complex I activity in experimental models.
- **Oxidative stress and neuroinflammation:** animal studies report oxidative damage, microglial activation, and loss of nigrostriatal dopaminergic neurons.
- **Protein handling and signaling:** experimental work reports alpha-synuclein accumulation, endolysosomal dysfunction, reduced autophagy, and increased LRRK2 kinase activity.

These mechanisms resemble recognized pathways in PD, but mechanistic plausibility is supportive evidence - not proof of individual causation.

Interpret the evidence carefully

- **Association is not the same as certainty.** Epidemiologic studies estimate group-level risk; they do not identify the cause of one person's disease.
- **Exposure mixtures matter.** Camp Lejeune water included multiple volatile organic compounds, even though modeled TCE levels were especially high.
- **Dose reconstruction is often uncertain.** Historical job titles alone are not reliable exposure measurements.
- **Long latency is plausible.** PD may be diagnosed decades after occupational exposure, so a complete chronology is essential.

MEDICAL RECORD GUIDE

Build the record around exposure, disease, and reasoning

A useful medical opinion connects person-specific facts to the literature and explains uncertainty.

1. Document the exposure

- **Agent identity:** TCE, trade name, product formulation, Safety Data Sheets, procurement or inventory records.
- **Work details:** employer/site, job title, tasks, dates, frequency, duration, proximity to source, and whether TCE was heated or sprayed.
- **Routes and controls:** inhalation, dermal contact, ingestion; ventilation, respirators, gloves, spills, confined spaces, and hygiene practices.
- **Corroboration:** industrial-hygiene sampling, exposure matrices, DOE/VA records, environmental reports, coworker statements, and incident reports.

2. Establish the neurologic diagnosis

- **Neurology records:** diagnostic assessment, examination findings, response to levodopa, progression, and movement-disorder specialist opinions.
- **Phenotype:** bradykinesia, rigidity, rest tremor, postural instability, asymmetry, prodromal symptoms, and nonmotor features.
- **Testing:** neuroimaging, DaTscan when performed, laboratory evaluation, neuropsychological testing, and therapy notes. No single test proves TCE causation.

3. Create a defensible timeline

- First exposure; periods of greatest intensity; last exposure; onset of prodromal symptoms; onset of motor symptoms; first medical documentation; diagnosis; and course over time.
- Distinguish contemporaneous records from later recollection. Explain gaps rather than ignoring them.

4. Address alternative explanations

- Family history/genetic risk; age; pesticide or other solvent exposure; traumatic brain injury; medication-induced parkinsonism; vascular disease; atypical neurodegenerative syndromes; manganese or carbon-monoxide exposure; and other relevant medical conditions.
- An alternative risk factor does not automatically exclude contribution by TCE, but it must be evaluated explicitly.

5. Structure the medical opinion

- State the diagnosis and the specific causal question. Describe exposure evidence and its limitations. Explain temporality, magnitude/duration, mechanistic plausibility, and consistency with human evidence. Analyze competing causes. State the applicable certainty standard and give a reasoned conclusion.
- Avoid unsupported statements such as “TCE always causes PD” or “no family history proves occupational causation.”

Clinical testing limitation
Blood or urine testing reflects recent exposure and generally cannot reconstruct a solvent dose from decades earlier. A normal current test does not rule out past exposure.

Selected references

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