Exhibit 127

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REPORT

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Summary

Parkinson's disease (PD) is a common neurodegenerative disease, mainly affecting motor functions, and causing rigidity, tremor at rest, slowness of movement, and impaired balance and coordination. These deficits are the consequence of the degeneration of dopaminergic neurons in the substantia nigra area of the brain. Symptoms of PD can be treated with pharmaceutical drugs, but eventually the efficacy of these treatments decreases; there is no cure for PD. Environmental factors are believed to be primarily responsible for the etiology of idiopathic PD. After a cluster of young addicts developed PD in the 1980s because of a contaminant (MPTP) found in a meperidine analog, research on environmental factors possibly involved in PD's etiology has blossomed. The main chemicals that are emerging from these studies are certain pesticides (e.g. paraquat, rotenone) and the volatile solvent trichloroethylene (TCE). The latter is the focus of this report, as members of the military, their families and civilian workers at Camp Lejeune, NC, were exposed to high levels of this compound for extended periods. The literature on TCE and PD shows a strong convergence of findings that range from human studies and observations, to controlled animal studies. Biological plausibility is also supported by mechanistic findings. Multiple epidemiological studies, in twins discordant for PD, and in Camp Lejeune veterans, indicate a strong association between exposure to TCE and PD. Additional human studies strengthen the relationship between TCE and PD. More than a dozen case reports establish associations between TCE exposure and the diagnosis of PD. Nine different controlled animal studies in rats and mice show that administration of TCE, at different dose levels and by different routes, causes loss of dopaminergic neurons, impairment of mitochondrial respiration, oxidative stress, microglia activation, neuroinflammation, and accumulation of alpha-synuclein, all hallmarks of PD. Mechanistic hypotheses focus on the role played by TaClo, a neurotoxic metabolite of TCE, on activation of LRRK2 kinase activity, and on a potential role of the microbiome/microbiota, which is affected by TCE and compromises the gut-brain axis. Considered altogether, the scientific evidence indicates that there is, more likely than not, a causal relationship between TCE exposure and PD.

Qualifications of Dr. Lucio G. Costa

I am a Professor Emeritus of Toxicology at the University of Washington in Seattle (WA, United States). After receiving a doctorate in Pharmacology from the University of Milano in 1977, serving in the Italian Army, and working as a postdoctoral fellow at the University of Milano, in 1980 I joined the laboratory of renowned toxicologist Prof. Sheldon D. Murphy at the University of Texas Medical School in Houston, TX, first as a Senior Fellow (1980-81), then as a Research Scientist (1982-83). Since 1983 I have been on the faculty of the Toxicology Program at the University of Washington, where I was promoted to full professor with tenure in 1992. Over the years I have served as the Director of the University of Washington Toxicology Program, Director of the Neurotoxicology Research Core of the NIEHS Center in Ecogenetics and Environmental Health, and as a Core Faculty of the Environmental and Occupational Epidemiology Training Program, the Environmental Pathology/Toxicology Training Program, and the Interdisciplinary Molecular and Cellular Biology Program, all at the University of Washington.

I am a member of several national and international professional organizations such as the U.S. Society of Toxicology (since 1982), the Neurotoxicology Specialty Section of the Society of Toxicology (past councilor and president), the Pacific Northwest Chapter of the Society of Toxicology (past councilor and president), the Society for Neuroscience (since 1983), the International Neurotoxicology Association (since 1985, founding member, past president, and past councilor). I am also a Fellow of the Academy of Toxicological Sciences (Certification in General Toxicology) (since 1993), and a European Certified Toxicologist (ERT; since 2006). I received a Special Emphasis Research Career Award from the National Institute of Occupational Safety and Health in 1986, and the Achievement Award in 1995 and the Zeneca Travelling Lectureship Award in 1997, both from the Society of Toxicology. In 1994 I was elected Fellow of the American Association for the Advancement of Science.

I am the Editor of the scientific publications “Current Protocols in Toxicology”, and “Advances in Neurotoxicology”, a past Associate Editor (1989-2023) of the scientific journal “Neurotoxicology”, and a current or past member of the editorial boards of several

toxicology and pharmacology journals. I also served as a manuscript reviewer for over seventy different scientific journals.

I have been a member of several grant review panels for the National Institutes of Health and the U.S. Environmental Protection Agency and have reviewed grant proposals for over 25 agencies worldwide. I have been a member of dozens of panels and committees at the national and international level dealing with toxicology and risk assessment issues. Among these, I was a member of review panels for the U.S. Environmental Protection Agency, the Institute of Medicine/National Academy of Sciences, the European Food Safety Agency, and the World Health Organization. I was also a member of the Pesticide Incident Reporting and Tracking (PIRT) panel of the State of Washington from 1989 to 2003, and a member of the Agricultural Pesticide Advisory Board of the State of Washington (Gubernatorial appointment) from 1989 to 2000.

Over the years, I have chaired and/or organized symposia at dozens of scientific meetings in the United States and internationally and have been invited to give lectures at over 80 national and international conferences. I was also invited to give seminars in several Universities and Research Centers in several countries.

I have been teaching classes in toxicology and neurotoxicology at the University of Washington for over 35 years and have supervised over 70 graduate students (PhD and MS) and 38 senior fellows and visiting scientists. I have kept an active research program in the area of neurotoxicology, with funding provided by the National Institutes of Health, the Environmental Protection Agency, the National Science Foundation, and the Department of Defense. The primary focus of my research relates to the toxicology and neurotoxicology of pesticides, solvents, metals, environmental contaminants, air pollution, and on various aspects of gene-environment interactions in relationship to neurotoxicity and neurodegeneration. I have authored over 320 papers published in peer-reviewed journals, in addition to 32 books, and over 135 book chapters. Please see the list of publications for the past ten years in the Appendix.

Methodology

The objective of this report is to determine whether there is a causal relationship between exposure to the solvents trichloroethylene (TCE) and perchloroethylene (PCE) and the etiology of Parkinson's disease (PD). This report and the opinions expressed are based on the review of information provided by studies in the open literature on TCE, PCE, and PD. Searches on PubMed were performed mostly with the keywords TCE, PCE, PD, neurotoxicity. This opinion considers human studies (epidemiological studies and case reports), controlled animal studies, and mechanistic information (usually deriving from animal and/or in vitro studies). The approach and methodology used were the same as in my research and scholarly activities.

What is Parkinson's disease (PD)?

Parkinson's disease (PD) is a movement disorder characterized by the loss of dopamine-producing neurons in the substantia nigra region of the midbrain. First described by Dr. James Parkinson in 1817, PD is the second most common neurodegenerative disease (after Alzheimer's disease), with an incidence of 10-20 cases per 100,000 per year, but it is now considered the fastest growing neurological disorder in terms of prevalence (Dorsey and Bloem, 2024). Global incidence in 2040 is projected at 20 million cases (De Miranda and Greenamyre, 2020). While aging represents a significant risk factor for PD development, the projected increase in PD incidence appears to outpace that of normal aging, suggesting that, given the primary environmental causes of the disease (see next section), global industrialization and pollution may be strongly involved. Parkinsonism is a general term that refers to a group of neurological disorders that share some of the same motor symptoms as PD but may have additional symptoms. There appears to be a gender difference in the incidence of PD, with a 1.5-3-fold male preponderance. PD can manifest as a rare, familial form and as a most common sporadic form, known as idiopathic PD. PD can be thought of as a disease of dopamine deficiency. Selective dopaminergic neuron death results in diminished ability to control voluntary movements,

and hence the cardinal symptoms for diagnosis include slowness of movement (bradykinesia), rigidity, tremor at rest, and impaired balance and coordination. Current therapeutic interventions, which diminish symptoms but do not “cure” the disease, are aimed at increasing dopamine levels or effects, and include the dopamine precursor levodopa, dopamine receptor agonists, and inhibitors of MAO-B and of COMT, two enzymes involved in dopamine metabolism. PD is also a disease of protein accumulation, as the protein alpha-synuclein accumulates in dopaminergic neurons in structures known as Lewy bodies. Neuroinflammation is also a cardinal event in PD. In addition to motor deficits, PD patients can display other symptoms including depression, sleep disturbances, decreased olfaction, and gastrointestinal issues. Some of these symptoms (e.g., olfactory deficit) may precede motor symptoms by several years.

What causes PD?

Environmental factors have long been thought to be fundamentally important in PD’s etiology (Dorsey and Bloem, 2024). The link between environmental factors and PD has been strengthened by two important findings in PD research: (1) Twin studies have not identified a major role for genetics in sporadic PD (Tanner et al. 1999). PD has been calculated to have a hereditability estimate of 0.34, almost at the bottom of the list of common disorders with mixed etiology (e.g., schizophrenia is 0.81), emphasizing the minimal genetic contribution to the etiology of the disease (Bogers et al. 2023). (2) A small cluster of cases of Parkinsonism was identified in the 1980s in Northern California, in which a contaminant of synthetic meperidine, called MPTP, was shown to be responsible for causing the same brain lesions that are found in PD/Parkinsonism (Langston et al. 1983). This episode is of utmost importance as it was the first one in which it was firmly established that an exogenous agent could damage the dopaminergic system and cause the same symptoms as in PD. The cases were young (most in their twenties) and had in common only the fact they were drug addicts and had consumed the same batch of synthetic meperidine. They displayed typical PD symptoms and responded to pharmacological therapies used in PD. Years of subsequent experimental work have defined that MPTP is rapidly absorbed and transfers to the brain, where it is converted

into the ultimate neurotoxic agent, MPP+, which accumulates in dopaminergic neurons and causes their demise by inducing oxidative stress and damaging mitochondria (Langston, 2017).

This episode prompted further studies investigating possible environmental risk factors for PD. Among environmental toxicants, pesticides, particularly the herbicide paraquat, which has a structure similar to MPTP, and the natural insecticide rotenone (Goldman et al. 2017), and some organochlorine compounds, in particular trichloroethylene (TCE) (De Miranda and Greenamyre, 2020), have emerged as strong links over the past few decades.

One important fact to consider is that exposure to a neurotoxic agent may precede the appearance of PD symptoms by decades. It has been calculated that PD symptoms manifest when brain dopamine content has decreased by 70-80%. Because of the normal aging process, there is a progressive loss of dopamine, but the “threshold” for clinical signs to be manifest has been estimated to be reached after the age of approximately 110 years. In individuals diagnosed with PD, one or more environmental agents, acutely or chronically, are believed to cause damage to the dopaminergic system, which adds to the normal decline due to aging. When the threshold is reached, then the clinical signs of PD begin to manifest. Indeed, in the cluster of MPTP-exposed individuals, there were subjects exposed to high levels of MPTP, who rapidly developed PD symptoms, and other subjects, who were exposed to lower doses of MPTP, who were asymptomatic at the time, but upon PET scan showed significant loss of dopaminergic neurons and were predicted to develop PD in the subsequent years (Calne et al. 1985).

What is trichloroethylene (TCE)?

TCE is a simple halogenated hydrocarbon (C_2HCl_3). It is clear and colorless, and highly volatile. It was first synthesized in 1836, but the actual discovery is thought to have occurred in 1864 in Germany by Emil Fischer. It was initially proposed as an anesthetic, as it was thought to be less toxic than chloroform, but its main use over the years has been for cleaning. Its powerful degreasing properties made it the staple solvent for the

dry-cleaning industry, later replaced by PCE. It has also been used as a solvent in the textile industry and in decaffeination processes. TCE is still used extensively in various industries and is released in the environment from the industrial sites. It is currently a contaminant in most Superfund sites. Human exposure occurs via ingestion of contaminated water, dermally, or by inhalation.

Can TCE cause PD?

As indicated by increasing evidence provided by studies in humans and animals, TCE can cause PD. TCE has been considered as a potential risk factor for PD since the late 1990s following the publication of a single case report (Guehl et al. 1999; see below) describing a woman who was diagnosed with PD several years after exposure to TCE. Additional case reports and clinical/epidemiological studies have since confirmed this association. In addition to the epidemiological findings, several controlled animal studies in rats and mice have shown that TCE targets dopaminergic neurons in the substantia nigra, causes mitochondrial toxicity, induces microglia activation and neuroinflammation, as well as alpha-synuclein accumulation (see below). These effects are found in individuals diagnosed with PD. Furthermore, potential mechanisms underlying the dopaminergic neurotoxicity of TCE have been proposed, involving products of TCE metabolism (TaClo), as well as effects mediated by the intestinal microbiota (see below). All this evidence is presented and discussed in greater detail in the following sections. Several reviews on the relationship between TCE and PD have been published in the past decade (Goldman, 2010; Caudle et al. 2012; Lock et al. 2013; De Miranda and Greenamyre, 2020; Chen et al. 2022; Dorsey et al. 2023a; Dorsey and Bloem, 2024; Dorsey et al. 2024). In 2009 the National Research Council (NRC) investigated the potential health effects of contaminated water supplies at Camp Lejeune (NRC, 2009). In their report, they concluded that “inadequate/insufficient evidence to determine whether an association exist” between TCE exposure and PD. Indeed, only a few human/epidemiological studies were available at that time, and no animal or other supporting study. The three studies considered by the NRC indicated a trend toward an association with solvent exposure whose significance increased with increasing exposure

time (McDonnell et al. 2003); no evidence of association with solvents except for individuals with certain genotypes, e.g. glutathione-S-transferase M-null individuals (Dick et al. 2007); a cluster of PD cases linked to exposure to TCE (Gash et al. 2008, also discussed in this report). All other evidence on a causal relationship between TCE and PD and discussed in the present report was published after 2009.

Human evidence: epidemiological studies

There are a few clinical/epidemiological studies which report of a strong association between TCE exposure and the development of PD, in addition to a recent retrospective study which also found an association between TCE and PD. Furthermore, a very recent study links mortality due to PD to TCE exposure.

Goldman et al. 2012. This is a case-control study in twins. Ninety-nine twin pairs, discordant for PD, were interviewed for lifetime occupations and leisure activities. Based on this information, a cumulative exposure index to TCE and other solvents was calculated. The OR (odds ratio, a measure of association between exposure and outcome) for TCE and PD was 6.1 (95% CI=1.2-33, $p<0.034$). TCE represented the strongest risk for PD, followed by PCE, which -like TCE- was also present in two wells at Camp Lejeune, NC, and by carbon tetrachloride. Twins are genetically very similar or identical and share many demographic and lifestyle factors; thus, using a discordant twin-pair designs (with or without PD) is considered more resistant to confounding factors. This study was the first epidemiological study which found an association between TCE exposure and PD.

Bove et al. 2014a; 2014b. These two studies examined mortality among marines and navy personnel (Bove et al. 2014a) and among civilian employees (Bove et al. 2014b) at Camp Lejeune, as compared to those at Camp Pendleton. Mortality hazard ratios at Camp Lejeune were significantly higher for many causes, including PD. The study estimated levels of TCE and PCE in water in the Hadnot Point system during the period 1975-1985 of 359 μg/L and 16 μg/L, respectively.

ATSDR (Agency for Toxic Substances and Disease Registry), 2018. This is a large morbidity study conducted on more than 200,000 marines and 8,000 civilians who were stationed at Camp Lejeune between 1972-1985, who were compared with marines and civilians at Camp Pendleton. Overall responses to the survey was only 31%, and medical confirmation was obtained in 50-60% of all cases. A significant increase in PD incidence was found in the civilian population exposed to TCE and PCE at Camp Lejeune, but not in the marines. This was due to the fact that PD is a disease of old age, and at the time of the survey only 7% of the marines were >65 years old, versus 49% of the civilians. The OR for PD in the civilian population was 2.78 (95% CI = 0.87-8.94) for individuals with a combined "low" TCE/PCE exposure (defined as less than 10,868 ppb-months for TCE and less than 457 ppb-months for PCE). The OR values were higher for higher TCE /PCE exposures.

Goldman et al. 2023. This population-based cohort study examined the risk for PD among soldiers and other personnel who resided in Camp Lejeune, NC, for at least three months between 1975 and 1985 (n=172,128), with a follow up in 1997-2021. Soldiers and other personnel who resided at Camp Pendleton, CA (n=168,361) served as controls. A total of 430 veterans had PD; 279 were in Camp Lejeune, and 151 in Camp Pendleton. Risk of PD was found to be 70% higher in Camp Lejeune veterans (OR=1.70; 95% CI=1.39-2.07; p<0.001). Camp Lejeune veterans also had a significant increase of prodromal PD diagnoses, such as tremors (OR=1.19; 95% CI=1.09-1.29, p<0.01). This study is considered, at least as of now, the strongest human evidence of an association between TCE (and PCE) exposure and the development of PD (Wadman, 2023; Dorsey et al. 2023b). Furthermore, the extent of the issue may have been underestimated, as family members of soldiers and other civilians working at the base were not fully considered in the study.

Dorsey et al. 2024. This is a retrospective study which identified a cluster of PD cases in attorneys working near a dry-cleaning facility, which used TCE and PCE as cleaning solvents, located in Rochester, NY. The proportion of PD in the exposed group was higher than expected based on age and sex (5.1% vs. 1.7%, p<0.01). The number of PD cases were significantly higher in the exposed group than in a reference group (4 vs. 1). Possibly

because of the small number of subjects, this result was not statistically significant ($p<0.21$).

Bove et al. 2024. This is a large study, follow-up to two previous studies by the same authors (Bove et al. 2014a; 2014b), aimed at evaluating mortality of Marines, Navy personnel and civilian workers at Camp Lejeune and at Camp Pendleton. The former were exposed to TCE and to PCE over a thirty-year period (1953-1985). The findings indicate a two-fold increase of mortality due to PD in Marines and Navy personnel at Camp Lejeune, compared to Camp Pendleton.

Goldman et al. 2024. This study is a follow-up of the previous study by the same authors on the incidence of PD in former service members stationed at Camp Lejeune between 1975 and 1985 (Goldman et al. 2023). The investigated cohort consisted of 84,824 veterans with an average duration of 25 months (range: 3-108 months) residence in the base, between 1975 and 1985. A total of 177 PD cases were exposed to levels of total volatile compounds (TVOCs) -including TCE- with a median of 4,970 (1,676 – 11,410) μL/L-months. Compared to individuals with PD not exposed to volatile organic compounds (VCOs), exposed personnel registered a significantly faster disease progression, as evidenced by a reduced time to psychosis, factures and falls. This study adds to the evidence that exposure to TCE, and perhaps other VOCs such as PCE, can cause a more rapid progression of PD.

Human evidence: case reports

Guehl et al. 1999. Case 1 was a 47-year-old woman who had a history of exposure to TCE in the cleaning and plastic industries. She developed problems in walking, and rigidity on the right side. She was subsequently diagnosed with PD, and later experienced severe on-off phenomena and levodopa-induced dyskinesia.

Gash et al. 2008. Case 1 was a 49-year-old man with a 25-year history of exposure to TCE used as a degreaser. He did not use personal protection equipment and exposure

was mainly dermal and by inhalation. Case 2 was a colleague of Case 1 who shared a similar job and was diagnosed with PD in his sixties and died at the age of 76. Case 3 was a 56-year-old woman with occupational exposure to TCE in the same plant as Cases 1 and 2. She was exposed to TCE dermally and by inhalation and was diagnosed with PD at the age of 53, though she had symptoms (freezing, rigidity, bradykinesia) for several years before diagnosis.

Reis et al. 2016. Case 1 was a 43-year-old man who had worked as a car painter since the age of 18. He developed the first signs of PD (tremors) at the age of 38 and was diagnosed with PD a few years later. He had been working with solvents, including TCE, for over 20 years. Case 2 was a 52-year-old man who also worked with solvents for twenty years and was diagnosed with PD at the age of 48. Case 3 was a 59-year-old car painter who had been working with solvents including TCE since the age of 18, who started to show signs of PD at the age of 59.

Dorsey et al. 2023a. Case 1 was a male professional athlete who developed signs of PD and received a PD diagnosis at the age of ~ 34. He had spent his early childhood at Camp Lejeune where he drank, bathed, and swam in TCE-contaminated water. Case 2 is a 57-year-old woman who was diagnosed with PD almost thirty years after serving four years as a captain in Camp Lejeune where she was exposed to TCE-contaminated water. Case 3 is a 48-year-old physician who was raised in a community heavily contaminated with TCE and had further TCE exposure in the following years. He was diagnosed with PD at the age of 38. Case 4 was an 85-year-old physicist who had been exposed occupationally to TCE for several years and was diagnosed with PD at the age of ~72. Case 5 was a 72-year-old retired teacher who had been working with TCE in a print shop and was diagnosed with PD 35 years later. Case 6 was diagnosed with PD at the age of 36. He had been using TCE in several jobs for about seven years. Case 7 was a politician diagnosed with PD at the age of ~70. He had been exposed to TCE while degreasing planes in the military for six years in his twenties.

These fourteen case reports provide strong supportive evidence of the relationship between TCE and the development of PD. Case reports have known advantages but also disadvantages. The first case report (Guehl et al. 1999) initiated a signal, while the case

series with multiple cases provides stronger evidence, particularly when considered in a full framework of other studies.

Animal evidence

Numerous experimental studies in animals strongly support the observations in humans of a causal relationship between TCE exposure and PD, and these are briefly summarized and commented on below.

Guehl et al. 1999. Adult male OF1 mice were treated by intraperitoneal (i.p.) injection daily for four weeks (5 days/week) with 40 mg/kg TCE or saline and sacrificed after another week with no treatment. TCE-treated mice did not display any motor abnormalities but presented a significant lower number (-50%) of tyrosine hydroxylase (TH)-positive neurons in the substantia nigra, indicating degeneration of dopaminergic neurons. Very similar results had also been seen with the potent dopaminergic neurotoxicant MPTP (Bezard et al. 1997).

Gash et al. 2008. Adult male Fischer 344 rats were treated by oral gavage with 1000 mg/kg TCE in olive oil for six weeks (5 days/week). The brain was dissected out and mitochondria were isolated for measurement of mitochondrial respiration and enzyme activity. TCE was found to selectively inhibit complex I activity in the substantia nigra, while sparing other brain areas and the liver. There was also a significant decrease in the number of TH-positive neurons in the substantia nigra. Cytoplasmic alpha-synuclein inclusions were present in the substantia nigra of TCE-treated rats but not in controls.

Liu et al. 2010. Adult male Fischer 344 rats were given 200, 500 or 1000 mg/kg TCE by oral gavage for six weeks (5 days/week), while controls received olive oil. TH histochemistry revealed a dose-dependent loss of dopaminergic neurons in the substantia nigra, i.e., 20.1% at 200 mg/kg, 25.6% at 500 mg/kg, and 45.6% at 1000 mg/kg. Additional staining indicated a real loss of dopaminergic neurons. The effects of TCE were selective for dopaminergic neurons in the substantia nigra, as other brain areas and other neurotransmitter systems were not affected. Striatal levels of DOPAC (a dopamine metabolite) were decreased by 33% in the 1000 mg/kg TCE group, and this

change was still present when rats were sacrificed two weeks after termination of TCE exposure. Rats exposed to the highest dose of TCE had a significant motor deficit on the rotarod, but did not differ from controls in open field behavior. Additional studies carried out in TCE-treated rats (1000 mg/kg) showed a significant reduction of mitochondrial complex I at week two of treatment. Cleaved caspase 3 immunostaining showed strong cytosolic-positive signals in TCE-treated rats, suggesting that apoptosis (a type of cell death) is involved in the loss of dopaminergic neurons. Oxidative and nitrosative stresses, as well as microglia activation were also increased in TCE-treated rats, together with an increase in alpha-synuclein. All findings are consistent with oral TCE exposure targeting dopaminergic neurons in the substantia nigra, the same that are affected in PD.

Sauerbeck et al. 2012. Male rats (Fischer 344) were treated with TCE in olive oil by oral gavage at the dose of 1000 mg/kg for one or two weeks. At either time-point rats were subjected to traumatic brain injury (TBI) with the purpose of investigating potential interactions between TCE and TBI. The short exposure to TCE caused some of the same effects previously reported (e.g. decreased mitochondrial respiration, decreased number of dopaminergic neurons), and these were significantly increased by TBI in a synergistic fashion. Of interest is that TBI alone has been shown to play a role in the development of PD (Bower et al. 2003).

Liu et al. 2018. Young adult male C57BL/6 mice were treated with TCE in olive oil at the dose of 400 mg/kg by oral gavage for eight months (5 days/week). The treatment resulted in a progressive significant loss of dopaminergic neurons in the substantia nigra (-34.1 at 3 months and -48.1% at 8 months), and significant losses of dopamine (-50.2%) and DOPAC (-72.1%) in the striatum at 8 months. Additional findings included decreased locomotor activity and rotarod performance, mitochondrial dysfunction, oxidative stress, alpha-synuclein misfolding, and inflammation in the substantia nigra. All these findings confirm previous results in rats but were observed after a longer exposure to TCE (8 months vs 6 weeks) at lower dose levels (400 mg/kg vs 1000 mg/kg). Of relevance in the present study are also the measurement of the brain levels (31 pg/g) of TaClo (1-trichloromethyl-1,2,3,4-tetrahydro-beta-carboline, a neurotoxic metabolite of TCE), which

was also shown to cause MPP+-like neurotoxicity in nigrostriatal organotypic cultures in vitro.

Keane et al. 2019. Young adult C57BL/6 male and female mice were treated with TCE (1000 mg/kg, twice weekly, for eight weeks, by i.p. injection). No effects were seen on motor activity in the accelerating rotarod. TCE decreased the number of TH-positive cells in the substantia nigra (by ~50%). The effects of TCE were also measured in A30P mutant alpha-synuclein mice (carrying a human mutation affecting alpha-synuclein processing), which would be expected to display enhanced susceptibility to TCE. Still no significant effects were seen in the behavioral experiments, but TCE decreased dopaminergic neurons by ~70% in A30P mutant mice. In another experiment, TaClo was also injected directly in mice, with the same protocol as TCE, and at the dose of 2 mg/kg. TaClo did not cause significant behavioral effects but decreased the number of dopaminergic neurons.

De Miranda et al. 2021. In various experiments, male and female adult Lewis rats were given TCE in olive oil at the doses of 50, 100, 200, or 400 mg/kg by oral gavage for one, three or six weeks. Administration of 200 mg/kg TCE to aged adult rats (15-month-old) for six weeks caused reduction in locomotor activity, a 30% reduction of TH staining and a 32% loss of dopaminergic neurons. This same treatment increased oxidative stress and increased activated microglia. Novel findings were an increase in endosomes in dopaminergic neurons leading to loss of endolysosomal function which is considered a pre-degenerative pathology in PD, and an increase in LRRK2 (leucine rich repeat kinase 2) activity which was found to precede dopaminergic neuron degeneration. The effect on LRRK2 was dose-dependent (50-800 mg/kg TCE) and may be related to the increased oxidative stress caused by TCE. This finding is of much relevance as the most commonly inherited mutations associated with elevated risk for PD are within the LRRK2 gene, and account for 1-2% of all cases. LRRK2 plays a relevant role in the autophagy-lysosomal pathway (ALP) and its overactivity due to specific mutation or to an environmental agent such as TCE would result in ALP dysfunction.

Adamson et al. 2023. While all previous studies involved administration of TCE by the oral or intraperitoneal routes, in this recent study adult male and female Lewis rats as well as adult C57BL/6 mice of both sexes, were exposed to TCE by inhalation. Rats were

exposed for 7 days/week for eight weeks, while mice were exposed for 7 days/week for twelve weeks. Exposure levels of 50 ppm for rats and 100 ppm for mice were equivalent to a human exposure of ~ 8 ppm. This level is lower than the TCE exposure limits set by U.S. regulatory agencies. For example, the Threshold Limit Value (TLV) for TCE is 10 ppm. In both rodent species TCE exposure caused a loss of dopaminergic neurons, microglia activation, accumulation of alpha-synuclein, and behavioral motor alterations (more in rats than in mice).

Ilieva et al. 2024. This study is a follow-up of that of De Miranda et al. (2021). Adult female rats were treated orally with TCE (200 mg/kg) for six weeks. Confirming previous findings, this treatment caused an increase of oxidative stress in dopaminergic neurons, mitochondrial damage, activation of microglia, a reduction of mitophagy (a process of removal of damaged mitochondria), increased neuroinflammation, and a 45% loss of dopaminergic neurons. Activity of LRRK2 was increased by TCE. Administration of MLi2, an inhibitor of LRRK2 activity, significantly attenuated all effects of TCE. Parallel in vitro experiments in gene-edited HEK 293-T cells confirmed these findings, suggesting a key role of LRRK2 in TCE-induced neurotoxicity and the protection afforded by LRRK2 inhibition.

These studies are consistent in showing that exposure to TCE causes dopaminergic toxicity, as observed in PD. Of relevance is the fact that TCE exerts its neurotoxicity in both rats and mice of both sexes, when administration occurred by the oral, intraperitoneal or inhalation routes. Also, the effects are dose- and time-dependent. The results clearly indicate alterations of mitochondrial functions (specifically of complex I in the respiratory chain), loss of dopaminergic neurons in the substantia nigra, increased oxidative stress and microglia activation, accumulation of alpha-synuclein, and some behavioral motor changes. In addition, a neurotoxic metabolite of TCE, TaClo, was detected in brain upon TCE administration, and was shown to cause the same neurotoxicity as TCE when injected directly to animals. Finally, TCE was shown to induce LRRK2 activity, mimicking the changes seen in individuals carrying specific LRRK2 mutations that may represent a risk factor for a small percentage of PD cases. Overall,

the controlled animal experiments provide very strong support to human findings, indicating that TCE can induce brain damage of the same nature as that observed in PD.

Biological plausibility: possible mechanisms of TCE-induced PD/Parkinsonism

The most studied and so far, most convincing potential mechanism for TCE-induced PD/Parkinsonism focuses on the role played by TCE metabolism. Because of its chemical properties (e.g. volatility, lipophilicity), TCE is readily absorbed across biological membranes, and is extensively metabolized. Specifically, TCE undergoes metabolism by two major pathways, cytochrome P450 (CYP)-dependent oxidation, and conjugation with glutathione (GSH) (Lash et al. 2000). The oxidative pathway is quantitatively by far the major pathway of TCE metabolism; it is mediated by various CYPs, particularly CYP2E1, and occurs primarily in the liver. While more than one of the oxidized metabolites of TCE may be involved in its dopaminergic neurotoxicity, most attention has been devoted to chloral (trichloroacetaldehyde). Chloral can react with the endogenous biogenic amine tryptamine to form 1-trichloro-1,2,3,4-tetrahydro-beta-carboline (TaClo). TaClo, which is structurally similar to the dopaminergic neurotoxicant MPTP, has emerged as an important factor that can explain the dopaminergic neurotoxicity of TCE (Riederer et al. 2002). TaClo impairs dopamine metabolism and inhibits mitochondrial respiration, causes oxidative stress and neuroinflammation in dopaminergic neurons and leads to their apoptotic cell death (Bringmann et al. 2000; 2002; Boulton et al. 2012; Keane et al. 2019; Yang et al. 2019). In addition, when given in vivo to mice, TaClo causes the same dopaminergic neurotoxic effects as seen with TCE (Liu et al. 2018). The mitochondria are the main target for compounds, such as MPTP, that cause PD. Inhibition of mitochondrial complex I causes an impairment of mitochondrial respiration with a resulting incomplete electron transfer within the mitochondrial respiratory chain. This leads to depletion of ATP (adenosine triphosphate) and promotion of oxidative stress and apoptosis of dopaminergic cells.

In addition to the role played by TaClo, the ability of TCE (and/or its metabolites) to stimulate LRRK2 kinase activity in dopaminergic neurons is considered of relevance, since this results in dopaminergic neuropathology and degeneration. Pharmacological inhibition of LRRK2 significantly reduces the dopaminergic neurotoxicity of TCE (Ilieva et al. 2024). Genetic mutations of the LRRK2 gene (resulting in a gain-of-function) are one of the most common genetic risk factors for PD, and individuals who carry such mutations, already at increased risk for PD, may be particularly susceptible to the neurotoxic effects of TCE (De Miranda and Greenamyre, 2020).

Finally, an alternative /complementary mechanism may be related to the gut microbiota (Chen et al. 2022). Like various other compounds (e.g. some pesticides), TCE has been shown to perturb the microbiome/microbiota. For example, in adult rats treated orally with 200 mg/kg TCE for six weeks there were changes in gut microorganisms reflective of the microbial changes found in patients with idiopathic PD, such as decreased abundance of short-chain fatty acid acid-producing *Blautia*, and elevated lactic acid-producing *Bifidobacteria* (Ilieva et al. 2022). Similar findings were also reported in mice upon long-term administration of TCE in drinking water (Khare et al. 2018). A recent paper by Kulcsarova et al. (2023) suggests that the increasing number of PD cases (which the authors define as the “Parkinson’s pandemic”) may indeed be fueled by effects of environmental contaminants on the gut. In this regard, it has been shown that LRRK2 variants are associated with both PD and with inflammatory bowel disease, and activation of LRRK2 kinase activity by TCE may influence the gut-brain axis governing PD (Peter and Strober, 2023).

Dose level considerations

During 1975-1985, the period of maximal contamination in Camp Lejeune, the estimated monthly median TCE levels were 366 μg/L (366 ppb) (Goldman et al. 2023). At the Hadnot Point drinking water plant in Camp Lejeune, reconstructed TCE concentration reached a maximum monthly average value of 783 μg/L during November 1983, with a one-time maximum measured value of 1400 μl/L (ATSDR, 2017; Maslia et al. 2016). In the periods 1975-1979 and 1980-1985, TCE levels in water averaged an estimated 328 μg/L and 446

μg/L, respectively (Bove et al. 2014a). Note that the Maximum Contaminant Level (MCL) set by the US Environmental Protection Agency for TCE is 5 μg/L. Thus, the levels of TCE in water at Camp Lejeune greatly exceeded this value, for an extended period.

Military personnel exposed to contaminated drinking water at Camp Lejeune between 1975 and 1985 had a 70% increased risk of PD, compared to military personnel at Camp Pendelton, where water was not contaminated (Goldman et al. 2023). In addition, military personnel at Camp Lejeune exposed to TCE also had a faster progression of PD compared to non-exposed individuals (Godman et al. 2024). Note that the duration of exposure at Camp Lejeune was on average 27.5 months, with a range of just 3 months to 108 months (Goldman et al. 2024).

Animal studies are often carried out at higher dose levels, as exposure times are shorter. Nevertheless, all animal studies in rodents indicate that TCE, given by the oral or i.p. routes, is neurotoxic, and causes the same alterations in the central nervous system (CNS) as present in PD. A study also examined the effects of TCE in rats and mice when given by inhalation (Adamson et al. 2023). This study is noticeable for two reasons: (1) low levels of TCE were utilized (50 ppm in rats and 100 ppm in mice, corresponding to a human exposure of 8 ppm, below the TLV value for TCE, which is 10 ppm); (2) longer duration of exposure were utilized (2 months in rats, 3 months in mice). Such exposure caused “potent dopaminergic neurodegeneration and recapitulated some of the observed neuropathology associated with PD” (Adamson et al. 2023). Overall, this latter experimental protocol more closely mimics real life human exposure in terms of dose level and duration of exposure.

In summary, it can be concluded that exposure of three months or longer to the contaminant TCE at the level of 366 ppb, or exposure to equivalent cumulative ppb of TCE, can, at least as likely as not, be a cause of PD. This does not exclude that shorter periods, or lower levels of exposure may also cause PD. Furthermore, other factors, including genetics, may increase susceptibility of certain individuals to TCE-induced PD. In addition, strong consistent and convergent experimental animal findings support the conclusion that TCE can cause the same pathological changes in the CNS as present in PD.

Is there a role for PCE in PD?

PCE (perchloroethylene or tetrachloroethylene) is a widely used solvent, particularly in dry-cleaning operations. Its structure (C_2Cl_4) is very similar to TCE (C_2HCl_3). There is evidence that Marines, Navy personnel and civilians, and civilian workers at Camp Lejeune were also exposed to PCE, in addition to TCE (Bove et al. 2024). In the twin study by Goldman et al. (2012), exposure to PCE was a nearly significant risk factor for PD. Indeed, while the OR for TCE was 6.1 (95% C.I.=1.2-33, p<0.034), the OR for PCE was 10.5 (95% C.I.=0.97-113, p<0.053), thus barely shy of statistical significance (p<0.050). When exposures to TCE and PCE were combined, the OR was 8.9 (95% C.I.=1.7-47, p<001), i.e. of higher statistical significance than TCE alone (Goldman et al. 2012). Exposure to PCE also likely contributed to the increased mortality of civilians employees at Camp Lejeune reported in the Bove et al. (2024) study. Furthermore, in the 2018 ATSDR morbidity study of civilian employees, significant OR values of 2.78 to 3.47 were found in individuals with low to moderate combined TCE/PCE exposure (ATSDR, 2018). Thus, the contribution of exposure to PCE to PD, while less studied than that of TCE, cannot be discounted. Though supporting animal studies directly assessing the dopaminergic neurotoxicity of PCE are not yet available, other lines of evidence suggest that exposure to PCE may cause PD. First, the overall neurotoxicity profiles of PCE and TCE are very similar (Bale et al. 2011). Second, PCE, like TCE, can be metabolized to the mitochondrial neurotoxicant TaClo (Riederer et al. 2002) which is believed to be the ultimate toxicant in TCE dopaminergic neurotoxicity. Third, in cells in culture, PCE - like TCE - causes activation of LRKK2 and ensuing oxidative stress. This latter pathway is emerging as relevant in TCE-mediated dopaminergic neurotoxicity (Ilieva et al. 2024). Thus, considering PCE a dopaminergic neurotoxicant and a cause of PD, is certainly plausible.

Conclusions

Findings of epidemiological studies in humans, human case reports, controlled animal studies, and mechanistic investigations on biological plausibility, converge in convincingly

establishing that TCE can cause the same type of brain damage as seen in PD. The evidence is strong, as it is provided by the three important areas of epidemiology, experimental toxicology, and mechanistic studies. Results from the epidemiological studies clearly establish an association between TCE exposure and PD. This human evidence is supported by a series of case reports, which similarly indicate a relationship between TCE exposure and PD. Animal studies provide a very strong support for TCE to be causally linked to PD, as the administration of TCE to naïve mice and rats causes the same type of brain damage as seen in PD. Finally, mechanistic findings provide evidence that there are plausible ways by which TCE can cause PD.

Altogether, these findings indicate that TCE is a strong candidate as one of the principal environmental factors responsible for PD. It is safe to state that, as to the link between TCE and PD, the field has already transitioned from an observed "association" to a verdict of "causation". To distinguish between causal and noncausal associations in observational studies, specific considerations (also referred to as viewpoints or criteria) have been proposed by Sir Austin Bradford Hill (Hill, 1965). These observations are derived from those developed by the U.S. Surgeon General for establishing a causal association between smoking and adverse health effects, and relate to strength of association, consistency, specificity, temporality, biological gradient, plausibility, coherence, experimental evidence, and analogy (Hill, 1965; Rothman and Greenland, 2005; Fedak et al. 2015). How do these considerations apply to the case of TCE and PD? Do they prove causation? The quick answer is yes. Strength of association and consistency are shown by the presence of multiple, statistically significant epidemiological studies linking TCE exposure to PD. Specificity (a much-debated consideration, which does not play a relevant role in causal inference; Rothman and Greenland, 2005) would not apply to this case, as TCE exposure causes many other adverse health effects in addition to PD. Smoking is another well-known situation in which specificity is not present. Considerations on temporality are believed to be the *sine qua non* for causality, as exposure must precede the disease, and this is indeed the case for TCE exposure and the development of PD. The presence of a biological gradient is indicated by increasing

PD incidence associated with higher and longer exposures to TCE. Considerations on plausibility are important, as they relate to the biological plausibility of a TCE-PD causal association, based on current scientific knowledge. The mechanistic studies discussed in the report clearly indicate that this is the case for TCE. Similarly, considerations on coherence also fully apply to the causality of TCE exposure and PD. Indeed, there is a clear and close relationship between the biochemical effects of TCE in the CNS and the known biochemical mechanisms involved in PD. Considerations on experimental evidence, obtained in several convergent animal studies in multiple species, are clearly showing that exposure to TCE can cause the same biochemical alterations as those found in PD. Finally, considerations on analogy also apply to TCE, as we know that other compounds that damage dopaminergic neurons and mitochondria are causally linked to PD (e.g. MPTP). Fulfilling the Bradford Hill considerations is considered the strongest framework for determining causal inference in epidemiological studies.

My conclusion, after reviewing the scientific literature, is that there is strong evidence that TCE can cause PD, and that a causal link between PCE and PD also exists, although based on less evidence. This is the same conclusion reached in the 2017 report of the Agency for Toxic Substances and Disease Registry (ATSDR, 2017). ATSDR reported that levels of TCE in water at Camp Lejeune were substantially higher than the EPA's maximum contaminant level for TCE (ATSDR, 2017). It is my opinion that ATSDR employed a sound methodology and that the categories of causal association set forth in the report were well defined and appropriate. The ATSDR report concludes that exposure to TCE has "Equipoise and above evidence for causation" of PD (ATSDR, 2017). After reviewing the literature, including several new studies published in the past few years which substantially strengthen the causal relationship between TCE and PD, it is my opinion, within a reasonable degree of scientific certainty, that there is, at least as likely as not, or even more likely than not, a causal relationship between exposure to TCE and PD/Parkinsonism.

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Abbreviations

ALP	Autophagy-lysosomal pathway
ATP	Adenosine triphosphate
ATSDR	Agency for Toxic Substances and Disease Registry
CI	Confidence intervals

CNS	Central nervous system
COMT	Catechol -O- methyltransferase
CYP	Cytochrome P450
DOPAC	3,4-Dihydroxyphenylacetic acid
GSH	Glutathione (reduced)
i.p.	Intraperitoneal
LRRK2	Leucine rich repeat kinase 2
MAO-B	Monoamine oxidase, Type B
MPP+	1-Methyl-4-phenylpyridinium
MPTP	1-Methyl-4-phenyl-1,2,3,6-tetrahydropyridine
OR	Odds ratio
PCE	Perchloroethylene (Tetrachloroethylene)
PD	Parkinson’s disease
PET	Positron emission tomography
Ppb	Parts per billion
Ppm	Parts per million
TaClo	1-trichloromethyl-1,2,3,4-tetrahydro-beta-carboline
TBI	Traumatic brain injury
TCE	Trichloroethylene
TH	Tyrosine hydroxylase
TLV	Threshold limit value
TVOCs	Total volatile organic compounds
VOC	Volatile organic compound

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Appendix

Dr. Lucio G. Costa

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Aschner M, **Costa LG** (eds.). Environmental Factors in Neurodevelopmental and Neurodegenerative Diseases. Elsevier, New York, 2015, pp. 436.

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Rocha JB, Aschner M, **Costa LG** (eds). Advances in Neurotoxicology, Vol. 9. Alternative Methods in Neurotoxicology. Elsevier/Academic Press, New York, 2023; pp. 342.

Kodavanti PRS, Aschner M, **Costa LG** (eds). Advances in Neurotoxicology, Vol. 10. Neurotoxicity of halogenated Organic Compounds. Elsevier/Academic Press, New York, 2023; pp. 443

Tinkov AA, Aschner M, **Costa LG** (eds). Advances in Neurotoxicology, Vol. 11. The microbiome and neurotoxicity. Elsevier/Academic Press, New York, 2024; pp. 209.

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Abstracts

Pizzurro DM, Dao K, **Costa LG**. Astrocytes protect against diazinon- and diazoxon-induced inhibition of neurite outgrowth by regulating neuronal glutathione. Toxicologist 2014; p. 461, #1753.

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Roque PJ, Bommarito P, **Costa LG**. Microglia activation by diesel exhaust particles mediates cerebellar neuronal death. Annual PANWAT Meeting, Bothell, WA, 2014.

de Laat R, Dao K, **Costa LG**. Hormetic effect of low-dose BDE-47 in mouse neurons. Annual PANWAT Meeting, Bothell, WA, 2014.

Chang R, Cole TB, **Costa LG**. Autism spectrum disorder (ASD) related behavioral assessment due to prenatal diesel exhaust (DE) exposure. Annual PANWAT Meeting, Bothell, WA, 2014.

Roque PJ, Bommarito P, **Costa LG**. Microglia mediate diesel exhaust particle-induced cerebellar neuronal death. Toxicologist 2015; p. 453, #2109.

Coburn JL, Cole TB, **Costa LG**. Diesel exhaust exposure suppresses adult neurogenesis in mice in a sex-and brain region-dependent manner. Toxicologist 2015; p. 323, #1504.

Costa LG, Cole TB, Coburn J, Chang YC, Dao K, Roque PJ. Neurotoxicity of acute diesel exposure in adult mice. Neurotoxicol. Teratol. 2015; 49: 100.

Coburn J, Cole TB, **Costa LG**. Acute diesel exhaust exposure induces microglial activation and suppresses adult hippocampal neurogenesis but does not induce apoptosis. Toxicologist 2016; #1206.

Roque PJ, **Costa LG**. Microglia modulate the effect of diesel exhaust particles on neuronal death: role of pr-inflammatory cytokines. Toxicologist 2016; #1207.

Chang YC, Kalata E, Cole TB, **Costa LG**. Developmental exposure to diesel exhaust induces an autism-related behavioral phenotype in mice. Toxicologist 2016; #1169.

Chang YC, Cole TB, Hickman E, **Costa LG**. Developmental exposure of mice to diesel exhaust is associated with dysregulation of the reelin pathway and an increase in autism-related behaviors. Toxicologist 2017; #2642

Chang YC, Cole TB, **Costa LG**. Developmental neurotoxicity of traffic-related air pollution: studies with diesel exhaust in mice. INA 17 meeting, Florianopolis, Brazil, May 2017.

Coburn JL, Cole TB, Dao KT, **Costa LG**. Acute exposure to diesel exhaust impairs adult neurogenesis in mice: prominence in males and protective effects of pioglitazone. Toxicologist 2018; #3482.

Chang YC, Cole TB, **Costa LG**. Developmental traffic-related air pollution exposure and autism-related neurotoxicity in mice. Toxicologist 2019; #1089.

Garrick JM, Dao K, Cole TB, **Costa LG**. Paraoxonase -2 deficiency in the aging brain: immunological and neurodegenerative consequences. Toxicologist 2020; #1436

Cases in which I testified at deposition or trial in the past 4 years

None

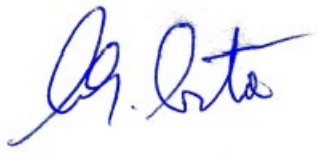
General compensation

$600/hour

$1200/hour (deposition, trial)

Date: December 6, 2024

Signature:



Lucio G. Costa

LITIGATION CONSULTATION

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2022: Law offices of Forsberg & Umlauf, Seattle, WA (Mr. Gibson, defendant's attorney). Expert consultation on alleged health effects related to toxic exposure. Wrote report. No deposition or testimony.

November 2024

CURRICULUM VITAE

LUCIO GUIDO COSTA

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PERSONAL
Born: October 14, 1954, Milano, Italy
Citizenship: Italian (permanent resident in USA)

EDUCATION

1973-77	University of Milano, Italy, Doctor in Pharmacy (Pharmacology), with Highest Honors; doctoral thesis on "L-Ascorbic Acid Activity on Arachidonic Acid Metabolism in Smooth Muscle"
1978	Postgraduate School of Experimental Pharmacology, University of Milano, Italy

PROFESSIONAL POSITIONS

2023- present	**Professor Emeritus**, Department of Environmental and Occupational Health Sciences, School of Public Health, University of Washington, Seattle, Washington
1992-2022	**Professor**, Department of Environmental and Occupational Health Sciences, School of Public Health, University of Washington, Seattle, Washington
1995-2022	**Director**, Neurotoxicology Research Core, NIEHS Interdisciplinary Center for Exposure, Diseases, Genomics & Environment (EDGE), University of Washington
1990-2022	**Core faculty**, Interdisciplinary Molecular and Cellular Biology Program, University of Washington
1989-2022	**Core faculty**, Environmental and Occupational Epidemiology Training Program, University of Washington
1985-2022	**Core faculty**, Environmental Pathology/Toxicology Training Program, University of Washington
1984-present	**Member**, Graduate Faculty, University of Washington
1988-2022	**Research Affiliate**, Alcoholism and Drug Abuse Institute, University of Washington
2004-2023	**Adjunct Professor**, University of Parma, Department of Medicine and Surgery, Parma, Italy
2007-2022	**Research Affiliate**, Center on Human Development and Disabilities, University of Washington

2013-2022	**Core Faculty**, Biostatistics, Epidemiologic and Bioinformatic Training in Environmental Health, University of Washington
1991-2000	**Director**, Toxicology Program, Department of Environmental Health, University of Washington
2002-2004	**Adjunct Professor**, University of Bari Medical School, Dept. of Pharmacology and Human Physiology, Bari, Italy
1998-2002	**Adjunct Professor**, University of Roma La Sapienza, Dept. of Pharmacology and Physiology, Roma, Italy
1986-1998	**Adjunct Professor**, University of Milano, Postgraduate School in Experimental Toxicology, Milano, Italy
1990-1998	**Adjunct Professor**, University of Pavia Medical School, Departments of Neurology and Pharmacology, Pavia, Italy
1991-1992	**Associate Professor**, Department of Environmental Health, School of Public Health and Community Medicine, University of Washington, Seattle, Washington
1987-1991	**Research Associate Professor**, Department of Environmental Health, School of Public Health and Community Medicine, University of Washington, Seattle, Washington
1983-1987	**Research Assistant Professor**, Department of Environmental Health, School of Public Health and Community Medicine, University of Washington, Seattle, Washington
1982-1983	**Research Scientist**, Division of Toxicology, Department of Pharmacology, University of Texas Medical School at Houston, Texas
1983	**Lecturer** in Neuropharmacology, University of Houston at Clear Lake, Texas
1980-81	**Postdoctoral Fellow**, Division of Toxicology, Department of Pharmacology, University of Texas Medical School at Houston, Texas
1978-1979	**Postdoctoral Research Associate**, Institute of Pharmacology and Pharmacognosy, University of Milano, Italy
1977-1978	**Lecturer** in Pharmacognosy, School of Pharmacy, University of Milano, Italy

FELLOWSHIPS, AWARDS, HONORS

1973-1977	**Graduate Fellowships**, INPDAI/FASDAI, Italy
1979	**Postdoctoral Fellowship**, Giovanni Lorenzini Foundation, Milano, Italy
1979	**Postdoctoral Fellowship**, Nutrition Foundation of Italy, Milano, Italy
1979	**Postdoctoral Fellowship**, University of Milano, Italy
1982	**NATO Travel Fellowship** to attend the NATO-ASI on "Principles and Methods in Receptor Binding", Urbino, Italy, September 8-18
1986	**Gordon Research Conferences Fellowship** to attend the Gordon Conference on Mechanism of Toxicity, Meriden, NH (declined).
1986	**NATO Fellowship** to attend the NATO-ASI on "Principles and Clinical Implications of Neuroreceptor Modulation", Corfu (Greece), June 16-27 (declined).
1986-1990	**Special Emphasis Research Career Award**, National Institute of Occupational Health.

1991	**Fellowship** to attend the "Summer School for a Multidisciplinary Assessment of Environmental Risks for Human Health", University of Siena, October 7-19 (declined).
1991	**Visiting Professor**, Chinese Academy of Preventive Medicine, Bejing, People's Republic of China.
1993-	**Fellow, ATS** (Academy of Toxicological Sciences). Certification in General Toxicology. Recertified in 1998, 2003, 2008, 2013, 2018; 2023
1994-	**Fellow**, American Association for the Advancement of Science.
1995	**Achievement Award**, Society of Toxicology
1997	**Zeneca Traveling Lectureship Award**, Society of Toxicology
1998	**Best Class of 1997-1998 award**, "ENVH 531, Neurotoxicology", Graduate Students, Dept. of Environmental Health, University of Washington
2006-	**European Certified Toxicologist (ERT)**. Certification in General Toxicology. Recertified in 2011, 2017, 2022.
2010	**Distinguished Faculty Lecture**, School of Public Health, University of Washington
2015	**Best Publication Award**, Reproductive and Developmental Toxicology Specialty Section, Society of Toxicology

PROFESSIONAL ORGANIZATIONS

Society of Toxicology, 1982-
Neurotoxicology Specialty Section, Society of Toxicology, 1984- ; **Councilor,** 1988-1990; **Vice-President Elect**, 2017-2018; **Vice-President**, 2018- 2019; **President**, 2019-2020; **Past-President**, 2020-2021; **Past Past-President**, 2021-2022
Pacific Northwest Association of Toxicologists, 1985- ; **Vice President,** 1989-1991; **President,** 1991-1992; **Councilor**, 1992-1993.
International Neurotoxicology Association 1984- (charter member); **Secretary,** 1990-1991; **President,** 1991-1995; **Councilor**, 2013-2015; 2015-2017
Academy of Toxicological Sciences, 1993-; **Member, Board of Directors**, 2010-2013
Society for Neuroscience, 1983-2018
International Brain Research Organization, 1983-2018
American Association for the Advancement of Science, 1983-; **Fellow**, 1994-
American Institute of Biological Sciences, 1997-
Italian Society of Pharmacology, 1986-
Italian Society of Toxicology, 1986-; **Councilor**, 2003-2006
Italian Association of Pharmacists, 1979-
Research Society on Alcoholism, 1993-2016
International Society for Biomedical Research on Alcoholism, 1993-2016
The New York Academy of Sciences, 1983-1990
Agrochemicals Division, American Chemical Society, 1986-1991
International Society of Occupational Medicine and Toxicology, 1992-1996

PROFESSIONAL ACTIVITIES

Editorial Responsibilities

Editor, Current Protocols in Toxicology, 1997-

Editor, Advances in Neurotoxicology, 2017-
Editor, Toxicology Section, BMC Pharmacology and Toxicology, 2012-2020
Editor, Special Issue on "Marine Neurotoxins", Marine Drugs, 2012- 2013; 2015-2016
Associate Editor, NeuroToxicology, 1989 -
Associate Editor, Toxicological Sciences, 2011- 2019
Associate Editor, Neurotoxicology Section, Encyclopedia of Neurological Sciences, 2011-
Associate Editor, Toxicology Section, Encyclopedia of Human Biology, 2012-
Member, Editorial Board, Journal of Clinical Toxicology, 2013-
Member, Editorial Board, Neurotoxicology and Teratology, 2011-
Member, Editorial Board, Biomedical and Environmental Sciences, 2003-
Member, Editorial Board, Frontiers in Bioscience, 1996-
Member, Editorial Board, Acta Biomedica, 2007-
Associate Editor, Journal of Toxicology, 2008-2018
Member, Editorial Board, Journal of Pharmacology and Experimental Therapeutics, 1999-2016
Member, Editorial Board, Current Enzyme Inhibition, 2008-2012
Member, Editorial Board, BMC Pharmacology, 2005-2012
Member, Editorial Board, Toxicology, 1994-2006
Member, Editorial Board, BIOMARKERS, 1995-2001
Member, Editorial Board, Washington Public Health, 1989-2000
Member, Editorial Board, Toxicology and Ecotoxicology News/Reviews, 1997-1998
Member, Editorial Board, Journal of Toxicology and Environmental Health, 1993-1998
Editor, PANWAT News, 1989-1991
Member, SciMat Editorial Advisory Board, The Alcoholism and Drug Research and Communication Center, Austin, TX, 1995-1996
Member, Editorial Board, DEH Newsletter, 1989-1994.
Reviewer for the journals:

Addiction Biology
Alcohol
Alcoholism: Clinical and Experimental Research
American Journal of Public Health
Annali dell'Istituto Superiore di Sanita`
Aquatic Toxicology
Archives of Toxicology
Biochemical Pharmacology
Biomedical and Environmental Sciences
Biomedicine and Pharmacotherapy
Blood
BMC Biochemistry
BMC Biology
BMC Neuroscience
BMC Pharmacology and Toxicology
Brain Research
Brain Research Bulletin
Cell Biochemistry and Function
Cell Biology and Toxicology

Cellular and Molecular Neurobiology
Chemical Reviews
Chemico-Biological Interactions
CHIMICAoggi
Chinese Journal of Oceanology and Limnology
Comparative Biochemistry and Physiology
Current Medicinal Chemistry
Drug and Chemical Toxicology
Ecotoxicology and Environmental Safety
Environmental Biotechnology
Environmental Health Perspectives
Environmental Science and Technology
Environmental Toxicology and Chemistry
European Journal of Neurology
Fish Physiology and Biochemistry
Free Radical Research
Frontiers in Biosciences
Fundamental and Applied Toxicology
Glia
Human and Experimental Toxicology
Immunopharmacology
International Journal of Food Science and Nutrition
International Journal of Immunopharmacology
International Journal of Environmental Research and Public Health
Journal of Biochemical Toxicology
Journal of Clinical Toxicology
Journal of Exposure Analysis and Environmental Epidemiology
Journal of Medicinal Chemistry
Journal of Neurochemistry
Journal of Neuroimmunology
Journal of Pharmacology and Experimental Therapeutics
Journal of the National Cancer Institute
Journal of Toxicology
Journal of Toxicology and Environmental Health
Lancet
Lancet Neurology
Life Sciences
Metallomics
Molecular Pharmacology
Neurology
Neuropharmacology
Neuroscience
Neurotoxicity Research
NeuroToxicology
Neurotoxicology and Teratology

Pharmacological Research
Pharmacology Biochemistry and Behavior
Psychopharmacology
Regulatory Pharmacology and Toxicology
Science
Teratology
Tissue and Cell
Toxicological Sciences
Toxicology
Toxicology and Applied Pharmacology
Toxicology in Vitro
Toxicology Letters
World Journal of Biological Psychiatry

Reviewer for the Annals of the New York Academy of Sciences
Reviewer for the American Chemical Society Books
Reviewer for the Agency for Toxic Substances and Disease Registry
Reviewer for the National Academy of Sciences

Membership in National and International Panels and Committees
Member, NIH Review Panel on Motivated Behavior and Alcohol, 2020b
Member, NIH Review Panel on Alcohol and Motivated Behaviors, 2020a
Member, NIH Review Panel on Alcohol and Motivated Behaviors, 2019
Member, NIH Review Panel on RIVER R35 grants, 2019
Member, NIH Review Panel on Neurotoxicology, 2019
Member, NIH Review Panel on Alcohol and Drugs, 2017
Member, NIH Review Panel on Alcohol and Neurotoxicology, 2017
Member, NIH Review Panel on VICTER Awards, 2017
Member (ad hoc), NIH Neurotoxicology and Alcohol Review Panel, 2016
Member, NIH Review Panel on VICTER Awards, 2016
Member, NIH Review Panel Loan Repayment Program, Pediatrics, 2016
Member, NIH, Special Emphasis Review Panel on Neurotoxicology, 2016
Member, Technical Qualification Review Board, US Environmental Protection Agency, 2015
Member, NIH Review Panel on Alcohol and Heavy Metals, 2015
Member (ad hoc), NIH Neurotoxicology and Alcohol Review Panel, 2015
Member, NIH Review Panel on Director's Early Independence Awards, 2015
Chair, NIH Review Panel on Neurotoxicology and Drugs, 2015
Member, Working Group on Amino Acids and Vitamins, FEEDAP Panel, EFSA, 2015-2018
Member, Working group on Colourings, FEEDAP Panel, EFSA, 2015-2018
Member, NIH Review Panel on Drugs, Alcohol and Heavy Metals, 2014b
Member (ad hoc), NIH Biobehavioral Regulation, Learning and Ethology Review Panel, 2014
Member, NIH Review Panel on Drugs, Alcohol and Heavy Metals, 2014a
Member, NIH Special Emphasis Review Panel on Neurotoxicology, 2014
Member, ECVAM's Scientific Advisory Committee (ESAC), JRC Ispra, 2013-2016
Member, FEEDAP Panel, European Food Safety Authority (EFSA), 2013-2015

Member, Working Group on Amino Acids, FEEDAP Panel, EFSA, 2013-2015
Member, Review panel "Toxicological Profile of Hexachlorobenzene", ATSDR, 2012
Member, Technical Qualification Review Board, US Environmental Protection Agency, 2012
Member, Review panel "Toxicological Profile of Toxaphene", ATSDR, 2010
Chair, Review panel on EPA IRIS on "Toxicological Review of Hexachloroethane", 2010
Member, Board of Directors, Academy of Toxicological Sciences, 2010-2013
Member, ECVAM's Scientific Advisory Committee (ESAC), JRC Ispra, 2010-2013
Member, FAO/WHO Expert group on Risk and Benefit of Fish, Rome, 2010
Member, Expert HENVINET Expert Panel on "Chlorpyrifos", Dresden, 2009
Member, NIH Special Emphasis Review Panel on Neurotoxicology, 2009c
Member, NIH Special Emphasis Review Panel on Neurotoxicology, 2009b
Member, NIH Review Panel on Challenge Grants, 2009
Member, WHO Expert Panel on "Brominated Flame Retardants", Copenhagen, 2009
Member, NIH Special Emphasis Review Panel on Neurotoxicology, 2009a
Chair, Review panel on EPA IRIS on "Toxicological review of thallium and compounds", 2008
Member, NIH Special Emphasis Review Panel on "Addiction", 2007
Member, Review Panel on EPA IRIS on "Toxicological Review of Polybrominated Diphenyl Ethers", 2006-2007
Member (ad hoc), NAL Study Section, National Institute of Health, 2006
Member, Peer Review Team, Neurotoxicology Division, US Environmental Protection Agency, 2006
Member (ad hoc), Technical Qualification Review Board, US Environmental Protection Agency, 2006
Member, NIH Special Emphasis Review Panel on "Neurotoxicology", 2005
Member, Review Panel on EPA IRIS on "Toxicological Review of n-Hexane", 2005
Member, NTP/CERHR Expert Panel Committee on "Reproductive and developmental Toxicity of amphetamine, methamphetamine and methylphenidate", 2004-2005
Member, Advisory Committee on "Polybrominated diphenyl ethers", State of Washington, Departments of Ecology and Health, 2004-2005
Member, NIH Special Emphasis Review Panel on "Neurotoxicology of heavy metals", 2003
Member (ad hoc), Technical Qualification Review Board, US Environmental Protection Agency, 2003
Member, WHO/ICPS planning committee for preparation of an Environmental Health Criteria document on "Scientific principles for evaluating health risks in children associated with exposure to chemicals", 2003
Member, External Review Panel, Life Sciences Task Force-Programs of Excellence, Texas A&M University, 2003
Member, Alcohol Research Center site visit team, National Institute of Alcoholism and Alcohol Abuse, 2003
Member, CONTAM Panel, European Food Safety Agency, 2003-2006
Member, Biomedical Research Review Subcommittee (ad hoc), National Institutes of Health, 2003
Member, ALTX-3 study section (ad hoc), National Institutes of Health, 2003
Member, "Neurotoxicity Task Force", European Center for the Validation of Alternative Methods, 2001-

Member, Special Review Panel on “Endocrine Disruptors”, Environmental Protection Agency, 1999
Member, Institute of Medicine/National Academy of Sciences Committee on Health Effects Associated with Service in the Persian Gulf War, 1998-2000.
Member, NIEHS Special Emphasis Review Panel on RFA 97-002, "Linking Environmental Agents, Oxidative Damage and Disease", 1997
Member, Special Peer Review Panel on “Endocrine Disruptors”, Environmental Protection Agency, 1997
Member, Panel on Gulf War Illnesses, American Institute of Biological Sciences/BioReview, 1997
Temporary Adviser, World Health Organization Joint Meeting on Pesticides, Core Assessment Group, Leicester, England, 1997.
Member, Scientific Group on Methodologies for the Safety Evaluation of Chemicals (SGOMSEC), Workshop on Alternative Testing Methodology, Ispra, Italy, 1997.
Member (ad hoc), Technical Qualification Review Board, Environmental Protection Agency, 1996
Member, Italian Panel of Experts in Pharmacology, European Agency for the Evaluation of Medicines (EMEA), 1996-
Member, Special Peer Review Panel on "Endocrine Disruptors", Environmental Protection Agency, 1996
Secretary, Scientific Program Committee, Eighth International Congress of Toxicology, Paris, 1996-1998.
Member, NIEHS Special Review Committee on RFA 94-009, "Mechanistically - based alternative methods in toxicology", 1995
Member, Program Committee, Fifth Meeting of the International Neurotoxicology Association, 1993-1995
Member, Local Arrangements Committee, Seventh International Congress of Toxicology, Seattle, WA 1995
Member, Health Effects Review Panel, Environmental Protection Agency, 1989 - 1993.
Member, Health and Environment Committee, Long Term Plan for Environmental Restoration, Department of Energy, 1990 - 1993.
Member, Pesticide Incident Reporting and Tracking Review Panel, State of Washington, 1989 – 2000; 2001- 2003 (Gubernatorial appointment).
Member, Agricultural Pesticide Advisory Board, State of Washington (Gubernatorial appointment), 1989-2000.
Member, Executive Committee, International Neurotoxicology Association, 1990 - 1998.
Member, Northwest Consumer Food Safety Council Advisory Board, 1990-1999.
Member, Task force on Control of the Russian Wheat Aphid, State of Washington, 1989-1993
Member, Citizens' Toxics Advisory Group, Seattle City Light, 1986-1993
Member, Integrated Pest Management Program Advisory Board, King County Department of Public Works, 1992-1996
Member, Alcohol and Drug Abuse Grant Review Committee, University of Washington, 1988-1990
Member, Scientific Committee, International Neurotoxicology Association, 1987-1990

Member, site-visit review team for the National Institute of Environmental Health Sciences, Program Project at the University of Maryland, 1991
Member, on-site review team for the National Science Foundation EPSCoR program at Mississippi State University, 1985

Other Professional Activities
Grant Reviewer for:
Action, Medical Research for Children, for life, UK
Agency for Toxic Substances and Disease Registry
Air Force Office of Scientific Research
Alcoholism and Drug Abuse Institute, University of Washington
Alzheimer's Disease Research Center, University of Washington
American Institute of Biological Sciences
Basic Research Foundation, Israeli Academy of Sciences and Humanities
CEEH/EDGE Center, University of Washington
Center for Alternatives to Animal Testing
Center for Environmental and Rural Health, Texas A & M University
Center for Indoor Air Research
Danish Agency for Science, Technology and Innovation
Department of Veteran Affairs
Diabetes Endocrinology Research Center, University of Washington
Düsseldorf Universität
Environment and Health Fund, Republic of Israel
Environmental Protection Agency
European Commission
Florida Department of Health
Graduate School Research Fund, Univ. of Washington
INBRE Developmental Research Project program, Puertorico
Industrial Hygiene Research Fund, Dept. of Environmental Health, University of Washington
Institut National de la Recerche Agronomique, Paris, France
Institute for Translational Health Sciences, University of Washington
Israeli Science Foundation
Johns Hopkins University, Center in Urban Environmental Health
Life Sciences task Force-Programs of Excellence, TexasA&M University
March of Dimes Birth Defects Foundation
Mississippi State University, Office of Research
MIUR (Ministero dell'Universita` e della Ricerca Scientifica e Tecnologica)
National Institute of Alcoholism and Alcohol Abuse
National Institute of Environmental Health Sciences
National Science Foundation
NATO (North Atlantic Treaty Organization)
Natural Sciences & Engineering Research Council of Canada, Strategic Grants Program
Netherland's Ministry of the Environment
NOAA (National Oceanic and Atmospheric Administration)

North Carolina Biotechnology Center
Portorico INBRE Program
Royalty Research Fund, University of Washington
Scottish Executive Health Department
Singapore National Medical Research Council
South Carolina EPSCoR/IDeA Program
Southern California Environmental Health Sciences Center, Pilot Projects Program
Swiss National Science Foundation
Swiss 3R Competence Center
The Wellcome Trust
UK Health Protection Agency
University of Central Oklahoma
US Civilian Research and Development Foundation
USDA SDE/GWIS Fellowship Program

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FUNDING

Previous Funding

NATO Grants for International Collaboration in Research, co-PI with Dr. F. Cattabeni, University of Urbino, Italy, "Developmental Neurotoxicity of Pesticides", 1.10.1985 - 9.30.1987, $10,000.

Industrial Hygiene Research Grant (DEH), "Biochemical Studies on the Neurotoxicity of Trimethyltin", PI, 6.1.1984 - 12.31.1985, $8,460.

Industrial Hygiene Research Grant (DEH), "Neurotoxic effects of developmental exposure to the organophosphate insecticide parathion", 1.1.1986 - 6.30.1987, $11,820.

Industrial Hygiene Research Grant (DEH), "Occupational and Environmental Exposures to Organotin Compounds, Evaluation of Erythrocyte's Na+/K+-ATPase as a Tool for Biological Monitoring", PI, 1.1.88 - 6.30.88, $6,082.

Industrial Hygiene Research Grant (DEH), "A pilot study on the levels of cholinergic muscarinic receptors in lymphocytes from asthmatic patients", PI, 09.01.1988 - 06.30.1989 $6,621.

Alcoholism and Drug Abuse Institute, University of Washington, "Interaction of Ethanol with the Phosphoinositide System During Development", PI, 1.1.87 - 9.30.1988, $13,000.

Milbank Memorial Fund, "Illnesses and Injuries among Washington State Agricultural Workers", (L. Rosenstock, PI), 11.1.1986 - 10.31.1988, $120,504.

USDA-Forest Service, Pacific Northwest Region, "Health Effects Implications of Pacific Northwest Region Vegetation Management", (S.D. Murphy, PI), 10.1.1986 - 9.30.1988, $204,867.

NIOSH, Special Emphasis Research Career Award (SERCA), "Peripheral Markers of Muscarinic Receptors", PI, 10.1.1986 - 3.30.1990, $90,000.

Industrial Hygiene Research Grants (DEH), "A pilot study to determine levels of hemoglobin adducts in laboratory workers exposed to acrylamide", PI, 9.1.89 - 6.30.90, $6,000.

NIEHS, "Factors affecting pesticide toxicity", co-PI and PI, 12.1.1985 - 11.30.1990, $586,187.

BSRG, "Molecular aspects of the developmental neurotoxicity of alcohol", PI, 10.1.90 - 3.31.91, $5,684.

Industrial Hygiene Research Grant (DEH), "Development of a rapid immunological technique for the determination of hemoglobin adducts in acrylamide-exposed laboratory workers", PI (C.J. Calleman, co-PI), 10.1.90 - 6.30.91, $6,000.

NIEHS Superfund Research Program, "Peripheral Markers of Potential Neurotoxic Chemicals", Project Director (S.D. Murphy, then D.L. Eaton, PI of Program Project), 10.1.1987 - 9.30.1992, $114,570 ADC.

EPA, CR 81676810, Supplement, Field trip to China, PI, 9.20.91 - 12.31.91, $86,844.

Charles A. Dana Foundation, "Program in Genetic and Environmental Health", co-investigator (A.G. Motulsky, PI), 7.1.1986 - 6.30.1992, $200,000 ADC.

NIEHS, "Human Serum Paraoxonase: role in pesticide metabolism", Co-investigator (C.E. Furlong PI), 7.1.89 - 6.30.92 $146,596 ADC.
NIEHS, "Primate developmental effects of methylmercury", Co-investigator (T. Burbacher, PI), 7.1.89 - 6.30.92, $220,024 ADC.
BSRG, "Development of in vitro models to investigate the role of glutathione in neurotoxicity", PI, 11.1.91 - 3.31.92, $4,300.
Industrial Hygiene Research Grant (DEH), "Glutathione as a modulator of the neurotoxicity of styrene oxide, " PI, 12.1.91 - 6.30.92, $5,888.
Industrial Hygiene Research Grant (DEH), "A Study of ethnic variations in the number of beta receptors", Co-investigator (J. Koenig, PI), 12.1.91 - 6.30.92, $6,000.
EPA, CR 816768010, "Determination of hemoglobin adducts following acrylamide exposure," PI, 9.2.90 - 3.20.93, $114,713 ADC.
Alcoholism and Drug Abuse Institute, University of Washington, "A mechanistic hypothesis for the developmental neurotoxicity of ethanol: the interaction with muscarinic receptor-stimulated phosphoinositide metabolism and calcium homeostasis", PI, 1.1.91 - 9.30.92, $14,883.
NIOSH, "Peripheral markers of styrene toxicity", Co-investigator (H. Checkoway, PI), 12.1.88 - 11.30.92 $114,245 ADC.
NIAAA, "Ethanol and phosphoinositides during brain development", PI, 9.29.91-9.28.96, $122,212 ADC.
NIEHS Superfund Research Program, "Peripheral Biomarkers of Potential Neurotoxic Chemicals", Project Director (D.L. Eaton, PI), 4.1.95 - 3.31.96, $60,000 ADC.
NIEHS, "Human serum paraoxonase: role in pesticide metabolism", Co-investigator (C.E. Furlong, PI), 4.1.93-2.28.97, $114,898 ADC.
Alcohol and Drug Abuse Institute, University of Washington, "Microglia as a cellular target for the developmental neurotoxicity of ethanol", PI, 2.1.95 - 1.31.97, $14,903.
NIEHS, "Exposure to EMF and astrocytoma: in vitro studies", Principal Investigator, 2.1.97-1.31.99, $48,992 ADC (10% FTE).
NIEHS, "Developmental neurotoxicity of toluene", Principal Investigator, 10.1.99-9.30.02, $49,500 ADC (5% FTE).
NIEHS, "Gene/environment interaction in Parkinson's disease", Co-investigator (H. Checkoway, PI), 7.1.00-6.30.05, $326,609 ADC.
NIEHS, Superfund Research Program, "Environmental and Biochemical Risk Factors for Parkinson's Disease", Co-investigator, (H. Checkoway, Project Director), 4.1.00 - 3.31.06, $182,797 ADC.
NIEHS, Center for Child Environmental Health Risks Research", (E. Faustman, PI), Director of Project on "Paraoxonase polymorphism: role in defining susceptibility to the developmental neurotoxicity of organophosphate", 5.7.04 – 5.6.09, $120,000 ADC.
NIEHS, "Paraoxonase (PON1): A biomarker of susceptibility to environmentally-induced diseases" (Superfund Program Project), Principal Investigator, 04.01.00 - 3.31.06, $196,234 ADC.
NIEHS, "Gene/environment interaction in Parkinson's disease", Co-investigator (H. Checkoway, PI), 7.1.00-6.30.05, $326,609 ADC.
NIEHS, "The FHCRC/UW Toxicogenomics Consortium", Project co-leader with C.E. Furlong (H. Zarbl, PI), 9.30.01-8.31.06, $1,214,300 ADC.

NIEHS, "Center for Ecogenetics and Environmental Health", Director, Neurotoxicology Core (D.L. Eaton, PI), 4.1.05 - 3.31.11, $1,100,000 ADC
NIEHS/EPA, Center for Child Environmental Health Risks Research", (E. Faustman, PI), Co-Director (with C.E. Furlong) of Project on "Paraoxonase polymorphism: role in defining susceptibility to the developmental neurotoxicity of organophosphate"; Co-Director (with E.M. Faustman) of Project on "Molecular Mechanisms", 5.7.04 – 5.6.09, $1,000,000 ADC.
NIEHS/NSF, Center for Human Health and Ocean Sciences (E.M Faustman, PI), Co-director (with E.M Faustman) of Project on "Mechanisms of toxicity", 9.1.09 – 8.31.11, $120,000 ADC (Project)
NIAAA, "Mechanisms of ethanol-induced neurodevelopmental effects", Co-investigator (M. Guizzetti, PI), 9.25.08-8.31.12, $225,000 ADC
Department of Defense, Manganese Health Research Program, "Effects of manganese on glial-neuronal interactions", Principal Investigator, 4.15.08-2.28.11, $90,000 ADC
NIAAA, "Developmental neurotoxicity of ethanol" Principal Investigator, 7/01/07-6/30/13, $225,000 ADC
NIEHS, "Structure and Function of the Human PON1 Polymorphisms", co-PI; Furlong, PI; 4/1/09 – 3/31/14, $449,884 ADC
NIEHS, "Paraoxonases as biomarkers of susceptibility to environmentally-induced diseases" Project Co-Director with C. Furlong; Checkoway, PI, 4/1/09-3/31/14, $263,693 ADC
NIEHS/EPA, "Center for Child Environmental Health Risk Research", co-Inv.; Project Director; Faustman, PI; 9/25/09-7/31/14, $1,000,000 ADC
DEOHS, "Diesel exhaust-induced neurotoxicity: genetic susceptibility and gender influence" Pilot Project, Costa, PI, 1/01/2011-6/30/2013, $82,449
DEOHS, "Developmental neurotoxicity of air pollution: biochemical, epigenetic and behavioral effects" Pilot Project, Costa, PI, 12/01/2012-6/30/2014, $73,276
CEEH/DEOHS, Technology Access Fund (Costa, PI) 3/1/2012-6/30/2013, $5,000
NIEHS, "Low level exposure to PBDEs: testing the hormetic and epigenetic hypotheses ", Costa PI, 4/01/2013-3/31/2016, $150,000 ADC.
NIEHS, "Paraoxonases as biomarkers of susceptibility to environmentally-induced diseases" (Superfund), Gallagher PI, 07/01/2015-03/31/2017, $165,000 ADC
NIEHS, "Traffic-related air pollution and neurodegenerative disorders: gene-environment interactions", Costa PI, Pilot Project, 5/01/2016-3/31/2017, $40,000
University of Washington, Office of the Provost, Bridge Funding for "UW Diesel Exposure Facility", PI, 06/01/17-05/31/18, $50,000 ADC
NIEHS, "Air pollution and the brain: gender as an important determinant of susceptibility", Costa, PI, 07/01/2013-03/31/2019, $225,000 ADC
NIEHS, "Center for Ecogenetics and Environmental Health", Costa, Director of ARE on Neurotoxicology and Neurodegeneration, TJ Kavanagh, PI, 4/1/16-3/31/21, $1,000,000 ADC.
NIEHS, "Role of Paraoxonases (PONs) in Modulating Cadmium and Manganese Neurotoxicity", (Superfund), Gallagher, PI, 04/01/2017-03/31/2022, $260,835 ADC (Project co-PI)
NIEHS, "Center for Ecogenetics and Environmental Health", Costa, Director of ARE on Neurotoxicology and Neurodegeneration, J Kaufman, PI, 4/1/21-3/31/26, $1,000,000 ADC.
NIEHS, "Gene-environment interactions in the developmental neurotoxicity of traffic-related air pollution", Costa PI, 09/01/2017-07/31/2023, $225,000 ADC

NIEHS, Supplement to "Gene-environment interactions in the developmental neurotoxicity of traffic-related air pollution", Costa PI, 09/01/2018-07/31/2023, $219,920 ADC
NIEHS, "The role of arachidonic acid metabolic pathways involved in resolution versus progression of PM-induced cardiometabolic toxicity, Costa, co-investigator (J. Araujo, PI), 02/01/2022-11/30/2022, $346,296 ADC

CONFERENCES AND SYMPOSIA

Participant, Gordon Research Conference on Mechanisms of Toxicity, Meriden, NH, 1985
Scientific Director, NATO-ASI on "Toxicology of Pesticides: Experimental, Clinical and Regulatory Perspectives", Riva del Garda, Italy, 1986
Organizer, Meeting on "Working with Pesticides: Health and Safety Issues", Seattle, WA, 1987
Organizer and Scientific Moderator, First meeting of the International Neurotoxicology Association, Lunteren, The Netherlands, 1987
Organizer and Chairman of Symposium, Poster session and Research Workshop, Second meeting of the International Neurotoxicology Association, Sitges, Spain, 1989.
Organizer and Chairman of session, International Symposium on "Exposure to Styrene: Risks, Toxicity and Criteria for Biological Monitoring", Pavia, Italy, 1990
Co-Chairman, Symposium on "Molecular Mechanisms of Neurotoxicity in Development," International Society for Developmental Neuroscience, 8th biennial meeting, Bal Harbour, FL, 1990
Co-Chair of Session, Eighth International Neurotoxicology Conference, "Role of Toxicants in Neurological Disorders", Little Rock, AK, 1990.
Course Director, Northwest Center for Occupational Health and Safety, course on "Pesticide Medicine", Seattle, WA 1991
Organizer and Chairman of session, International Symposium on "Current Topics in Alcoholism", Pavia, Italy, 1991
Organizer and Chairman of symposium, Eighth Annual Meeting of the Pacific Northwest Association of Toxicologists, Seattle, WA, 1991
Co-Chair of Symposium, "Ecogenetics: Genetic Susceptibility to Environmental Agents", Society of Toxicology 31st Annual Meeting, Seattle, WA 1992.
Co-Chair of Platform Session, "Receptors and Signal Transduction", Society of Toxicology 31st Annual Meeting, Seattle, WA, 1992.
Course Director, Northwest Center for Occupational Health and Safety, course on "Pesticide Medicine", Yakima, WA, 1992.
Organizer and Chairman of symposium, Tenth International Neurotoxicology Conference, "Mechanisms of Developmental Neurotoxicity", Little Rock, AK, 1992.
Chair of Poster Sessions, "Central Nervous System" and "Peripheral Nervous System", Society of Toxicology 32nd Annual Meeting, New Orleans, LA, 1993.
Course Director, Northwest Center for Occupational Health and Safety, course on "Pesticide Medicine: Are Children at risk?", Bellingham, WA, 1993.

Co-Chair of Symposium, "Nutrition and Neurotoxicity", Fourth meeting of the International Neurotoxicology Association, Helsingor, Denmark, 1993
Co-Chair of International Symposium on "Occupational Neurotoxicology", Pavia, Italy, 1993
Course Director, "Toxicology", Summer Institute of Public Health Practice, Seattle, WA 1993
Course Director, Northwest Center for Occupational Health and Safety, course on "Pesticide Medicine", Seattle, WA, 1994.
Chair of Symposium, "Biological Indicators of Exposure to Toxicants", Tenth National meeting of the Italian Society of Toxicology, Pavia, Italy, 1994.
Chair of Symposium, "Glial cells and Mechanisms of Neurotoxicity", Fifth Meeting of the International Neurotoxicology Association, Port Ludlow, WA, 1995.
Chair of Session, "Synaptogenesis", NIEHS Workshop on Developmental Neurotoxicology, Research Triangle Park, NC, 1995
Panel Discussant, "Neurotoxins and mechanisms of neuronal injury", NIEHS Workshop on The Role of the Environment in Parkinson's disease, Research Triangle Park, NC, 1995
Organizer and Speaker, Course on "Mechanisms of Neurotoxicity", Central University of Venezuela, Caracas, Venezuela, 1995.
Facilitator, Session on "Targets/Receptor", Workshop on "Chlorpyrifos Neurotoxicity", Research Triangle Park, NC, 1996
Course Director, Northwest Center for Occupational Health and Safety, course on "Pesticide Medicine", Yakima, WA, 1996.
Chair, Session on "Mechanisms of Toxicity", Eleventh National Meeting of the Italian Society of Toxicology, Milano, Italy, 1996
Chair, Symposium on "Role of Genetic Polymorphism in Neurotoxicity", Sixth Meeting of the International Neurotoxicology Association, Szeged, Hungary, 1997
Chair, Symposium on “Genetic predisposition and individual sensitivity”, Sixteenth International Neurotoxicology Conference “Pesticides and susceptible populations: who is at risk and when?” Little Rock, AR, 1998.
Chair, Symposium on “Mechanisms of developmental neurotoxicology: molecular and cellular targets”, Seventeenth International Neurotoxicology Conference “Children’s Health and the Environment: Mechanisms and Consequences of Developmental Neurotoxicology”, Little Rock, AR, 1999.
Course Director, Northwest Center for Occupational Health and Safety, Course on “Pesticide Medicine”, Yakima, WA, 2000.
Chair, Symposium on “Recent Advances in Developmental Neurotoxicology”, Eight meeting of the International Neurotoxicology Association, Estoril, Portugal, 2001
Chair, Symposium on “Environmental Risk Factors”, Nineteenth International Neurotoxicology Conference “Parkinson’s Disease, Environment and Genes” Colorado Springs, 2001
Course Director, Northwest Center for Occupational Health and Safety, Course on "Pesticide Medicine", Seattle, WA, 2002
Chair, Symposium on “Genetic polymorphisms in toxicology”, Thirteenth meeting of the Italian Society of Toxicology, Urbino, 2003
Chair, Symposium on “Food neurotoxicants and nutritional aspects of neurodegeneration”, Nineth meeting of the International Neurotoxicology Association, Dresden, 2003
Member, International Advisory Board, NEUROTOX 2003, Nottingham, 2003
Co-Chair, Poster Sessions, Fortyfirst EUROTOX meeting, Firenze, 2003

Member, Scientific Committee, First International Meeting on Paraoxonases, Ann Arbor, MI, 2004
Member, Scientific Committee, Tenth meeting of the International Neurotoxicology Association, Helsinki, 2005
Member, Scientific Committee, Second International Meeting on Paraoxonases, Debrecen, Hungary, 2006
Chair, Symposium on "Developmental neurotoxicity and food contaminants", Fortythird EUROTOX Meeting, Cavtat/Dubrovnik, Croatia, 2006
Member, Scientific Organizing Committee, International Conference on Food Contaminants and Neurodevelopmental Disorders, Valencia, Spain, 2006
Member, Scientific Committee, Eleventh meeting of the International Neurotoxicology Association, Asilomar, CA, 2007
Member, Scientific Committee, Third International Meeting on Paraoxonases, Los Angeles, CA, 2008
Chair, Symposium on "Algal biotoxins", Fifteenth Meeting of the Italian Society of Toxicology, Verona, 2009
Member, Scientific Committee, Fourth International Meeting on Paraoxonases, Reus, Spain, 2010
Member, Scientific Steering Committee, Third Symposium on Developmental Neurotoxicity (DNT 3), Varese, Italy, 2011
Chair, Symposium on "DNT assays and non-mammalian species", Third Symposium on Developmental Neurotoxicity (DNT 3), Varese, Italy, 2011
Member, Scientific Committee, Fifth International Meeting on Paraoxonases, Columbus, OH, 2012
Member, Program Committee, 15th meeting of the International Neurotoxicoloy Association (INA15), Montreal, Canada, 2015
Member, Scientific Committee, Sixth International Meeting on Paraoxonases, Alicante, Spain, 2015
Member, International Advisory Committee, Second International Conference on the Neurotoxicity and Prevention of Manganese Adverse Effects (MANGANESE2016), New York, NY, 2016
Member, Scientific Committee, DNT 5, Konstanz, Germany 2019-20 (postponed)

Invited Speaker

Invited Lectures at National and International Meetings

NATO Advanced Study Institute on "Toxicology of the Nervous System", Belgirate, Italy, 1984
Northwest Center for Occupational Health and Safety, Course on "Health and Safety at Hazardous Waste Sites", Seattle, WA, 1984
International Society for the Study of Xenobiotics, meeting on "Foreign Compound Metabolism", Malta, 1985
Northwest Center for Occupational Health and Safety, Course on Maritime Occupational Health and Safety, Seattle, WA, 1985
Marine Chemist Association, 28th Annual Meeting, Portland, OR, 1986
ASPET/SOT joint meeting, Symposium on "Noncholinergic Effects of Organophosphates", Baltimore, MD, 1986

Italian Society of Neuroscience, Symposium on "Neurotoxicity of Environmental Pollutants", Pisa, Italy, 1986
Pacific Northwest Association of Toxicologists, Annual Meeting, Symposium on "Recent Advances in Pesticide Toxicology", Fort Warden, WA, 1987
NATO Advanced Research Workshop on "The acetylcholine nicotinic receptor in the central nervous system", Venice, Italy, 1988
SOT Annual meeting, Symposium on "Neurotoxicant-induced alterations in cellular interactions", Atlanta, GA, 1989
Alcohol and Drug Abuse Institute Research Symposium, Seattle, WA, 1989
Seminars in Medical Toxicology, Centro Antiveleni Ospedale di Niguarda, Milano, Italy, 1989; 1990
Symposium on "Management of the Pesticide Poisoned Patient," Ronan, MT, 1989
Northwest Center for Occupational Health and Safety, Course on "Advances in Pesticide Medicine", Yakima, WA 1989
Charles A. Dana Foundation Conference on "Environmental factors influencing health and disease in aging", Rye, NY, 1989
NIEHS Superfund Basic Research Program, Meeting on "Application of Molecular Biomarkers in Epidemiology", Research Triangle Park, NC, 1990
American Chemical Society, 199th National Meeting, Symposium on "Organophosphates: Chemistry, Fate and Effects," Boston, MA, 1990
International Symposium on "Exposure to Styrene: Risks, Toxicity and Criteria for Biological Monitoring", Pavia, Italy, 1990
International Society for Developmental Neuroscience, 8th biennial meeting, Symposium on "Molecular Mechanisms of Neurotoxicity," Bal Harbour, FL, 1990
City of Seattle, Pesticide Recertification Seminar, Seattle, WA, 1990
International Symposium on "Current Topics in Alcoholism", Pavia, Italy, 1991
Northwest Center for Occupational Health and Safety, Course on "Pesticide Medicine", Seattle, WA, 1991
Pacific Basin Conference on Hazardous Waste, Symposium on "Health effects of hazardous waste", Bangkok, Thailand, 1992
International Meeting on "Toxicological Risks of Pesticides", Pavia, Italy, 1992
Northwest Center for Occupational Health and Safety, Course on "Pesticide Medicine", Yakima, WA 1992
IUTOX 92 Satellite Symposium on "Neurotoxicological Aspects of Pesticides and Neurotoxins", Soverato, Italy, 1992
Tenth International Neurotoxicology Conference on "Mechanisms of Developmental Neurotoxicity", Little Rock, AK, 1992
Learning Disability Association, Symposium on "Tots and Toxins", San Francisco, CA, 1993
International Symposium on "Occupational Neurotoxicology", Pavia, Italy, 1993
Eleventh International Neurotoxicology Conference on "Drugs of Abuse and Developmental Neurotoxicity", Little Rock, AK, 1993
American College of Toxicology, Fourteenth Annual Meeting, Symposium on "Receptor - mediated neurotoxicity", New Orleans, LA, 1993
Northwest Center for Occupational Health and Safety course on "Pesticide Medicine", Seattle, WA, 1994

Second PERC Symposium on "Early detection and risk factors in Parkinson's disease and other neurodegenerative disorders", Santa Cruz, CA, 1994 (declined)
XIIth International Congress of Pharmacology, Symposium on "Alterations in cell signalling and cytotoxicity", Montreal, Canada, 1994
Italian Society of Toxicology, Tenth national meeting, Symposium on "From the receptor to the gene in neurotoxicology", Pavia, Italy, 1994
International Symposium on "Human Health and Environment: Mechanisms of Toxicity and Biomarkers to assess adverse effects of chemicals," Salsomaggiore, Italy, 1994
Meeting on "Environmental Human Biomonitoring: from exposure to health effects", Duesseldorf, Germany,1995.
Italian Society of Toxicology, Eleventh National Meeting, Symposium on "Mechanisms of Neurodegeneration", Milano, Italy, 1996
Fourteenth International Neurotoxicology Conference: Part 2, “Neurotoxicity and Neurodegeneration: Biological Links”, Symposium on "Genetic and Environmental Interactions in Neurotoxicity and Neurodegeneration", Soverato, Italy 1997
Sixth Meeting of the International Neurotoxicology Association, Symposium on “Role of Genetic Polymorphisms in Neurotoxiciy”, Szeged, Hungary, 1997
International Conference on "Volatile Organic Compounds in the Environment, Risk Assessment and Neurotoxicity", Pavia, Italy, 1997
Third International Meeting on “Esterase Reacting with Organophosphorus Compounds”, Dubrovnik, Croatia, 1998
Ninth Congress of the International Society for Biomedical Research on Alcoholism, Symposium on “Alcohol Action on Glial Cells”, Copenhagen, Denmark, 1998
Eigth Congress of the International Union of Toxicology (ICT VIII), Symposium on “Addressing Uncertainties in Risk Assessment for Neurotoxicants”, Paris, France, 1998
Sixteenth International Neurotoxicology Conference, Symposium on “Genetic Predisposition and Individual Sensitivity”, Little Rock, AK, 1998
Seventh Meeting of the International Neurotoxicology Association, Symposium on “Susceptible sub-populations”, Leicester, UK, 1999.
Seventeenth International Neurotoxicology Conference, Symposium on “Mechanisms of Developmental Neurotoxicology: Molecular and Cellular Targets”, Little Rock, AR, 1999.
Eleventh Congress of the Brasilian Society of Toxicology and International Congress of Clinical Toxicology, Plenary Lecture on “Biochemical and Molecular Neurotoxicology” and Symposia on “Genetic susceptibility” and “Biomarkers in Toxicology”, Guaruja, Brasil, 1999
Twentysixth International Congress of Occupational Health, Symposium on “Polymorphic Metabolizing Enzymes and Biological Monitoring”, Singapore, 2000
Eigth Meeting of the International Neurotoxicology Association, Symposium on “Recent Advances in Developmental Neurotoxicology”, Estoril, Portugal, 2001
Thirtynineth Congress of the European Societies of Toxicology (EUROTOX), Symposium on “Age susceptibility in neurotoxicology”, Istanbul, 2001
Workshop on "Developmental neurotoxicity of pyrethroids", European Commission, Health & Consumer Safety Protection Directorate General, Brussels, 2001
Canadian Federation of Biological Sciences, 45th Annual Meeting, Symposium on “Neurotoxicology”, Montreal, 2002

Thirteenth Meeting of the Italian Society of Toxicology, Symposium on "Genetic polymorphisms in toxicology", Urbino, 2003
Nineth Meeting of the International Neurotoxicology Association, Symposium on "Food neurotoxicants and nutritional aspects of neurodegeneration", Dresden, 2003
Thirtyfirst Meeting of the Italian Society of Pharmacology, Symposium on "Substances of abuse and nervous system development", Trieste, 2003
Fortyfirst Meeting of the European Societies of Toxicology (EUROTOX), Symposium on "Alteration of signal transduction in neurotoxicity", Firenze, 2003
Sixtyseventh National Congress, Italian Society of Occupational Medicine and Industrial Hygiene, Bari, 2003
American Association for the Advancement of Sciences Annual Meeting, Symposium on "The Human Genome Project and Public Health: Gene-environment interactions", Seattle, 2004
First International meeting on Paraoxonases, Ann Arbor, MI, 2004
Sixth International Symposium on Biological Monitoring in Occupational and Environmental Health, Keynote Lecture on "Biochemical and molecular markers in neurotoxicology", Heidelberg, 2004
Advanced Course on Occupational Cancer and Chemical Risk, Parma, 2005
Italian Society of Epidemiology, Meeting on Environmental Epidemiology, Taranto, 2005
Sixtyeighth National Congress, Italian Society of Occupational Medicine and Industrial Hygiene, Parma, 2005
Seventeenth National Congress, Italian Society of Preventive and Social Pediatrics, Parma, 2005
Twentyeigth International Congress on Occupational Health, Symposium on "New trends in Occupational Medicine", Milano, 2006
International Course on Neurotoxicants and Neurodegenerative Disorders, Symposium on "Neurotoxicity of Pesticides", Venezia, 2006
Second International Meeting on Paraoxonases, Debrecen, Hungary, 2006
Fortythird Meeting of the European Societies of Toxicology (EUROTOX), Symposium on "Developmental neurotoxicity and Food Contaminants", Cavtat/Dubrovnik, Croatia, 2006
Meeting on "Food and Nutrition in Pediatrics", Parma, 2006
International Conference on "Food contaminants and neurodevelopmental disorders", Valencia, 2006
International Symposium on "Algal Toxins", Trieste, 2007
Mid America Toxicology Course, Kansas City, MO, 2007-2011
Eleventh Meeting of the International Neurotoxicology Association, Symposium on "Application of in vitro neurotoxicity testing for regulatory purposes", Asilomar, CA, 2007
Third International Meeting on Paraoxonases, Los Angeles, CA, 2008
Second Symposium on Developmental Neurotoxicity (TestSmart DNT II), Reston, VA, 2008
Italian Society of Toxicology (SITOX), Fifteenth Meeting, Symposium on "Toxicogenetics and pharmacogenetics", Verona, 2009
Seventh World Congress on Alternatives and Animal Use in the Life Sciences, Symposium on "Neuroscience", Roma, 2009
International Society for the Study of Xenobiotics, North America Annual Meeting, Symposium on "Metabolism, environmental chemicals and health", Baltimore, 2009 (declined).
Fourth International Meeting on Paraoxonases, Symposium on "Toxicology", Reus, Spain, 2010

Meeting on "Toxic substances in foods: a multidisciplinary approach", Italian Society of Nutrition, Parma, 2010
Fortyfirst Congress of the Portoguese Society of Pharmacology, Keynote speaker "The nervous system as a target for toxicity: mechanisms, biomarkers and testing", Coimbra, 2011
Twentieth Annual Meeting, Italian Association for in Vitro Toxicology (CellTox), Roma, Italy, 2011
Fifth International Meeting on Paraoxonases, Columbus, OH, 2012
Fourteenth meeting of the International Neurotoxicology Association (INA14), Symposium on "Genetic modulation of neurotoxicity and neuroprotection: state-of-the-art developments surveying selected neurotoxins", Egmond aan Zee, The Netherlands, 2013
Annual meeting of the Neurobehavioral Teratology Society, Symposium on "Unmasking silent neurotoxicity following developmental exposure to environmental toxicants", Bellevue, WA, 2014 (declined).
Fifteenth meeting of the International Neurotoxicolog yAssociation (INA15), Symposium on "Neurotoxicants are in the air: neurotoxicity of air pollution", Montreal, 2015
Sixth International Meeting on Paraoxonases, Alicante, Spain, 2015
Second International Conference on the Neurotoxicity and Prevention of Manganese Adverse Effects (MANGANESE2016), New York, NY, 2016 (declined)
Sixteenth meeting of the International Neurotoxicology Association (INA16), Symposium on "Environmental Factors in Autism Spectrum Disorders", Florianopolis, Brazil, 2017 (declined)
MilanoPsichiatria, Symposium on "Environmental Chemicals and Neurodevelopmental Disorders", Milano, Italy, 2019
Health Effects Institute Annual Meeting, Boston, 2020 (declined, meeting was cancelled)

Invited Seminars
Hoechst AG, Department of Toxicology, Frankfurt am Main, West Germany, 1982
University of Texas Medical Branch at Galveston, Department of Pharmacology and Toxicology, 1983
University of Washington, Department of Environmental Health, 1983; 1985; 1988; 1993
University of Milano, Department of Pharmacology, 1984, 1985
Farm Accident Rescue Program, Recognition of Pesticide Toxicology, Quincy, WA, 1984
University of Padova, Department of Pharmacology and Institute of Occupational Medicine, 1985
Battelle, Geneva Research Center, Geneva, Switzerland, 1986
US Environmental Protection Agency, Research Triangle Park, NC, 1986
University of Milano, Department of Agricultural Chemistry, 1989
Institute of Occupational Medicine, Chinese Academy of Preventive Medicine, Bejing, People's Republic of China, 1991.
West China University of Medical Sciences, School of Public Health, People's Republic of China, 1991
US Environmental Protection Agency, HERL, Research Triangle Park, NC, 1992
California Parkinson Foundation, San Jose, CA, 1992

Institute of Neurological Sciences, C. Mondino Foundation, University of Pavia, Pavia, Italy, 1993
Virginia Polytechnic Institute and State University, Molecular Cell Biology and Biotechnology Program, Blacksburg, VA, 1994
University of Bari, Institute of Occupational Medicine, Bari, Italy, 1995
Universidad Central de Venezuela, Facultad de Farmacia, Caracas, Venezuela, 1995
University of Urbino, School of Environmental Sciences, Urbino, Italy, 1997
University of Geneva, Department of Pharmacology, Geneva, Switzerland, 1997
Nestle Research Center, Lausanne, Switzerland, 1997
University of Heidelberg, School of Medicine, Heidelberg, Germany, 1997
Zeneca, Central Toxicology Laboratory, Macclesfield, England, 1997
University of Padova, Institute of Occupational Medicine, 1998
University of Roma, Institute of Pharmacology and Pharmacognosy, 1998
University of Pavia, Dept. of Pharmacology, School of Pharmacy, 1999
University of Connecticut, School of Pharmacy, 2000
University of Lisbon, School of Pharmacy, 2000
Sumitomo Chemical Company, Osaka, Japan, 2003-2010
Vanderbilt University, Center in Molecular Toxicology, 2005
University of Texas at Austin, 2016
University of Washington, DEOHS, 2017
University of Washington, Neurology GrandRounds, 2018
University of Washington, Center for Human Development and Disabilities, 2019
University of Rochester, 2019 (declined)
Columbia University, 2019 (declined)

UNIVERSITY SERVICE

Chair, Auxiliary Faculty Review Committee, 2019-2022
Member, PhD Exam Committee, 2019-2022
Faculty Sponsor and Mentor, Toby Cole (Clinical Assistant Professor salaried), 2019-2022
Faculty Co-Mentor, Judit Marsillach (Clinical Assistant Professor salaried), 2020-2021; Assistant Professor, 2021-2022
Member, Auxiliary Faculty Review Committee, 2018-2019; 2022
Member, Faculty Teaching Peer Review Committee, DEOHS, 2016-2017
Member, Diversity Committee, DEOHS, 2016-2020
Member, PhD Oversight Committee, DEOHS, 2012-2019
Member, Research Strategy Task Force, DEOHS, 2016-2018
Member, Admissions Committee, DEOHS, 2014-2015
Member, Faculty Search Committee, DEOHS, 2010-2012
Member, PhD Exam Committee, DEOHS, 2008-2011
Member, Faculty Council, School of Public Health and Community Medicine, University of Washington, 1999-2003
Chair, PhD Exam Committee, DEOHS, 2003-2006

Member, Faculty Senate, University of Washington, 1995-97
Member, Adjudication Panel, Faculty Senate, University of Washington, 1995-00
Member, Steering Committee, Interdisciplinary Molecular and Cellular Biology Program, University of Washington, 1992-2000
Member, DEH Admissions Committee, 1991-1999 (Chair: 1992-1993)
Member, DEH Curriculum and Teaching Policy Committee, 2000-2003
Chair, Sheldon D. Murphy Endowed Chair in Toxicology and Environmental Health Search Committee, 1995-1996
Chair, DEH Development Committee, 1991-1995
Director, Toxicology Program, DEH, 1991-2000
Member, DEH Ph.D. Committee, 1991-1995
Member, Radiological Sciences Review Committee, University of Washington, 1994-1995
Member, DEH Biomonitoring Laboratory Committee, 1990-1993.
Member, DEH Promotion, Tenure and Academic Policy Committee, 1988-1993; 1998-2003 (Chair: 1999-2000; 2002-2003)
Member, DEH Newsletter Committee, 1989-1993.
Director, DEH Animal Facilities, 1987-2022
Member, DEH Industrial Hygiene Research Committee, 1984-1991
Member, DEH Ad Hoc Committee for Open House, 1985
Chair, DEH Search Committee, Toxicology faculty, 1990
Chair, DEH Field Research and Consulting Group Advisory Committee, 1986-1988
Organizer, DEH Journal Club, 1985-1988
Member, DEH Seminar Committee, 1987-1988

TEACHING

Past Classroom Teaching

University of Washington

Lecturer in Toxicology, Department of Environmental and Occupational Health Sciences, University of Washington School of Public Health, Seattle, WA, 1984-
Courses:
ENVH 531, Neurotoxicology, 3 credits (50% responsibility, with Toby Cole).
ENVH 516, Toxic Agents: effects and Mechanisms (Environmental and Occupational Toxicology III), 3 credits (50% responsibility, with Toby Cole).

Guest Lectures in ENVH 515 (Environmental and Occupational Toxicology II), EPI/ENVH 571 (Neuroepidemiology and Environmental Risk Factors) DEOHS, ENVH 583, Environmental Health Readings, 1 credit (50% responsibility), 2004

ENVH 505, Fundamentals of Environmental and Occupational Toxicology, 4 credits (2020), with Rebekah Petroff

Other

Lecturer in Pharmacology and Toxicology, University of Parma Medical School, 2004-
Lecturer in Pharmacognosy, University of Milano, School of Pharmacy, 1977-1978

Lecturer in Neurotoxicology, University of Texas Graduate School of Biomedical Sciences and School of Public Health at Houston, Texas, 1981-1983
Lecturer in Neuropharmacology, University of Houston at Clear Lake, Texas, 1983
Course on Principles of Toxicology for EPA personnel, Seattle, WA, 1984-1985
Training course in Toxicology for EPA personnel and state and local environmental health practitioner, Tacoma, WA, June 1985; Portland, OR, August 1985 (Course organizer and instructor)
Lecturer in Toxicology, Neurotoxicology and Neuropharmacology, Postgraduate Schools in Neurophysiopathology, Pharmacology and Medical Toxicology, University of Pavia School of Medicine, 1990-1998
Lecturer in Toxicology, University of Milano, School of Pharmacy, 1989-1997
Lecturer in Toxicology, Postgraduate School in Experimental Toxicology, University of Milano, 1985-1998
Lecturer in Toxicology, University of Roma La Sapienza, School of Pharmacy, 1998-2002
Lecturer in Pharmacology and Toxicology, University of Bari Medical School, 2002-2004

Students and Postdoctoral Fellows Precepted or Advised
Graduate students, Ph.D. degree
James Meador, Univ. of Washington, School of Fisheries, Member of Thesis Committee and Graduate School Representative, 1985-1988.
Emma Bergmark, on leave from Dept. of Radiobiology, University of Stockholm, Sweden, Thesis Advisor, 1989-1992.
Carol Trenga, University of Washington, Dept. of Environmental Health, Thesis Advisor, 1990-1992.
Lynnda Reid, University of Washington, DEH, Advisor, 1991
Mara Seeley, University of Washington, DEH, Member of Thesis Committee, 1991-1996
Tammy McCullough, University of Washington, School of Business Administration, Member of Thesis Committee and Graduate School Representative 1991-1994.
Michelle Catlin, University of Washington, DEH, Thesis Advisor, 1992-1999.
Thomas McHugh, University of Washington, DEH, Rotation Advisor, 1993
Wan Fen Li, University of Washington, DEH, Thesis Advisor, 1993-1999
Gina Stivahtis, University of Washington, Molecular and Cellular Biology Program, Rotation Advisor, 1994
Lisa Jean Madden, University of Washington, Department of Psychology, Member of Thesis Committee and Graduate School Representative, 1995-1998.
Dolores Diaz, University of Washington, DEH, Rotation Advisor, 1995
Hailing Lu, University of Washington, DEH, Thesis Advisor, 1996-2001
Min Wei, University of Washington, DEH, Thesis Advisor, 1997-1999.
Shaw-Ree Chen, University of Washington, Molecular and Cellular Biology Program, Member of Thesis Committee and Graduate School Representative, 1998- 2002
Kyle J. Norton, University of Washington, Department of Pathology, Member of Thesis Committe and Graduate School Representative, 1999-2002.
Chantra Eskes, University of Lausanne, Faculty of Sciences, External expert, 2000
Samir Kelada, University of Washington, DEH, Thesis Advisor, 2001-2006

Francisco Dieguez, University of Washington, DEH, Member of Thesis committee, 2001-2003
Julia Hoeft, University of Washington, DEH, Member of Thesis Committee, 2001-2004
Kathryn VandeMark, University of Washington, DEH, Thesis Advisor, 2003-2009
Nadia Moore, University of Washington, DEH, Thesis Advisor, 2004-2008
Emma-Jane Poulton, University of Washington, DEOHS, Rotation Advisor, 2005
Heather Klinsworth, University of Washington, DEOHS, Rotation Advisor, 2006; Member, Thesis Committee, 2006-2011
Jing Chen, University of Washington, DEOHS, Rotation Advisor, 2005; Thesis Advisor, 2006-2010
Lewis Chang, Cancer Prevention Program, Fred Hutchinson Cancer Research Center, Graduate School Representative, 2004-2007
Cassandra Fok, University of Washington, DEOHS, Rotation Advisor, 2007
Devasmita Chakraverty, University of Washington, DEOHS, Rotation Advisor, 2007
Roshan Tofighi, Institute of Environmental Medicine, Karolinska Institute, Stockholm, External expert, 2007
Pamela Roque`, University of Washington, DEOHS, Rotation Advisor, 2007; Thesis Advisor, 2007- 2013
Daniella Pizzurro, University of Washington, DEOHS, Rotation Advisor, 2008; Thesis Advisor, 2008- 2013
Sean Harris, University of Washington, DEOHS, Rotation Advisor, 2009
Antoinette Defaux, University of Lausanne, Faculty of Sciences, External Expert, 2010
Travis Cook, University of Washington, DEOHS, Member of Thesis Committee, 2011-2014
Megan Cartwright, University of Washington, DEOHS, Thesis Co-Advisor, 2011-2013
Shih-Yu (Shelley) Chang, University of Washington, DEOHS, Rotation Advisor, 2011
Anna Engstrom, University of Washington, DEOHS, Rotation Advisor, 2012; Member of Thesis Committee, Member of Reading Committee, 2014-2016
Susanna Wegner, University of Washington, DEOHS, Member of Thesis Committee, 2012-2014
Ian Stanaway, University of Washington, DEOHS, Rotation Advisor, 2013
Brittany Weldon, University of Washington, DEOHS, Rotation Advisor, 2013
Jacki Coburn, University of Washington, DEOHS, Rotation Advisor, 2013; Thesis Advisor, 2013-2018
Yu-Chi (Rachel) Chang, University of Washington, DEOHS, Thesis Advisor, 2013-2018
Jacqueline Garrick, University of Washington, DEOHS, Rotation Advisor, 2014; Thesis Advisor, 2014-2021
Julie Park, University of Washington, DEOHS, Member of Thesis Committee, 2015- 2017
Hao Wang, University of Washington, DEOHS, Member of Thesis Committee, 2015- 2018
Rachel Shaffer, University of Washington, DEOHS, Member of Thesis Committee, 2017- 2020
Mustafa Ramadan, DEOHS, Rotation Advisor, 2018; Thesis Advisor, 2018 - 2019

Graduate Students, Master's degree

Stevin H. Zorn, Univ. of Texas Graduate School in Biomedical Sciences at Houston, Member of Thesis Committee, 1983
Paul L. Roney, Univ. of Texas School of Public Health at Houston, Adjunct Member of Thesis Committee, 1983

Brian McDonald, Univ. of Washington, Dept. of Environmental Health, Advisor and Thesis Advisor, 1984-1986
Priscilla Anderson, Univ. of Washington, DEH, Member of Thesis Committee, 1985-1986
William E. Maier, Univ. of Washington, DEH, Thesis Advisor, 1987-1988
Catherine Karr, Univ. of Washington, DEH, Thesis Advisor, 1987-1989
Denise M. Hamel, Univ. of Washington, DEH, Member of Thesis Committee, 1987-1988
Jin Li, Univ. of Washington, DEH, Member of Thesis Committee, 1988-1989
Eugene Gallagher, Univ. of Washington, DEH, Member of Thesis Committee, 1988-1989
Lonie Swenson, University of Washington, DEH, Member of Thesis Committee, 1988-1989
Jonathan Gastel, University of Washington, DEH, Advisor, 1988-1989
Carol Trenga, University of Washington, DEH, Thesis Advisor, 1988-1990
Denise LaFlamme, University of Washington, DEH, Member of Thesis Committee, 1989-1990.
Laurence S. Wechsler, University of Washington, DEH, Member of Thesis Committee, 1989-1990
Barbara Fitzgerald, University of Washington, DEH, Thesis Advisor, 1989-1991
Cheryl Rohwein, University of Washington, DEH, Member of Thesis Committee, 1990-1991
Nancy Beck, University of Washington, DEH, Advisor, 1990-1991; Member of Thesis Committee, 1991-1992
Kathryn Kovacs, University of Washington, DEH, Thesis Advisor, 1990-1992
Jennifer Reasoner, University of Washington, DEH, Member of Thesis Committee, 1992-1993
Wan Fen Li, University of Washington, DEH, Thesis Advisor, 1992-1993
Aoki Yutaka, University of Washington, DEH, Member of Thesis Committee, 1992-1993
John Kushleika, University of Washington, DEH, Member of Thesis Committee, 1993-1994
Min Wei, University of Washington, DEH, Thesis Advisor, 1995-1997
Sue Lee, University of Washington, DEH, Thesis Advisor, 1996-1998
Susan Leaman, University of Washington, DEH, Thesis Advisor, 1999-2001
Mark Burry, University of Washington, DEH, Thesis Advisor, 1999-2001
Julia Hoeft, University of Washington, DEH, Member of Thesis Committee, 1999-2001
Jenna Fisher, University of Washington, DEH, Member of Thesis Committee, 2001-2003
Kathleen Newhouse, University of Washington, Dept. of Environmental and Occupational Health Sciences, Member of Thesis Committee, 2002-2003
Betsy Walter, University of Washington, DEOHS, Thesis Advisor, 2003-2005
Jing Chen, University of Washington, DEOHS, Thesis Advisor, 2004-2005
Heather Klinsworth, DEOHS, Member of Thesis Committee, 2004-2005
Karen Jensen, DEOHS, Thesis Advisor, 2005-2008
Li Li, DEOHS, Thesis Advisor, 2005-2007
Candice Huang, DEOHS, Thesis Advisor, 2007- 2009
Travis Cook, DEOHS, Member of Thesis Committee, 2007-2009
Cassandra Fok, DEOHS, Member of Thesis Committee, 2008-2009
Chunyan Zhou, DEOHS, Thesis Advisor, 2009-2011
Leah Tait, DEOHS, Thesis Advisor, 2009-2011
Dan Miao, DEOHS, Thesis Advisor, 2013-2015
Kevin Heffern, DEOHS, Member of Thesis Committee, 2017-2018
Ashley Phillips, DEOHS, Thesis Advisor (with Toby Cole), 2020-2022

Undergraduate Students

Rini Sulaiman, Univ. of Washington, Institute of Environmental Studies, Supervisor for Undergraduate Thesis Project, 1984-1985
Kymberly Anable, High School Minority Students Biomedical Research Apprenticeship Program and Seattle University, Supervisor, 1986-1990
Karimah Hudda, University of Washington, Supervisor for Pre-Med Thesis Project, 1994-1995
Diane Wing, University of Washington, Supervisor for Pre-Med Thesis Project, 1995
Destia A. Johnson, University of Washington, Supervisor for Pre-Med Thesis Project, 1995
Clifford Lee, University of Washington, Supervisor for Undergraduate Research Projects, 1995-1996
Abby Close, University of Pennsylvania, Supervisor for Summer Research Project, 1996
Shahed Vahabpour, University of Washington, Supervisor for Undergraduate Research Project, 1999
Yunie Kim, University of Washington, Supervisor of Undergraduate Research Project, 2002-2003
Ahran Jang, University of Washington, Supervisor of Undergraduate Research Project, 2009-2010
Josh Matlock, University of Washington, Supervisor of Undergraduate Research Project, 2009-2010
Lisa Xiao, St. John's University, Environmental Health Research Experience Program, 2011
Zohal Sarwary, University of Washington, Supervisor of Undergraduate Research Project, 2012
Mitra Geier, Western Washington University, Environmental Health Research Experience Program, 2012
Mitwa Patel, University of Washington, Supervisor of Undergraduate Research Project, 2013-2015
Paige Bommarito, University of Michigan, Environmental Health Research Experience Program, 2014
Elzbieta Kalata, University of Illinois, Environmental Health Research Experience Program, 2015
Elise Hickman, University of California at Davis, Environmental Health Research Experience Program, 2016
Natalya Matlashchuk, University of Washington, Supervisor of Undergraduate Research Project, 2016-2017
Lea Barny, Dartmouth College, Environmental Health Research Experience Program, 2017

High School Students

From the Seattle Public School "Summer Work Training Program":
Marlena J. Foxx, Summer 1988
Anthony Gillard, Summer 1990
Thyda Chhom, Summer 1991
Cristina Davis, Summer 1992

From the University of Washington "GenOM Program":
Priscilla Perez, Summer 2008

Postdoctoral Fellows

Colleen Stamper, Ph.D., Postdoctoral Fellow, University of Washington, Department of Environmental Health, Advisor, 1985-1986
Walter Balduini, Ph.D., Senior Postdoctoral Fellow, University of Washington, DEH, Advisor, 1986-1989; 1993
Giovanni Olibet, M.D., Senior Research Fellow, University of Washington, DEH, Advisor, 1986-1988
Teresa Coccini, Ph.D., Postdoctoral fellow, University of Washington, DEH, Advisor, 1988-1990
Carl Calleman, Ph.D., Senior Fellow, University of Washington, DEH, Advisor, 1988-1993
Lydia Burrell, Ph.D., Fellow, University of Washington, DEH, Advisor, Summer 1988
Stefano Candura, M.D., Senior Fellow, University of Washington, DEH, Advisor, 1989-1992
Anna F. Castoldi, Ph.D., Senior Fellow, University of Washington, DEH, Advisor, 1990-1993
Paola Costa-Mallen, Ph.D., Research Scientist, University of Washington, DEH, Advisor, 1992-2006
Darrell Jackson, Ph.D., Senior Fellow, University of Washington, DEH, Advisor, 1992-1993
Liliana Intropido, Ph.D., Senior Fellow, University of Washington, DEH, Advisor, 1993-1994
Filippo Renò, Ph.D., Senior Fellow, University of Washington, DEH, Advisor, 1993-1994
Kevin Yagle, Ph.D., Senior Fellow, University of Washington, DEH, Advisor, 1993-2000
Marina Guizzetti, Ph.D., Research Scientist, University of Washington, DEH, Advisor, 1994-2010
Jan Oberdoester, Ph.D., Senior Fellow, University of Washington, DEH, Advisor, 1999-2000
Federica Bordi, Ph.D., Senior Fellow, University of Washington, DEH, 1999-2000
Wan Fen Li, Ph.D., Senior Fellow, University of Washington, DEH, 1999-2001
Annabella Vitalone, Ph.D., Senior Fellow, University of Washington, DEH, 2000-2003
Brian Thompson, Ph.D., Senior Fellow, University of Washington, DEOHS, 2003
Gennaro Giordano, Ph.D., Research Scientist, University of Washington, DEOHS, 2004- 2012
Rian de Laat, Ph.D., Senior Fellow, University of Washington, DEOHS, 2012- 2014
Toby B. Cole, Ph.D., Research Scientist, University of Washington, DEOHS, 2013-2019
Pamela Roque`, Ph.D., Senior Fellow, University of Washington, DEOHS, 2013- 2016
Jacqueline Garrick, Ph.D., Senior Fellow, University of Washington, DEOHS, 2021-2022

Visiting Scientists

DeSheng Wu, Ph.D., West China University of Medical Sciences, Chengdu, People's Republic of China, 1987-1988
Deng Hai, M.D., Institute of Occupational Medicine, Chinese Academy of Preventive Medicine, Beijing, People's Republic of China, 1990-1991
Cuye Tang, Ph.D. West China University of Medical Sciences, Chengdu, People's Republic of China, 1990-1991
Xiaoxia Tan, M.D., Department of Clinical Epidemiology and Medicine, Hunan Medical University, Changsha, People's Republic of China, 1991-1995
Shi Qing Xiao, M.D., Ministry of Public Health, Bejing, People's Republic of China, 1991-1993
Marion Ehrich, Ph.D., Virginia Polytechnic Institute and State University, Blacksburg, Virginia, 1993
Enrico Alleva, Ph.D., Istituto Superiore di Sanita`, Rome, Italy, 1998

Eriko Kashiwada, M.D., Osaka City University, Osaka, Japan, 1998
Ryozo Tsuji, Ph.D., Sumitomo Chemical Company, Osaka, Japan, 2001-2002
Vittorio Fattori, Ph.D., Dept. of Human Physiology and Pharmacology, University of Bari Medical School, 2006
Alessandra Amadori, Ph.D., University of Urbino, 2008
Sara Tagliaferri, Ph.D., University of Parma, 2009-2010.
Byung-Sun Choi, MD, PhD, Dept. of Preventive Medicine, School of Medicine, Chung-Ang University, Seoul, Republic of Korea, 2012-2013
Shuying Gao, PhD, Harbin Medical University, People's Republic of China, 2013-2014