Exhibit 123

GENERAL CAUSATION REPORT: TETRACHLOROETHYLENE AND PARKINSON'S DISEASE

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1. EXECUTIVE SUMMARY OF CONCLUSIONS

1.1 Tetrachloroethylene (PCE) is at least as likely as not a cause of Parkinson's disease (PD) based upon the following scientific evidence:

1.1.1 Structural similarity and structural activity relationships to trichloroethylene (TCE), which is a known PD risk factor based upon collective epidemiological and neurotoxicological data. There is also a lack of scientific support for the alternate hypothesis that 1 additional chlorine atom and 1 less hydrogen atom (PCE vs TCE) would be protective in preventing PD risk.

1.1.2 Epidemiology of chlorinated ethylene solvents (inclusive of PCE) and PD.

1.1.3 Direct experimental evidence that PCE toxicity is mediated by critical PD pathogenic pathways.

1.1.4 Hill considerations met directly for PCE, or by scientifically relevant analogy for TCE.

1.1.4.1 Strength of association

1.1.4.2 Consistency

1.1.4.3 Temporality

1.1.4.4 Biological plausibility

1.1.4.5 Coherence

1.1.4.6 Experiment

1.1.4.7 Analogy

2. QUALIFICATIONS

2.1 Education:

2.1.1 **Postdoctoral Fellowship (2006-2011),** Pittsburgh Institute for Neurodegenerative Diseases, University of Pittsburgh, Pittsburgh, PA 15260. Focus of training on mechanisms of environmentally induced neurodegeneration.

2.1.2 **Doctorate of Philosophy (Ph.D.) in Toxicology (December, 2006**), University of Michigan, Ann Arbor, MI, 48109. Focus of specialization (coursework and research) in neurotoxicology.

2.1.3 **Bachelor of Science (B.S.) with Honor, Physiology (May, 1998),** Lyman Briggs School of Science, Michigan State University, East Lansing, MI, 48824

Experience I have been internationally recognized in neurotoxicology. My research focuses on the adverse effects of environmental and occupational exposures on the nervous system. Since 2012, I have led a research lab with research foci across biological scale, from basic science studies on mechanisms of action, neurotoxic damage and the subcellular, cellular, and tissue levels, through elucidation of phenotypic effects. I have led and am currently leading studies in a variety of model systems and in clinical populations. My research has led to the development and advancement of novel cellular and animal model systems to discover how environmental exposures perturb molecular and biochemical mechanisms to induce neuropathology, resulting in phenotypic effects relevant to human neurodegenerative diseases. I am providing a copy of my most recent CV which demonstrates my qualifications and experience in greater detail, including publications, extramural funding, and consultations on a number of topics research interests. Of note and relevance to this matter, a considerable focus of my scholarship has been on the etiopathogenesis of Parkinson's disease (PD).

2.2 Select evidence of external recognition (see full CV for specific details)**.**

2.2.1 Elected Counselor of the International Neurotoxicology Association

2.2.2 Former President of the Neurotoxicology Specialty Section, Society of Toxicology

2.2.3 Standing Member of the Neurotoxicology and Alcohol Study Section, National Institutes of Health (extensive other grant and paper review shown in CV).

2.2.4 Acting Head, School of Health Sciences, Purdue University

2.2.5 Former recipient of the NIH Pathway to Independence Award (K99/R00)

3. METHODOLOGY

3.1 Materials reviewed:

3.1.1 2017 ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases. See reference list.

3.1.2 Scientific literature (PubMed – Table 1). All relied upon material within the reference list of this report.

Table 1. PubMed search terms.

Primary search term	**Secondary search term (+ "X")**
Tetrachloroethylene/perchloroethylene/PCE/PERC	• Parkinson's • Neurotoxicity • Dopamine • Brain • Synuclein • LRRK2 • Mitochondria
Trichloroethylene/TCE (focus on since 2017 ATSDR report)	• Parkinson's • Neurotoxicity • Dopamine • Brain • Synuclein • LRRK2 • Mitochondria
Chlorinated solvents* (focus on since 2017 ATSDR report) **This search is limited to chlorinated solvents because of the totality of research implicating this specific use class and chemical structure class (ATSDR report and other epidemiological studies cited throughout). "Solvents" alone are a broad chemical use class that does not, in itself specify any chemical structure of expected biological activity.*	• Parkinson's • Neurotoxicity • Dopamine • Brain • Synuclein • LRRK2 • Mitochondria

3.1.3 Chemical properties (PubChem). See reference list.

3.2 Establishment of causation:

In general, a similar strategy used by the ATSDR was utilized here[1]:

3.2.1 Sufficient: The evidence is sufficient to conclude that a causal relationship exists.

3.2.2 Equipoise and Above*: The evidence is sufficient to conclude that a causal relationship is at least as likely as not, but not sufficient to conclude that a causal relationship exists.

3.2.3 Below Equipoise: The evidence is not sufficient to conclude that a causal relationship is at least as likely as not, or is not sufficient to make a scientifically informed judgment.

3.2.4 Against: The evidence suggests the lack of a causal relationship.

Relative to legislation in Camp Lejeune cases, the minimum threshold was as follows[1]:

*Equipoise and above evidence for causation: The evidence is sufficient to conclude that a causal relationship is at least as likely as not, but not sufficient to conclude that a causal relationship exists. This category would be met, for example, if:

3.2.5 A meta-analysis does not provide convincing evidence (e.g., the summary risk estimate is close to the null value of 1.0, i.e., ≤1.1), or if the meta-analysis observes a non-monotonic exposure-response relationship) but there is at least one epidemiological study considered to be of high utility occurring after the meta-analysis has been conducted, in which an association between the exposure and increased risk of the disease of interest has been found and in which chance and biases can be ruled out with reasonable confidence.

3.2.6 A meta-analysis has not been conducted, but there is at least one epidemiological study considered to be of high utility in which an association between the exposure and increased risk of the disease of interest has been found and in which chance and biases can be ruled out with reasonable confidence.

Toxicological analyses:

3.2.7 For the purpose of this expert analysis, toxicological plausibility is also considered. That is, whether or not relevant neurotoxicology data support an at least as likely as not etio-pathogenic relationship between PCE and PD.

Table 2. Bradford Hill for assessing causation (with modernized language; importantly, the Hill considerations were originally developed based upon epidemiology and there have since been a number of advances in numerous relevant criteria, including toxicology)[2,3]:

Criterion	**Bradford Hill 1965 Criteria Description**[1]
Strength of association	The strength of a supposed association between an intervention and an outcome is determined by the appropriate statistic used to measure the protective effect of an intervention (e.g., relative risk or OR).
Consistency	Has it been repeatedly observed by different persons, in different places, circumstances and times?
Specificity	Specificity is present when the intervention is exclusive to the outcome and when the outcome has no other known cause or associated risk factors; cautions that this criterion should not be overemphasized and that if specificity is not apparent, this does not preclude causation **For toxicological analyses, the "intervention" would be the exposure.*
Temporality	Refers to temporal relationship of association between exposure and disease outcome; to infer causality, exposure must precede outcome
Dose-response	If the association is one in which a dose–response curve or biological gradient can be observed, this adds to the case for causality.
Biological plausibility	A likely biological mechanism linking the intervention to the observed findings helps to explain causality; plausibility depends on biological knowledge of the day **For toxicological analyses, the "intervention" would be the exposure.*
Coherence	When the evidence from different disciplines sources 'hangs well together' and does not

[1] The criteria proposed by Sir Bradford Hill in 1965 are in reality considerations to be used to assess available evidence and draw conclusions on causal inference and causation. Hill put it this way: "[h]ere then are nine different viewpoints from all of which we should study association before we cry causation. What I do not believe – and this has been suggested -is that we can usefully lay down some hard-and-fast rules of evidence that *must* be obeyed before we accept cause and effect. . . . none can be required as a *sine qua non.* What they can do, with greater or less strength, is to help us make up our minds . . ." Hill, "The Environment of Disease: Association or Causation. Proceedings of the Royal Society of Medicine, Section of Occupational Medicine, 295, 299 (1965).

	conflict with other generally known facts, this criterion is met
Experiment	Experimental evidence from laboratory studies or RCTs could potentially provide strongest support for causation. this criterion often provides the strongest support for causation and describes whether there is empirical evidence for the association.
Analogy	Causality is supported by analogy if there are similar associations or causal relationships in other areas of relevance, weakest form of evidence of causality.

4. RESULTS OF EXPERT ANALYSIS

4.1 Overview of relevant aspects of the 2017 ATSDR report (relevant portions receive additional qualitative and quantitative analysis and commentary in section 4.2)

4.1.1 Summary of overarching issues and report excepts.

PCE, also known as perchloroethylene (PERC) was a known chemical exposure at Camp Lejeune, along with trichloroethylene (TCE), benzene, 1,2-dichloroethylene (DCE) and vinyl chloride. During the 1982 sampling, measured levels of TCE and PCE in the distribution system of Hadnot Point were as high as 1,400 ppb and 100 ppb, respectively, well above the Environmental Protection Agency's (EPAs) 5 ppb maximum contaminant level (MCL). The Tarawa Terrace treatment plant provided drinking water to the Tarawa Terrace housing area at the base. Of note, during the July 1982 distribution system sampling, PCE was measured as high as 104 ppb and reached a maximum of 215 ppb during the February 1985 sampling[1].

2017 ATSDR report determined TCE was Equipoise and above evidence for causation and PCE was below equipoise evidence for causation[1].

Direct excerpts of 2017 ATSDR report[1] (shown in brackets and quotes):

Epidemiology:

["The epidemiological evidence for TCE or PCE exposures and Parkinson disease is very limited because few studies have been conducted. On the other hand, there is mechanistic information based on animal studies. Therefore, ATSDR's assessment of the evidence for causation placed high weight on studies and review articles that provided mechanistic information. High weight was also given to a well-conducted twin study although the study was limited by a small number of exposed cases. One study has evaluated TCE and PCE exposure and Parkinson disease; two studies have evaluated chlorinated solvents and aromatic solvents separately, and several studies have evaluated any solvents. A meta-analysis evaluated 16 studies that evaluated any solvents and obtained a summary OR of 1.35 that increased to 1.58 when the

analysis was restricted to six higher quality studies (Pezzoli et al. 2013). The two studies of chlorinated and aromatic solvents had mixed findings (Brouwer et al. 2015 and Van der Mark et al. 2015). The key study is the Goldman et al. 2012 twin study which found high elevations in risk for both PCE and TCE with evidence of an exposure-response relationship for exposure duration and cumulative exposure. This study used rigorous methods to ensure diagnostic accuracy and to assess exposures. The twin design had the advantage of controlling for potential confounders due to genetic and shared environmental factors. A limitation was the small number of exposed cases which resulted in wide confidence intervals.

Parkinson disease mortality was evaluated in the Camp Lejeune mortality study of civilian workers and an elevated risks were observed when comparing these workers to the U.S. population (SMR=2.2, 95% CI: 0.7-5.1) and civilian workers at Camp Pendleton (RR=3.1, 95% CI: 0.8, 12.9). Although limited by the small number of deaths due to Parkinson disease, the study found that four of the five deaths occurring among the Camp Lejeune workers had above the median cumulative exposure to TCE and PCE (Bove et al. 2014b). Parkinson disease mortality could not be evaluated in the Camp Lejeune mortality study of Marines.

There have been a few case reports of Parkinson disease and Parkinsonism among TCE-exposed workers that are described in a review article of TCE and Parkinson disease by Zaheer and Slevin 2011."][4-10]

Expert notes on the above ATSDR text: PD mortality could not be assessed in the Bove study because only 14% of the cohort had died by analyses. The above studies are inclusive of multiple types of human studies, for example, retrospective concordance study in a twin cohort (PD risk), retrospective cohort mortality study (compared to a base without known high chlorinated solvent exposure), case-control, and individual case reports. The studies cited above on chlorinated solvents that are said to have mixed results did not specify which chlorinated solvents were expected, nor specific exposure levels.

Hill consideration met: Strength of association. Human studies suggest a relationship between PCE and PD that is at minimum, as likely as not. Additional context in section 4.2.

Hill consideration met: Consistency. Human studies suggest a relationship between PCE and PD that is at minimum, as likely as not. These studies cited above have been conducted in multiple human cohorts. Additional context in section 4.2.

Hill consideration met: Temporality: Epidemiology studies show that exposure occurs prior to increased risk.

Hill consideration met: Analogy. Human studies show a clear relationship between TCE and PD, without ruling out similar effects for PCE. In contrast, a similar relationship is highly suggested by studies examining the entire chemical class, or PCE individually. These studies cited above have been conducted in multiple human cohorts. Overall, the chemical structure

similarities between PCE and TCE predict similar biological effects (see section 4.2.1). There is also a lack of scientific support for the alternate hypothesis that 1 additional chlorine atom and 1 less hydrogen atom (PCE vs TCE) would be protective in preventing PD risk.

Animal and mechanistic information (again, direct excerpt from the 2017 ATSDR report in brackets and quotes):
["TCE has been found to be a mitochondrial neurotoxin in animal studies, and mitochondrial dysfunctions in substantia nigra dopamine neurons is considered to cause the disease (Gash et al. 2008; Zaheer and Slevin 2011). Studies in rats have shown that TCE exposure causes selective loss of dopaminergic neurons in the substantia nigra pars compacta (SNpc), a pattern consistent with human pathological staging of Parkinson disease (Goldman et al. 2011). A systematic review of the toxicological and epidemiological evidence made several observations: (1) it is uncertain whether inhalation of TCE can cause similar damage since the animal studies involved oral administration of TCE; (2) it is uncertain whether TCE or its metabolites cause the damage to dopaminergic neurons in the SNpc; and (3) if a TCE metabolite is the cause of the damage, it is also possible that PCE could cause similar damage since

TCE and PCE have some common metabolites (Lock et al. 2013). The review concluded: "On balance, the convergence of toxicological and epidemiological research suggests a plausible association between TCE exposure and PD [Parkinson disease]." A recent report by the IOM echoes this conclusion:

"…Parkinson disease is a neurobehavioral effect that may result from exposure to TCE and/or PCE." (IOM 2015)."][4,8,11,12]

Hill consideration met: Biological Plausibility: The above conclusions show PCE and TCE overlap with respect to PD, and extends beyond Analogy, where biological plausibility is supported by overlapping metabolites. Additional context and weight of evidence for both mechanistic and animal studies are further explained in section 4.2

Conclusion: ["Positive associations have been observed for TCE and PCE and Parkinson disease in a well-conducted twin study (Goldman et al. 2012). The Camp Lejeune study of civilian workers also found a positive association for Parkinson disease (Bove et al. 2014b). Because only two studies have focused on TCE exposure (Goldman et al. 2012; Bove et al. 2014b), the epidemiological evidence for causation for TCE and Parkinson disease is very limited. However, the findings from animal studies indicating a plausible mechanism for TCE exposure and Parkinson disease that is relevant to humans provides important supplemental evidence for causation. ATSDR concludes that the epidemiological evidence for causality by itself is currently too weak to achieve equipoise and above. However, given the strong supporting mechanistic evidence for TCE, ATSDR concludes that there is equipoise and above evidence for causation for TCE and Parkinson disease.

For PCE, the epidemiological evidence is very limited and there is no available information on a plausible mechanism as there is for TCE. However, this may change if a metabolite of TCE that is common to PCE is found to be the agent causing damage to the dopaminergic neurons. Given what is presently known, ATSDR concludes that there is below equipoise evidence for causation for PCE and Parkinson disease".][4,9].*

*These conclusions were drawn as of 2017 by ATSDR report. Additional weight of evidence is addressed throughout this report. It is worth noting that even as of this 2017 ATSDR report, available data (or evidence?) supports that TCE and PCE are at least as likely as not to be a cause of PD. Based upon my review of the evidence as set out in this report, I have reached the same conclusion.

4.1.2 Overall expert commentary on the 2017 ATSDR report.

As of the 2017 ATSDR report, collective scientific data had already reached the threshold of equipoise and above evidence for causation for TCE and Parkinson disease. However, PCE had not reached this threshold, though data were deemed to be suggestive. Section 4.2 more broadly examines mechanistic data for PCE that had occurred both prior to this the 2017 report and since. Moreover, the strength of the analogy of PCE neurotoxicity to TCE neurotoxicity is also examined.

4.2 Relevant neurotoxicological evidence that was not considered in the 2017 ATSDR report or that requires additional context. With respect to the role of PCE in PD, there are additional interpretations (points of relevance that should be expounded upon) and data (either new since 2017 or not fully considered in the ATSDR report) that are relevant.

4.2.1 Chemical properties comparison. It is a well-known toxicological principle that chemicals sharing structural features may have similar biological effects (structure activity relationships)[13]. Moreover, in toxicology, including the neurotoxicology of PD, a single chemical within a chemical class often receives preferential research focus. Such focus can be due to use, awareness, or even precipitated by initial results driving future focus (Table 3).

Table 3. Known PD relevant toxicants (both compelling epidemiology and toxicology studies), along with other, less well studied members of the same chemical class.

Established PD toxicant/toxin	Chemical class	Additional chemicals that have received less research attention on PD relevance
Paraquat	Viologen	Diquat
Rotenone	Crystalline isoflavone/rotenonoid	Deguelin, dehydrodeguelin, rotenol, tephrosin and sumatrol from Indigofera tinctoria
Trichloroethene	Chloroethylene	Vinyl chloride, dichloroethylene, tetrachloroethylene

TCE and PCE differ by a single atom, TCE having 1 less chlorine and 1 more hydrogen than PCE. Specifically for PCE and TCE, where since the 1970s it has been know that because the chloroethylenes are each members of the same chemical family (within chlorinated olefins), certain similarities in biologic response were expected and found (this early study focused on metabolic overlap and biological membrane damage)[14]. Also shown are 1,2-dichloroethylene, DCE and vinyl chloride, VC, as additional chloroethylenes for reference. Note that the addition of each chlorine increases lipophilicity (increased logP, also known as log Kow) and decreases water solubility. Lipophilicity means "fat-loving", where in general, increased lipophilicity results in increased biological accumulation (Table 4, also noted in in section 4.2.5 showing higher biological half-life of PCE vs TCE). This relationship is shown in Figure 1, where the bioconcentration factor increases as the logP increases (up until logP ≥6, where chemicals become insoluble)[15-17].

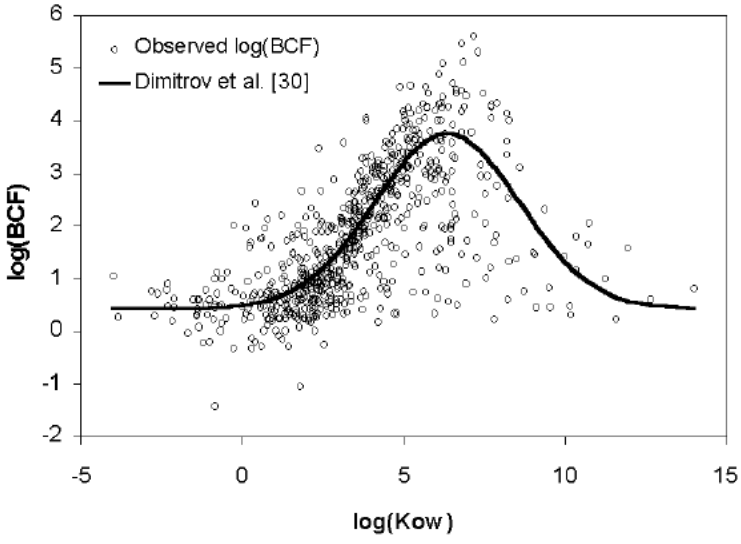
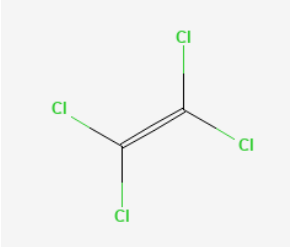
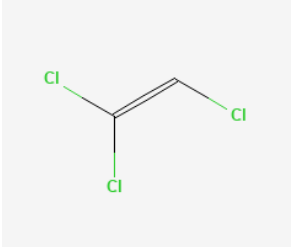
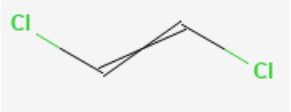
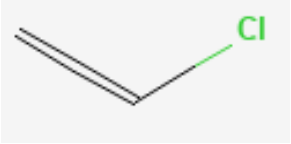


Figure 1. Bioconcentration (BCF) increases as logP increases (also known as log Kow) (modified from[15]). All chloroethylenes shown in Table 4 have logPs within the range where increasing logP would be expected to increase bioconcentration.

Table 4. Relevant chemical properties comparison of PCE and TCE (1,2-dichloroethylene, DCE and vinyl chloride, VC, also shown as chloroethylenes for reference) (from PubChem[18,19]).

Chemical property	**PCE**	**TCE**	**DCE***	**VC**
Molecular formula	C_2Cl_4 $Cl_2C=CCl_2$	C_2HCl_3 $ClCH=CCl_2$	$C_2H_2Cl_2$ ClC=CCl	C_2H_3Cl H2C=CHCl
2D structure				
Molecular weight (g/mol)	165.8	131.38	96.94	62.5
XLogP3 XLogP3 3.0 PubChem release 2021.10.14	3.4	2.6	1.9	1.5
Solubility in water (mg/L at 25 °C)	206	1280	Isomer dependent	8800
Vapor pressure (mm Hg at 25 °C)	18.5	69	200	2980
Odor threshold (from AIHA (ppm)	47 *Note: PCE odor does not necessarily provide adequate warning because PCE quickly desensitizes olfactory responses; persons can suffer exposure to vapor concentrations in excess of TLV limits without smelling it.*	82	17[20]	10

*DCE comes in multimer isomers. *Mixed isomer form* is shown for reference.

Interpretation of key chemical differences: The additional chlorine results in a heavier molecule, though both PCE and TCE would be considered small molecules. PCE is excepted to be more lipophilic due to a higher LogP, meaning it may cross biological membranes more efficiently[21].

> **Hill consideration met: Analogy**. Chemicals with highly similar structures are expected to produce similar biological effects, unless there is scientific evidence to suggest otherwise (structure activity relationships). The chemical structures of PCE and TCE are highly similar.

4.2.2 Metabolic pathways of PCE. Importantly, in some environments PCE may be converted to TCE (Figure 2). Moreover, PCE and TCE share or have similar metabolites (Figure 3).

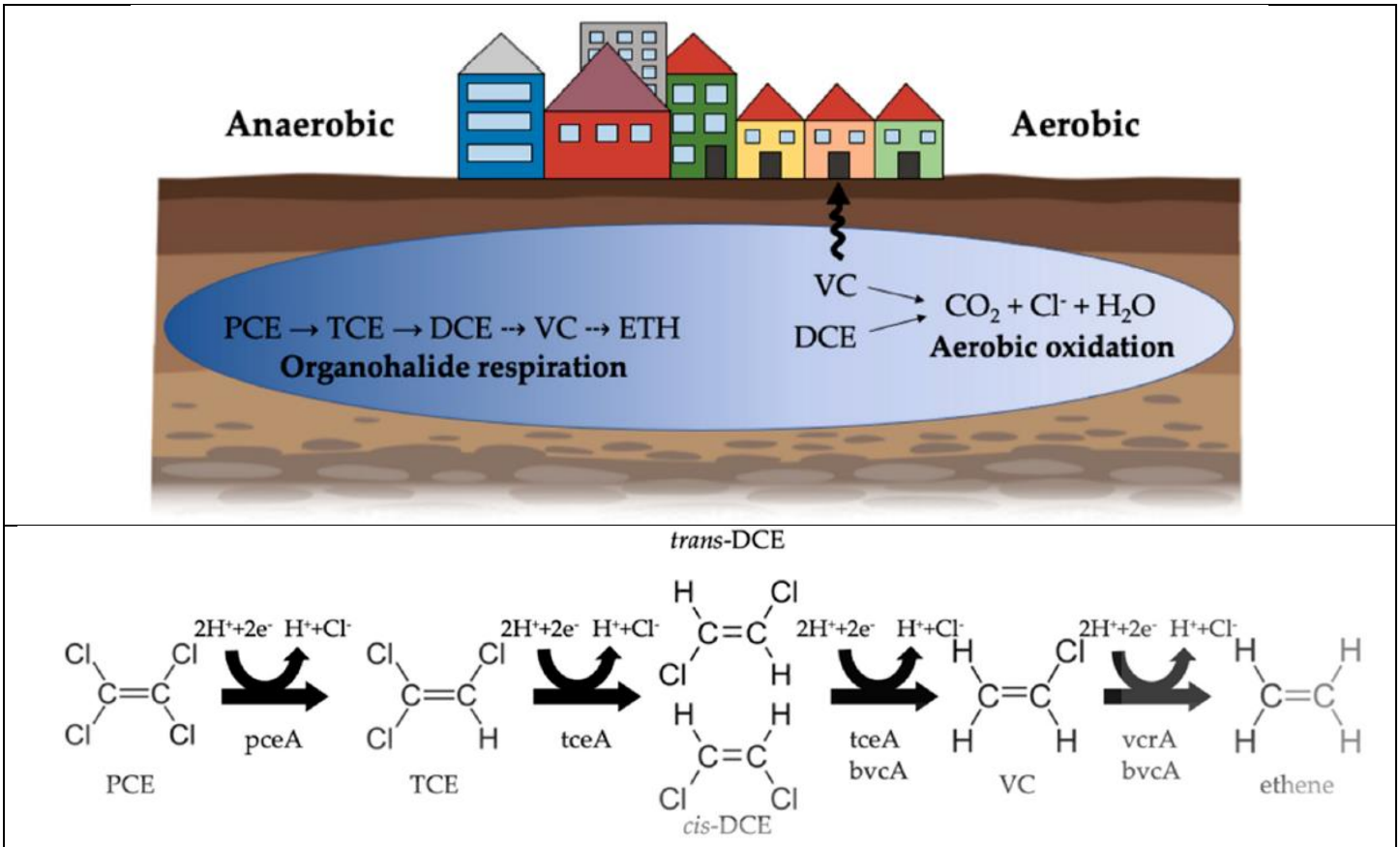
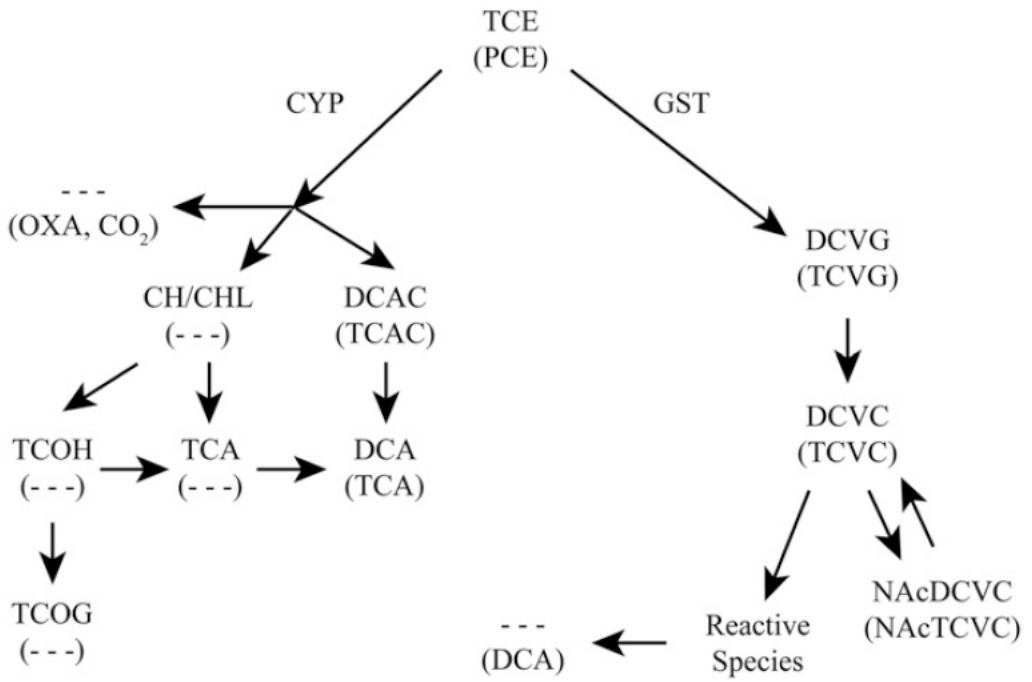


Figure 2. Under anaerobic conditions PCE can be converted to TCE (modified from[22]).



Tissue specificity of TCE and PCE metabolites

Target Tissue	Oxidative Pathway		GSH Conjugation Pathway	
	TCE	PCE	TCE	PCE
Liver: local formation	CH/CHL TCA TCOH TCOG DCAC DCA	 TCA TCAC 	DCVG DCVC NAcDCVC	TCVG TCVC NAcTCVC
Kidney: local formation	—	—	DCVG DCVC NAcDCVC Reactive metabolites	TCVG TCVC NAcTCVC Reactive metabolites DCA
Lung: local formation	CH/CHL TCA TCOH DCAC DCA	 TCA TCAC 	—	—
Testes: local formation	CH/CHL TCA TCOH DCAC DCA	 TCA TCAC 	—	—
Systemically available	CH/CHL TCA TCOH TCOG DCA	 TCA 	DCVG DCVC NAcDCVC	TCVC TCVC NAcTCVC DCA

TCOG, trichloroethanol *O*-glucuronide.

Figure 3. PCE and TCE have similar metabolic pathways, producing similar metabolites (in some cases the same) (modified from[23]).

Oxidative metabolism of both PCE and TCE is likely through CYP2E1, which is expressed in the brain[23]. Figure 4 shows that not only does CYP2E1 occur in the brain, but it is also specifically detectable within the substantia nigra, where the dopaminergic neurons are located that are lesioned in PD.

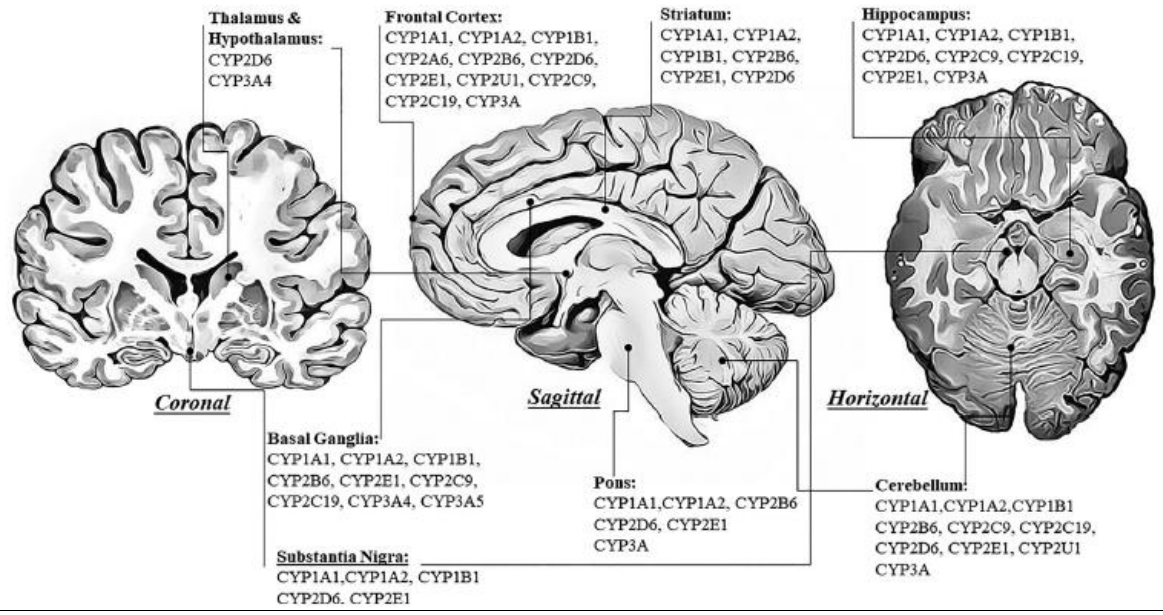


Figure 4. CYP2E1 is present in the substantial nigra, where it is plausible that local metabolism of chlorinated solvents could occur. (modified from[24]).

Hill consideration met: Biological Plausibility: The above conclusions show PCE and TCE overlap with respect to PD extends beyond Analogy, where biological plausibility is supported by overlapping and metabolic pathways metabolites.

Overall Hill consideration comment: PCE may be converted to TCE under some environmental conditions. Under such a scenario, PCE would be relevant to all TCE met Hill considerations.

4.2.3 Epidemiology of PCE and TCE.

Etiological links between TCE and Parkinson's are stronger than PCE, though "data are suggestive for PCE" as well[25]. Additionally, not only are volatile organic compounds (inclusive of PCE and TCE) a PD risk factor, they also increase the rate of disease progression/severity of symptoms[25]. As of the 2017 ATSDR report, the epidemiology of TCE plus the animal data was already equipoise sufficient. The epidemiology specific to PCE and PD requires additional context. A well-designed 2012 study noted, PCE is a solvent that "has been previously linked to Parkinson's in case reports or analytic studies"[4]. This paper aimed to verify this link using large-scale epidemiology, where the authors examined a cohort of 99 pairs of twins discordant for PD (one of the twins had PD and the other did not). They then calculated each twin's PCE exposure, utilizing "occupational exposure assessment methods" that probed whether the twin had been exposed to PCE via his job or hobby. Only 6% of the twins had any exposure to PCE, which

meant that the study had limited power and therefore wide confidence intervals (CIs). Here, the study showed a strong link between PCE exposure and Parkinson's Disease. For the twin with any exposure to PCE, the odds ratio for this analysis was 10.5 (95% CI = 0.97-113), indicating that the twin exposed to PCE was more than 10 times as likely to develop PD than the twin who was not exposed to PCE. Even given the small sample size, the results showed very near statistical significance for PCE, *i.e.*, they "tended toward significantly increased risk.". Of note, typical scientific statistical testing requires $p<0.05$, meaning ≥95% chance that the effect was not detected due to random variation (in this case $p = 0.053$, or 94.7% chance that the effect was not detected due to random variation). Similar results were shown for duration of PCE exposure and cumulative PCE exposure. TCE exposure was associated with significantly increased risk of PD with an odds ratio of 6.1 (95% CI= 1.2–33). While the magnitude of effect for TCE was lower than PCE, the odds ratio for TCE reached statistical significance. Given this, it is reasonable to assume that PCE did not reach statical significance due to the smaller sample size (only 5% of PD cases were exposed to PCE, while 10% of cases were exposed to TCE and 12% of cases were exposed to both PCE and TCE). Moreover, pooled exposure to either TCE or PCE was associated with markedly increased PD risk, odds ratio of 8.9(95% CI = 1.7 – 47)[4]. Again, based upon these data, it is reasonable to assume, and highly likely, that an increased sample size would have reached statistical significance between PCE and PD (Figure 5).

Solvent	Case$^-$/ Control$^-$	Case$^+$/ Control$^-$	Case$^-$/ Control$^+$	Case$^+$/ Control$^+$	Ever/Never Exposed, OR (95% CI)	*p*
Toluene	72	11	9	7	1.3 (0.5–3.3)	>0.2
Xylene	88	6	2	3	2.2 (0.4–12)	>0.2
n-Hexane	85	6	7	1	1.3 (0.4–4.1)	>0.2
CCl_4	74	14	9	2	2.3 (0.9–6.1)	0.088
PERC	93	5	1	0	10.5 (0.97–113)	0.053
TCE	87	9	2	1	6.1 (1.2–33)	0.034
TCE or PERC	85	11	2	1	8.9 (1.7–47)	0.010
Any of 6 solvents	51	19	14	15	1.7 (0.8–3.7)	0.16
Any of 4 excluding TCE and PERC	53	18	15	13	1.5 (0.7–3.1)	>0.2

[a]Ever exposure, adjusted for respondent type and smoking.
CCl_4 = carbon tetrachloride; CI = confidence interval; OR = odds ratio; PD = Parkinson disease; PERC = perchloroethylene (tetrachloroethylene); TCE = trichloroethylene.

Figure 5. Solvent exposure as a risk factor in PD. TCE and PCE+TCE reached statistical significance. While PCE was just below reaching statistical significance, it had the highest calculated odds ratio. It is critical to note that typical scientific statistical testing requires $p<0.05$, meaning ≥95% chance that the effect was not detected due to random variation. This is a far greater burden of scientific proof than ≥50% to establish equipoise (as likely as not) and above evidence for causation.

An additional study also showed a link between PCE exposure and PD in civilians who worked at Camp Lejeune, which showed that civilians with "above median" exposure to PCE had a

hazard ratio of 2.68 (0.22-33.28), interpreted as nearly triple the risk of PD as those who had below median exposure[9]. Again, the risk ratio (this example as hazard ratio) was higher for PCE than TCE. The confidence intervals were wide given the small sample size. However, this study still indicates that 22.3 micrograms/liter-years of PCE exposure is enough to increase the risk of Parkinson's Disease, Here again, it is also worth noting that the risks for civilians exposed to above-median amounts of TVOC—a total measure of exposure to all the chemicals—were 2.52 (0.21-30.83)[9] (Figure 6).

Contaminant	≥ Median Exposure	Cumulative Exposure	Log_{10} Cumulative Exposure
PCE	2.68 (0.22, 33.28) N = 4	0.0199 (0.0005, 0.0393) p = .04	1.9718 (−0.8134, 4.7569) p = .16
TCE	2.51 (0.21, 30.76) N = 4	0.0009 (0.0001, 0.0017) p = .04	2.6244 (−0.7668, 6.0156) p = .13
Vinyl Chloride	2.81 (0.23, 34.11) N = 4	0.0129 (0.0005, 0.0253) p = .04	2.0982 (−0.7936, 4.9900) p = .15
Benzene	2.52 (0.20, 31.59) N = 4	0.0490 (0.0008, 0.0971) p = .05	2.0910 (−0.7578, 4.9398) p = .15
TVOC	2.52 (0.21, 30.83) N = 4	0.0005 (<0.0001, 0.0011) p = .04	2.6729 (−0.7448, 6.0905) p = .12

Exposure lagged 10 years. Adjusted by sex, race, occupation (blue collar vs white collar) and education. Selected causes of death. Camp Lejeune cohort (N = 4,647).

Figure 6. hazard ratios (95% CIs) for PD for categorized [<median (ref.) ≥median) maximum cumulative exposure and coefficients (95% CIs) for continuous cumulative exposure (μg/L/year) (modified from[9]).

Note: An odds ratio (OR) compares the odds of an event occurring in one group compared to another at a single point in time, while a hazard ratio (HR) compares the instantaneous risk of an event occurring between two groups over the study period (useful in analyzing time to event data).

Overall, the evidence for the role of solvents in PD continues to increase, where much of the research focus continues to be on TCE. Here, military site-specific exposures have received much attention. It is notable that military service itself has been repeatedly investigated as a risk factor for neurodegenerative diseases. Thus, a recent study comparing PD prevalence in military personnel from Camp Pendelton vs. Camp Lejeune (known PCE and TCE contamination above regulatory limits) was a highly powerful epidemiological approach using a cohort design. The results showed that those at Camp Lejeune exposed to TCE had a 70% increased risk of developing PD[26]. The study notes that during the exposure period, maximum contaminant levels for drinking water at Camp Lejeune were exceeded for at least 3 chloroethylenes, including PCE, TCE, and vinyl chloride. Many of the conclusions drawn were relative to TCE and PD because in part of the extensive research focus on TCE and PD in both epidemiological studies and animal studies. However, other VOCs noted above were also present and above regulatory thresholds. Moreover, while similar mechanisms of action are expected, potency differences may occur (noted in mitochondrial studies discussed in this report). The authors note the following limitation in this highly powerful study: "*Finally, although TCE was the VOC present in the Camp Lejeune water supply at the highest concentrations, the water also contained high levels of PCE, vinyl chloride, and benzene*."[26]. Thus, contributions to PD risk from PCE cannot be fully separated in this study. Overall, this study collectively increases the weight of evidence of chloroethylenes as PD risk factors (benzene is in a different chemical class and not linked to PD). This study received commentary in the journal Science, where multiple prominent

neurotoxicologists not involved in the study commented on the importance, noting the impact to understanding PD risk and PD mechanisms[27].

Hill consideration met: Strength of association. The totality of human studies suggest a causative relationship between PCE and PD that is at minimum, as likely as not. Studies point to PCE as an individual risk factor or part of collective VOC exposures that are risk factors.

Hill consideration met: Consistency. The totality of human studies suggest a causative relationship between PCE and PD that is at minimum, as likely as not. These studies cited above have been conducted in multiple human cohorts.

Hill consideration met: Temporality: Epidemiology studies show that exposure occurs prior to increased risk.

4.2.4 **Hill consideration met: Analogy**. Human studies show a clear relationship between TCE and PD, without ruling out similar effects for PCE. In contrast, a similar relationship is highly suggested by studies examining the entire chemical class, or PCE individually. These studies cited above have been conducted in multiple human cohorts. Overall, the chemical structure similarities between PCE and TCE predict similar biological effects (see section 4.2.1). There is also a lack of scientific support for the alternate hypothesis that 1 additional chlorine atom and 1 less hydrogen atom (PCE vs TCE) would be protective in preventing PD risk.

4.2.5 **TCE neurotoxicology and relevance to PCE.** The 2017 ATSDR report states: "it is also possible that PCE could cause similar damage since TCE and PCE have some common metabolites (Lock et al. 2013). The review concluded: "On balance, the convergence of toxicological and epidemiological research suggests a plausible association between TCE exposure and PD [Parkinson disease]." A recent report by the IOM echoes this conclusion: "…Parkinson disease is a neurobehavioral effect that may result from exposure to TCE and/or PCE.".[1,12]. My collective review of the epidemiology and toxicology leads to an opinion, to a reasonable degree of scientific certainty, that PCE is at least as likely as not a cause of PD relevant neurotoxicity.

Updated toxicology studies on chlorinated solvents and PD.

Overview and new animal studies

Early work on TCE and PD stemmed from both a case report and preliminary animal studies[28], which have now developed into a strong body of scientific literature that confirms the relationship. Important publications in animal models had suggested a strong link between oral TCE exposure and a PD phenotype, where initial reports of 1000 mg/kg/day, 5 days/week, for 6 months (expected blood levels, ~35x that of industrially exposed workers)[10]. A follow-up study across multiple doses, showed detectable loss of nigral dopamine neurons (key PD neuropathology hallmark) could be achieved as low as 500 mg/kg and that at doses 200-1000mg/kg, the magnitude of effect was dose dependent[29]. These papers found a number of other key markers of a PD relevant phenotype, including striatal dopamine depletion, decreased mitochondrial complex I activity, oxidative stress, alpha-synuclein accumulation, and motor

deficits. Further studies, while in typically non-PD relevant brain regions found treatment induced alterations in glutathione redox homeostasis, and methylation potential, which are broadly important in neurodegeneration, inclusive of PD[30,31].

Since these initial animal studies, the scientific literature has been significantly strengthened. A more recent paper, conducted across a wider dose range and mimicking a 'living' exposure (daily, instead of 5-day workweek simulation) found a PD relevant phenotype (similar to the studies above, at an even lower dose (200 mg/kg/day, which would be expected to produce blood levels ≥7x exposed workers (well within risk assessment uncertainty factors)[32]. Notably, beyond the phenotypic markers already noted, this paper additionally found evidence of neuroinflammation, endolysosomal dysfunction, additional markers of protein aggregation, LRRK2 protein-protein interactions, and LRRK2 kinase activation prior to cell death, showing that not only does TCE produce a PD phenotype, but it likely does so through mechanistic pathways known to be involved in human PD. Furthermore, another follow-up paper showed that LRRK2 inhibition is protective in limiting PD relevant TCE neurotoxicity, again supporting the relevance of TCE in acting through a key PD mechanistic pathway[33]. Finally, there are systemic aspects (outside the brain) to PD, including changes to the microbiome. TCE exposure in rodents also produces microbiome changes consistent with human PD[34]. Translational value of these mechanistic, neuropathology, and neurobehavioral studies is deemed to be very high.

In a 2013 paper, a weakness in the TCE PD literature was stated: "it is not possible at this stage to give a clear view on the risk to humans as inhalation a major route of absorption for humans has not been examined"[12]. There had been some preliminary comparisons suggesting inhalation was more potent in in inducing motor phenotypes in rats[35]. Importantly, it is worth noting that there have been considerable recent advances in inhalation exposure modeling, including inhalation studies on TCE and PD endpoints of direct relevance to human PD risk. In this study, inhalation of TCE, the primary route of human exposure (vapor intrusion from water supplies) was administered at only 50-100 ppm (time weighed average). It is worth noting further that these levels are modestly above regulatory levels (Figure 7) and the difference between animal exposures and human regulatory limits is well below the typical ≥100x adjustment (10x adjustment for intraspecies variability and 10x adjustment for interspecies variability)[36]. Moreover, this study developed the doses based upon a low human equivalent dose (HED) of TCE in rodents, using allometric scaling to normalize TCE dose to body surface area between humans and rats or mice, where the HED for 50 ppm in rats and 100 ppm in mice was determined to be approximately equivalent to 8 ppm in a human. This study found yet again neuropathology highly relevant to human PD (in both rats and mice), including nigrostriatal dopamine neuron degeneration and alpha synuclein accumulation (Figure 8)[36]. The multiple other mechanistic studies noted above conducted in animal models further support the fact that TCE exposure can both reproduce key PD pathology, as well as activate pathogenic pathways. Collectively these studies both show that TCE induced PD relevant neurotoxicity is achieved in animal models across multiple rat strains and multiple routes of exposure[10,29,32-34,36]. These data further support a relationship that as of the 2017 ATSDR was deemed to be at least as likely as not for TCE as a PD risk factor.

Agency	Exposure	Regulated Level (TCE/PCE)	Regulation Details
American Conference of Government Industrial Hygienists	Air: Workplace	10/25 ppm	Threshold limit value-time-weighted-average (TLV-TWA)
		25/100 ppm	Threshold limit value-short-term-exposure-limit (TLV-STEL)
National Institute for Occupational Health and Safety	Air: Workplace	25/50 ppm	10-h time-weighted-average (TWA)
Occupational Safety and Health Administration	Air: Workplace	100/100 ppm	Permissible exposure limit (PEL) over 8-h workday
		300/300 ppm	Single exposure for up to 5 min in any 2 h
Environmental Protection Agency	Air: Environment	N/A	Regulation
	Drinking water	5/5 ppb	
Food and Drug Administration	Food: Bottled water	5/5 mcg/l	Maximum permissible level

Figure 7. US Governmental regulations for TCE and PCE (modified from[36]). Limited differences in regulatory levels show an expectation of similar overall toxicity thresholds. Moreover, animal studies[36] conducted near these levels likely have high translational value.

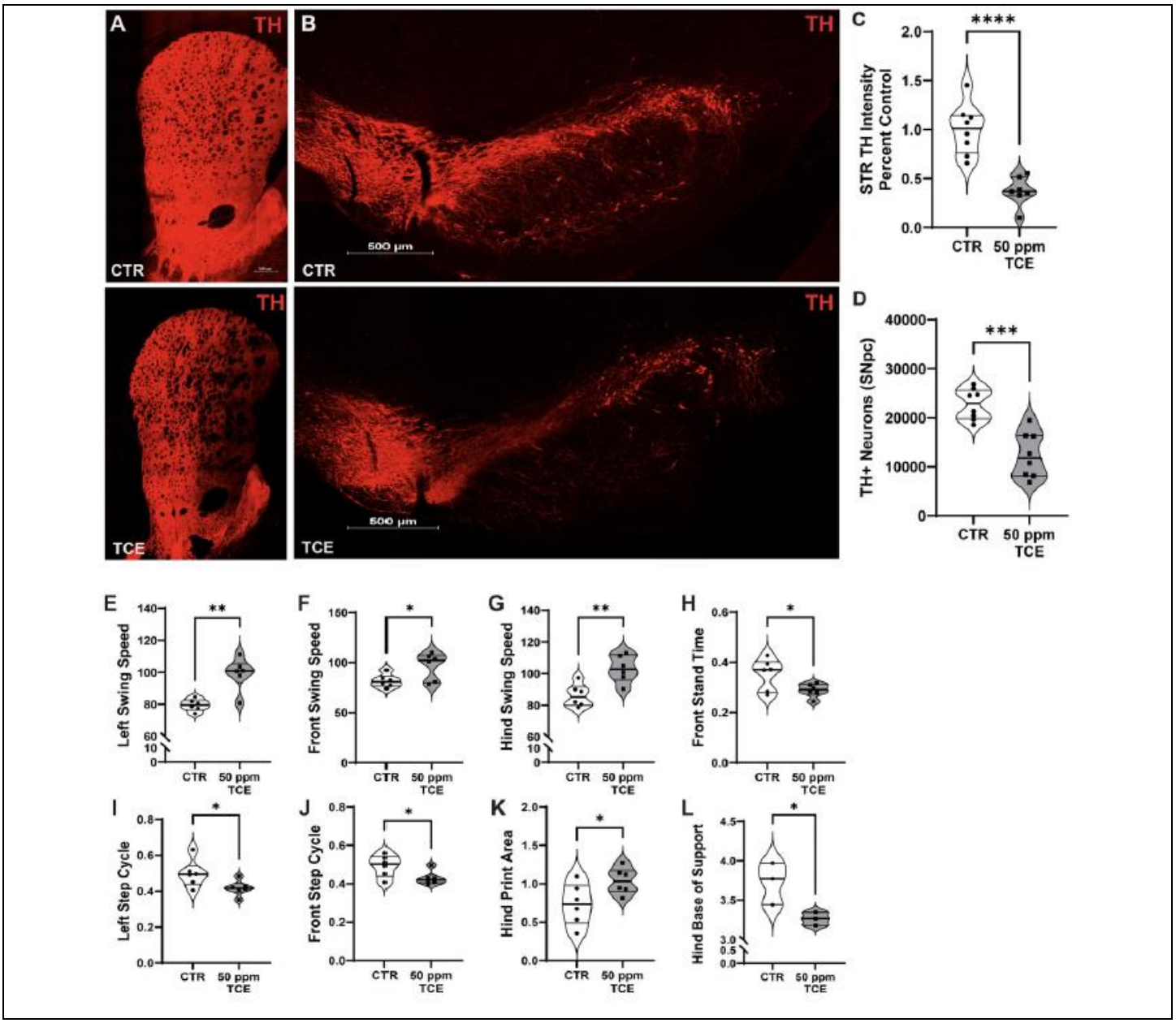


Figure 8. TCE inhalation (50 ppm for 8 weeks) in rats produced neuropathology and neurobehavioral deficits consistent with PD (modified from[36]). Compared to control (A), treated rats (B) exhibit significant loss of striatal dopamine terminals (left panels of A,B, quantified in C) and loss of dopamine neuron cell bodies in the substantia nigra (right panels of A,B, quantified in D). Moreover, several neurobehavioral measures (E-L) show motor deficits relevant to PD.

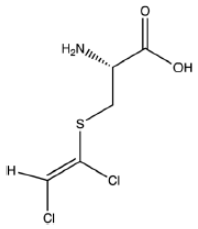
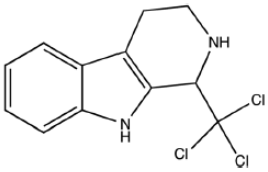
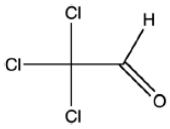
Hill consideration met: Analogy. Chemicals with highly similar structures are expected to produce similar biological effects, unless there is scientific evidence to suggest otherwise (structure activity relationships). The chemical structures of PCE and TCE are highly similar. Animal data has strengthened for TCE exposure (multiple exposure routes, multiple animal models) producing PD relevant neurochemical alterations, neuropathology and motor deficits. PCE to a reasonable degree of scientific certainty would produce similar findings in studied under similar conditions. Moreover, there is also a lack of scientific support for the alternate hypothesis that 1 additional chlorine atom and 1 less hydrogen atom (PCE vs TCE) would be protective in preventing PD relevant neurotoxicity. Thus, at present, the most plausible scientific conclusion is that PCE would be expected to produce a PD phenotype in animal models similar to TCE.

Metabolism and mitochondrial toxicity

A recent literature review strongly suggests that mitochondrial toxicity is a likely mechanism of action for TCE-induced dopaminergic neurotoxicity, a pathological hallmark in PD. Of note, TCE metabolism (shown in Figure 2) produces several putative mitochondrial toxicants[37] (Figure 8A). Here, it is important to reiterate that the loss of dopaminergic neurons in the substantia nigra results in many of the PD symptoms and that this subpopulation of neurons is especially sensitive to mitochondrial toxicity[31]. Thus, mitochondrial toxicity has been extensively studied as a primary mechanism of neurotoxic action in PD, so much so, that is it a published adverse outcome pathway (mitochondrial dysfunction is an upstream event in the adverse outcome pathway[38]).

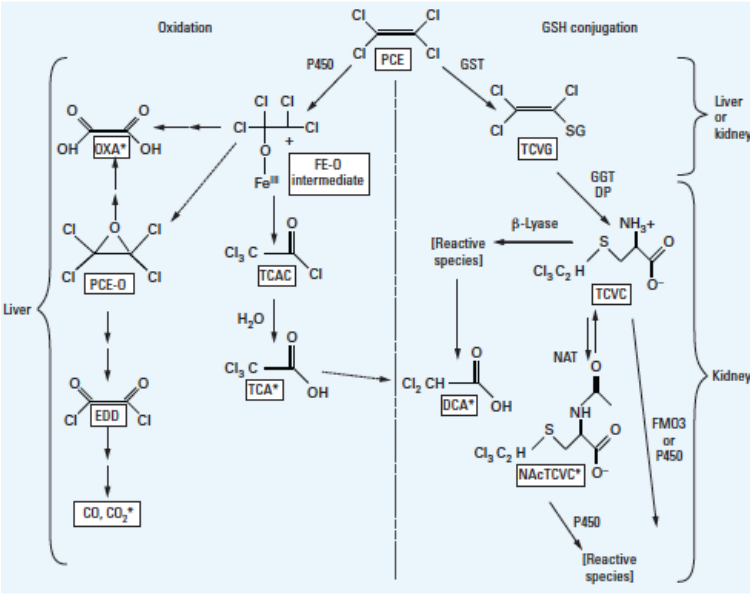
PCE produces several structurally similar metabolites (Figures 3 and 9B). While several TCE metabolites are putative mitochondrial toxicants, PCE metabolites have yet to be sufficiently tested for this mechanism. For both PCE and TCE, it is worth noting that there are considerable differences in metabolism across species, where humans may have different metabolic flux[39]. Both PCE and TCE may produce TaClo (Figure 9C), a mitochondrial toxicant (complex I inhibitor) with similar properties to MPP+, which is known to cause a PD like syndrome[4,40,41]. Here, it is worth noting that TaClo is >10x more potent than MPP+ in inhibiting mitochondrial complex I, a well-known PD mechanism for multiple PD toxicants[42]. While there is some controversy on whether TaClo is formed in mammals exposed to chloroethylenes, there are reports of detection in the blood of humans exposed to known chloroethylene metabolites[20]. Thus, multiple chloroethylenes potentially act through well-known PD mechanism with high potency. Both PCE and TCE can deplete cellular thiols (cysteine conjugation via GST shown in Figures 3 and 9B). Thiol depletion is an important mechanism in PD relevant pathogenesis[43].

A.

Structure	Metabolite name	Synonyms	Molecular formula
	*S*-(1,2-Dichlorovinyl)-L-cysteine	DCVC	$C_5H_7Cl_2NO_2S$
	1-Trichloromethyl-1,2,3,4-tetrahydro-beta-carboline	TaClo	$C_{12}H_{11}Cl_3N_2$
	Chloral	Trichloroacetaldehyde (TCAH), trichloroethanal	C_2HCl_3O

[a] Chemical structures were created using ChemOffice Professional (Version 18, CambridgeSoft).

B.



C.

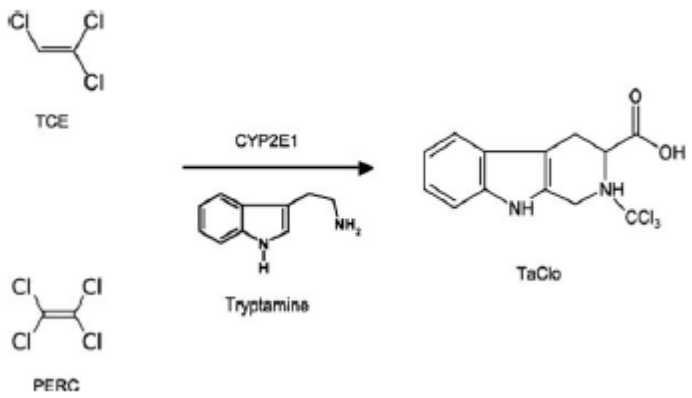


Figure 9. PCE and TCE may produce putative mitochondrial toxicants. A. Putative mitochondrial toxicants produced by TCE metabolism (modified from)[37] and B. Structurally similar metabolites produced by PCE metabolism (modified from[44]). It is also notable that both PCE and TCE may deplete cellular thiols through cysteine conjugation (A,B) through cysteine conjugation. C. Both PCE and TCE may produce TaClo, a mitochondrial toxicant (complex I inhibitor) with similar properties to MPP+, which is known to cause a PD like syndrome (modified from[4]).

Hill consideration met: Biological Plausibility: The above conclusions show PCE and TCE overlap with respect to PD extends beyond Analogy, where biological plausibility is supported by overlapping and metabolic pathway metabolites. Here, specifically, mitochondrial toxicants and mitochondrial toxicity (important primary PD pathogenic mechanism) are expected products. Again, serving as further evidence that PCE neurotoxicity to a degree of reasonable scientific certainty is expected to be highly similar to TCE. There is also a lack of scientific support for the alternate hypothesis that 1 additional chlorine atom and 1 less hydrogen atom (PCE vs TCE) would be protective in preventing PD relevant mechanisms of neurotoxicity.

4.2.6 PCE vs TCE: research focus and potency

Importantly, a recent review notes that TCE continues to receive significant research due to TCE remaining one of the most significant environmental contaminants in the US, and extensive research suggests TCE to be a PD risk factor[37]. The widespread TCE contamination of municipal waters systems noted in the paper cited above, and other papers likely explains the research focus being heavily weighted towards TCE vs PCE, which can be a more localized exposure. It is worth noting, that PCE levels local to specific exposures can be quite high, for example, near dry cleaners. Thus, while the weight of evidence for PCE in PD is currently less than TCE in PD, this is not due to scientifically documented lack of effect or lower magnitude of effect. To date, PCE simply has not been investigated as a PD risk factor as thoroughly or rigorously as TCE.

One study found that PCE had an even greater effect (equimolar comparison) on mitochondrial pathways than TCE. Here, the authors found that mitochondria-related transcriptional pathways are strongly affected, in dose-response manner, in both liver and kidney by PCE but not TCE[45]. Their results were supported by prior observations showing that PCE had a much stronger effect on uncoupling mitochondrial oxidative phosphorylation than TCE[46]. Such mitochondrial effects may be mediated through decreased electron flow at the susceptible portion in the mitochondrial inner membrane[47]. As stated above, mitochondrial toxicity is a critical mechanism of PD. Moreover, both PCE and TCE have been tested together for inhibition of mitochondrial respiration (Figure 10), where PCE (relative to TCE) was both more lipophilic and more potent[48].

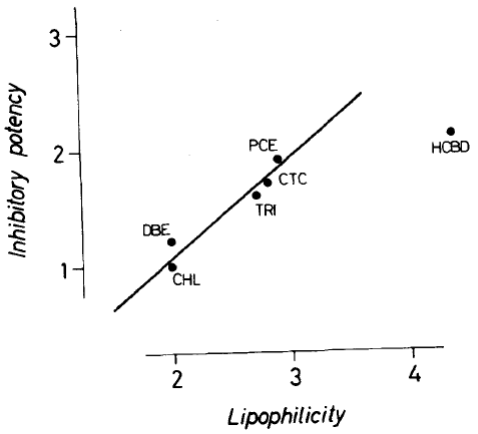


Figure 10. PCE is both more potent in inhibiting mitochondrial respiration and is more lipophilic (expected increased efficiency in crossing biological membranes) than TCE (here denoted as TRI) (modified from[48]).

Similarly, both PCE and TCE affect calcium signaling, a critical aspect of neurophysiology; PCE being far more potent than TCE in inhibiting whole cell calcium currents[49].

Another study that was a meta- and reanalysis of data on multiple solvents, including both PCE and TCE focused on neurobehavioral outcomes in rats. It found that tested solvents did not differ significantly in potency on any outcome measure when dose was expressed as molar brain concentration (Figure 11)[50] .

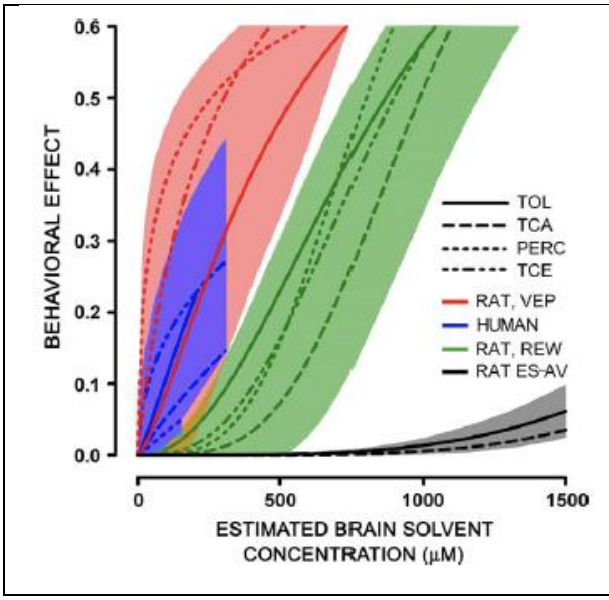


Figure 11. Estimated brain solvent concentration vs expected neurobehavioral effects. Broad markers of effects on neurological function include visual evoked potential (VEP), medium motivation involving food reward (REW), and avoidance of electrical shock (ES-AV) (modified from[50]).

Research has shown that PCE and TCE act similarly in living systems. However, it is notable that the additional chlorine molecule on PCE results in a more lipophilic molecule (see XLogP3 in Table 2), likely to result in higher bioaccumulation. Indeed, a study in both exposed humans and animals determined that indicating that PCE accumulates in the body at 3 to 4 times the rate of TCE under the same conditions of re-peated exposures and that the half-life for PCE = 144 hours and for TCE = 41 hours[51]. Indeed, in experimental studies PCE accumulates in mammalian brain (rats), with high accumulation compared to other organs (Figure 12A,B)[52]. Notably, in higher order species such as dogs, bioaccumulation is weighted even more heavily towards the brain (Figure 12C)[53]. Such differences in potency with respect to producing PD specific endpoints have yet to be adequately compared for PCE

and TCE. However, it is scientifically plausible that even at similar potency, a lower level of PCE exposure (vs TCE) could result in at least similar magnitude of neurotoxicity, given the experimentally determined higher bioconcentration and half-life of PCE vs TCE.

A.

Pharmacokinetic Parameters Estimates for a 2-hr Inhalation Exposure of Rats to 500 ppm PCE[a]

Tissue	Area under curve $\int_0^\infty$ (μg · min/ml)	Half-life (min)	C_{max} (μg/g)
Liver	31247	423	152.4
Kidney	25868	425	107.5
Fat	1493190	578	1536.3
Heart	23179	328	106.6
Lung	18596	406	94.6
Muscle	24458	335	87.3
Brain	32975	455	173.9
Blood	8464	322	44.9

[a] Each value represents the value for tissues of five rats pooled at each of 16 time points, ranging from 15 min after the initiation of exposure to 72 hr postexposure.

B.

Partition coefficients	
Blood:air	19.8
Fat:blood	152.5
Lung:blood	2.47
Liver:blood	5.25
Muscle:blood	2.98
Brain:blood	4.37
Heart:blood	2.68
Kidney:blood	4.45
Rest of body:blood	2.98

C.

PHARMACOKINETIC PARAMETERS IN THE DOG FOLLOWING ORAL ADMINISTRATION OF 10 mg PCE/kg BODY WT[a]

Tissue	Area under curve (μg · min/ml)	Half-life (min)	C_{max} (μg/g)	T_{max} (min)
Liver	1,851 ± 757	2448 ± 922	6.3 ± 0.6	60
Kidney	1,606 ± 621	1572 ± 262	4.9 ± 0.4	60
Fat	55,838 ± 9,640	494 ± 77	42.8 ± 3.5	720
Heart	1,849 ± 620	1775 ± 464	5.7 ± 1.6	60
Lung	1,001 ± 681	2289 ± 863	2.4 ± 0.5	60
Muscle	1,907 ± 1,564	1625 ± 886	3.1 ± 0.1	60
Brain	3,238 ± 1,153	4641 ± 1547	11.4 ± 8.2	60
Blood	782 ± 146	865 ± 385	1.5 ± 0.3	60

[a] Each value represents the mean ± SD for three dogs at six time points ranging from 1 to 72 hr.

Figure 12. Brain accumulation and partition coefficients for PCE following a single exposure (modified from[52]). A, B. Rat brain has the highest area under the curve of organs (only fat is higher)

and a partition coefficient near liver and kidney indicating high brain entry. C. The half-life is even longer in higher order species such as dog (modified from[53]).

Overall rationale for drawing PCE neurotoxicological conclusions based upon TCE and other chlorinated solvent data.

Given the collective, chemical property similarities, metabolic profiles, and known biological overlap, the neurotoxicity of PCE and TCE are highly likely to be similar. TCE has received more research given more widespread contamination as noted above. An expert review by the National Center for Environmental Assessment, Office of Research and Development, United States Environmental Protection Agency reached similar conclusions, stating the following with respect to PCE, TCE and other chlorinated solvents, especially with respect to neurochemical receptor interactions and adverse neurological outcomes[54]:

["Commonalities of neurobehavioral and neurophysiological changes for the chlorinated solvents in in vivo studies suggest that there is a common mechanism(s) of action in producing resultant neurotoxicological consequences."

"Collectively, TCE, PERC, and DCM have been reported to interact directly with several different classes of neuronal receptors by generally inhibiting excitatory receptors/channels and potentiating the function of inhibitory receptors/channels. Given this mechanistic information and available studies for TCE, DCM, and PERC, we provide hypotheses on primary targets (e.g. ion channel targets) that appear to be most influential in producing the resultant neurological effects."]

Since this 2011 review, much has been discovered on the relationship between TCE and PD. Here again, given the rather consistent overlapping biological effects amongst chlorinated solvents, overlapping PD relevant neurotoxicity is also highly likely.

While the ability to produce similar adverse neurological outcomes is highly likely, potency comparisons cannot yet be made with respect to PCE induced PD relevant neurotoxicity. Given known half-life differences and potency in engaging biological mechanisms noted above, it is reasonable to conclude, and indeed likely, that PCE would be of similar or greater neurotoxic potency.

Hill consideration met: Biological Plausibility: The above conclusions show PCE and TCE overlap with respect to PD extends beyond Analogy, where biological plausibility is supported by multiple overlapping and PD relevant mechanisms. Notably, PCE may be more potent in some measures of mitochondrial toxicity (a known human PD and PD model neurotoxicological cascade) and alterations in calcium signaling.

Hill consideration met: Experiment: The above conclusions show that in a laboratory setting, PCE induces PD relevant mechanisms. Notably, PCE may be more potent in some measures of mitochondrial toxicity (a known human PD and PD model neurotoxicological cascade) and alterations in calcium signaling.

Hill consideration met: Analogy. Chemicals with highly similar structures are expected to produce similar biological effects, unless there is scientific evidence to suggest otherwise (structure activity relationships). The chemical structures of PCE and TCE are highly similar. In contrast, there is a lack of scientific support for the alternate hypothesis that 1 additional chlorine atom and 1 less hydrogen atom (PCE vs TCE) would be protective in preventing PD relevant neurotoxicity.

4.2.7 PCE specific neurotoxicological data. Despite the lower devoted research focus on PCE vs TCE, there are some compelling data that PCE is also a neurotoxicant and similar to TCE, is directly relevant to PD. Early studies in rats exposed to 300-600 ppm for up to 12 weeks found broad markers of neurotoxicity, such as decreases in brain growth, total protein, and DNA[55]. Similarly, in another study, rats exposed up to 800 ppm for up to 13 weeks with a limited endpoint scope (evoked signaling) found evidence of flash evoked potential in the visual cortex[56]. It is important to note that such studies were done in an attempt to mimic a workday exposure scheme, whereas a notable recent TCE inhalation exposure utilized a novel, passive whole body inhalation chamber[36]. An additional set of studies with rather narrow scopes in gerbils showed loss of specific phospholipids, neurochemical alterations, and effects on glial cells in brains after chronic treatment[57-59]. Some of these studies examined both PCE and TCE, where, in general findings were in agreement, though some similar studies found PCE to be more potent[60]. The overall conclusion was that PCE is a potent neurotoxicant[59,61]. Importantly, these studies did not focus on PD relevant brain regions.

Moreover, the U.S. Environmental Protection Agency (EPA) completed a toxicological review of PCE in February 2012 in support of the Integrated Risk Information System (IRIS) and a review of key findings and scientific issues regarding the human health effects of PCE described in the U.S. EPA's Toxicological Review of PCE found the following[62]:

> ["Neurotoxicity was identified as a sensitive noncancer health effect, occurring at low exposures: a conclusion supported by multiple studies. Evidence was integrated from human, experimental animal, and mechanistic data sets in assessing adverse health effects of PCE."]
>
> The source material the EPA based its conclusions on to make the statement above relied upon the same or similar research and data that I reviewed for this report. In fact, throughout this report, I note significant recent human and laboratory studies that strengthen the statement above.

In a recent basic science study, PCE was found to induce reactive oxygen species (ROS) at a similar magnitude as TCE, where ROS are a well-known PD mechanism[33]. This paper also found that that both PCE and TCE toxicity were dependent on LRRK2, a kinase that has a key pathogenic role in both sporadic and familial PD (Figure 12). These findings specifically show that wild-type LRRK2 (important in sporadic PD cases) has an important role in both PCE and

TCE toxicity at similar magnitudes. In TCE treated rats, LRRK2 activation also occurs (inhibition is also protective in animal models), where based on all available evidence, a similar effect would be expected for PCE[32,33]. With respect to the role of wild-type LRRK2, well established PD risk factors such as paraquat and rotenone were also included in this study, where similar effects were found. This study suggests again, the PCE interacts with a key PD relevant pathogenic pathway in a highly similar fashion to TCE (Figure 13).

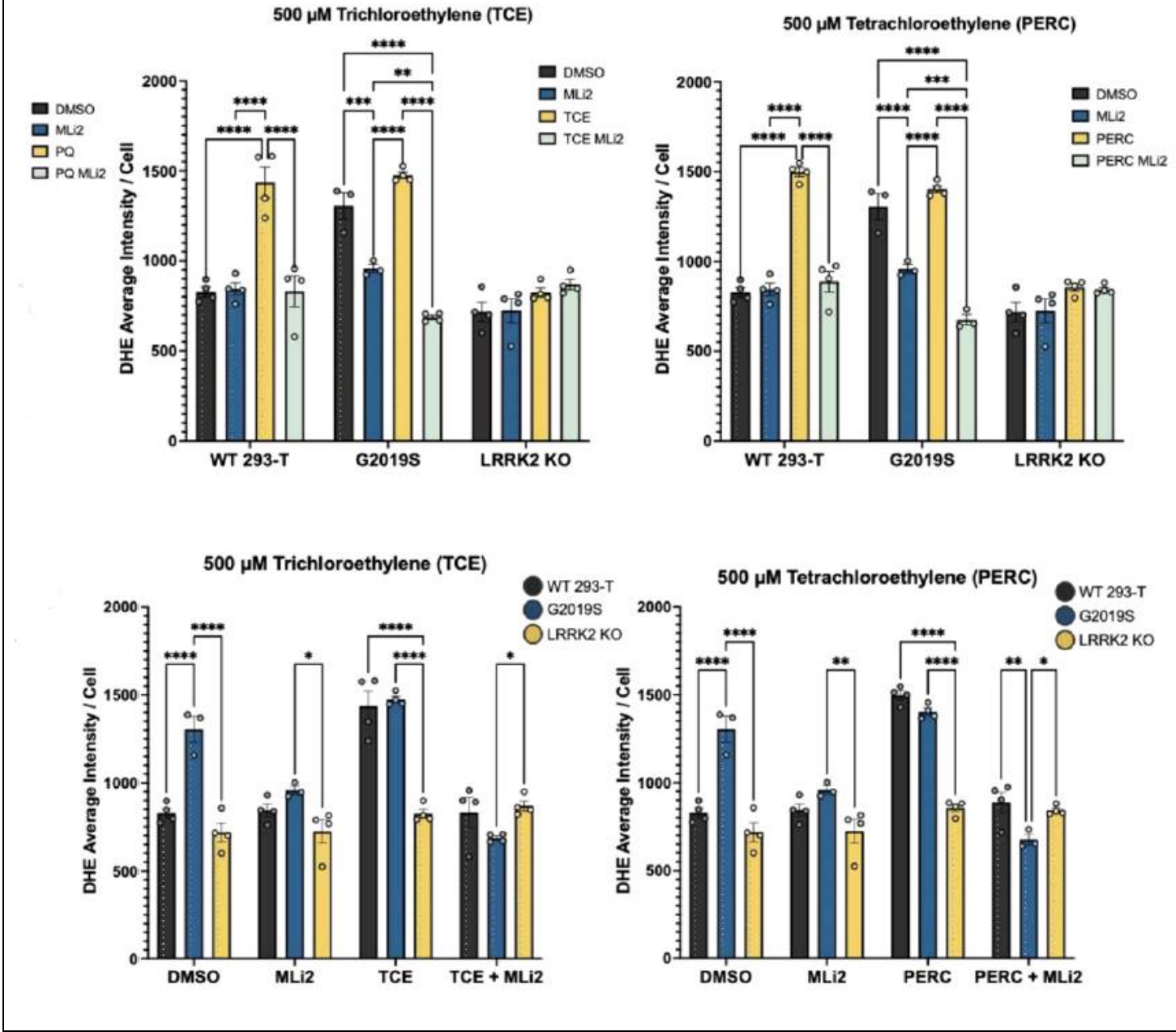


Figure 13. Cellular treatment of both PCE and TCE produces oxidative stress that is LRRK2 dependent. Equimolar PCE and TCE treatment in wild-type (WT) or in a PD causing LRRK2 mutation (G2019S) produces a similar magnitude of reactive oxygen species (assessed by DHE). The LRRK2 inhibitor is protective and a lack of effect on LRRK2 knockout (KO) cells further supports involvement of LRRK2 (modified from[33]). Of note, the effects and mechanistic inferences related to the importance of LRRK2 kinase activity were similar to well established PD risk factors, such as

paraquat and rotenone, also included in this study (data not shown). These findings show that both PCE and TCE toxicity is mediated through a well-known PD mechanistic pathway.

Hill consideration met: Biological Plausibility: PCE can toxicity produces ROS, which is LRRK2 dependent, a key PD mechanistic pathway.

Hill consideration met: Experiment: PCE can activate key PD relevant pathogenetic pathways, including ROS and LRRK2 in a laboratory experiments.

Hill consideration met: Analogy. PCE can activate key PD relevant pathogenetic pathways, including ROS and LRRK2. Moreover, it does so in a highly similar fashion to TCE and at similar potency. Highly relevant LRRK2 inhibition was protective in a TCE animal model, further supporting PD relevance. Given the chemical and mechanistic overlap, by analogy, PCE induced neurotoxicity in animal models would also be expected to be LRRK2 dependent. There is also a lack of scientific support for the alternate hypothesis that 1 additional chlorine atom and 1 less hydrogen atom (PCE vs TCE) would be protective in preventing PD relevant mechanisms of neurotoxicity.

4.2.8 Hill consideration met directly for PCE and PD. Table 5 summarizes Hill considerations met in this report. Specific applications are described in sections 4.1-4.2.6 from individual research foci. There are also additional considerations that are collectively met:

Hill consideration met: Coherence: Epidemiological and toxicological data for PCE and PD are coherent for potential risk (at least as likely as not) plus induction of PD relevant mechanisms.

4.2.9 Table 5. Bradford Hill consideration met directly for PCE and PD. See sections 4.1-4.2.7 for specific data supporting Hill consideration application and detailed commentary.

Consideration	Hill consideration met/not met
Strength of association	Met: Multiple epidemiology studies suggest a link between PCE and PD, implicating PCE at minimum, as likely as not a PD risk factor.
Consistency	Met: Multiple epidemiology studies suggest a link between PCE and PD, implicating PCE at minimum, as likely as not a PD risk factor. These multiple studies are from different human cohorts.
Specificity	Not directly met: This consideration is virtually impossible to meet for an environmental PD risk factor. Other known PD risk factors (i.e. rotenone and paraquat) can cause other non-neurologic

	health issues and there are multiple known risk factors for PD..
Temporality	Met: Epidemiological studies show that PCE exposure precedes elevated PD risk.
Dose-response	Not directly met: While, by analogy and in view of the data provided by the research thus far discussed above, a dose response is expected, additional data are required. Broad measures of neurotoxicity in animal studies show dose response. PD specific endpoint studies are needed across a wide range similar to what has been conducted with TCE.
Biological plausibility	Met: PCE and TCE have similar metabolites, extending the weight of evidence beyond simple analogy to biological plausibility when considering the role of chloroethylenes in PD relevant neurotoxicity. The commonalities extend to putative mitochondrial toxicants, an important exposure in PD. A number of other key biochemical mechanisms are engaged specifically by PCE.
Coherence	Met: Epidemiological and toxicological data are coherent for potential risk and induction of PD relevant mechanisms.
Experiment	In laboratory studies, with controlled exposures, PCE can activate key PD relevant pathogenetic pathways, including ROS, mitochondrial toxicity, and LRRK2 in a laboratory experiments. Each of these pathways are relevant to PD mechanisms.
Analogy	PCE and TCE are both chloroethylenes with highly similar chemical structures. Moreover, PD risk has been studied for both individual chloroethylenes, for chlorinated solvents, and solvents in general. Overall, the weight of evidence suggests that PCE induced PD relevant neurotoxicity would be analogous to TCE induced PD relevant neurotoxicity.

5. SUMMARY OF SCIENTIFIC CONCLUSIONS

5.1 Tetrachloroethylene (PCE) is at least as likely as not a cause of Parkinson's disease (PD) based upon the following scientific evidence:

BASED ON THE FOREGOING ANALYSIS, AND BASED UPON MY EDUCATION, TRAINING AND EXPERIENCE, IT IS MY OPINION TO A REASONSABLE DEGREE OF SCIENTIFIC CERTAINTY THAT TETRACHLOROETHYLENE (PCE) IS AT LEAST AS LIKELY A CAUSE OF PARKINSON'S DISEASE (PD). MY ANANLYSIS INLCUDES THE FOLLOWING:

5.1.1 Structural similarity and structural activity relationships to trichloroethylene (TCE), which is a known PD risk factor based upon collective epidemiological and neurotoxicological data. There is also a lack of scientific support for the alternate hypothesis that 1 additional chlorine atom and 1 less hydrogen atom (PCE vs TCE) would be protective in preventing PD risk.

5.1.2 Epidemiology of chlorinated ethylene solvents (inclusive of PCE) and PD.

5.1.3 Direct experimental evidence that PCE toxicity is mediated by critical PD pathogenic pathways.

5.1.4 Hill considerations met directly for PCE, or by scientifically relevant analogy for TCE.

I AM BEING COMPENSATED $500 AN HOUR FOR MY TIME DEVOTED TO INVESTIGATING THE RELEVANT ISSUES AND DRAFTING THIS REPORT.

12/08/2024

Jason Cannon, Ph.D.

6. REFERENCES (cited directly in this report, or reviewed)

1. ATSDR. ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases. Agency for Toxic Substances and Disease Registry, Department of Health and Human Services 2017.

2. Boniface S, Scannell JW, Marlow S. Evidence for the effectiveness of minimum pricing of alcohol: a systematic review and assessment using the Bradford Hill criteria for causality. BMJ open. 2017;7(5):e013497.

3. Cox Jr LA. Modernizing the Bradford Hill criteria for assessing causal relationships in observational data. Critical reviews in toxicology. 2018;48(8):682-712.

4. Goldman SM, Quinlan PJ, Ross GW, Marras C, Meng C, Bhudhikanok GS, Comyns K, Korell M, Chade AR, Kasten M, Priestley B, Chou KL, Fernandez HH, Cambi F, Langston JW, Tanner CM. Solvent exposures and Parkinson disease risk in twins. Annals of neurology. 2012;71(6):776-84. doi: 10.1002/ana.22629. PubMed PMID: 22083847; PMCID: 3366287.

5. Pezzoli G, Cereda E. Exposure to pesticides or solvents and risk of Parkinson disease. Neurology. 2013;80(22):2035-41. Epub 2013/05/29. doi: 10.1212/WNL.0b013e318294b3c8. PubMed PMID: 23713084.

6. Brouwer M, Koeman T, van den Brandt PA, Kromhout H, Schouten LJ, Peters S, Huss A, Vermeulen R. Occupational exposures and Parkinson's disease mortality in a prospective Dutch cohort. Occupational and environmental medicine. 2015;72(6):448-55. Epub 2015/02/26. doi: 10.1136/oemed-2014-102209. PubMed PMID: 25713156.

7. van der Mark M, Vermeulen R, Nijssen PC, Mulleners WM, Sas AM, van Laar T, Huss A, Kromhout H. Occupational exposure to solvents, metals and welding fumes and risk of Parkinson's disease. Parkinsonism & related disorders. 2015;21(6):635-9. Epub 2015/04/24. doi: 10.1016/j.parkreldis.2015.03.025. PubMed PMID: 25903042.

8. Zaheer F, Slevin JT. Trichloroethylene and Parkinson disease. Neurol Clin. 2011;29(3):657-65. Epub 2011/08/02. doi: 10.1016/j.ncl.2011.05.001. PubMed PMID: 21803216.

9. Bove FJ, Ruckart PZ, Maslia M, Larson TC. Mortality study of civilian employees exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study. Environ Health. 2014;13:68. Epub 2014/08/15. doi: 10.1186/1476-069x-13-68. PubMed PMID: 25115749; PMCID: PMC4237831.

10. Gash DM, Rutland K, Hudson NL, Sullivan PG, Bing G, Cass WA, Pandya JD, Liu M, Choi DY, Hunter RL, Gerhardt GA, Smith CD, Slevin JT, Prince TS. Trichloroethylene: Parkinsonism and complex 1 mitochondrial neurotoxicity. Annals of neurology. 2008;63(2):184-92. Epub 2007/12/25. doi: 10.1002/ana.21288. PubMed PMID: 18157908.

11. Populations BotHoS, Legislation CotRoCGftCoHCIbtCL. Review of VA clinical guidance for the health conditions identified by the Camp Lejeune legislation: National Academies Press; 2015.

12. Lock EA, Zhang J, Checkoway H. Solvents and Parkinson disease: a systematic review of toxicological and epidemiological evidence. Toxicology and applied pharmacology. 2013;266(3):345-55. Epub 2012/12/12. doi: 10.1016/j.taap.2012.11.016. PubMed PMID: 23220449; PMCID: PMC3621032.

13. Banerjee A, Kar S, Roy K, Patlewicz G, Charest N, Benfenati E, Cronin MTD. Molecular similarity in chemical informatics and predictive toxicity modeling: from quantitative read-across (q-RA) to quantitative read-across structure-activity relationship (q-RASAR) with the application of machine learning. Crit Rev Toxicol. 2024;54(9):659-84. Epub 2024/09/03. doi: 10.1080/10408444.2024.2386260. PubMed PMID: 39225123.

14. Reynolds ES, Moslen MT. Damage to hepatic cellular membranes by chlorinated olefins with emphasis on synergism and antagonism. Environmental health perspectives. 1977;21:137-47. Epub 1977/12/01. doi: 10.1289/ehp.7721137. PubMed PMID: 612438; PMCID: PMC1475335.

15. Dimitrov S, Dimitrova N, Walker J, Veith G, Mekenyan O. Predicting bioconcentration factors of highly hydrophobic chemicals. Effects of molecular size. Pure and Applied Chemistry. 2002;74(10):1823-30.

16. Meylan WM, Howard PH, Boethling RS, Aronson D, Printup H, Gouchie S. Improved method for estimating bioconcentration/bioaccumulation factor from octanol/water partition coefficient. Environmental Toxicology and Chemistry: An International Journal. 1999;18(4):664-72.

17. Dimitrov S, Mekenyan O, Walker J. Non-linear modeling of bioconcentration using partition coefficients for narcotic chemicals. SAR and QSAR in Environmental Research. 2002;13(1):177-84.

18. PubChem Compound Summary for CID 31373, Tetrachloroethylene.: National Center for Biotechnology Information (2024); 2024 [cited 2024 Retrieved November 23, 2024]. Available from: https://pubchem.ncbi.nlm.nih.gov/compound/Tetrachloroethylene.

19. PubChem Compound Summary for CID 6575, Trichloroethylene. : National Center for Biotechnology Information (2024); 2024 [cited 2024 Retrieved November 23, 2024]. Available from: https://pubchem.ncbi.nlm.nih.gov/compound/Trichloroethylene.

20. Bringmann G, God R, Fähr S, Feineis D, Fornadi K, Fornadi F. Identification of the dopaminergic neurotoxin 1-trichloromethyl-1,2, 3,4-tetrahydro-beta-carboline in human blood after intake of the hypnotic chloral hydrate. Analytical biochemistry. 1999;270(1):167-75. Epub 1999/05/18. doi: 10.1006/abio.1999.4088. PubMed PMID: 10328779.

21. Klaassan CD. Casarett & Doull's toxicology: the basic science of poisons. 8th ed. New York: McGraw Hill Medical; 2013.

22. Bertolini M, Zecchin S, Cavalca L. Sequential Anaerobic/Aerobic Microbial Transformation of Chlorinated Ethenes: Use of Sustainable Approaches for Aquifer Decontamination. Water. 2023;15(7):1406. PubMed PMID: doi:10.3390/w15071406.

23. Cichocki JA, Guyton KZ, Guha N, Chiu WA, Rusyn I, Lash LH. Target Organ Metabolism, Toxicity, and Mechanisms of Trichloroethylene and Perchloroethylene: Key Similarities, Differences, and Data Gaps. The Journal of pharmacology and experimental therapeutics. 2016;359(1):110-23. Epub 2016/08/12. doi: 10.1124/jpet.116.232629. PubMed PMID: 27511820; PMCID: PMC5034707.

24. Kuban W, Daniel WA. Cytochrome P450 expression and regulation in the brain. Drug metabolism reviews. 2021;53(1):1-29. doi: 10.1080/03602532.2020.1858856.

25. Goldman SM, Weaver FM, Gonzalez B, Stroupe KT, Cao L, Colletta K, Brown EG, Tanner CM. Parkinson's disease progression and exposure to contaminated water at camp lejeune. Movement Disorders. 2024.

26. Goldman SM, Weaver FM, Stroupe KT, Cao L, Gonzalez B, Colletta K, Brown EG, Tanner CM. Risk of Parkinson Disease Among Service Members at Marine Corps Base Camp Lejeune. JAMA neurology. 2023;80(7):673-81. Epub 2023/05/15. doi: 10.1001/jamaneurol.2023.1168. PubMed PMID: 37184848; PMCID: PMC10186205 Edward Hines, Jr VA Hospital during the conduct of the study and outside the submitted work. Dr Brown reported receiving grants from the Michael J. Fox Foundation and the National Institute on Aging and personal fees from Gateway Consulting, LLC, outside the submitted work. Dr Tanner reported receiving personal fees from Lundbeck Pharma, CNS Ratings, Adamas, Cadent, and Evidera; serving on advisory boards for Kyowa Kirin, Acorda, Australia Parkinson's Mission; serving on a clinical trial steering committee for Jazz Pharmaceuticals/Cavion; and receiving grants from the National Institutes of Health, Biogen Idec, Parkinson Foundation, Michael J. Fox Foundation, Department of Defense Parkinson's Research Program, Roche, Genentech, BioElectron, and Gateway Institute for Brain Research, LLC, outside the submitted work. No other disclosures were reported.

27. Wadman M. Solvent exposure strongly linked to Parkinson's. Science. 2023;380(6646):683. Epub 2023/05/18. doi: 10.1126/science.adi7660. PubMed PMID: 37200442.

28. Guehl D, Bezard E, Dovero S, Boraud T, Bioulac B, Gross C. Trichloroethylene and parkinsonism: a human and experimental observation. European journal of neurology : the official journal of the European Federation of Neurological Societies. 1999;6(5):609-11. Epub 1999/08/24. doi: 10.1046/j.1468-1331.1999.650609.x. PubMed PMID: 10457397.

29. Liu M, Choi DY, Hunter RL, Pandya JD, Cass WA, Sullivan PG, Kim HC, Gash DM, Bing G. Trichloroethylene induces dopaminergic neurodegeneration in Fisher 344 rats. Journal of neurochemistry. 2010;112(3):773-83. Epub 2009/11/20. doi: 10.1111/j.1471-4159.2009.06497.x. PubMed PMID: 19922440.

30. Blossom SJ, Melnyk S, Cooney CA, Gilbert KM, James SJ. Postnatal exposure to trichloroethylene alters glutathione redox homeostasis, methylation potential, and neurotrophin

expression in the mouse hippocampus. Neurotoxicology. 2012;33(6):1518-27. Epub 2012/03/17. doi: 10.1016/j.neuro.2012.02.017. PubMed PMID: 22421312; PMCID: PMC3383874.

31. Currim F, Tanwar R, Brown-Leung JM, Paranjape N, Liu J, Sanders LH, Doorn JA, Cannon JR. Selective dopaminergic neurotoxicity modulated by inherent cell-type specific neurobiology. Neurotoxicology. 2024;103:266-87. Epub 2024/07/05. doi: 10.1016/j.neuro.2024.06.016. PubMed PMID: 38964509; PMCID: PMC11288778.

32. De Miranda BR, Castro SL, Rocha EM, Bodle CR, Johnson KE, Greenamyre JT. The industrial solvent trichloroethylene induces LRRK2 kinase activity and dopaminergic neurodegeneration in a rat model of Parkinson's disease. Neurobiol Dis. 2021;153:105312. Epub 2021/02/27. doi: 10.1016/j.nbd.2021.105312. PubMed PMID: 33636387; PMCID: PMC8026730.

33. Ilieva NM, Hoffman EK, Ghalib MA, Greenamyre JT, De Miranda BR. LRRK2 kinase inhibition protects against Parkinson's disease-associated environmental toxicants. Neurobiol Dis. 2024;196:106522. Epub 2024/05/06. doi: 10.1016/j.nbd.2024.106522. PubMed PMID: 38705492; PMCID: PMC11332574.

34. Ilieva NM, Wallen ZD, De Miranda BR. Oral ingestion of the environmental toxicant trichloroethylene in rats induces alterations in the gut microbiome: Relevance to idiopathic Parkinson's disease. Toxicology and applied pharmacology. 2022;451:116176. Epub 2022/08/02. doi: 10.1016/j.taap.2022.116176. PubMed PMID: 35914559; PMCID: PMC10581445.

35. Waseem M, Ali M, Dogra S, Dutta KK, Kaw JL. Toxicity of trichloroethylene following inhalation and drinking contaminated water. Journal of applied toxicology : JAT. 2001;21(6):441-4. Epub 2001/12/18. doi: 10.1002/jat.778. PubMed PMID: 11746189.

36. Adamson A, Ilieva N, Stone WJ, De Miranda BR. Low-dose inhalation exposure to trichloroethylene induces dopaminergic neurodegeneration in rodents. Toxicological sciences : an official journal of the Society of Toxicology. 2023;196(2):218-28. Epub 2023/09/05. doi: 10.1093/toxsci/kfad090. PubMed PMID: 37669148; PMCID: PMC11491929.

37. De Miranda BR, Greenamyre JT. Trichloroethylene, a ubiquitous environmental contaminant in the risk for Parkinson's disease. Environ Sci Process Impacts. 2020;22(3):543-54. Epub 2020/01/31. doi: 10.1039/c9em00578a. PubMed PMID: 31996877; PMCID: PMC7941732.

38. Bal-Price A, Leist M, Schidknecht S, Tschudi-Monnet F, Terron A. Inhibition of the mitochondrial complex I of nigro-striatal neurons leads to parkinsonian motor deficits: Society for Advancement of AOPs; 2023 [cited 2024 11/21/2024]. Available from: https://aopwiki.org/aops/3.

39. Valdiviezo A, Brown GE, Michell AR, Trinconi CM, Bodke VV, Khetani SR, Luo YS, Chiu WA, Rusyn I. Reanalysis of Trichloroethylene and Tetrachloroethylene Metabolism to Glutathione Conjugates Using Human, Rat, and Mouse Liver in Vitro Models to Improve Precision in Risk Characterization. Environmental health perspectives. 2022;130(11):117009. Epub 2022/11/30. doi: 10.1289/ehp12006. PubMed PMID: 36445294; PMCID: PMC9707501.

40. Keane PC, Hanson PS, Patterson L, Blain PG, Hepplewhite P, Khundakar AA, Judge SJ, Kahle PJ, LeBeau FEN, Morris CM. Trichloroethylene and its metabolite TaClo lead to degeneration of substantia nigra dopaminergic neurones: Effects in wild type and human A30P mutant α-synuclein mice. Neuroscience letters. 2019;711:134437. Epub 2019/08/20. doi: 10.1016/j.neulet.2019.134437. PubMed PMID: 31422098; PMCID: PMC6892271.

41. Liu M, Shin EJ, Dang DK, Jin CH, Lee PH, Jeong JH, Park SJ, Kim YS, Xing B, Xin T, Bing G, Kim HC. Trichloroethylene and Parkinson's Disease: Risk Assessment. Molecular neurobiology. 2018;55(7):6201-14. Epub 2017/12/23. doi: 10.1007/s12035-017-0830-x. PubMed PMID: 29270919.

42. Janetzky B, Gille G, Abdel-Mohsen M, God R, Rausch WD, Bringmann G, Reichmann H. Effect of highly halogenated β-carbolines on dopaminergic cells in culture and on mitochondrial respiration. Drug Development Research. 1999;46(1):51-6.

43. Garcia-Garcia A, Zavala-Flores L, Rodriguez-Rocha H, Franco R. Thiol-redox signaling, dopaminergic cell death, and Parkinson's disease. Antioxidants & redox signaling. 2012;17(12):1764-84. Epub 2012/03/01. doi: 10.1089/ars.2011.4501. PubMed PMID: 22369136; PMCID: PMC3474187.

44. Guyton Kathryn Z, Hogan Karen A, Scott Cheryl S, Cooper Glinda S, Bale Ambuja S, Kopylev L, Barone S, Makris Susan L, Glenn B, Subramaniam Ravi P, Gwinn Maureen R, Dzubow Rebecca C, Chiu Weihsueh A. Human Health Effects of Tetrachloroethylene: Key Findings and Scientific Issues. Environmental health perspectives. 2014;122(4):325-34. doi: 10.1289/ehp.1307359.

45. Zhou YH, Cichocki JA, Soldatow VY, Scholl EH, Gallins PJ, Jima D, Yoo HS, Chiu WA, Wright FA, Rusyn I. Editor's Highlight: Comparative Dose-Response Analysis of Liver and Kidney Transcriptomic Effects of Trichloroethylene and Tetrachloroethylene in B6C3F1 Mouse. Toxicological sciences : an official journal of the Society of Toxicology. 2017;160(1):95-110. Epub 2017/10/04. doi: 10.1093/toxsci/kfx165. PubMed PMID: 28973375; PMCID: PMC5837274.

46. OGATA M, HASEGAWA T. Effects of chlorinated aliphatic hydrocarbons on mitochondrial oxidative phosphorylation in the rat with reference to the effects of chlorinated aromatic hydrocarbons. Industrial health. 1981;19(2):71-5.

47. Miyazaki Y, Takano T. Impairment of mitochondrial electron transport by tetrachloroethylene. Toxicology letters. 1983;18(1-2):163-6. Epub 1983/08/01. doi: 10.1016/0378-4274(83)90088-7. PubMed PMID: 6623542.

48. Fiorucci L, Monti A, Testai E, Ade P, Vittozzi L. In vitro effects of polyhalogenated hydrocarbons on liver mitochondria respiration and microsomal cytochrome P-450. Drug Chem Toxicol. 1988;11(4):387-403. Epub 1988/01/01. doi: 10.3109/01480548809018110. PubMed PMID: 3243187.

49. Shafer TJ, Bushnell PJ, Benignus VA, Woodward JJ. Perturbation of voltage-sensitive Ca2+ channel function by volatile organic solvents. The Journal of pharmacology and experimental

therapeutics. 2005;315(3):1109-18. Epub 2005/08/20. doi: 10.1124/jpet.105.090027. PubMed PMID: 16109744.

50. Benignus VA, Bushnell PJ, Boyes WK, Eklund C, Kenyon EM. Neurobehavioral effects of acute exposure to four solvents: meta-analyses. Toxicological sciences : an official journal of the Society of Toxicology. 2009;109(2):296-305. Epub 2009/04/03. doi: 10.1093/toxsci/kfp063. PubMed PMID: 19339666.

51. Ikeda M, Imamura T. Biological half-life of trichloroethylene and tetrachloroethylene in human subjects. Internationales Archiv für Arbeitsmedizin. 1973;31(3):209-24. doi: 10.1007/BF00539241.

52. Dallas CE, Chen XM, O'Barr K, Muralidhara S, Varkonyi P, Bruckner JV. Development of a physiologically based pharmacokinetic model for perchloroethylene using tissue concentration-time data. Toxicology and applied pharmacology. 1994;128(1):50-9. Epub 1994/09/01. doi: 10.1006/taap.1994.1179. PubMed PMID: 8079354.

53. Dallas CE, Chen XM, Muralidhara S, Varkonyi P, Tackett RL, Bruckner JV. Use of tissue disposition data from rats and dogs to determine species differences in input parameters for a physiological model for perchloroethylene. Environ Res. 1994;67(1):54-67. Epub 1994/10/01. doi: 10.1006/enrs.1994.1064. PubMed PMID: 7925194.

54. Bale AS, Barone S, Jr., Scott CS, Cooper GS. A review of potential neurotoxic mechanisms among three chlorinated organic solvents. Toxicology and applied pharmacology. 2011;255(1):113-26. Epub 2011/05/26. doi: 10.1016/j.taap.2011.05.008. PubMed PMID: 21609728.

55. Wang S, Karlsson JE, Kyrklund T, Haglid K. Perchloroethylene-induced reduction in glial and neuronal cell marker proteins in rat brain. Pharmacol Toxicol. 1993;72(4-5):273-8. Epub 1993/04/01. doi: 10.1111/j.1600-0773.1993.tb01649.x. PubMed PMID: 8372046.

56. Mattsson JL, Albee RR, Yano BL, Bradley G, Spencer PJ. Neurotoxicologic examination of rats exposed to 1,1,2,2-tetrachloroethylene (perchloroethylene) vapor for 13 weeks. Neurotoxicology and teratology. 1998;20(1):83-98. Epub 1998/03/25. doi: 10.1016/s0892-0362(97)00074-3. PubMed PMID: 9511173.

57. Kyrklund T, Alling C, Kjellstrand P, Haglid KG. Chronic effects of perchloroethylene on the composition of lipid and acyl groups in cerebral cortex and hippocampus of the gerbil. Toxicology letters. 1984;22(3):343-9. Epub 1984/09/01. doi: 10.1016/0378-4274(84)90112-7. PubMed PMID: 6485008.

58. Kyrklund T, Kjellstrand P, Haglid KG. Lipid composition and fatty acid pattern of the gerbil brain after exposure to perchloroethylene. Archives of toxicology. 1987;60(5):397-400. Epub 1987/07/01. doi: 10.1007/bf00295762. PubMed PMID: 3662814.

59. Rosengren LE, Kjellstrand P, Haglid KG. Tetrachloroethylene: levels of DNA and S-100 in the gerbil CNS after chronic exposure. Neurobehav Toxicol Teratol. 1986;8(2):201-6. Epub 1986/03/01. PubMed PMID: 3713968.

60. Kyrklund T, Kjellstrand P, Haglid KG. Effects of exposure to Freon 11, 1,1,1-trichloroethane or perchloroethylene on the lipid and fatty-acid composition of rat cerebral cortex. Scandinavian journal of work, environment & health. 1988;14(2):91-4. Epub 1988/04/01. doi: 10.5271/sjweh.1942. PubMed PMID: 3387963.

61. Briving C, Jacobson I, Hamberger A, Kjellstrand P, Haglid KG, Rosengren LE. Chronic effects of perchloroethylene and trichloroethylene on the gerbil brain amino acids and glutathione. Neurotoxicology. 1986;7(1):101-8. Epub 1986/01/01. PubMed PMID: 2872637.

62. Guyton KZ, Hogan KA, Scott CS, Cooper GS, Bale AS, Kopylev L, Barone S, Makris SL, Glenn B, Subramaniam RP, Gwinn MR, Dzubow RC, Chiu WA. Human health effects of tetrachloroethylene: key findings and scientific issues. Environmental health perspectives. 2014;122(4):325-34. Epub 2014/02/18. doi: 10.1289/ehp.1307359. PubMed PMID: 24531164; PMCID: PMC3984230 necessarily represent the views or policies of the U.S. EPA. The authors declare they have no actual or potential competing financial interests.

63. *See Generally* Bove Dep. Oct. 17, 2024.

64. *See Generally* Bove Dep. Oct. 18, 2024.

65. Bove Frank J. *Evaluation of cancer incidence among Marines and Navy personnel and civilian workers exposed to contaminated drinking water at USMC Base Camp Lejeune: a cohort study*. January 29, 2024. medRxiv preprint doi: https://doi.org/10.1101/2024.01.27.24301873.

66. Camp Lejeune Justice Act. Pub. L. 117-168, title VIII, §804, Aug. 10, 2022, 136 Stat. 1802.

67. Hill AB. The Environment and Disease: Association or Causation? Proc R Soc Med. 1965 May;58(5):295-300. doi: 10.1177/003591576505800503. PMID: 14283879; PMCID: PMC1898525.

68. Lash, L. H., 2024. Trichloroethylene: An Update on an Environmental Contaminant with Multiple Health Effects. Annu Rev Pharmacol Toxicol.

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Lyman Briggs School of Science, Michigan State University, East Lansing, MI, 48824

PROFESSIONAL EXPERIENCE and ACADEMIC APPOINTMENTS

08/2024-05/2025	**Fellow**, Big Ten Academic Alliance Academic Leadership Program (BTAA-ALP)
11/2023-present	**Acting Head**, School of Health Sciences, Purdue University
08/2023-present	**Assistant Vice Provost** for Interdisciplinary Graduate Programs, Purdue University
08/2022-present	**Professor of Toxicology** (tenured), School of Health Sciences, Purdue University
08/2022-present	**Consultant (toxicology)**, Forensic Psychology Consultants, LLC
08/2021-present	**Co-leader**, Healthy Lifestyles and Vital Longevity – College of Health and Human Sciences Signature Area, Purdue University
06/2021-08/2024	**Mentor**, National Institute of Health (NIH) funded Toxicology Mentoring and Skills Development Training Program (ToxMSDT)
07/2020-present	**Courtesy Appointment**, Department of Public Health, Purdue University
07/2020-present	**Member**, Neurotoxicity Technical Working Group, Botanical Safety Consortium (BSC), Health and Environmental Sciences Institute (HESI)
04/2019-04/2024	**Member**, Fulbright Specialist Roster, U.S. Department of State's Bureau of Educational and Cultural Affairs (ECA) and World Learning
12/2018-12/2019	**Fellow**, Faculty Leadership Academy for Interdisciplinary Research, Office of the Executive Vice President for Research and Partnerships, Purdue University
07/2017-09/2023	**Head**, Purdue University Interdisciplinary Life Science Program (PULSe)
09/2016-08/2017	**Chair, Integrative Neuroscience Training Group**, Purdue University Interdisciplinary Life Science Program (PULSe)
08/2016-10/2023	**Director of Toxicology Graduate Program**, School of Health Sciences, Purdue University
08/2016-12/2018	**Director of Graduate Studies**, School of Health Sciences, Purdue University
08/2016-08/2022	**Associate Professor of Toxicology** (tenured), School of Health Sciences, Purdue University
01/2016-present	**Faculty Associate**, Integrative Neuroscience Center, Purdue University
06/2013-present	**Faculty Associate**, Center on Aging and Life Course, Purdue University
02/2012-present	**Administrative Member**, Integrative Neuroscience Training Group, Purdue University Interdisciplinary Life Science Ph.D. program (PULSe)
01/2012-07/2016	**Assistant Professor of Toxicology**, School of Health Sciences, Purdue University
09/2010-12/2011	**Research Associate**, Pittsburgh Institute for Neurodegenerative Diseases, Department of Neurology, University of Pittsburgh
09/2010-12/2011	**Research Associate**, Department of Veteran's Affairs, VA Pittsburgh Healthcare System
09/2006-09/2010	**Postdoctoral Associate**, Pittsburgh Institute for Neurodegenerative Diseases, Department of Neurology, University of Pittsburgh

09/2005-08/2006 **Graduate Student Research Assistant**, Department of Neurosurgery, University of Michigan

09/2001-08/2005 **NIEHS Predoctoral Research Trainee**, Environmental Health Sciences, Toxicology Program, University of Michigan

09/2001-05/2003 **Polysomnographic Research Analyst**, University of Michigan School of Nursing

07/2000-08/2001 **Lead Research Polysomnographic Technologist**, General Clinical Research Center, Medical School, University of Michigan

08/1999-01/2000 **Teaching Assistant**, Capstone laboratory in Physiology, Physiology Department, Michigan State University

06/1998-07/2000 **Polysomnographic Technologist**, Ingham Regional Medical Center, Lansing, MI

ACADEMIC AND PROFESSIONAL HONORS

Awards

2022 Travel Award ($1,000), Incoming Mobility Commission, Office of Science and Art, University of Rijeka

2019 Purdue Research Foundation International Travel Grant

2017 University Faculty Scholar (2017-2022), total award of $100k in discretionary funds

2017 Showalter Faculty Scholar (2017-2022) – subset of University Faculty Scholars (excellence in life sciences)

2016 Seed for Success Award, Purdue University (external sponsor award >$1M)

2015 Travel award ($1700), Elucidating Environmental Dimensions of Neurological Disorders and Disease: Understanding New Tools from Federal Chemical Testing Programs, Environmental Defense Fund, NIEHS/NTP

2015 Outstanding Reviewer – Elsevier (top 10th percentile, number of reviews completed for *Neurobiology of Disease* in the past two years)

2014 Early Career Reviewer (2nd selection), Clinical Neuroplasticity and Neurotransmitters Study Section, Center for Scientific Review, National Institutes of Health

2013 Early Career Reviewer 1st selection, Clinical Neuroplasticity and Neurotransmitters Study Section, Center for Scientific Review, National Institutes of Health

2013 Appointed as Faculty Associate, Center on Aging and Life Course, Purdue University

2013 Certificate of Excellence in Reviewing, *Experimental Neurology*

2011 NIH (NIEHS) Individual Career Development Award (K99/R00)

2011 AstraZeneca Travel Award (100% funding for travel and attendance), Gordon Research Conference, Cellular & Molecular Mechanisms of Toxicity Understanding Innovative Mechanistic Toxicology in the Post-Genomic Era

2011 Abstract chosen for oral presentation. Gordon Research Conference, Cellular & Molecular Mechanisms of Toxicity Understanding Innovative Mechanistic Toxicology in the Post-Genomic Era

2011 1st place in poster competition. Gordon Research Conference, Cellular & Molecular Mechanisms of Toxicity Understanding Innovative Mechanistic Toxicology in the Post-Genomic Era

2010 Best Overall Poster, 2010 Annual Spring Meeting, Allegheny-Erie Society of Toxicology

2008 Postdoctoral Fellowship, American Parkinson's Disease Association, Inc.

2007 Institutional Postdoctoral Training Fellowship, NIMH Training Grant the Neurobiology of Psychiatric Disorders, University of Pittsburgh

2006 Rackham Travel Award, Society of Toxicology's 45th annual meeting, Rackham Graduate School, University of Michigan

2005 Rackham Travel Award, Society of Toxicology, Society of Toxicology's 44th annual meeting, Student Scholarship, 13th International Symposium on Brain Edema and Conference on Intracerebral Hemorrhage

2004 Rackham Travel Award, Society of Toxicology's 43rd annual meeting, Rackham Graduate School, University of Michigan

2003 Rackham Travel Award, Society of Toxicology's 42nd annual meeting, Rackham Graduate School, University of Michigan
2001 Institutional Predoctoral Training Fellowship (3 competitive renewals), NIEHS Environmental Toxicology Research Training Grant, The University of Michigan
1998 Bachelor of Science Degree, *with honor*
1996 Tower Guard: Sophomore Honor Service Society, Michigan State University

Society Memberships

2006-Present Society for Neuroscience
2002-Present Society of Toxicology, Neurotoxicology Specialty Section
2002-Present International Neurotoxicology Association

Professional Activities

Associate Editor

Frontiers in Toxicology (2019-)

NeuroToxicology (2019-)

Toxicological Sciences (2023-)

Editorial Board Membership

Journal of Biochemical and Molecular Toxicology (2021- present)

Toxicology, (2019-present)

Toxics, Editorial Board Member (2019 – present)

NeuroToxicology (2018-2019)

Neurotoxicology & Teratology (2018-present)

Frontiers in Environmental Science, Toxigogenomics section, Review Member, Editorial Board (2017 – 2019)

Frontiers in Genetics, Toxicogenomics section, Review Member, Editorial Board (2017 – present)

Toxicological Sciences, Editorial Board Member (2015 – 2023)

Experimental Biology and Medicine, Member, Pharmacology & and Toxicology Section (2013-2016)

Guest Editor

Neurotoxicology and Teratology (2019-2020), Special Issue entitled, *"Leveraging non-mammalian models for developmental neurotoxicity testing"*

Governmental Document Review

National Center for Environmental Health (NCEH)/Agency for Toxic Substances and Disease Registry (ATSDR), Office of Science, US Centers for Disease Control and Prevention

Editorial Review for Scientific Journals

Aging Cell
Analytical Methods
Archives of Toxicology
Biochemical Pharmacology
Biological Trace Element Research
Biomedicine & Pharmacotherapy
BMC Neurology
BMC Neuroscience
Brain Research
Cell Death & Disease
Cells
Chemical Communications
Chemosphere
Clinical Neurology & Neurosurgery
Current Cancer Drug Targets
Disease Models & Mechanisms
Eco-Environment and Health
Environmental Health Perspectives
Environmental Pollution
Environment International
Experimental Biology and Medicine
Experimental Brain Research
Experimental Neurology
Food & Function
Frontiers in Genetics
Frontiers in Immunology
Frontiers in Neuroscience
Free Radical Biology and Medicine
Glia
Gerontology & Geriatric Medicine
IBRO Reports
International Journal of Developmental Neuroscience
International Journal of Environmental Research and Public Health
Journal of Dietary Supplements
Journal of Functional Foods
Journal of Integrative Neuroscience
Journal of Neural Transmission
Journal of Neurochemistry
Journal of Neurogenetics
Journal of Neuroinflammation
Journal of the Neurological Sciences
J Neuropath and Experimental Neurology
Journal of Nervous and Mental Disease
Journal of Neuroscience Research
Journal of Toxicology
Marine Pollution Bulletin
Meat Science
Metabolic Brain Disease
Metallomics
Molecular and Cellular Neuroscience
Neurobiology of Aging
Neurobiology of Disease
Neurochemical Research
Neurochemistry International
Neuropharmacology
Neuroscience
Neuroscience Letters
Neurotoxicity Research
Neurotoxicology
Neurotoxicology & Teratology
npj Biomedical Innovations
npj Clean Water
Organic & Biomolecular Chemistry
Pesticide Biochemistry and Physiology
Pharmacology & Therapeutics
Physiology & Behavior
PloS ONE
PNAS
PNAS Nexus
Psychopharmacology
Science Signaling
Scientific Reports
Toxicology
Toxicology & Applied Pharmacology
Toxicology Research
Toxicological Sciences

Editorial Review for Textbooks

Jones and Bartlett Learning

Grant Review

2024 Initiate Programme, Luxembourg National Research Fund
2024 Research project review, Croatian Science Foundation, Summer, 2024
2024 Chair, Peripheral Neuropathy Panel, Congressionally Directed Medical Research Programs, Department of Defense, Summer, 2024
2023 Chair, Neurotoxicology Panel, Congressionally Directed Medical Research Programs, Department of Defense, Winter, 2023
2023 Peripheral Neuropathy, Congressionally Directed Medical Research Programs, Department of Defense, Winter, 2023
2023 Austrian Science Fund, ad hoc reviewer, Summer, 2023
2023- Standing member of *Neurotoxicology and Alcohol* (NAL) Study Section, Center for Scientific

Review, National Institutes of Health, begins 07/2023 and ends 06/2029; service (10/2023)
2023 2023/05 ZNS1 SRB-D (26) F, NST2 Overflow SEP, NINDS Post-Doc Career Development and Research Training, Center for Scientific Review, National Institutes of Health, Winter, 2023
2023 2023/05 NST-2 L, NINDS Post-Doc Career Development and Research Training, Center for Scientific Review, National Institutes of Health, Winter, 2023
2023 Toxic Exposures Research Program, Congressionally Directed Medical Research Programs, Department of Defense
2022 Purdue Reviewer, Overseas Visiting Doctoral Fellowship (OVDF) Program, Purdue and India's Science and Engineering Research Board
2022 F03A-E (20) L, *Fellowships: Neurodevelopment, Synaptic Plasticity and Neurodegeneration*, Center for Scientific Review, National Institutes of Health, Fall, 2022
2022 Dutch research foundation ParkinsonNL, Fall, 2022
2022 ZRG1 F03B-L (20) L, *Fellowships: Biophysical, Physiological, Pharmacological and Bioengineering Neuroscience*, Center for Scientific Review, National Institutes of Health, Summer, 2022
2022 ZRG1 F03B-L (20) L, *Fellowships: Biophysical, Physiological, Pharmacological and Bioengineering Neuroscience*, Center for Scientific Review, National Institutes of Health, Winter, 2022
2021 *Open Competition Domain Science*, Dutch Research Council, Netherlands, Fall, 2021
2021 NIEHS P42 Superfund Research Program – Phase I and Phase II review, National Institutes of Health, Fall, 2021
2021 ZRG1 F03B-R (20) L, *Fellowships: Biophysical, Physiological, Pharmacological and Bioengineering Neuroscience,* Center for Scientific Review, National Institutes of Health, *ad hoc,* Summer, 2021
2021 *Showalter Review Panel*, Purdue Research Foundation, Spring, 2021
2021 Core Pilot review, Translational Research Development Program, Indiana Clinical and Translational Sciences Institute (CTSI), Spring, 2021
2021 National Centre for the Replacement, Refinement and Reduction of Animals in Research (NC3Rs), United Kingdom, *Ad hoc* Reviewer, Spring, 2021
2020 Investigating Environmental Risk Factors, The Michael J. Fox Foundation, Fall, 2020
2020 ZRG1 F03A-E (20) L, *Fellowships: Neurodevelopment, Synaptic Plasticity and Neurodegeneration Fellowship Panel* (F03A), Center for Scientific Review, National Institutes of Health, *ad hoc,* Summer, 2020
2020 *Showalter Review Panel*, Purdue Research Foundation, Spring, 2020
2020 ZRG1 F03A-E (20) L, *Fellowships: Neurodevelopment, Synaptic Plasticity and Neurodegeneration Fellowship Panel*, Center for Scientific Review, National Institutes of Health, *ad hoc,* Spring, 2020
2019 K99/R00 Pathway to Independence Award Panel, National Institute of Environmental Health Sciences, National Institutes of Health, *ad hoc,* Summer, 2019
2019 IMM-K (50) *US-Brazil Collaborative Research Program*, National Institutes of Health, Summer, 2019
2019 *Swiss National Science Foundation*, *ad hoc* reviewer
2019 *Early Life Stressors and Alcohol Use Disorders* [ZRG1 IFCN-C (07) S] Study Section, Center for Scientific Review, National Institutes of Health, *ad hoc,* Spring, 2019
2019 *Neurotoxicology and Alcohol* (NAL) Study Section, Center for Scientific Review, National Institutes of Health, *ad hoc,* Spring, 2019
2018 *Environmental Factors (EF),* peer review panel of the 2018 Parkinson's Disease Research Program (PRP) for the Department of Defense Congressionally Directed Medical Research Programs (CDMRP)
2018 K99/R00 Pathway to Independence Award Panel, National Institute of Environmental Health Sciences, National Institutes of Health, *ad hoc,* Fall, 2018
2018 *Neurobiology – E,* VA Merit Review Panel, Summer 2018
2018 *Neurobiology of Alcohol Toxicity and Chemosensation member conflict Special Emphasis Panel* Study Section [2018/05 ZRG1 IFCN-N (03) M], Center for Scientific Review, National Institutes of Health, *ad hoc,* Spring, 2018
2017 Department of Defense Congressionally Directed Medical Research Programs, Metals Toxicology, Teleconference
2017 *Neurotoxicology and Alcohol* (NAL) Study Section, Center for Scientific Review, National Institutes of Health, *ad hoc,* Fall, 2017
2017 Department of Defense Congressionally Directed Medical Research Programs, Metals Toxicology, FP-MT
2017 Indiana Alzheimer Disease Center (IADC) Pilot Project Grant Review

2017 Department of Defense Congressionally Directed Medical Research Programs, Discovery Metals Toxicology Metals Toxicology
2017 Department of Defense Congressionally Directed Medical Research Programs, Pre-application Metals Toxicology Metals Toxicology
2017 *Neurotoxicology and Alcohol* (NAL) Study Section, Center for Scientific Review, National Institutes of Health, *ad hoc,* Summer, 2017
2017 Reviewer, New R01 Incentive Program, Office of the Executive Vice President for Research and Partnerships
2017 *Neuroplasticity & Compensation/Progression & Heterogeneity (NPC-PH)* peer review panel of the 2016 Parkinson's Disease Research Program (PRP) for the Department of Defense Congressionally Directed Medical Research Programs (CDMRP)
2016 *Neurobiology – E,* VA Merit Review Panel, Winter 2016
2016 *Systemic Injury and Environmental Exposures* (SIEE), Study Section, Center for Scientific Review, National Institutes of Health, *ad hoc,* Fall, 2016
2016 Department of Defense Congressionally Directed Medical Research Programs Metals Toxicology Metals Toxicology (Discovery Award)
2016 Department of Defense Congressionally Directed Medical Research Programs, Pre-application Metals Toxicology Metals Toxicology [Investigator-Initiated Research Award (IIRA), Technology/Therapeutic Development Award (TTDA)]
2016 *Clinical Neuroplasticity and Neurotransmitters Study Section*, Center for Scientific Review, National Institutes of Health, *ad hoc,* Summer, 2016
2016 Target Advancement Panel, The Michael J Fox Foundation
2016 Health Research Council of New Zealand
2015 Department of Defense Congressionally Directed Medical Research Programs, Metals Toxicology Metals Toxicology [Investigator-Initiated Research Award (IIRA), Technology/Therapeutic Development Award (TTDA)]
2015 Department of Defense Congressionally Directed Medical Research Programs, Metals Toxicology (Discovery Award)
2015 Department of Defense Congressionally Directed Medical Research Programs, Pre-Application Metals Toxicology [Investigator-Initiated Research Award (IIRA), Technology/Therapeutic Development Award (TTDA)]
2015 Parkinson's disease Society – *UK; ad hoc* grant reviewer, Summer, 2015
2015 Indiana Spinal Cord and Brain Injury Fund, Indiana State Department of Health, Spring, 2015
2014 *Clinical Neuroplasticity and Neurotransmitters Study Section*, Center for Scientific Review, National Institutes of Health, *ad hoc,* Summer, 2014
2013 Joint Research Actions, The French Community of Belgium, University of Liège, *ad hoc* Spring, 2013
2013 *Clinical Neuroplasticity and Neurotransmitters Study Section*, Center for Scientific Review, National Institutes of Health, *ad hoc,* Spring, 2013
2012 The Medical Research Council (MRC) of South Africa – External Grant Reviewer
2012 Collaborative Incentive Research Grant (CIRG), CUNY – *ad hoc* External Reviewer, 5/2012
2010 Parkinson's disease Society – *UK; ad hoc* grant reviewer, Fall, 2010

Program/other External Review

2022 External Reviewer/Focus Group Member, Strategic Plan Review, Lyman Briggs College, Michigan State University

Consortium Memberships

2012-2015 LRRK2 Biology Program, the Michael J. Fox Foundation

ACTIVE/PENDING RESEARCH SUPPORT

NAME OF INDIVIDUAL		
Project Number (Principal Investigator) Source Title of Project *(or Subproject)* Major goals	Dates of Approved/Proposed Project Annual Direct Costs	Person Months (Cal/Academic/ Summer)

ACTIVE*

R01 ES035019-A1 (Cannon and Foti - mPIs) 01/01/2024 – 08/31/2028 2.0 Summer
NIEHS/NIH ~$351,153
PFAS induced alterations in reward processing
The goal is to determine whether PFAS exposure may be a risk factor for anhedonia through translationally connected animal and human studies. Role = PI. Total cost = $2,694,050.

R01ES025750-06A1 (Cannon, PI) 09/15/2023 – 09/14/2026* 1.0 Academic
NIEHS/NIH $382,579 3.0 Summer
Mechanisms of PhIP-induced dopaminergic neurotoxicity
The major goals are to test whether the heterocyclic amine PhIP induces selective dopaminergic toxicity and determine mechanisms of action. In this cycle, we aim discover how human relevant neuromelanin-neurotoxicant interactions modulate dopaminergic neurotoxicity Role: PI. Total cost = $1,563,395. *Due to current economic and political climates, the 1st 3 years are awarded as lumps some, with years 4,5 subject to Type 4 (non-competing) continuation applications to be submitted 90 days prior to the current end-date.

PD211037 [mPIs, Cannon (contact) and Wells] 09/30/2022 – 09/29/2025 1.8 Summer
DOD ~$250,000
Role Of Military Relevant Chlorpyrifos Exposure In Parkinson's Disease Relevant Dopaminergic Neurotoxicity.
The goal is to understand whether military-related chlorpyrifos exposure may influence PD risk. Role = PI. Total cost = $1,199,999.

1R01AG080917 (Bowman Yuan, and Zhang, mPIs) 09/22/2022 – 05/31/2027 0.23 Academic
NIA/NIH ~$479,223 0.07 Summer
Modeling functional genomics of susceptibility to the persistent effects of environmental toxins in an elderly rural Indiana neurodegenerative cohort
The goal is to advance understanding of how gene-environment interactions influence neurodegeneration in rural patients. Role = co-I. Total cost = 3,737,946, $264,306 to Cannon lab.

PR211366 (PI, Little) 09/15/2022 – 09/14/2026 0.5 Academic
DOD $400,000 0.5 Summer
Role Of Comorbid Military-Relevant Stressors In Osteoarthritis.
The goal is to investigate psychological stress-induced mechanisms of accelerated development of end-stage post-traumatic knee osteoarthritis (OA). Role = co-I. Total cost = $2,431,591, $363,735 to Cannon lab.

SUBMITTED/PENDING
P42ESXXXXX (MPIs Sepulveda and Freeman) 07/01/2024 - 06/30/2029 1.0 Academic
NIEHS/NIH $1,999,985
Center for Health Impacts and Remediation of PFAS (CHIRP) 2.0 Summer
Role = Lead, P2 (*Adverse Neuropsychiatric Outcomes Induced by GL-specific Neurotoxicity*); Lead RETCC (*Research Experience and Training Coordination Core*) Total cost = $15,315,127, ~$3,846,447 to Cannon lab.

PREVIOUS RESEARCH SUPPORT
1937986 (Webb, PI) 02/15/2020 - 01/31/2024
NSF
Super-resolution in vivo optical imaging as a window to Parkinson's disease pathogenesis. The goals are to identify and image novel pathogenetic mechanisms to PD. Role = co-I. Total cost = $400,000, ~$105,000 to Cannon lab.

2120200-DBI (Umulis, PI) 09/01/2021- 08/31/2026
BII: Emergent Mechanisms in Biology of Robustness, Integration, & Organization (EMBRIO). Create an institute that advances understanding of basic biology and robustness if signaling across biological scale. Role = co-I. Total cost = $12,000,000, ~$40,000 to Cannon lab (role in this grant ended 08/30/23).

R21AG068787S-1 (Cannon, PI) 09/01/2021 – 05/31/2023
NIA/NIH
PFOS-induced dopaminergic neurodegeneration across nematode, amphibian, and rodent models
The goal was to assess relevance of PFAS neurotoxicity to Alzheimer's disease. Role = PI. Total cost = $308,499.

R21AG068787 (Cannon, PI) 09/01/2020 – 05/31/2023 (NCE)
NIA/NIH
PFOS-induced dopaminergic neurodegeneration across nematode, amphibian, and rodent models
The goal was to advance understanding of PFAS neurotoxicity through comparative biology approaches. Role = PI. Total cost = $409,222.

No number (Rochet, PI) 07/01/2021-12/31/2022
Branfman Foundation
Neuroprotective efficacy of XJB-5-131 in rodent Parkinson's disease models.
The goal is to test a novel therapeutic approach in PD. Role = co-I. Total cost = $112,019, $60,071 to Cannon lab.

R03NS108229 (Rochet, PI) 05/15/2020-04/30/2022
NINDS/NIH
Role of endosulfine-alpha expression and phosphorylation in Parkinson's disease
The goal is to understand the neurobiology of endosulfine, relative to Parkinson's disease. Role: co_I. Total Cost = 155,000. $8,613 to Cannon lab.

R01ES025750 (Cannon, PI) 06/01/2016 – 05/31/2022
NIEHS/NIH
Mechanisms of PhIP-induced dopaminergic neurotoxicity
The major goals are to test whether the heterocyclic amine PhIP induces selective dopaminergic toxicity and determine mechanisms of action. Role: PI. Total cost = $1,683,647.

R01ES025750-S1 (Cannon, PI) 09/01/2018 – 05/31/2022
NIA,NIEHS/NIH
Mechanisms of PhIP-induced dopaminergic neurotoxicity – Alzheimer's disease supplement
The major goals are to test whether heterocyclic amines may produce neuropathology indicative of Alzheimer's disease. Role: PI. Total cost = $336,582

No Number (Cannon, PI) 07/01/2019 – 12/31/2021
Office of the Executive Vice President for Research and Partnerships, Purdue University
NIH Competing Renewal Program - Mechanisms of PhIP-induced dopaminergic neurotoxicity
The goal is to develop a novel animal model to elucidate mechanisms of heterocyclic amine neurotoxicity. Development of this model is expected to increase competitiveness of NIH applications. Total cost = $30,000.

No Number (Rochet, PI) 08/01/2019 - 12/31/2020
Branfman Family Foundation
Role of alpha-synuclein-mediated membrane permeabilization in the propagation of PD neuropathology
The goal was to determine how aSyn aggregates in Parkinson's disease. Role: co-I. Total cost = $101,638; $30,762 to Cannon Lab.

R21 NS105048 (Webb, PI) 10/01/2018 – 09/30/2021
NINDS/NIH
In Vivo Optical Imaging of Alpha-Synuclein Aggregation
This project entails the application of a high-resolution whole brain optical molecular imaging method to determine the pathogenic mechanism involved in the temporal and spatial development of Parkinson's disease (PD). Role = co-I. Total cost = $403,204, $48,614 to Cannon lab.

R21NS106319 (Tantama, PI) 09/15/2018 – 08/31/2020
NINDS/NIH
LRRK2 Kinase Activity and Mitochondrial Oxidative Stress
The goal was to utilize novel probes to image mitochondrial mechanisms of Parkinson's disease relevant neurodegeneration. Role = Co-I (Purdue site PI). Total cost = $424,301, $95,380 to Cannon.

No Number (Rochet, PI) 09/01/2018 - 08/31/2019
Branfman Family Foundation
Role of alpha-synuclein-mediated membrane permeabilization in the propagation of PD neuropathology
The goal is to determine how aSyn aggregates in Parkinson's disease. Role: co-I. Total cost = $50,000; $8,232 to Cannon Lab.

No number (Webb, PI) 05/01/2018 - 12/31/2018
NIH-targeted Funding Opportunities Initiative
Office of the Executive Vice President for Research and Partnerships, Purdue University
In Vivo Optical Imaging to Solve Mysteries of Parkinson's Disease
The major goal is to collect preliminary data for an extramural submission on novel imaging approaches to visualize Parkinson's disease pathology. Role: co-I. Total cost = $30,000. No direct funds to Cannon lab.

No Number (Rochet, PI) 06/01/2018 – 07/31/2019
Michael J. Fox Foundation
Neuroprotective effects of NFE2L1 in PD models
The goal is to test whether NFE2L1 modulation is protective in PD models.
Role: co-I. Total cost = $57,000. ~$3,000 to Cannon lab.

No Number (Rochet, PI) 11/01/2016 – 06/30/2019
Michael J. Fox Foundation
Neuroprotective effects of endosulfine-alpha in PD models
The goal is to test whether endosulfine-alpha alleviates aSyn-mediated neurodegeneration by inhibiting aSyn self-assembly at membrane surfaces. Role: co-I. Total cost = $66,706. $3,200 to Cannon lab.

No Number (Rochet, PI) 08/01/2015 - 01/31/2018
Branfman Family Foundation
Vesicle permeabilization associated with membrane-induced aSyn aggregation: Role in Parkinson's disease
The goal is to determine how aSyn aggregates in Parkinson's disease. Role: co-I. Total cost = $200,000; $41,989 to Cannon Lab.

No Number (Tantama, PI) 07/01/2015 – 06/30/2018
Showalter Trust
Imaging mitochondrial oxidative stress in Parkinson's disease
The major goal was to develop and test novel in vitro and in vivo probes for assessing PD-relevant oxidative stress. Role: co-I. Total cost = $75,000; $7,500 to Cannon lab.

No Number (Rochet, PI) 05/01/2015 – 12/31/2016
Purdue University, new R01 program
Membrane-induced aSyn aggregation in Parkinson's disease
The goal was to collect preliminary data on mechanisms of neurodegeneration for an R01 submission. Role: co-I. Total cost = $30,000; $7,500 to Cannon lab.

R03ES022819 (Cannon, PI) 01/17/2014 - 12/31/2016
NIEHS/NIH
PhIP-induced neurodegeneration: mechanisms and relevance to Parkinson's disease
The goal of this proposal was to preliminarily examine the neurotoxicity of PhIP. A major goal is to produce preliminary data for this more expansive R01 proposal to mechanistically examine PD-relevant neurotoxicity. Role: PI. Total cost = $154,000

No Number; The Michael J. Fox Foundation; 11/01/2012-10/31/2015; PI (Cannon)
Parkinson's and inflammatory bowel diseases: interaction in LRRK2 transgenic rats
The goal was to identify immunological links between Parkinson's disease and inflammatory bowel disease mediated by disease causing mutations in LRRK2. Total cost: $250,000
No number; Showalter Research Trust; 07/01/2013-06/30/2014; PI (Cannon)
Mechanisms of PhIP-mediated neurotoxicity and relevance to Parkinson's disease
The goal of this proposal is to preliminarily examine the neurotoxicity of PhIP and generate data for more expansive future studies. Total cost = $75,000

R00ES019879 (Cannon, PI) 02/10/2012 - 01/31/2017
NIH/NIEHS
New Approaches to Gene-environment Interaction Modeling in Parkinson's Disease
The major goals of the project were to develop and characterize new *in vivo* gene-environment interaction models of Parkinson's disease to identify new mechanisms of interactions and therapeutic targets. Role: PI. Total Cost = $783,978

No number; 08/01/2011-07/31/2013; PI (Cannon)
Phenotypic Characterization of BAC LRRK2 Transgenic Pre-clinical Models
The University of Pittsburgh (subcontract from Michael J. Fox Foundation to Greenamyre)
The main goals of this work were to characterize the behavioral, neurochemical, and pathological features of rats expressing LRRK2 mutations. Total cost: $95,900

1 K99 ES019879; 06/01/2011-02/09/2012; PI (Cannon)
NIEHS/NIH
New Approaches to Gene-environment Interaction Modeling in Parkinson's Disease
The purpose of this grant was to develop new-gene environment interaction models of PD and transition Cannon to an independent faculty position. Total cost: $90,000 utilized, $180,000 awarded (early transition to independence)

No number; 7/1/2008-12/31/2009; PI (Cannon)
Postdoctoral Fellowship, American Parkinson Disease Association, Inc.
Genetic and environmental interactions in Parkinson's disease: potential for new therapeutic pathways
The goal of this project was to develop and test gene-therapy vectors in the rotenone model of Parkinson's disease. Total cost: $35,000

T32 MH18273; 6/29/2007-6/30/2008; PI (Zigmond)
Institutional Training Grant, NIH
The purpose of this training grant was to support the trainee's postdoctoral training and research.

T32 ES07062; 9/1/2001-8/31-2005; PI (Richardson)
Institutional Training Grant, NIEHS
The purpose of this training grant was to support the trainee's doctoral training and research.

ACTIVE/PENDING SUPPORT FOR OTHER ACTIVITIES

ACTIVE

PENDING
T32ES036148 (Cannon, MPI – Contact, Bowman, MPI) 08/01/2024 – 07/31/2029
NIEHS/NIH
Toxicology training in bidirectional translation across biological scale
The goal is to innovatively train graduate students and postdoctoral fellows in translational toxicology using the adverse outcome pathway as a template. Total cost = $2,579,894. Impact score = 40. Resubmission in preparation.

COMPLETED

No number (Cannon, PI) 09/20/2019 – 07/31/2023
International Program and School of Health Sciences, Purdue University
Study Abroad Intercultural Learning (SAIL) Subsidy Grant
Neuroscience and Toxicology in Croatia
This grant reduces student costs for this study abroad. Total award = $10,666

No number (Cannon, PI) 07/01/2017 – 09/01/2023
Office of Interdisciplinary Graduate Programs
Discretionary funding for effort as Head of Purdue University Interdisciplinary Life Science Program (PULSe). Award: $3,750/year
Discretionary funding deposited to my research incentives account that I use to support new collaborative research initiatives.

No number (Cannon, PI) 10/30/2022 – 10/29/2023
International Program and School of Health Sciences, Purdue University
Study Abroad Intercultural Learning (SAIL) Subsidy Grant
Neuroscience and Toxicology in Croatia
This grant reduces student costs for this study abroad. Total award = $8,000

No number (Cannon, PI) 07/01/2017 – 06/30/2022
Office of the Provost/Showalter Trust
Discretionary funding as *Showalter Faculty Scholar/University Faculty Scholar*. Total award = $50,000 ($10,000 dispersed/year)
Discretionary funding that I use to support new collaborative research initiatives.

No number (Cannon, PI) 09/20/2019 – 09/19/2020
International Program
Study Abroad Intercultural Learning (SAIL) Intercultural Pedagogy Grant (IPG)
Neuroscience and Toxicology in Croatia
This grant provides discretionary funding to add intercultural learning objectives to a study abroad. Total award = $2,000

No number (Cannon, PI) 09/24/2018 – 08/01/2019
International Program and College of Health and Human Sciences, Purdue University
Exploratory Study Abroad Intercultural Learning (SAIL) grant
Neuroscience and Toxicology in Croatia
This grant funds exploratory travel to Croatia to develop of a study abroad program focused on neuroscience and toxicology. Total award = $4,000

PUBLICATIONS

#Figure chosen for cover art

Peer-reviewed publications

1. Tanwar, R., Doepeker, A., Wells, E., Cannon, J., Parkinson's disease risk and mechanisms from military relevant organophosphate exposures. 8th Rijeka Forum On Neurodegenerative Diseases: The Regulation Of Gene Expression In Neurological Disease And Neuroimmunology. 26-27.
2. Currim, F., Tanwar, R., Brown-Leung, J. M., Paranjape, N., Liu, J., Sanders, L. H., Doorn, J. A., and Cannon, J. R. (2024). Selective dopaminergic neurotoxicity modulated by inherent cell-type specific neurobiology. *Neurotoxicology* doi: https://doi.org/10.1016/j.neuro.2024.06.016.
3. Currim, F., Shukla, S., Singh, J., Gohel, D., Mane, M., Shinde, A., Roy, M., Goyani, S., Vasiyani, H., Chandran, A., Rochet, J. C., Cannon, J.*, and Singh, R.* (2024). Neuronal exosomal miRNAs modulate mitochondrial functions and cell death in bystander neuronal cells under Parkinson's disease stress conditions. *Neurotoxicology* doi: 10.1016/j.neuro.2024.02.005. *Co-corresponding authors.
4. *Bellamri, M., Brandt, K., Cammerrer, K., Syeda, T., Turesky, R. J., and Cannon, J. R. (2023). Nuclear DNA and Mitochondrial Damage of the Cooked Meat Carcinogen 2-Amino-1-methyl-6-phenylimidazo[4,5-b]pyridine in Human Neuroblastoma Cells. *Chemical research in toxicology* **36**(8), 1361-1373. **ACS Editor's Choice; NIEHS Extramural Paper of the Month**
5. De Marchi, F., Franjkic, T., Schito, P., Russo, T., Nimac, J., Chami, A. A., Mele, A., Vidatic, L., Kriz, J., Julien, J.-P., Apic, G., Russell, R. B., Rogelj, B., Cannon, J. R., Baralle, M., Agosta, F., Hecimovic, S., Mazzini, L., Buratti, E., and Munitic, I. (2023). Emerging Trends in the Field of Inflammation and Proteinopathy in ALS/FTD Spectrum Disorder. *Biomedicines* **11**(6), 1599.
6. Enkh-Amgalan, S., Brown-Leung, J. M., Syeda, T., Nolan, R. M., Cannon, J. R., and Chester, J. A. (2023). Paraquat exposure produces sex-dependent reduction in binge-like alcohol drinking in high alcohol-preferring mice. *Food and Chemical Toxicology*, 113685.
7. Sammi, S. R., Syeda, T., Conrow, K. D., Leung, M. C. K., and Cannon, J. R. (2023). Complementary biological and computational approaches identify distinct mechanisms of chlorpyrifos versus chlorpyrifos-oxon-induced dopaminergic neurotoxicity. *Toxicological sciences : an official journal of the Society of Toxicology* **191**(1), 163-178.
8. Fernandez, R. F., Wilson, E. S., Diaz, V., Martínez-Gardeazabal, J., Foguth, R., Cannon, J. R., Jackson, S. N., Hermann, B. P., Eells, J. B., and Ellis, J. M. (2023). Lipid metabolism in dopaminergic neurons influences light entrainment. *Journal of neurochemistry* **165**(3), 379-390.
9. Cannon, J. (2022). Invited Perspective: Long-Lasting Legacy of Banned Contaminants in Alzheimer's Disease Etiology-Convergence of Epidemiological and Toxicological Findings. *Environmental health perspectives* **130**(8), 81303.
10. Brown-Leung, J. M., and Cannon, J. R. (2022). Neurotransmission Targets of Per- and Polyfluoroalkyl Substance Neurotoxicity: Mechanisms and Potential Implications for Adverse Neurological Outcomes. *Chemical research in toxicology* **35**(8), 1312-1333. **Selected for special virtual issue International Day of Women and Girls in Science**, see: Bryant-Friedrich, A., Kraegeloh, A., and Sturla, S. J. (2023). A Virtual Issue of Chemical Research in Toxicology in Celebration of the International Day of Women and Girls in Science. *Chemical research in toxicology* **36**(2), 123-128.

11. Sammi, S. R., Jameson, L. E., Conrow, K. D., Leung, M. C. K., and Cannon, J. R. (2022). Caenorhabditis elegans Neurotoxicity Testing: Novel Applications in the Adverse Outcome Pathway Framework. *Front Toxicol* **4**, 826488.
12. Syeda, T., and Cannon, J. R. (2022). Potential Role of Heterocyclic Aromatic Amines in Neurodegeneration. *Chemical research in toxicology* **35**(1), 59-72.
13. Adamson, S. X., Zheng, W., Agim, Z. S., Du, S., Fleming, S., Shannahan, J., and Cannon, J. (2021). Systemic Copper Disorders Influence the Olfactory Function in Adult Rats: Roles of Altered Adult Neurogenesis and Neurochemical Imbalance. *Biomolecules* **11**(9).
14. Syeda, T., and Cannon, J. R. (2021). Environmental exposures and the etiopathogenesis of Alzheimer's disease: The potential role of BACE1 as a critical neurotoxic target. *J Biochem Mol Toxicol* **35**(4), e22694.
15. Lawana, V., Um, S. Y., Foguth, R. M., and Cannon, J. R. (2020). Neuromelanin formation exacerbates HAA-induced mitochondrial toxicity and mitophagy impairments. *Neurotoxicology* **81**, 147-160.
16. Foguth, R., Sepulveda, M. S., and Cannon, J. (2020). Per- and Polyfluoroalkyl Substances (PFAS) Neurotoxicity in Sentinel and Non-Traditional Laboratory Model Systems: Potential Utility in Predicting Adverse Outcomes in Human Health. *Toxics* **8**(2).
17. Foguth, R. M., Hoskins, T. D., Clark, G. C., Nelson, M., Flynn, R. W., de Perre, C., Hoverman, J. T., Lee, L. S., Sepulveda, M. S., and Cannon, J. R. (2020). Single and mixture per- and polyfluoroalkyl substances accumulate in developing Northern leopard frog brains and produce complex neurotransmission alterations. *Neurotoxicology and teratology* **81**, 106907.
18. Syeda, T., Foguth, R. M., Llewellyn, E., and Cannon, J. R. (2020). PhIP exposure in rodents produces neuropathology potentially relevant to Alzheimer's disease. *Toxicology* **437**, 152436.
19. Bentz, B. Z., Mahalingam, S. M., Ysselstein, D., Montenegro, P. C., Cannon, J. R., Rochet, J. C., Low, P. S., and Webb, K. J. (2020). Localization of fluorescent targets in deep tissue with expanded beam illumination for studies of cancer and the brain. *IEEE Trans Med Imaging* doi: 10.1109/TMI.2020.2972200.
20. Lawana, V., Um, S. Y., Rochet, J. C., Turesky, R. J., Shannahan, J. H., and Cannon, J. R. (2020). Neuromelanin Modulates Heterocyclic Aromatic Amine-Induced Dopaminergic Neurotoxicity. *Toxicological sciences : an official journal of the Society of Toxicology* **173**(1), 171-188.
21. Sammi, S. R., Foguth, R. M., Nieves, C. S., De Perre, C., Wipf, P., McMurray, C. T., Lee, L. S., and Cannon, J. R. (2019). Perfluorooctane Sulfonate (PFOS) Produces Dopaminergic Neuropathology in Caenorhabditis elegans. *Toxicological sciences : an official journal of the Society of Toxicology* **172**(2), 417-434, 10.1093/toxsci/kfz191.
22. Patel, S. H., Yue, F., Saw, S. K., Foguth, R., Cannon, J. R., Shannahan, J. H., Kuang, S., Sabbaghi, A., and Carroll, C. C. (2019). Advanced Glycation End-Products Suppress Mitochondrial Function and Proliferative Capacity of Achilles Tendon-Derived Fibroblasts. *Sci Rep* **9**(1), 12614, 10.1038/s41598-019-49062-8.
23. Foguth, R. M., Flynn, R. W., de Perre, C., Iacchetta, M., Lee, L. S., Sepulveda, M. S., and Cannon, J. R. (2019). Developmental exposure to perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) selectively decreases brain dopamine levels in Northern leopard frogs. *Toxicology and applied pharmacology* **377**, 114623, 10.1016/j.taap.2019.114623.
24. Sanyal, A., Dutta, S., Camara, A., Chandran, A., Koller, A., Watson, B. G., Sengupta, R., Ysselstein,

D., Montenegro, P., Cannon, J., Rochet, J. C., and Mattoo, S. (2019). Alpha-Synuclein Is a Target of Fic-Mediated Adenylylation/AMPylation: Possible Implications for Parkinson's Disease. *Journal of molecular biology* **431**(12), 2266-2282, 10.1016/j.jmb.2019.04.026.

25. Weera, M. M., Agim, Z. S., Cannon, J. R., and Chester, J. A. (2019). Genetic correlations between nicotine reinforcement-related behaviors and propensity toward high or low alcohol preference in two replicate mouse lines. *Genes Brain Behav* **18**(3), e12515, 10.1111/gbb.12515.
26. Fernandez, R. F., Kim, S. Q., Zhao, Y., Foguth, R. M., Weera, M. M., Counihan, J. L., Nomura, D. K., Chester, J. A., Cannon, J. R., and Ellis, J. M. (2018). Acyl-CoA synthetase 6 enriches the neuroprotective omega-3 fatty acid DHA in the brain. *Proceedings of the National Academy of Sciences of the United States of America* **115**(49), 12525-12530, 10.1073/pnas.1807958115.
27. Wise, J. P., Jr., Price, C. G., Amaro, J. A., Cannon, J. R., (2018). Autophagy Disruptions Associated With Altered Optineurin Expression in Extranigral Regions in a Rotenone Model of Parkinson's Disease. *Front Neurosci*. **12,** 289.
28. Adamson, S. X., Shen, X., Jiang, W., Lai, V., Wang, X., Shannahan, J. H., Cannon, J. R., Chen, J., and Zheng, W. (2018). Subchronic Manganese Exposure Impairs Neurogenesis in the Adult Rat Hippocampus. *Toxicological sciences : an official journal of the Society of Toxicology* **163**(2), 592-608, 10.1093/toxsci/kfy062.
29. Agim, Z. S., and Cannon, J. R. (2018). Alterations in the nigrostriatal dopamine system after acute systemic PhIP exposure. *Toxicology letters* **287**, 31-41, 10.1016/j.toxlet.2018.01.017.
30. Cruz-Hernandez, A., Agim, Z. S., Montenegro, P. C., McCabe, G. P., Rochet, J. C., and Cannon, J. R. (2018). Selective dopaminergic neurotoxicity of three heterocyclic amine subclasses in primary rat midbrain neurons. *Neurotoxicology* **65**, 68-84, 10.1016/j.neuro.2018.01.009.
31. #Sammi, S. R., Agim, Z. S., and Cannon, J. R. (2018). From the Cover: Harmane-Induced Selective Dopaminergic Neurotoxicity in Caenorhabditis elegans. *Toxicological sciences : an official journal of the Society of Toxicology* **161**(2), 335-348, 10.1093/toxsci/kfx223.
32. Park, J., Lee, J. W., Cooper, S. C., Broxmeyer, H. E., Cannon, J. R., and Kim, C. H. (2017). Parkinson disease-associated LRRK2 G2019S transgene disrupts marrow myelopoiesis and peripheral Th17 response. *J Leukoc Biol* doi: 10.1189/jlb.1A0417-147RR, 10.1189/jlb.1A0417-147RR.
33. #Wise, J. P., Jr., and Cannon, J. (2016). From the Cover: Alterations in Optineurin Expression and Localization in Pre-clinical Parkinson's Disease Models. *Toxicological sciences : an official journal of the Society of Toxicology* **153**(2), 372-81, 10.1093/toxsci/kfw133.
34. Dukes, A. A., Bai, Q., Van Laar, V. S., Zhou, Y., Ilin, V., David, C. N., Agim, Z. S., Bonkowsky, J. L., Cannon, J. R., Watkins, S. C., Croix, C. M., Burton, E. A., and Berman, S. B. (2016). Live imaging of mitochondrial dynamics in CNS dopaminergic neurons in vivo demonstrates early reversal of mitochondrial transport following MPP+ exposure. *Neurobiol Dis* **95**, 238-249, 10.1016/j.nbd.2016.07.020.
35. Fu, S., Jiang, W., Gao, X., Zeng, A., Cholger, D., Cannon, J., Chen, J., and Zheng, W. (2016). Aberrant Adult Neurogenesis in the Subventricular Zone-Rostral Migratory Stream-Olfactory Bulb System Following Subchronic Manganese Exposure. *Toxicological sciences : an official journal of the Society of Toxicology* **150(2)**, 347-368, 10.1093/toxsci/kfw007.
36. Lee, J. W., and Cannon, J. R. (2015). LRRK2 mutations and neurotoxicant susceptibility. *Experimental biology and medicine* **240**(6), 752-9, 10.1177/1535370215579162.

37. Agim, Z. S., and Cannon, J. R. (2015). Dietary factors in the etiology of Parkinson's disease. *BioMed research international* **2015**, 672838, 10.1155/2015/672838.
38. Zharikov, A. D.*, Cannon, J. R.*, Tapias, V.[1], Bai, Q., Horowitz, M. P., Shah, V., El Ayadi, A., Hastings, T. G., Greenamyre, J. T., and Burton, E. A. (2015). shRNA targeting alpha-synuclein prevents neurodegeneration in a Parkinson's disease model. *The Journal of clinical investigation* **125**(7), 2721-35, 10.1172/JCI64502. *Joint 1st authors.
39. Wirbisky, S. E., Weber, G. J., Sepulveda, M. S., Xiao, C., Cannon, J. R., and Freeman, J. L. (2015). Developmental origins of neurotransmitter and transcriptome alterations in adult female zebrafish exposed to atrazine during embryogenesis. *Toxicology* **333**, 156-167, 10.1016/j.tox.2015.04.016.
40. Robison, G., Sullivan, B., Cannon, J. R., and Pushkar, Y. (2015). Identification of dopaminergic neurons of the substantia nigra pars compacta as a target of manganese accumulation. *Metallomics : integrated biometal science* **7**(5), 748-55, 10.1039/c5mt00023h.
41. Lee, J. W., Tapias, V., Di Maio, R., Greenamyre, J. T., and Cannon, J. R. (2015). Behavioral, neurochemical, and pathologic alterations in bacterial artificial chromosome transgenic G2019S leucine-rich repeated kinase 2 rats. *Neurobiology of aging* **36**(1), 505-18, 10.1016/j.neurobiolaging.2014.07.011.
42. Di Maio, R., Cannon, J. R., and Timothy Greenamyre, J. (2015). Post-status epilepticus treatment with the cannabinoid agonist WIN 55,212-2 prevents chronic epileptic hippocampal damage in rats. *Neurobiol Dis* **73C**, 356-365, 10.1016/j.nbd.2014.10.018.
43. O'Neal, S. L., Lee, J. W., Zheng, W., and Cannon, J. R. (2014). Subacute manganese exposure in rats is a neurochemical model of early manganese toxicity. *Neurotoxicology* **44C**, 303-313, 10.1016/j.neuro.2014.08.001.
44. Griggs, A. M., Agim, Z. S., Mishra, V. R., Tambe, M. A., Director-Myska, A. E., Turteltaub, K. W., McCabe, G. P., Rochet, J. C., and Cannon, J. R. (2014). 2-Amino-1-methyl-6-phenylimidazo[4,5-b]pyridine (PhIP) is selectively toxic to primary dopaminergic neurons in vitro. *Toxicological sciences : an official journal of the Society of Toxicology* **140**(1), 179-89, 10.1093/toxsci/kfu060.
45. Wirbisky, S. E., Weber, G. J., Lee, J. W., Cannon, J. R., and Freeman, J. L. (2014). Novel dose-dependent alterations in excitatory GABA during embryonic development associated with lead (Pb) neurotoxicity. *Toxicology letters* **229**(1), 1-8, 10.1016/j.toxlet.2014.05.016.
46. Wang, Y., Lee, J. W., Oh, G., Grady, S. R., McIntosh, J. M., Brunzell, D. H., Cannon, J. R., and Drenan, R. M. (2014). Enhanced synthesis and release of dopamine in transgenic mice with gain-of-function alpha6* nAChRs. *Journal of neurochemistry* **129**(2), 315-27, 10.1111/jnc.12616.
47. Tapias, V., Cannon, J. R., and Greenamyre, J. T. (2014). Pomegranate juice exacerbates oxidative stress and nigrostriatal degeneration in Parkinson's disease. *Neurobiology of aging* **35**(5), 1162-76, 10.1016/j.neurobiolaging.2013.10.077.
48. Cannon, J. R., and Greenamyre, J. T. (2013). Gene-environment interactions in Parkinson's disease: Specific evidence in humans and mammalian models. *Neurobiol Dis* **57C**, 38-46, 10.1016/j.nbd.2012.06.025.

49. *Cannon, J. R.**, Geghman, K. D.**, Tapias, V.[1], Sew, T., Dail, M. K., Li, C., and Greenamyre, J. T. (2013). Expression of human E46K-mutated alpha-synuclein in BAC-transgenic rats replicates early-stage Parkinson's disease features and enhances vulnerability to mitochondrial impairment. *Exp Neurol* **240**, 44-56, 10.1016/j.expneurol.2012.11.007. ***Received editorial commentary**. **Joint 1st authors.
50. #Milanese, C., Sager, J. J., Bai, Q., Farrell, T. C., Cannon, J. R., Greenamyre, J. T., and Burton, E. A. (2012). Hypokinesia and reduced dopamine levels in zebrafish lacking beta- and gamma1-synucleins. *The Journal of biological chemistry* **287**(5), 2971-83, 10.1074/jbc.M111.308312.
51. #Cannon, J. R., and Greenamyre, J. T. (2011). The role of environmental exposures in neurodegeneration and neurodegenerative diseases. *Toxicological sciences : an official journal of the Society of Toxicology* **124**(2), 225-50, 10.1093/toxsci/kfr239. ***Received editorial commentary.**
52. *Cannon, J. R., Sew, T., Montero, L., Burton, E. A., and Greenamyre, J. T. (2011). Pseudotype-dependent lentiviral transduction of astrocytes or neurons in the rat substantia nigra. *Exp Neurol* **228**(1), 41-52, 10.1016/j.expneurol.2010.10.016.
53. Tapias, V., Cannon, J. R., and Greenamyre, J. T. (2010). Melatonin treatment potentiates neurodegeneration in a rat rotenone Parkinson's disease model. *J Neurosci Res* **88**(2), 420-7, 10.1002/jnr.22201.
54. Greenamyre, J. T., Cannon, J. R., Drolet, R., and Mastroberardino, P. G. (2010). Lessons from the rotenone model of Parkinson's disease. *Trends Pharmacol Sci* **31**(4), 141-2; author reply 142-3, 10.1016/j.tips.2009.12.006.
55. Cannon, J. R., and Greenamyre, J. T. (2010). Neurotoxic in vivo models of Parkinson's disease recent advances. *Prog Brain Res* **184**, 17-33, 10.1016/S0079-6123(10)84002-6.
56. Cannon, J. R., and Greenamyre, J. T. (2009). NeuN is not a reliable marker of dopamine neurons in rat substantia nigra. *Neurosci Lett* **464**(1), 14-7, 10.1016/j.neulet.2009.08.023.
57. Drolet, R. E., Cannon, J. R., Montero, L., and Greenamyre, J. T. (2009). Chronic rotenone exposure reproduces Parkinson's disease gastrointestinal neuropathology. *Neurobiol Dis* **36**(1), 96-102, 10.1016/j.nbd.2009.06.017.
58. Cannon, J. R., Tapias, V., Na, H. M., Honick, A. S., Drolet, R. E., and Greenamyre, J. T. (2009). A highly reproducible rotenone model of Parkinson's disease. *Neurobiol Dis* **34**(2), 279-90.
59. Cannon, J. R., Hua, Y., Richardson, R. J., Xi, G., Keep, R. F. and Schallert, T. (2007). The effect of thrombin on a 6-hydroxydopamine model of Parkinson's disease depends on timing. *Behav Brain Res* **183**(2), 161-8, 10.1016/j.bbr.2007.06.004.
60. Cannon, J. R., Xi, G., and Keep, R. F. (2007). Recent research on changes in genomic regulation and protein expression in intracerebral haemorrhage. *International journal of stroke : official journal of the International Stroke Society* **2**(4), 265-9, 10.1111/j.1747-4949.2007.00160.x.
61. Cannon, J. R., Keep, R. F., Schallert, T., Hua, Y., Richardson, R. J. and Xi, G. (2006). Protease-activated receptor-1 mediates protection elicited by thrombin preconditioning in a rat 6-hydroxydopamine model of Parkinson's disease. *Brain Res* **1116**(1), 177-86, 10.1016/j.brainres.2006.07.094.
62. Cannon, J. R., Nakamura, T., Keep, R. F., Richardson, R. J., Hua, Y. and Xi, G. (2006). Dopamine changes in a rat model of intracerebral hemorrhage. *Acta Neurochir Suppl* **96**, 222-6.

63. Cannon, J. R., Keep, R. F., Hua, Y., Richardson, R. J., Schallert, T., and Xi, G. (2005). Thrombin preconditioning provides protection in a 6-hydroxydopamine Parkinson's disease model. *Neurosci Lett* **373**(3), 189-94, 10.1016/j.neulet.2004.10.089.
64. Lukacs, J. L., Chilimigras, J. L., Cannon, J. R., Dormire, S. L., and Reame, N. E. (2004). Midlife women's responses to a hospital sleep challenge: aging and menopause effects on sleep architecture. *J Womens Health (Larchmt)* **13**(3), 333-40, 10.1089/154099904323016491.

Submitted

1. Currim, F., Brown-Leung, J. M., Syeda, T., Corson, M., Schumann, S., Qi, W., Baloni, P., Shannahan, J., Rochet, J., Singh, R., and Cannon, J. Rotenone induced acute exosomal miRNA alterations in rat cerebrospinal fluid and serum induce mitochondrial dysfunction and cell death. *NPJ Parkinsons Dis.* In revision.

Book Chapters

1. Brown-Leung, J. M., and Cannon, J. R. (2023). Neurochemical mechanisms of perfluoroalkyl substances (PFAS) neurotoxic action. In Advances in Neurotoxicology (doi: https://doi.org/10.1016/bs.ant.2023.08.002. Academic Press.
2. Foguth, R., and Cannon, J. (2022). Emerging Contaminants as Contributors to Parkinsonism: Heterocyclic Amines. In Parkinsonism and the Environment (N. M. Filipov, Ed.) doi: 10.1007/978-3-030-87451-3_2, pp. 19-37. Springer International Publishing, Cham.
3. Lawana, V., and Cannon, J. R. (2020). Rotenone Neurotoxicity: Relevance to Parkinson's Disease. *Advances in Neurotoxicology*; In *Neurotoxicity of Pesticides* (M. Aschner, and L. G. Costa, Eds.), Vol. 4. Elsevier. 209-254.
4. Agim, Z. S., and Cannon, J. R. (2017). Dietary Anti-, Pro-Oxidants in the Etiology of Parkinson's Disease. In *Oxidative Stress and Redox Signaling in Parkinson's Disease* (R. Franco, J. A. Doorn, and J. C. Rochet, Eds.) doi: http://dx.doi.org/10.1039/9781782622888, pp. 447-504. Royal Society of Chemistry Croydon, UK.
5. Wise, J. P., Jr., Cannon, J. R. (2016). Recent Advances in the Exploration of Gene-Environment Interactions in Parkinson's Disease. In: L. Qi, (Ed.), Gene-Environment Interactions and Human Diseases. Nova Publishers, New York, 2016, pp. 171-208.
6. Cannon, J. R., Greenamyre, J. T. (2014). Rotenone as preclinical model compound in Parkinson disease. In: R. M. Kostrzewa, (Ed.), Handbook of Neurotoxicity Springer, New York, pp. 995-1012.
7. Cannon, J. R., Greenamyre, J. T. (2012). Environmental Toxins and Parkinson's Disease. In: R. Pfeiffer, et al., Eds.), Parkinson's disease. CRC Press, Boca Raton

EDITORIAL (inclusive of non-peer reviewed)

1. Volz, D. C., Cannon, J., and Tal, T. (2021). Introduction to leveraging non-mammalian models for developmental neurotoxicity testing. In (Vol. 87, pp. 107001.

ABSTRACTS

1. Tanwar, R., Doepeker, A., Wells, E., Cannon, J., Parkinson's disease risk and mechanisms from military relevant organophosphate exposures. 8th Rijeka Forum On Neurodegenerative Diseases: The Regulation Of Gene Expression In Neurological Disease And Neuroimmunology. 26-27.
2. Sammi, S. R., Cannon, J. R., 2024. Gamma-glutamyl-cysteine is critical for glutathione-mediated alleviation of PFOS neurotoxicity. The Toxicologist. 198, 4078.
3. Brown-Leung, J. M., H., P., Currim, F., Syeda, T., Cannon, J. R., 2024. Chronic Perfluorooctanesulfonic Acid (PFOS) Induces Feminized Neurobehavior in Male Mice. The Toxicologist. 198, 3574a.
4. Currim, F. M., Brown-Leung, J. M., Syeda, T., Rochet, J., Singh, R., Cannon, J., 2024. Exosomal miRNAs in biofluids of the rotenone-induced rat model of Parkinson's Disease induce dopaminergic neuron cell death in primary midbrain neurons. The Toxicologist. 198, 3134.
5. Cannon, J. R., Brown-Leung, J. M., 2023. Per- and polyfluorinated substances (PFAS) neurotoxicity and potential public health implications. International Conference on Pollutant Toxic Ions and Molecules. 5th 109.
6. Cannon, J.R., Currim, F. 2023. Exosomal miRNA alterations in rotenone models of Parkinson's Disease, MOL.07, p. 49, Book of Abstracts, Slovenian Neuroscience Association (SiNAPSA) Neuroscience Conference '23, Ljubljana, Slovenia
7. Brown-Leung, J. M., Syeda, T., Currim, F. M., Cannon, J. R., 2023. Chronic Perfluorooctanesulfonic Acid (PFOS) Induces Hyperactivity and Deficits in Nonassociative Learning in Male but Not Female Mice. The Toxicologist. 192, 3765.
8. Sammi, S., Cannon, J., 2023. Critical Role of Mitochondrial Carrier Protein in PFOS-led toxicity The Toxicologist. 192, 4020.
9. Syeda, T., Sammi, S., Cannon, J., 2023. Heterocyclic aromatic amines (HAAs) target mitochondrial physiology The Toxicologist. 192, 3913.
10. Rivera, A., Stephens, A., Jameson, L., Griffith, S., Sammi, S., Cannon, J., Leung, M., 2023. Potential Interaction of Cannabinoids and Insecticides in Causing Dopaminergic Neurotoxicity. The Toxicologist. 192, 4550.
11. Rubinchik, A. B., Tukker, A. M., Nolan, J. K., Gupta, K., Lake-Speers, M. A., Sammi, S. R., Cannon, J. R., Wereley, S. T., Lee, H., Bowman, A. B., 2023. Chlorpyrifos and Chlorpyrifos-Oxon Exposures Lead to the Dysregulation of Basal Glutamate Release in Human-Induced Pluripotent Stem Cell–Derived Glutamatergic Neurons. The Toxicologist. 192, 2017.
12. Enkh-Amgalan, S., Brown, J., Syeda, T., Foguth, R., Cannon, J., Chester, J., EFFECTS OF THE ENVIRONMENTAL TOXICANT, PARAQUAT, ON ALCOHOL-INDUCED LOCOMOTOR SENSITIZATION IN HIGH AND LOW-ALCOHOL-PREFERRING MICE. ALCOHOLISM-CLINICAL AND EXPERIMENTAL RESEARCH, Vol. 46. WILEY 111 RIVER ST, HOBOKEN 07030-5774, NJ USA, 2022, pp. 188A-188A.
13. Syeda, T., Min, S., Rochet, J., Cannon, J. R., 2022. Perfluorooctane Sulfonate (PFOS) Induce Alzheimer's Disease-Relevant Neurotoxicity The Toxicologist. 186, 2057.
14. Sammi, S. R., Cannon, J. R., 2022. Mitochondrial Targets of Perfluorooctane Sulfonic Acid (PFOS)-Mediated Neurotoxiciy. The Toxicologist. 186, 3071.
15. Brown, J. M., Syeda, T., Cannon, J. R., 2022. Perfluorooctanesulfonic Acid (PFOS)-Induced Catecholaminergic Neurotransmission Changes in Female Mice. The Toxicologist. 186, 3074.
16. Rubinchik, A. B., Tukker, A. M., Sammi, S. R., Cannon, J. R., Bowman, A. B., 2022. Chlorpyrifos and Chlorpyrifos-Oxon Exposure Leads to the Dysregulation of Basal Glutamate Release in Human-Induced Pluripotent Stem Cell-Derived Glutamatergic Neurons. The Toxicologist. 186, 3854.
17. Jameson, L., Rivera, A., Conrow, K., Pinhasova, N., Jourabchin, S., Johnson, A., Davis, A., Wiegers, T., Sammi, S., Mattingly, C., Afia, I., Orser, I., Cannon, J., Leung, M., 2022. Mechanism of Neurological Hazards from Insecticide Exposure in Cannibus The Toxicologist. 186, 3861.
18. Sammi, S. R. , Syeda, T., Cannon, J. R., 2021. Chlorpyriphos and Chlorpyriphos-Oxon Produce Dopaminergic Toxicity in C. elegans. The Toxicologist. 180, 2653.
19. Syeda, T., Min, S., Rochet, J. C., Cannon, J. R., 2021. Heterocyclic Aromatic Amines Promote Protein Aggregation and Induce Alzheimer's Disease-Relevant Neurotoxicity. The Toxicologist. 180, 2655.
20. Brown, J. M., Hoskins, T. D., Foguth, R. M., Syeda, T., Perez, M. S., Sepulveda, M. M., Cannon, J. R., 2021. Characterization of the Gray Tree Frog (Hyla versicolor) as a Potential Model for Developmental Neurotoxicity. The Toxicologist. 180, 2436.

21. Foguth, R., Hoskins, T. D., Clark, G. C., Nelson, M., de Perre, C., Lee, L., Sepulveda, M. M., Cannon, J. R., 2020. Effects of Developmental Exposure to Perfluoroalkyl Substances on Brain and Heart Innervation in Northern Leopard Frogs. The Toxicologist. 174**,** 2439.
22. Lawana, V., Um, S., Chandran, A., Shannahan, J. H., Cannon, J. R., 2020. Mitochondrial and Autophagolysosomal Dysfunction Induced by Heterocyclic Aromatic Amine Exposure Is Modulated by Neuromelanin Formation. The Toxicologist. 174**,** 1450.
23. Sammi, S. R., Nieves, C. S., Cannon, J. R., 2020. Mitochondrial Mechanisms of Harmane-Induced Dopaminergic Neurotoxicity in C. elegans. The Toxicologist. 174**,** 1442.
24. Syeda, T., Foguth, R., Cannon, J., 2020. PhIP Exposure: Alzheimer's Disease Relevant Neurotoxicity. The Toxicologist. 174**,** 1429.
25. Lawana, V., Um, S. Y., Shannahan, J., Cannon, J. R., 2019. The potential role of neuromelanin in modulating heterocyclic amine-induced dopaminergic neurotoxicity. Gordon Research Conference, Parkinson's disease: Pathogenesis, Pathophysiology and Experimental Therapeutics in Parkinson's Disease.
26. Syeda, T., Foguth, R., Cannon, J., 2020. PhIP Exposure: Alzheimer's Disease Relevant Neurotoxicity. The Toxicologist. 174**,** 1429.
27. Sammi, S. R., Cannon, J. R., 2019. PFOS produces selective dopaminergic neurotoxicity in C. elegans. Gordon Research Conference, Parkinson's disease: Pathogenesis, Pathophysiology and Experimental Therapeutics in Parkinson's Disease.
28. Foguth, R., Flynn, R., Sepulvada, M. M., Cannon, J. R., 2019. Developmental exposure to PFOS and PFOA produces selective dopamine decreases in leopard frogs. Gordon Research Conference, Parkinson's disease: Pathogenesis, Pathophysiology and Experimental Therapeutics in Parkinson's Disease.
29. Chandran, A., Montenegro-Larrea, P. C., Chandrasekaran, C., Hensel, M., Dehay, B., Moon, J., Zhang, M., Bezard, E., Cannon, J., Rochet, J. C., 2019. Neuroprotective effects of the transcription factor Nfe2L1 in PD models. Cerebbral Symposium, Purdue University. 06.
30. Chandrasekaran, C., Montenegro-Larrea, P. C., Hensel, J., Dutta, S., Faggiani, E., Acosta, G., Herr, S., Tang, J., Shi, R., Dehay, B., Bezard, E., Cannon, J., Rochet, J. C., 2019. Effects of the alpha-synuclein-interacting protein endosulfine-alpha on PD-related neuropathology. Cerebbral Symposium, Purdue University. 04.
31. Foguth, R. M., Flynn, R. W., Sepulvada, M. S., Cannon, J. R., 2019. Developmental exposure to PFOS and PFOA produces selective dopamine decreases in Northern leopard frogs. Cerebbral Symposium, Purdue University 03.
32. Syeda, T., Foguth, R. M., Cannon, J. R., 2019. Systemic PhIP exposure produces neurotoxicity of potential that is relevant to Alzheimer's disease. Annual Meeting of the Greater Indiana Society for Neuroscience. 2, 42.
33. Sammi, S. R., Cannon, J. R., 2019. PFOS produces selective dopaminergic neurotoxicity in C. elegans. Annual Meeting of the Greater Indiana Society for Neuroscience. 2, 38.
34. Lawana, V., Um, S. E., Shannahan, J., Cannon, J., 2019. The potential role of neuromelanin in modulating heterocyclic amineinduced dopaminergic neurotoxicity. Annual Meeting of the Greater Indiana Society for Neuroscience. 1**,** 49.
35. Sammi, S. R., Cannon, J. R., 2019. Perfluorooctanesulfonic Acid (PFOS) Is Selectively Neurotoxic to Dopaminergic Neurons in C. elegans. The Toxicologist. 168**,** 1722.
36. Foguth, R. M., Cannon, J. R., 2019. Neurobehavioral and Neurochemical Effects of Acute to Subchronic PhIP Exposure. The Toxicologist. 168**,** 1310.
37. Malek, E., Agim, Z. S., Foguth, R. M., Cannon, J. R., 2018. The Effects of Heterocyclic Amines on Dopamine Metabolism. 2018 Greater Indiana Society for Neuroscience Annual Meeting. 86.
38. Foguth, R. M., Agim, Z. S., Cannon, J. R., 2018. Heterocyclic Amine-Induced Alterations in Neuronal Bioenergetics. 2018 Greater Indiana Society for Neuroscience Annual Meeting. 81.
39. Fernandez, R. F., Kim, S., Zhao, Y., Counihan, J. L., Nomura, D. K., Chester, J. A., Cannon, J. R., Ellis, J. M., 2018. Long-chain Acyl-CoA Synthetase 6 regulates brain docosahexaenoic fatty acid metabolism and confers neuroprotection. 2018 Greater Indiana Society for Neuroscience Annual Meeting. 80.
40. Sammi, S. R., Agim, Z. S., Cannon, J. R., 2018. Mechanisms of harmane-induced selective dopaminergic neurotoxicity in C. elegans. 2018 Greater Indiana Society for Neuroscience Annual Meeting. 70.

41. Montenegro, P., Ysselstein, D., Agim, S., Dutheil, N., Hensel, J., Dehay, B., Bezard, E., Cannon, J. R., Rochet, J. C., 2018. Effects of the A53E substitution on alpha synuclein aggregation and neurotoxicity in Parkinson's disease models. 2018 Greater Indiana Society for Neuroscience Annual Meeting 68.
42. Cannon, J. R., 2018. Potential for autophagy as a primary mechanism of environmentally induced neurodegeneration. The Toxicologist. 162, 3232.
43. Wise, J. P., Price, C., Amaro, J., Nkera, B., Cannon, J. R., 2018. Autophagic disruption in brainstem nuclei in a preclinical Parkinson's disease model. 2018 Greater Indiana Society for Neuroscience Annual Meeting.
44. Wise, J. P., Price, C., Amaro, J., Nkera, B., Cannon, J. R., 2018. Autophagic disruption in brainstem nuclei in a preclinical Parkinson's disease model. The Toxicologist. 162, 2226.
45. Sammi, S. R., Agim, Z. S., Cannon, J. R., 2018. Mechanisms of harmane-induced selective dopaminergic neurotoxicity in Caenorhabditis elegans. The Toxicologist. 162, 2222.
46. Foguth, R. M., Agim, Z. S., Cannon, J. R., 2018. Heterocyclic amine induced bionenergetics alterations. The Toxicologist. 162, 1460.
47. Foguth, R. M., Agim, Z. S., Cannon, J. R., 2017. Heterocyclic amine-induced bioenergetics alterations. Ohio Valley Society of Toxicology Annual Meeting. 46.
48. Agim, Z. S., Cannon, J. R., 2017. Alterations in the nigrostriatal dopamine system after acute systemic PhIP exposure. Ohio Valley Society of Toxicology Annual Meeting. 42.
49. Sammi, S. R., Agim, Z. S., Cannon, J. R., 2017. Mechanisms of harmane-induced selective dopaminergic neurotoxicity in C. elegans. Ohio Valley Society of Toxicology Annual Meeting. 18.
50. Wise, J. P., Jr., Price, C., Amaro, J., Nkera, B., Cannon, J. R., 2017. Autophagic dysfunction in brainstem nuclei in a preclinical Parkinson's disease model. Ohio Valley Society of Toxicology Annual Meeting. 16.
51. Wise, J. P., Jr., Rochet, J. C., Cannon, J. R., 2017. Autophagy disruptions in preclinical to end-stage animal models of Parkinson's disease. EMBO Conference. Autophagy: From Molecular Principles to Human Disease. P3-047.
52. Nkera, B. Wise, J. Cannon, J.R., 2017. Evaluation of alpha-synuclein and optineurin colocalization in extranigral regions of end-stage Parkinson's disease. 2017 Summer Research Opportunity Poster Presentation. Purdue University. #29
53. Mestres-Villanueva, M. A., Wise, J. P., Jr., Cannon, J. R., 2017. Optineurin Distribution in Extranigral Nuclei: Altered Expression in Parkinson's Disease. The Toxicologist. 156, 2875.
54. Agim, Z. S., Jacquet, A., Rochet, J. C., Cannon, J. R., 2017. Mechanisms of PhIP-Induced Dopaminergic Neurotoxicity in Primary Midbrain Cultures. The Toxicologist. 156, 1423.
55. Wise, J., Cannon, J., 2017. The Role of Optineurin in Golgi Fragmentation during Parkinson's Disease. The Toxicologist. 156, 1421.
56. Cruz-Hernandez, A. A., Agim, Z. S., Montenegro, P., Rochet, J. C., Cannon, J. R., 2017. Comparative Dopaminergic Neurotoxicity Comparative Dopaminergic Neurotoxicity. The Toxicologist. 156, 1420.
57. Fu, X., Shen, X., Jiang, W., Lai, V., Wang, X., Cannon, J., Chen, J., Zheng, W., 2017. Subchronic Manganese (Mn) Exposure Impairs Hippocampal Neurogenesis in Adult Rats. The Toxicologist. 156, 1400.
58. Amaro, J.A., Wise, J.P., Jr., Cannon. J.R. 2017. Changes in optineurin distribution in dopaminergic neurons in a rotenone model of Parkinson's disease. College of Health and Human Sciences, Research Symposium, Purdue University.
59. Wise, J. P., Jr., Cannon, J. R., 2016. Optineurin expression in the nigrostriatal dopamine system: normal expression and response to rotenone treatment. XIV International Congress of Toxicology. PP21.6.
60. Agim, Z. S., Cannon, J. R., 2016. Dopaminergic neurotoxicity of PHIP in vivo. XIV International Congress of Toxicology. PP21.2.
61. Agim, Z. S., Jacquet, A., Rochet, J. C., Cannon, J. R., 2016. Mechanism of PHIP_induced doapminergic toxicity in primary midbrain cultures. XIV International Congress of Toxicology PP21.1.
62. Montenegro-Larrea, P. C., Wise, J., Head, K. J., Dehay, B., Bezard, E., Jelinek, J., Liu, Y., Cannon, J. R., Nephew, K., Rochet, J. C., March, 2016. Role of aberrant expression of neuroprotective genes in DLB and other synucleinopathy disorders. Indianapolis Society for Neuroscience Annual Meeting.

63. Wise, J., Cannon, J. R., 2016. Optineurin Expression in the Nigrostriatal Dopamine System in Normal and Rotenone-Treated Rats. The Toxicologist. 150, 2376.
64. Agim, Z. S., Jacquet, A., Rochet, J. C., Cannon, J. R., 2016. Mechanisms of PhIP-Induced Dopaminergic Neurotoxicity. The Toxicologist. 150, 2372.
65. Fu, X., Jiang, W., Gao, X., Zeng, A., Cholger, D., Cannon, J., Chen, J., Zheng, W., 2016. Aberrant Adult Neurogenesis in the Subventricular Zone (SVZ)-Rostral Migratory Stream (RMS)-Olfactory Bulb (OB) System Following Subchronic Manganese (Mn) Exposure. The Toxicologist. 150, 2366.
66. Cannon, J. R., 2016. PhIP Exposure and Dopaminergic Neuron Toxicity. The Toxicologist. 150, 1022.
67. Cannon, J. R., Turteltaub, K. W., 2016. Dietary Exposures to Heterocyclic Amines as a Potential Risk Factor for Neurological Disease. The Toxicologist. 150, 1019.
68. Wise, J., Agim, Z. S., Tambe, M. A., Mishra, V., Rochet, J. C., Cannon, J. R., 2015. Altered Optineurin Expression in Cellular and Rodent Models of Parkinson's Disease. Proceedings of Health and Disease: Science, Culture and Policy Research Poster Session. Purdue Research Day.
69. Agim, Z. S., Lee, J.-W., Rochet, J. C., Cannon, J. R., 2015. 2-Amino-1-methyl-6-phenylimidazo(4, 5-B)pyridine (PhIP) Dopaminergic Neurotoxicity in Primary Midbrain Cultures and In Vivo. Proceedings of Health and Disease: Science, Culture and Policy Research Poster Session. Purdue Research Day.
70. Wirbisky, S. E., Weber, G. J., Sepulvada, M. M., Xiao, C., Cannon, J. R., Freeman, J. L., Serotonin and Transcriptome Alterations in Brain Tissueof Adult Female Zebrafish Exposed to Atrazine During Embryogenesis. The Toxicologist. 144, 1700. Society of Toxicology Annual Meeting.
71. Lee, J.-W., Park, J., Kim, C. H., Cannon, J. R., Differential Effects of Hapten-Induced Experimental Colitis on G2019S LRRK2 Transgenic and Wild-Type Rats. The Toxicologist. 144, 1536. Society of Toxicology Annual Meeting.
72. Wise, J., Agim, Z. S., Tambe, M. A., Mishra, V., Rochet, J. C., Cannon, J. R., 2015. Altered Optineurin Expression in Cellular and Rodent Models of Parkinson's Disease. The Toxicologist. 144, 1525. Society of Toxicology Annual Meeting.
73. Agim, Z. S., Lee, J.-W., Rochet, J. C., Cannon, J. R., 2015. 2-Amino-1-methyl-6-phenylimidazo(4, 5-B)pyridine (PhIP) Dopaminergic Neurotoxicity in Primary Midbrain Cultures and In Vivo. The Toxicologist. 144, 1522. Society of Toxicology Annual Meeting.
74. Agim, Z. S., Griggs, A. M., Mishra, V., Turteltaub, K. W., Director-Myska, A. E., Rochet, J. C., Cannon, J. R., 2014. 2-Amino-1methyl-6-phenylimidazo(4,5-b)pyridine (PhIP) neurotoxicity in primary midbrain cultures. Health and Disease: Science, Culture and Policy graduate student poster competition, Purdue University.
75. Horin, A., Agim, Z. S., Lee, J.-W., Wise, J., Cannon, J. R., 2014. Neurotoxicity and gene-environment interactions in Parkinson's disease. Twenty second Annual Undergraduate Research Day. Department of Biological Sciences. Purdue University. #13.
76. Zheng, W., Pushkar, Y., Cannon, J. R., 2014. Accumulation of manganese in the subtantia nigra and alterations in brain neurochemistry following subchronic manganese exposure in rats. The Toxicologist. 138, 2107.
77. O'Neal, S. L., Lee, J.-W., Cannon, J. R., Zheng, W., 2014. Altered neurotransmitter levels in rats exposed to manganese: relevance to dopaminergic dysfunction and neurodegeneration. The Toxicologist. 138, 1361.
78. Wirbiski, S. E., Weber, G. J., Lee, J.-W., Cannon, J. R., Freeman, J. L., 2014. Lead-induced alterations within the excitatory GABAergic pathway during early embryonic zebrafish development. The Toxicologist. 138, 1338.
79. Lee, J.-W., Park, J., Kim, C. H., Cannon, J. R., 2014. LRRK2 transgenic rats exhibit heightened sensitivity in a colitis model. The Toxicologist. 138, 933.
80. Agim, Z. S., Griggs, A. M., Mishra, V., Turteltaub, K. W., Director-Myska, A. E., Rochet, J. C., Cannon, J. R., 2014. 2-Amino-1methyl-6-phenylimidazo(4,5-b)pyridine (PhIP) neurotoxicity in primary midbrain cultures. The Toxicologist. 138, 923.
81. Wise, J., Agim, Z. S., Tambe, M. A., Rochet, J. C., Cannon, J. R., 2014. Optineurin expression in dopaminergic neurons and response to Parkinson's disease relevant insults. The Toxicologist. 138, 378h.
82. Greenamyre, J. T., Cannon, J. R., Sanders, L. H., Tapias, V., Bai, Q., Volpicelli-Daley, L., Lee, V. M., and Burton, E. A. (2014). Synuclein-Mitochondrial Interactions in Substantia Nigra Dopamine Neurons A2 - Eiden, Lee E. In *Catecholamine Research in the 21st Century* (doi: https://doi.org/10.1016/B978-0-12-800044-1.00111-2, pp. 126-127. Academic Press, Boston.

83. Wang, Y., Lee, J.-W., Mcintosh, J. M., Brunzell, D. H., Cannon, J. R., Drenan, R. M., 2013. Increased activity of nicotinic acetylcholine receptors containing alpha6 subunits promotes dopamine tissue content and enhances dopamine release in nucleus accumbens. Program# 32.02. 2013 Neuroscience Meeting Planner. San Diego, CA: Society for Neuroscience, 2013. Online.
84. O'Neal, S. L., Cannon, J. R., Zheng, W., 2013. Altered Neurotransmitter Levels in Rats Subchronically Exposed to Manganese: Relevance to Dopaminergic Dysfunction and Neurodegeneration. Ohio Valley Society of Toxicology Annual Meeting.
85. Agim, Z. S., Griggs, A. M., Mishra, V., Rochet, J. C., Cannon, J. R., 2013. 2-Amino-1-methyl-6-phenylimidazo(4,5-b)pyridine (PhIP) neurotoxicity in primary midbrain cultures. Ohio Valley Society of Toxicology Annual Meeting
86. Griggs, A. M., Lee, J., Rochet, J. C., Turteltaub, K. W., Director-Myska, A. E., Cannon, J. R., 2013. Neurotoxicity of 2-amino-1-methyl-6-phenylimidazo[4,5-b]pyridine (PhIP). The Toxicologist. 132 Supplement, 2696.
87. Xiao, C., Weber, G. J., Watson, S. E., Freeman, J. L., Cannon, J. R., 2013. Linking Developmental Atrazine Exposure in Zebrafish to Long-Term Neurotransmission Alterations. The Toxicologist. 132, 1408.
88. Weber, G. J., Xiao, C., Watson, S. E., Freeman, J. L., Cannon, J. R., Developmental atrazine exposure in zebrafish leads to long-term neurotransmitter deficits. Program No. 354.23. Neuroscience Meeting Planner. New Orleans, LA: Society for Neuroscience, 2012. Online
89. Burton, E. A., Cannon, J., Bai, Q., Horowitz, M., Tapias, V., Shah, V., Sew, T., Ayadi, A. E., Greenamyre, J. T., Short hairpin RNA targeting endogenous α-synuclein prevents degeneration of dopaminergic neurons in the rotenone model of Parkinson's disease. Program No. 153.08. Neuroscience Meeting Planner. New Orleans, LA: Society for Neuroscience, 2012. Online.
90. Horowitz, M. P., Cannon, J. R., Sew, T., Montero, L., Bai, Q., Burton, E. A., Greenamyre, J. T., 2012. Transcriptional regulation of alpha-synuclein by GATA2 in substantia nigra. Program No. 53.26. Neuroscience Meeting Planner. New Orleans, LA: Society for Neuroscience, 2012. Online
91. Cannon, J. R., Geghman, K. D., Tapias, V., Sew, T., Dail, M., Nelson, J., Li, C., Greenamyre, J., 2012. Pathophysiology of BAC-transgenic rats expressing Disease Causing Mutations. The Toxicologist. 126, 979.
92. DiMaio, R., Cannon, J. R., Montero, L., Greenamyre, J., 2011. Cannabinoid 1 receptor as therapeutic target in chronic epilepsy. Program No. 560.09. Neuroscience Meeting Planner. Washington, DC: Society for Neuroscience, 2011. Online.
93. Cannon, J. R., Hoffman, E. K., Sew, T., Montero, L., Burton, E. A., Greenamyre, J. T., 2011. In vivo characterization and modulation of peroxidasin homolog (Drosophila)-like (PXDNL). Program No. 556.07. 2011 Neuroscience Meeting Planner. Washington, DC: Society for Neuroscience, 2011. Online.
94. Hoffman, E. K., DiMaio, R., Cannon, J. R., Greenamyre, J. T., 2011. Gene structure and functional characterization of a novel peroxidase (pxdnl) that confers resistance to oxidative stress in a human dopaminergic cell line. Program No. 556.03. Neuroscience Meeting Planner. Washington, DC: Society for Neuroscience, 2011. Online.
95. Horowitz, M. P., Cannon, J. R., Sew, T. J., Montero, L. M., Burton, E. A., Greenamyre, J. T., 2011. Targeting GATA2 to transcriptionally ameliorate alpha-synuclein and iron pathologies in Parkinson's disease. Program No. 357.14. Neuroscience Meeting Planner. Washington, DC: Society for Neuroscience, 2011. Online.
96. Greenamyre, J. T., Sew, T., Tapias, V., Montero, L., Geghman, K. D., Li, C., Cannon, J. R., 2011. Transgenic rats expressing Parkinson's disease genes: Characterization and toxicant sensitivity. Program No. 357.03. Neuroscience Meeting Planner. Washington, DC: Society for Neuroscience, 2011. Online.
97. Cannon, J. R., Sew, T., Tapias, V., Montero, L., Li, C., Geghman, K. D., Greenamyre, J. T., 2011. Transgenic rats expressing Parkinson's disease genes: characterization and toxicant sensitivity. Gordon Research Conference, Cellular & Molecular Mechanisms of Toxicity Understanding Innovative Mechanistic Toxicology in the Post-Genomic Era
98. Geghman, K. D., Cannon, J. R., Tapias, V. M., Sew, T., Li, C., Greenamyre, J. T. 2010. Characterization of BAC transgenic rats expressing mutated α-synuclein Program No. 857.5. Neuroscience Meeting Planner. San Diego, CA: Society for Neuroscience, 2010. Online.

99. Cannon, J. R., Sew, T., Montero, L., Greenamyre, J. T., Burton, E. A. 2010. Modulation of Parkinson's disease genes in a sporadic disease model Program No. 251.11. Neuroscience Meeting Planner. San Diego, CA: Society for Neuroscience, 2010. Online.
100. Drolet, R., Cannon, J. R., Montero, L. M., Greenamyre, J. T., 2009. Rotenone Recapitulates Parkinson's Disease-Related Gastrointestinal Motility Deficits and Alpha-Synuclein Pathology in the Enteric Nervous System. GASTROENTEROLOGY. 136, A578-A579.
101. Saporiti, F., Hu, X., Cannon, J. R., Hoffman, E. K., Greenamyre, J. T. 2009. Role of CD200-CD200R in rotenone model of Parkinson's disease Program No. 630.21. 2009 Neuroscience Meeting Planner. Chicago, IL: Society for Neuroscience, 2009. Online.
102. Drolet, R., Montero, L. M., Cannon, J. R., Greenamyre, J. T. 2009. Chronic rotenone reproduces Parkinson's disease-related alpha-synuclein pathology in the enteric nervous system and defects in GI motility Program No. 629.12. 2009 Neuroscience Meeting Planner. Chicago, IL: Society for Neuroscience, 2009. Online.
103. Tapias, V., Cannon, J. R., Greenamyre, J. T. 2009. Melatonin treatment potentiates neurodegeneration in a rat rotenone Parkinson's disease model Program No. 144.13. 2009 Neuroscience Meeting Planner. Chicago, IL: Society for Neuroscience, 2009. Online.
104. Cannon, J. R., El-Ayadi, A., Bai, Q., Shah, V., Greenamyre, J. T., Burton, E. A. 2009. Design and characterization of in vivo α-synuclein shRNA delivery Program No. 46.15. 2009 Neuroscience Meeting Planner. Chicago, IL: Society for Neuroscience, 2009. Online.
105. Geghman, K. D., Cannon, J. R., Ashrafi, S., Reddy, S., Schaffer, S., Liu, W., Greenamyre, J. T., Li, C. 2009. Characterization of bacterial artificial chromosome mediated transgenic rat models of parkinson's disease. Program No. 629.14. 2009 Neuroscience Meeting Planner. Chicago, IL: Society for Neuroscience, 2009. Online
106. Cannon, J. R., Na, H.M., Honick, A.S., Drolet, R.E., Greenamyre, J.T. Highly reproducible rotenone models of Parkinson's disease for neuroprotection studies. Program No. 48.22. 2008 Neuroscience Meeting Planner. Washington, DC: Society for Neuroscience, 2008. Online.
107. Greenamyre, J.T., Hu, X., Mastroberardino, P.G., Cannon, J. R., Vergun, O.V. Different sensitivity of substantia nigral and cortical mitochondria to calcium. Program No. 51.12. 2008 Neuroscience Meeting Planner. Washington, DC: Society for Neuroscience, 2008. Online.
108. Saporiti, F., Na, H.M., Hu, X., Cannon, J., Hoffman, E., Greenamyre, J. Role of CD200-CD200R in rotenone-induced degeneration of dopaminergic neurons. Program No.52.17. 2008 Neuroscience Meeting Planner. Washington, DC: Society for Neuroscience, 2008. Online.
109. Drolet, R., Cannon, J., Montero, L., Honick, T., Na, H., Greenamyre, J. Rotenone recapitulates Parkinson's disease-related gastrointestinal motility deficits and alpha-synuclein pathology in the enteric nervous system. Program No. 48.3. 2008 Neuroscience Meeting Planner. Washington, DC: Society for Neuroscience, 2008. Online.
110. Cannon, J. R., Honick, A.S., Na, H.M., Drolet, R.E., Greenamyre, J.T. Neuropathological characterization of chronic daily intraperitoneal rotenone in the rat. Program No. 904.14. 2007 Neuroscience Meeting Planner. San Diego, CA: Society for Neuroscience, 2007. Online.
111. Drolet, R., Cannon, J.R., Masa, R.M., Honick, A.S., Na, H., Greenamyre, J.T. Characterization of autonomic and enteric nervous system deficits in the rotenone model of Parkinson's Disease. Program No. 904.1. 2007 Neuroscience Meeting Planner. San Diego, CA: Society for Neuroscience, 2007. Online.
112. Cannon, J. R., Keep, R. F., Hua, Y., Schallert, T., Richardson, R. J., and Xi, G. Thrombin in experimental Parkinson's disease: administration with or after 6-OHDA. Program No. 755.4 2006 Neuroscience Meeting Planner. Atlanta, GA: Society for Neuroscience, 2006. Online.
113. Cannon, J.R., Xi, G., Hua Y, Schallert, T., Keep, R.F. 2006. Activation of the protease-activated receptor-1 mediates the protective effects of thrombin preconditioning in a Parkinson's disease model. The Toxicologist, 90, 1103
114. Cannon, J.R., Xi, G., Hua, Schallert, T., Keep., R.F. 2005. Thrombin preconditioning prevents dopaminergic terminal loss, but not dopamine depletion in a 6-hydroxydopamine Parkinson's disease model. The Toxicologist, 84, 1528
115. Cannon, J.R., Hua, Y., Keep, R.F. Schallert, T. Xi, G. 2004. Protection elicited by thrombin preconditioning in a Parkinson's disease model may be mediated through activation of the protease activated receptor 1, while inhibition may be neurotoxic. Michigan Society of Toxicology Fall Meeting

116. Cannon, J.R., Xi, G., Hua, Y. Schallert, T. Keep, R.F. 2004. Thrombin preconditioning provides non-dopaminergic mediated protection against 6-hydroxydopamine. Michigan Society of Toxicology Spring Meeting
117. Cannon, J.R., Xi G., Hua Y., Schallert T., and Keep R.F. 2004. Thrombin preconditioning protects against 6-hydroxydopamine, while large doses result in behavioral deficits. The Toxicologist, 78, 302
118. Cannon, J.R., Xi G., Schallert T., Hua Y., Keep R.F. 2003. Thrombin preconditioning provides neurobehavioral protection against a unilateral 6-hydroxydopamine lesion. The Toxicologist, 72, 348
119. Bernard, R.A., Goran, D.A., Carr, T.H., McFarlane, D.K., Bailey, M.L., Cannon, J.R., Cooper, T.G., Potchen, E.J. 1998. Effect of force on cortical activation produced by finger movement: an fMRI study. NeuroImage 7, S931.
120. Goran, D.A., Bernard, R.A., Carr, T.H., McFarlane, D.K., Bailey, M.L., Cannon, J.R., Cooper, T.G., Potchen, E.J. 1998. Comparison of acute pain and motor activation of second somatic sensory cortex: an fMRI study. NeuroImage 7, S427.

PRESS

"DO 'FOREVER CHEMICALS' PFOS AND PFOA SAP DOPAMINE?" *Futurity*. February 5th, 2020. https://www.futurity.org/pfos-pfoa-forever-chemicals-2272732-2/

"What the brain really thinks about forever chemicals". *Purdue Today*. Purdue University. February 4th, 2020; https://purdue.edu/newsroom/releases/2020/Q1/what-the-brain-really-thinks-about-forever-chemicals.html

"Americas: Purdue University's carcinogen research receives grant". *Food News International*. July 21st, 2016
https://foodnewsinternational.com/2016/07/21/americas-purdue-universitys-carcinogen-research-receives-grant/?platform=hootsuite

"Grant to Fund Purdue Study on Parkinson's". *Inside Indiana Business*. July 15th, 2016. https://www.insideindianabusiness.com/story/32454399/grant-to-fund-purdue-study-on-parkinsons

"Health sciences prof receives $1.68 million to study if dietary factors may have a role in Parkinson's disease", *Purdue Today*. Purdue University. July 12th, 2016;
https://www.purdue.edu/newsroom/releases/2016/Q3/health-sciences-prof-receives-1.68-million-to-study-if-dietary-factors-may-have-a-role-in-parkinsons-disease.html

INVITED PRESENTATIONS/SEMINARS/SESSION LEADERSHIP

09/18/2024 "*Inherent dopaminergic neurobiology modulates neurotoxicity*", Department of Biotechnology, University of Rijeka

09/17/2024 "*Parkinson's disease risk and mechanisms from military relevant organophosphate exposures*", 8th Rijeka Forum On Neurodegenerative Diseases: The Regulation Of Gene Expression In Neurological Disease And Neuroimmunology

08/22/2024 "*Meeting the PFAS Challenge: Adverse effects of PFAS on the nervous system*", Indiana Water Summit, Indianapolis, IN

03/29/2024 "*PFAS induced adverse neurological outcomes modulated by neurotransmission alterations*", PFAS lunch and learn, Institute for a Sustainable Future, Purdue University

11/10/2023 "*Translational mechanisms of heterocyclic aromatic amine induced neurotoxicity*", Research Institute for Medicines/Department of Pharmaceutical Sciences and Medicines, University of Lisbon, Lisbon, Portugal

11/06/2023 "*Per- and polyfluorinated substances (PFAS) neurotoxicity and potential public health implications*", International Conference on Pollutant Toxic Ions and Molecules, Caparica, Portugal

09/29/2023 "*Exosomal miRNA alterations in rotenone models of Parkinson's Disease*", Slovenian Neuroscience Association (SiNAPSA) Neuroscience Conference '23, Ljubljana, Slovenia

05/26/2023 "*Comparative biology approaches to identify neurological targets of PFAS toxicity*", Department of Neurology and Integrated Toxicology and Environmental Health Program, Duke University

05/25/2023 "*Environmentally-induced neurodegeneration overview and graduate programs at Purdue*" (dual research overview and HBCU recruiting presentation), College of Health and Sciences, North Carolina Central University

05/21/2023 "*Neuromelanin-neurotoxicant interactions underlie selective dopaminergic neuron sensitivity*", in "*Selective dopaminergic neurotoxicity modulated by inherent neurobiology*" (Cannon, Co-Chair) at the International Neurotoxicology Association Meeting, Durham, NC, 05/20/2023 – 05/25/2023

04/17/2023 "*Neurological targets of PFAS-induced toxicity*", Department of Pharmacology and Toxicology, University of Connecticut.

03/02/2023 "*Mechanistic neurotoxicology to translationally address neurodegenerative diseases*", Department of Environmental and Occupational Health, Indiana University

02/01/2023 "*Adverse neurological outcomes of PFAS-induced monoamine alterations*", Department of Environmental Sciences, University of California, Riverside.

11/18/2022 "*Critical roles of neuromelanin in the neurobiology and neurotoxicology of Parkinson's disease*", Department of Anatomy and Neurobiology, Virginia Commonwealth University

09/21/2022 "*Translational impact of neurotoxicant-neuromelanin interactions critical to catecholaminergic neurotoxicity*", Department of Environmental Medicine, University of Rochester.

07/03/2022 "*Role of environmentally induced mitophagy alterations in neurodegeneration*", invited speaker at: Inflammation and Proteinopathy in ALS FTD spectrum Disorder, Joint International

Center for Genetic Engineering and Biotechnology (ICGEB) and ALS Society of Canada meeting, Rijeka, Croatia.

07/03/2022 Session Chair, Awarded Young Researcher Talks and Online Selected Speed Talks at: Inflammation and Proteinopathy in ALS FTD spectrum Disorder, Joint International Center for Genetic Engineering and Biotechnology (ICGEB) and ALS Society of Canada meeting, Rijeka, Croatia.

10/01/2021 "*Linking primary mechanisms of environmentally induced neurotoxicity to human neurological disease relevance*", Health and Environmental Sciences Institute (HESI)/Combined Interdisciplinary and Translational Expertise (CITE) Keynote Lecture at EUROTOX 2021

10/01/2021 "*Translation of mechanistic data into in vivo systems to predict risk for neurodegeneration*", Symposium entitled "*Predictive systems to identify etiological factors and pathogenic mechanisms of neurodegeneration*"; served as co-Chair, EUROTOX 2021

06/17/2021 "*C elegans neurodegeneration/neurotoxicity assays*", Neurotoxicity Technical Working Group, Botanical Safety Consortium (BSC), Health and Environmental Sciences Institute (HESI)

01/19/2021 "*C elegans in neurotoxicity screening*", Neurotoxicity Technical Working Group, Botanical Safety Consortium (BSC), Health and Environmental Sciences Institute (HESI)

02/15/2020 "*Neurodegenerative diseases: identifying risk factors and new treatments*", Purdue President's Council, Back to Class, Naples, FL

02/07/2020 "*Mechanisms of environmentally induced neurodegeneration*". Purdue University Center for the Environment; Chemical Exposures Signature Research Area Lunch Group Meetings

01/31/2020 "*Per- and polyfluoroalkyl substances (PFAS) neurotoxicity in laboratory and sentinel models*". Department of Biomedical Sciences, Grand Valley State University

11/06/2019 "*Mechanisms of heterocyclic aromatic amine-induced dopaminergic neurotoxicity*". Department of Molecular pharmacology & Neuroscience, Loyola University

10/03/2019 Chair, Session at the 2019 International Neurotoxicology Association Meeting. Entitled, "*Immune dysregulation as a primary mechanism of early neurotoxicity – relevance to disease*". Individual talk entitled, "*Interactions between neuroinflammation and mitophagy in Parkinson's disease models*".

04/11/2019 "*Environmentally-induced Parkinson's disease: unique features and overlap with other neurodegenerative diseases*", Department of Biotechnology, University of Rijeka

04/08/2019 "*Parkinson's disease: environmental factors and pathogenic mechanisms*", Croatian Institute for Brain Research and Croatian Society for Neuroscience, University of Zagreb

04/08/2019 "*Neurotoxicity of per- and polyfluoralkyl substances (PFAS)*", Institute for Medical Research and Occupational Health and Croatian Society of Toxicology, University of Zagreb

06/14/2018 "Neurotoxicity of Dietary Heterocyclic amines and potential relevance to Parkinson's disease", Department of Pharmacological and Biomolecular Sciences, University of Milan

06/11/2018 "*Neurotoxicity of Heterocyclic Amines: Potential Relevance to Parkinson's Disease*", Plenary Speaker, World Summit on Toxicology, Rome, Italy

06/04/2018 "*Neurotoxicity of Heterocyclic Amines*", Department of Pharmacology and Toxicology, Michigan State University

03/14/2018 "*Potential for Autophagy as a Primary Mechanism of Environmentally-Induced Neurodegeneration*", Symposium at 2018 Annual Society of Toxicology Meeting – "*Mechanisms of Autophagic Function and Dysfunction in Neurotoxicity and Neurodegeneration*"

03/05/2018 "*Dopaminergic neurotoxicity of heterocyclic amines*", Environmental Toxicology Department, University of California, Davis

01/09/2018 "*Heterocyclic amine-induced dopaminergic neurotoxicity*", Graduate Seminar, School of Health Sciences, Purdue University

12/16/2017 "*Neurotoxicology of Heterocyclic Amines*", Department of Environmental Health Sciences and Brain Behavior & Environment-FIU Emerging Preeminent Program, Florida International University

05/18/2017 "*Identification of new etiological factors and new targetable mechanisms in Parkinson's disease*", *Inaugural Retreat, Purdue Institute for Integrative Neuroscience*, Saint Joseph, MI

03/24/2017 "*Environmental and mechanistic Investigations of Early-stage Parkinson's Disease*", Center for Urban Responses to Environmental Stressors, Institute of Environmental Health Sciences, Wayne State University

09/09/2016 *"Optineurin in preclinical to end-stage Parkinson's disease models"*, Department of Pharmaceutical Sciences Seminar Series, Northeast Ohio Medical University

07/13/2016 "*Mechanisms of environmentally-induced dopaminergic neurodegeneration*", NeuroNetworking, Purdue Institute for Integrative Neuroscience.

03/14/2016 Chair, Workshop at the 2016 Society of Toxicology Annual Meeting. Entitled, "*Dietary exposures to heterocyclic amines as a potential risk factor for neurological disease*". Individual talk entitled, "*PhIP exposure and dopaminergic neuron toxicity*".

02/05/2016 "*Developmental TCE exposure and Parkinson's disease*", P42 External Advisory Team and Members of the P42 team.

01/25/2016 "*Behavioral Core at Purdue: Some Possibilities*", Integrative Neuroscience Center Kickoff, Purdue University

12/12/2015 "*Dr. Schallert's Legacy in One LAB: How Lesioned Rats Behave and…How Scientists Should Behave*", SchallertFest, Symposium honoring Dr. Tim Schallert, University of Texas at Austin

03/31/2015 *"Environmentally-induced dopaminergic neurotoxicity"*, Medicinal Chemistry & Molecular Pharmacology Seminar Series, Purdue University

02/06/2015 *"Environmental mechanisms of Parkinson's disease*", College of Health and Human Sciences Dean's Visit, School of Health Sciences Faculty Meeting.

01/23/2015 *"Training for Success: Getting the Most Out Of Your Ph.D. and Postdoctoral Fellowship"*, Exposure to Mixtures and the Exposome Symposium, Department of Environmental Health Sciences, The University of Michigan

11/19/2014 "*Development and utilization of preclinical models of Parkinson's disease*", Behavioral Neuroscience Seminar, Department of Psychological Sciences, Purdue University

11/04/2014 "Dietary factors in the development of Parkinson's disease", Confronting Our Environmental Health Risks, Ted[x]PurdueU

09/17/2014 "*PhIP-mediated Neurotoxicity and Relevance to Parkinson's Disease*", Showalter Selection Committee Annual Purdue Meeting

04/05/2014 "*Neurodegeneration, Neurotoxicity, Gene-Environment Interactions*", Purdue Student Pugwash, Midwest Regional Conference

03/26/2014 "*Accumulation of Manganese in Substantia Nigra and Alterations in Brain Neurochemistry following Subchronic Manganese Exposure in Rats*", 2014 Society of Toxicology Annual Meeting, Workshop Session - Is Manganese-Induced Parkinsonism Mediated via Dopamine Neuron Degeneration or Dysfunction?

02/21/2014 "*The Role of Aging in Susceptibility to Neurotoxic Exposures and Neurodegenerative Diseases*". Center on Aging and the Life Course Colloquium, Purdue University

10/17/2013 "*Parkinson's and inflammatory bowel diseases: interaction in LRRK2 transgenic rats*". The Michael J. Fox Foundation, LRRK2 Awardee Meeting, New York, NY, USA.

09/27/2013 "*Neurotoxicity of 2-Amino-1-methyl-6-phenylimidazo [4,5-b]pyridine (PhIP)*", Department of Biological Sciences, Duquesne University

03/29/2013 "Neurotoxicity of 2-amino-1-methyl-6-phenylimidazo[4,5-*b*]pyridine (PhIP)". Biochemistry Seminar Series, Purdue University

09/25/2012 "*The Role of Alpha-Synuclein in Gene-Environment Interactions: Pathogenesis and Protection in Parkinson's Disease*". Purdue School of Health Sciences Seminar: HSCI 696.

09/18/2012 *"Potentiation and Protection in Gene-Environment Model of Parkinson's Disease"*. Molecular, Cellular and Integrative Neuroscience Program Seminar, Colorado State University, Fort Collins, CO, USA.

05/18/2012 *"Modeling gene-environment interactions in Parkinson's disease"*. Midwest Regional Chapter, Society of Toxicology, Chicago, IL, USA. Spring, 2012 meeting.

01/31/2012 "Neurotoxicant, genetic, and gene-environment interaction models of Parkinson's disease". Purdue School of Health Sciences Seminar: HSCI 696.

08/11/2011 *"Transgenic rats expressing Parkinson's disease genes: characterization and toxicant sensitivity"*. Gordon Research Conference, Cellular & Molecular Mechanisms of Toxicity Understanding Innovative Mechanistic Toxicology in the Post-Genomic Era

05/08/2009 "*Modeling Parkinson's disease: systems to test gene-environment interactions*", 22nd Annual Spring Meeting, Allegheny-Erie Society of Toxicology, Morgantown, WV, Host: Nicolas A. Stewart, Ph.D., President of AESOT, Research Instructor, University of Pittsburgh, Center for Clinical Pharmacology

09/05/2007 "*Improving the rotenone model*", Data Club, Pittsburgh Institute for Neurodegenerative Diseases

04/13/2006 "*Mechanisms of thrombin preconditioning in a 6-hydroxydopamine model of Parkinson's disease*", National Institute on Drug Abuse Training Program, The University of Chicago, Host: Un Jung Kang, M.D., Associate Professor of Neurology

04/03/2006 "*Mechanisms of thrombin preconditioning in a 6-hydroxydopamine model of Parkinson's disease*", Laboratory Meeting of Wei Zheng, Ph.D., Professor and University Faculty Scholar, School of Health Sciences, Purdue University

12/20/2005 "*Thrombin preconditioning, PARs and Parkinson's disease*", Neurosurgery Laboratory Conference, University of Michigan

12/14/2004 "*Protease-activated receptor-1 activation mediates the protective effects of thrombin preconditioning in a model of Parkinson's disease*", Current Topics in Toxicology, EHS 728, The University of Michigan, School of Public Health

01/27/2004 "*Thrombin preconditioning provides protection against 6-OHDA*". Current Topics in Toxicology, EHS 728, The University of Michigan, School of Public Health

03/18/2003 "*Neuroprotection in Animal Models of Parkinson's Disease*", Current Topics in Toxicology, EHS 728, The University of Michigan, School of Public Health

02/11/2003 "*Thrombin preconditioning in a 6-OHDA Parkinson's disease model*", Neurosurgery Laboratory Conference, University of Michigan

EXTERNAL CONSULTING

11/2024-present Expert Witness, Plaintiffs' Leadership in the Camp Lejeune litigation in the USDC-EDNC. Services included to date: initial literature review on the role of perchloroethylene (tetrachloroethylene) in Parkinson's disease; general causation report.

11/2024-12/2024 Individual consultation with a farmer reportedly exposed to chlorpyrifos. Provided professional written scientific opinion on the potential adverse health effects in cattle and humans.

08/2024-present Expert Witness, Johnson and Bell. Services included to date: medical and toxicology file review on alleged medical malpractice. Case No. 2023L006556; Cook County Circuit Court, Chicago, Illinois.

07/2022-05/2023 Expert Witness, BUNGER & ROBERTSON. Services included: discussion on delta-8 tetrahydrocannabinol (THC) – formulation, detection, adverse effects; especially in relation to how contamination and use may relate to assault; expert toxicological analyses of law enforcement, EMS, and hospital records; development and submission of expert witness scientific report. Case No. 53C02-2201-F3-000043; Monroe County Circuit Court II, Indiana.

05/2021-08/2021 Expert Witness, CIYOU & DIXON, P.C.; Analytical toxicology expertise relative to screen results for drugs of abuse. Services included: drug screen results review; literature review; determination of likelihood of use cessation relative to urine, oral fluid, and hair (head and body) screen results; determination of whether video evidence of alleged drug use was supported by screen data; pre-trial conferences with attorneys and clients; expert testimony in court on 08/19/2021 on the above items and also adverse effects during cross-examination. Case No. 53C04-1601-DR-000031; Monroe County Circuit Court VI, Indiana.

11/2020-04/2022 Expert witness. Perkins Coie/Winston & Strawn/Boeing. Services included: complaint review; expertise on neurotoxicology relevant to possible etiology of an amyotrophic lateral sclerosis case; literature review; medical and scientific records review; plaintiff deposition review; plaintiff disclosure review; pre-trial conferences; development and submission of expert witness scientific report; deposition; trial slide development and input; and mock direct and cross

examinations. Case settled prior to trial. Case No. 18 L 8347; Circuit Court of Cook County, Illinois.

04/2019 GLG Group. Provided consultation on biomarkers of exposure and neurodegenerative disease development.

05–06/2017 Expert witness. Lewis & Brisbois/Womble Carlyle Sandridge & Rice [*now Womble Bond Dickinson*]/Goodyear Tire and Rubber Company. Provided expertise on neurotoxicology relevant to possible etiology of an amyotrophic lateral sclerosis case. Services included: complaint review; pretrial consultation, and preparation as an expert witness. Case settled prior to trial. Case No. 15CV2760; County of Multnomah, Circuit Court for the State of Oregon.

TEACHING

Classroom:

2024

Course Description	*Course Code*	*Credit*	*Role*	*Semester*
Analytical Toxicology and Path[a]	HSCI562	3	Course Master	Spring
Intro to Environmental Health[b]	HSCI575	3	Guest Lecturer	Spring
Intro Occupat&Environ Health Sci[c]	HSCI345	2	Guest Lecturer	Spring
Professionalism[c]	HSCI613	1	Guest Lecturer	Spring
Everyday Toxicology[c]	HSCI360	2	Guest Lecturer	Spring
Professionalism[c]	HSCI590	1	Guest Lecturer	Spring

[a] Instructor of record
[b] Delivered 2 lectures
[c] Delivered 1 lecture

2023

Course Description	*Course Code*	*Credit*	*Role*	*Semester*
Biochemical Toxicology[a]	HSCI671	2	Course Master	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Spring
PULSe Lab Rotations[a]	GRAD590	2	PULSe Head	Spring
PULSe Dissertation Res (1st year)[a]	GRAD699	6	PULSe Head	Spring
Intro to Environmental Health[b]	HSCI575	3	Guest Lecturer	Spring
Everyday Toxicology[c]	HSCI360	2	Guest Lecturer	Spring
Professionalism[c]	HSCI590	1	Guest Lecturer	Spring
Neuroimmunology[d]	EBIL164	3	Guest Lecturer	Summer
Neuroscience in Croatia/ International Topics[a]	SA10222/ HSCI400	3	Course Master	Summer
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Fall
Fellowship and Grant Application Writing	GRAD590	1	Course Master	Fall
Intro Occupat&Environ Health Sci[c]	HSCI345	2	Guest Lecturer	Fall
Data Manag/Record Keeping[c]	GRAD590	1	Guest Lecturer	Fall
Preparing Future Faculty[c]	GRAD590	2	Guest Lecturer	Fall
Toxicology[b]	HSCI560	3	Guest lecturer	Fall
Grant writing for Health Sciences	[a]HSCI625	1	Guest lecturer	Fall

[a] Instructor of record
[b] Delivered 2 lectures
[c] Delivered 1 lecture
[d]Delivered 3 lectures to 4th year undergraduates and masters students in the Department of Biotechnology at the University of Rijeka, Croatia; Students on Purdue University Study Abroad, and students from St. Cloud State University also visiting the University of Rijeka on study abroad.

2022

Course Description	*Course Code*	*Credit*	*Role*	*Semester*
Analytical Tox and Path[a]	HSCI562	3	Course Master	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Spring
PULSe Lab Rotations[a]	GRAD590	2	PULSe Head	Spring
PULSe Dissertation Res (1st year)[a]	GRAD699	6	PULSe Head	Spring
Intro to Environmental Health[b]	HSCI575	3	Guest Lecturer	Spring
Everyday Toxicology[c]	HSCI360	2	Guest Lecturer	Spring
Neuroimmunology[d]	EBIL164	3	Guest Lecturer	Summer
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Fall
PULSe Lab Rotations[a]	GRAD590	2	PULSe Head	Fall
PULSe Dissertation Res (1st year)[a]	GRAD699	6	PULSe Head	Fall
Intro Occupat&Environ Health Sci[c]	HSCI345	2	Guest Lecturer	Fall
Toxicology[b]	HSCI560	3	Guest lecturer	Fall

[a] Instructor of record

[b] Delivered 2 lectures

[c] Delivered 1 lecture

[d]Delivered 2 lectures to 4th year undergraduates and masters students in the Department of Biotechnology at the University of Rijeka, Croatia.

2021

Course Description	*Course Code*	*Credit*	*Role*	*Semester*
Biochemical Toxicology[a]	HSCI671	2	Course Master	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Spring
PULSe Lab Rotations	GRAD590	2	PULSe Head	Spring
PULSe Dissertation Res (1st year)	GRAD699	6	PULSe Head	Spring
Intro to Environmental Health[b]	HSCI575	3	Guest Lecturer	Spring
Professionalism[c]	HSCI590	1	Guest Lecturer	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Fall
Toxicology[b]	HSCI560	3	Guest lecturer	Fall
PULSe Lab Rotations[a]	GRAD590	2	PULSe Head	Fall
PULSe Dissertation Res (1st year)[a]	GRAD699	6	PULSe Head	Fall
Health In The Time Of Pandemics: An Introduction[c]	PUBH202	3	Guest Lecturer	Fall
Intro Occupat&Environ Health Sci[c]	HSCI345	2	Guest Lecturer	Fall

[a] Instructor of record

[b] Delivered 2 lectures

[c] Delivered 1 lecture

2020

Course Description	*Course Code*	*Credit*	*Role*	*Semester*
Analytical Tox and Path[a]	HSCI562	3	Course Master	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Spring
PULSe Lab Rotations[a]	GRAD590	2	PULSe Head	Spring
PULSe Dissertation Res (1st year)[a]	GRAD699	6	PULSe Head	Spring
Intro to Environmental Health[b]	HSCI575	3	Guest Lecturer	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Fall
Toxicology[b]	HSCI560	3	Guest lecturer	Fall
PULSe Lab Rotations[a]	GRAD590	2	PULSe Head	Fall
PULSe Dissertation Res (1st year)[a]	GRAD699	6	PULSe Head	Fall
Health In The Time Of Pandemics: An Introduction[c]	PUBH202	3	Guest Lecturer	Fall
Intro Occupat&Environ Health Sci[c]	HSCI345	2	Guest Lecturer	Fall

[a] Instructor of record

[b] Delivered 2 lectures

[c] Delivered 1 lecture

2019

Course Description	*Course Code*	*Credit*	*Role*	*Semester*
Biochemical Toxicology[a]	HSCI671	2	Course Master	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Spring
PULSe Lab Rotations	GRAD590	2	PULSe Head	Spring
PULSe Dissertation Res (1st year)	GRAD699	6	PULSe Head	Spring
Intro to Environmental Health[b]	HSCI575	3	Guest Lecturer	Spring
(PET) training programme[d]			Guest Lecturer	Spring
PULSe Lab Rotations	GRAD590	2	PULSe Head	Fall
PULSe Dissertation Res (1st year)	GRAD699	6	PULSe Head	Fall
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Fall
Toxicology[b]	HSCI560	3	Guest lecturer	Fall
Intro Occupat&Environ Health Sci[c]	HSCI345	2	Guest Lecturer	Fall
Neurol & Neuropsych Dis Seminar[c]	BIOL695	2	Guest lecturer	Fall

[a] Instructor of record

[b] Delivered 2 lectures

[c] Delivered 1 lecture

[d]Developed one electronic lecture, entitled, "*Neurodegenerative effects of toxic metals*" for the Postgraduate Education in Toxicology (PET) training programme offered by the Netherlands Society of Toxicology for registration as a professional expert in toxicology (European Registered Toxicologist, ERT). The aim of this course is to familiarize participants with consequences of neurotoxicity, mechanisms of neurotoxicity and neurotoxicity testing methods. The course will consist of e-lectures and webinars that allow for offsite participation as well as (active) classes that require physical attendance of participants for 3 days. As the course will be accredited by Eurotox, it will be accessible for participants from across Europe. It is expected to be accessible for participants worldwide.

2018

Course Description	*Course Code*	*Credit*	*Role*	*Semester*
Analytical Tox and Path[a]	HSCI562	3	Course Master	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Spring
PULSe Lab Rotations	GRAD590	2	PULSe Head	Spring
PULSe Dissertation Research (1st year)	GRAD699	6	PULSe Head	Spring
Intro to Environmental Health[b]	HSCI575	3	Guest Lecturer	Spring
Intro Occupat&Environ Health Sci[c]	HSCI345	2	Guest Lecturer	Fall
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Fall
Toxicology[b]	HSCI560	3	Guest lecturer	Fall
Neurol & Neuropsych Dis Seminar[c]	BIOL695	2	Guest lecturer	Fall
PULSe Lab Rotations	GRAD590	2	PULSe Head	Fall
PULSe Dissertation Research (1st year)	GRAD699	6	PULSe Head	Fall

[a] Instructor of record
[b] Delivered 2 lectures
[c] Delivered 1 lecture

2017

Course Description	*Course Code*	*Credit*	*Role*	*Semester*
Biochemical Toxicology[a]	HSCI671	2	Course Master	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Spring
Intro to Environmental Health[b]	HSCI575	3	Guest Lecturer	Spring
Toxicology[b]	HSCI560	3	Guest lecturer	Fall
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Fall
Intro Occupat&Environ Health Sci[c]	HSCI345	2	Guest Lecturer	Fall
PULSe Lab Rotations	GRAD590	2	PULSe Head	Fall
PULSe Dissertation Research (1st year)	GRAD699	6	PULSe Head	Fall

[a] Instructor of record
[b] Delivered 2 lectures
[c] Delivered 1 lecture

2016

Course Description	*Course Code*	*Credit*	*Role*	*Semester*
Analytical Tox and Path[a]	HSCI562	3	Course Master	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Spring
Intro to Environmental Health[b]	HSCI575	3	Guest Lecturer	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Fall
Intro Occupat&Environ Health Sci[c]	HSCI345	2	Guest Lecturer	Fall
Toxicology[d]	HSCI560	3	Guest lecturer	Fall

[a] Instructor of record
[b] Delivered 2 lectures

[c] Delivered 1 lecture
[d] Delivered 3 lectures

2015

Course Description	*Course Code*	*Credit*	*Role*	*Semester*
Biochemical Toxicology[a]	HSCI671	2	Course Master	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Spring
Principles of Public Health Science[b]	HSCI201	3	Guest Lecturer	Spring
Toxicology[c]	HSCI560	3	Guest lecturer	Fall
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Fall
Intro Occupat&Environ Health Sci[b]	HSCI345	2	Guest Lecturer	Fall

[a] Instructor of record
[b] Delivered 1 lecture
[c] Delivered 3 lectures

2014

Course Description	*Course Code*	*Credit*	*Role*	*Semester*
Analytical Tox and Path[a]	HSCI562	3	Course Master	Spring
Intro to Environmental Health[b]	HSCI575	3	Guest Lecturer	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Spring
Principles of Public Health Science[b]	HSCI201	3	Guest Lecturer	Spring
Freshman Scholars Project Seminar[b]	HSCI195	1	Guest Lecturer	Fall
Intro Occupat&Environ Health Sci[c]	HSCI345	2	Guest Lecturer	Fall
HSCI Graduate Seminar[a]	HSCI696	1	Guest lecturer	Fall
Toxicology[d]	HSCI560	3	Course Master	Fall

[a] Instructor of record
[b] Delivered 1 lecture
[c] Delivered 2 lectures
[d] Delivered 3 lectures

2013

Course Description	*Course Code*	*Credit*	*Role*	*Semester*
Analytical Tox and Path[a]	HSCI562	3	Course Master	Spring
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Spring
Intro to Environmental Health[b]	HSCI575	3	Guest Lecturer	Spring
Toxicology[a]	HSCI560	3	Course Master	Fall
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Fall
Intro Occupat&Environ Health Sci[b]	HSCI345	2	Guest Lecturer	Fall
Special Lectures in Neuroscience	BIOL695	2	Instructor	Fall
Freshman Scholars Project Seminar[c]	HSCI195	1	Guest Lecturer	Fall

[a] Instructor of record
[b] Delivered 2 lectures

[c] Delivered 1 seminar

2012

Course Description	*Course Code*	*Credit*	*Role*	*Semester*
HSCI Graduate Seminar[a]	HSCI696	1	Course Master	Fall
Toxicology[b]	HSCI560	3	Guest Lecturer	Fall

[a] Instructor of record

[b] Delivered 2 lectures

2011 *Survival Skills and Ethics Workshop on Grant Writing*, University of Pittsburgh, Discussion leader, Ethics over lunch Session

2004 ENVIRON 310/NRE 310, *Environmental Chemicals and Disease*, 3.0 hrs, School of Natural Resources and Environment, University of Michigan, 1 lecture

1999 Physiology 475, *Capstone Laboratory in Physiology*, 2.0 hrs, Department of Physiology, Michigan State University, Teaching Assistant

MENTORSHIP

Postdoctoral Fellows, as Primary Mentor

Mohammed Jakaria, Ph.D. (University of Melbourne). 04/2024-present

Fatema Currim, Ph.D. (MS University of Baroda, India). 02/2024-present

Vivek Lawana, Ph.D. (Iowa State University) 01/2019-11/2019
Current position: Toxicology Study Director, American Preclinical Services, Minneapolis, MN

Tauqeerunnisa Syeda begum, Ph.D. (The Center for Research and Advanced Studies of the National Polytechnic Institute, Mexico City, Mexico) 11/2018-03/2022. Current position: Study Director, Corteva. Agriscience

Shreesh Raj Sammi, Ph.D. (Life Sciences CSIR-Central Drug Research Institute, Lucknow, India) 11/2016-01/2023. Current position: Assistant Professor, Department of Translational Neuroscience, Michigan State University.

Amy Griggs, Ph.D. (Chemistry, Purdue University) 12/2012-5/2013
Current Position: Lead Clinical Scientist, Cook MED Institute, West Lafayette, IN

Jang-Won Lee, Ph.D. (Toxicology, UC Davis) 04/2012-12/2014
Current position: Assistant Professor, Graduate School of Integrated Bio-industry, Sejong University, Seoul, Korea

Changhe Xiao, Ph.D. (Chemistry, Rutgers University) 01/2012-10/2012
Current Position: Staff Scientist, Abbott, Minneapolis, MN

Doctoral Students, as Major Professor

Jahidul Islam, predoctoral student, Toxicology (M.Sc., Toxicology, University of Rajshahi) 08/2024-present

Reeya Tanwar, predoctoral student, Integrative Neurosciences and Toxicology (B. Tech., New Delhi University) 04/2023-present

Josephine Brown, predoctoral student, Toxicology (M.S., Toxicology, University of Cincinnati) 08/2020-present

Emily K. McDonald, predoctoral student, Integrative Neurosciences and Toxicology (B.S., Biochemistry, Purdue University) 04/2018-09/2018
Current position: Decided to withdraw from Ph.D. study for family reasons.

Rachel M. (Foguth) Nolan, predoctoral student, PULSe Integrative Neurosciences and Toxicology (B.S., Biochemistry, Benedictine College) 04/2016-10/2020 (Graduation, 12/2020)
Current position: Senior Toxicologist, Cook Biotech, West Lafayette, IN

Johnny P. Wise, Jr., predoctoral student, Toxicology (B.S., Biology, University of Southern Maine) 08/2013-6/2018
Current position: Assistant Professor, Pediatric Research Institute, Department of Pediatrics, University of Louisville

Zeynep Sena Ağim, predoctoral student, Integrative Neurosciences and Toxicology (M.Sc., Molecular Biology and Genetics, Boğaziçi University, Turkey) 04/2013-12/2017
Current position: Scientific Managing Editor, Elsevier

Masters Students, as Major Professor

Madison Langley, M.S.-thesis, Toxicology (B.S., Forensic Chemistry, Sam Houston State University) 08/2024-present

Angela Cruz-Hernandez, M.S. – thesis, Toxicology (B.A., Chemistry, Florida International University) 08/2015 – 05/2017. Current position: Senior Scientist – Toxicologist, L'Oreal

Menghan Liu, M.S. – non-thesis, Toxicology (B.S., Biology, Purdue University) 08/2013-05/2015
Current position: Statistical Analyst, Fred Hutchinson Cancer Research Center

Xindi Ding, M.S. – non-thesis, Toxicology (B.S., Public Health, Capital Medical University, China) 08/2013-05/2015. Current position: Medical Science Liaison at Janssen Inc., Beijing City, China

Visiting Scholars, as site Mentor

Safreena Narukkottil, Ph.D. Student at Inter-University Center for Biomedical Research & Super Specialty Hospital. Overseas Visiting Doctoral Fellowship (OVDF) Program, Purdue and India's Science and Engineering Research Board (SERB). Mentor – Mentee team amongst 25/200 applicants chosen. 08/2024-present

Fatema Currim, Ph.D. Student at MS University of Baroda, India. Overseas Visiting Doctoral Fellowship (OVDF) Program, Purdue and India's Science and Engineering Research Board (SERB). Mentor – Mentee team amongst 25/127 applicants chosen. 02/2022-02/2024

Purdue School of Health Sciences Undergraduate Honors Program (as research mentor):

Lorraine Prevost, 2021-2021
Krista Snyder, 2021
Claudia Nieves, 2018-2020
Niharika Kaul, 2016-2018
Charles Price, 2016-2020. Med Student, IU School of Medicine
Morgan Kramer, 2014-2016
Joey Amaro, 2013-2017
Samantha Watson, 2012

Additional undergraduate researcher mentorship (Purdue University, unless otherwise noted)

2022-	Matthew Corson, Biomedical Health Sciences
2021-	Sofia Schuman, Biomedical Health Sciences
2021-	Hurshal Pol, Biomedical Health Sciences (began as a high school student)
2020-2021	Leah Van Zant, Biology, Purdue University
2020-2021	Alexis Wazniak, Biology, Purdue University
2020-2022	Mia Utayde, Biology, Purdue University
2019-2021	Hannah Welp, Biology
2019	Se Young Um, Biology
2019	Claudia Nieves, Purdue University, Purdue Summer Research Opportunities Program
2019	Georgia 'Cali' Clark, Morehead State University, Purdue Summer Research Opportunities Program. Recently Accepted to the University of Kentucky Medical School.
2019	Emily Llewellyn, Utah Valley University, Purdue Summer Research Opportunities Program
2018-2019	Madison Nelson, Health Sciences, Pre-med. Accepted to Lincoln Scholars Program. Doctor of Medicine track for Southern Illinois University School of Medicine.
2018-2020	Benjamin Clarke, Health Sciences, Pre-med.
2017	Bahati Nkera, University of Massachusetts, Purdue Summer Research Opportunities Program
2016	Mariella A Mestres Villanueva, University of Puerto Rico, Purdue Summer Research Opportunities Program. Current position: Ph.D. student at Ohio State University
2016	Erika Kischuk, Summer Internship Student, DePauw University
2016-2018	Eva Yezerets. Biomedical engineering
2015	Nickolas Anderson, Chemistry undergraduate student (Boston University)
2014	Saerom Kim, Chemistry undergraduate student
2013	Kyung-Min Lee, Pharmacy undergraduate student
2013-2014	Ker Ming Chew, Biochemistry undergraduate student
2013-2015	Adam Horin, Biology undergraduate student
2012	Vasin Dumrongprechachan, Health Sciences undergraduate student
2012	Monica Bomber, Biochemistry undergraduate student

Laboratory rotations

Purdue University Interdisciplinary Life Sciences Ph.D. Program/Toxicology

2020 Josephine Brown (Toxicology)
2018 Emily Malek (Integrative Neuroscience)
2018 Yiming Miao (Integrative Neuroscience)
2017 Chandnee Chandrasekaran (Integrative Neuroscience)
2017 Jennifer Hensel (Integrative Neuroscience)
2016 William Saloom (Integrative Neuroscience)
2016 Cynthia Alvarado (Integrative Neuroscience)
2016 Lisa Kobos (Toxicology)
2015 Rachel Foguth (Integrative Neuroscience)
2013 Sasha Vega Alvarez (Integrative Neuroscience)
2013 Marcus Weera (Integrative Neuroscience)
2013 Zeynep Sena Agim (Integrative Neuroscience)

University of Pittsburgh

2010 Paras Minhas, Neuroscience undergraduate/GA medical (University of Pittsburgh)
2010-2011 Salik Malik, Biological Sciences undergraduate student (University of Pittsburgh)
2008-2011 Laura Montero B.S. (West Virginia University), Technician
2008 Rupali Kumar, Neuroscience undergraduate student (University of Pittsburgh)
2008 Jayesh Madrecha, Neuroscience undergraduate student (University of Pittsburgh)
2008-2011 Nestor Tomycz, M.D., (University of Pittsburgh)
2009-2011 Thomas Sew, Neuroscience undergraduate student (University of Pittsburgh)

Awards won by students/postdocs while being mentored by Cannon:

Schumann, Sofia

- 2nd place undergraduate poster, 2024 HHS Life Inspired poster session

Pol, Hurshal

- 1st place 2024 Purdue undergrad 3-minute thesis competition and the crowd favorite award, Undergraduate Research Society.
- 1st place 2023 Research Talk (College of Health and Human Sciences), Undergraduate Research Expo

Currim, Fatema

- 1st Place Poster Presentation (Toxicology). 4th HSCI Annual Research Retreat, 2022

Utayde, Mia

- 3rd Place poster at the Spring Undergraduate Research Conference, Office of Undergraduate Research, Purdue University, 2022

Brown, Josephine

- 1st Place Poster Presentation (Toxicology). 3rd HSCI Annual Research Retreat, 2022

Sammi, Shreesh

- Postdoctoral Travel Grant, Purdue Postdoctoral Association, 2018
- Abstract chosen for oral presentation at the Society of Toxicology Annual Meeting. 2019 Scientific Program Committee Highlights Emerging Scientists: Adverse effects of Perfluorinated Alkyl Substances
- Postdoctoral Supplemental Travel Grant, Purdue Postdoctoral Association, 2019
- 3rd place in the Society of Toxicology, Neurotoxicology Specialty Section Poster Competition, 2019
- 3rd place, Postdoctoral Research Blitz Presentation, 2019 Purdue School of Health Sciences Retreat.
- Neurotoxicology Specialty Section (NTSS) Narahashi Travel Award to the Society of Toxicology (SOT) 2020 meeting
- 2rd place in the Society of Toxicology, Neurotoxicology Specialty Section Postdoctoral Poster Competition, 2020

- NIH/NIEHS Pathway to Independence Award (K99/R00), 2021-2026

Vivek Lawana
- 2nd place, Postdoctoral Research Blitz Presentation, 2019 Purdue School of Health Sciences Retreat.

Tauqeerunnisa Syeda
- 1st place, Postdoctoral Research Blitz Presentation, 2019 Purdue School of Health Sciences Retreat.

Foguth, Rachel
- 2018 Travel Grant, Purdue Institute for Integrative Neuroscience – to SOT 2019.
- 3rd place, Graduate Student Research Blitz Presentation, 2019 Purdue School of Health Sciences Retreat.
- 3rd place Neurotoxicology Specialty Section Graduate Student Poster Competition, 2020

Wise, J.
- Frederick N. Andrews Fellowship (2 years tuition and annual $18,000 stipend), Purdue Graduate School, 2013
- Compton Travel Award ($500), to 2015 Society of Toxicology Annual Meeting
- Purdue Research Foundation Fellowship (2016-2017), total award = $28,662
- Purdue Institute of Integrative Neuroscience Travel Award ($500), to 2016 SOT Annual Meeting
- Purdue Graduate Student Government Travel Grant ($250), to 2016 SOT Annual Meeting
- Bilsland Dissertation Fellowship (2017-2018), total award = valued >$62,000 due to forgiven tuition remits
- Winner of the Abstract Competition/travel award for Greater Indiana Chapter of the Society for Neuroscience's annual meeting; #1 graduate student abstract out of 122 submissions; "*Autophagic dysfunction in brainstem nuclei in a preclinical rotenone Parkinson's disease model*"
- Chair, of selected symposium at the 2018 Society of Toxicology Annual Meeting. Symposium entitled, "*Mechanisms of Autophagic Function and Dysfunction in Neurotoxicity and Neurodegeneration*"

Agim, Z.S.
- Women in Science Programs Travel Grant ($500), to 2014 Society of Toxicology Annual Meeting
- Purdue University Interdisciplinary Life Sciences Program Travel Grant ($150) to 2014 Society of Toxicology Annual Meeting
- Honorable mention (top 20% - ~70 contestants), Health and Disease: Science, Culture and Policy graduate student poster competition, Purdue University.
- Society of Toxicology Travel Award ($1000) to 2015 annual meeting
- Purdue Research Foundation Fellowship (2015-2016), total award = $28,662
- Compton Graduate Travel Award ($500) to 2016 SOT Annual Meeting
- Andrews Environmental Travel Grant ($1500) to 2016 IUTOX Annual Meeting
- A. H. Ismail Interdisciplinary Program Doctoral Research Travel Award ($1500) to 2016 SOT Annual Meeting
- Purdue University Interdisciplinary Life Sciences Program Travel Grant ($350) to 2017 SOT Annual Meeting
- Purdue Student Government Travel Grant ($500) to 2017 SOT Annual Meeting

Villanueva, M.A.
- 2017 Pfizer SOT Undergraduate Student Travel Award. Full funding for travel and all expenses to 2017 SOT Annual Meeting.

Amaro, J.A.
- 1st Place Poster, College of Health and Human Sciences, 2017 Undergraduate Research Symposium

Nieves, Claudia
- 2018 Paul L. Ziemer for Outstanding Freshmen Scholastic Performance

Student Committees:

Ph.D. Dissertation Committees

2020- Xueqi Tang, Purdue University Interdisciplinary Life Science Ph.D. Program
2019-2022 Saeed Alqahtani, Toxicology, School of Health Sciences, Purdue University
2018- Janiel Ahkin Chin Tai, Tox, Purdue University Interdisciplinary Life Science Ph.D. Program
2018- Jennifer Hensel, Purdue University Interdisciplinary Life Science Ph.D. Program
2018- Luqing Liu, Toxicology, School of Health Sciences, Purdue University
2016-2022 Cynthia Alvarado, Integrative Neurosciences, Purdue University Interdisciplinary Life Science, PhD. Program converted to M.S.
2016- 2019 Kaushik Muralidharan, Department of Biological Sciences, Purdue University
2016-2020 Saranya Radhakrishnan, Integrative Neurosciences, Purdue University Interdisciplinary Life Science Ph.D. Program
2016-2022 Chandnee Chandrasekaran, Integrative Neurosciences, Purdue University Interdisciplinary Life Science Ph.D. Program
2016-2022 Aswathy Chandran, Integrative Neurosciences, Purdue University Interdisciplinary Life Science Ph.D. Program
2015-2018 Paola Montenegro, PULSe/MCMP
2015-2019 David Edmondson, Imaging Sciences and Toxicology, School of Health Sciences, Purdue University
2015-2019 Daniel Cholger, Integrative Neurosciences, Purdue University Interdisciplinary Life Science Ph.D. Program
2014-2016 Sara Wirbisky, Toxicology, School of Health Sciences. Current position: Sr. Toxicologist, WIL Research
2014-2018 Xinxin Liu, Health Sciences, School of Health Sciences
2014-2018 Katharine Horzmann, Toxicology, School of Health Sciences, Purdue University.
2014-2018 Kathryn Thompson, Purdue University Interdisciplinary Life Science, Ph.D. Program, Molecular Signaling and Cancer Biology
2014-2019 Dennis Claddis, Nutrition
2013-2016 Jinyoung Lee, Toxicology, School of Health Sciences, Purdue University
2013-2016 Ruoyun Ma, Medical Physics, School of Health Sciences, Purdue University
2013-2014 Gyeon Oh, Medicinal Chemistry and Molecular Pharmacology
2013-2017 Sasha Vega Alvarez, Purdue University Interdisciplinary Life Science, Ph.D. Program, Integrative Neuroscience
2012 Hilary Broderick, Purdue University Interdisciplinary Life Science, Ph.D. Program, Integrative Neuroscience
2012-2015 Stefanie O'Neil, Purdue University Interdisciplinary Life Science Ph.D. Program, Integrative Neuroscience. Current position: Sr. Associate, S.C. Johnson

Ph.D. Preliminary Exam Committees

2023-2024 Purba Mandal, Integrative Neurosciences, Purdue University Interdisciplinary Life Science Ph.D. Program
2023-2024 Zahraa Alawadly, Integrative Neurosciences, Purdue University Interdisciplinary Life Science Ph.D. Program
2022 Alishia Aroor, Psychological Sciences, Ph.D. Program
2021-2022 Ruilin Yu, Integrative Neurosciences, Purdue University Interdisciplinary Life Science Ph.D. Program
2019 Lisa Kobos, Toxicology, School of Health Sciences, Purdue University
2016 Daniel Cholger, Integrative Neurosciences, Purdue University Interdisciplinary Life Science Ph.D. Program
2016 David Edmondson, Imaging Sciences and Toxicology, School of Health Sciences, Purdue University (Committee Chair)
2015 Amy Godfrey, Molecular Signaling and Cancer Biology, Purdue University Interdisciplinary Life Science Ph.D. Program

2015 Kathryn Thompson, Molecular Signaling and Cancer Biology, Purdue University Interdisciplinary Life Science Ph.D. Program
2015 Katharine Horzmann, Toxicology, School of Health Sciences, Purdue University.
2014 Sasha Vega Alvarez, Integrative Neurosciences, Purdue University Interdisciplinary Life Science Ph.D. Program
2013-2014 Stefanie O'Neil, Integrative Neurosciences, Purdue University Interdisciplinary Life Science Ph.D. Program (Committee Chair)
2012-2013 Glen Acosta, Integrative Neurosciences, Purdue University Interdisciplinary Life Science Ph.D. Program (Committee Member)

M.S. Committees

2018- Li Xia, Toxicology, School of Health Sciences
2012-2013 Sara Wirbisky, Toxicology, School of Health Sciences

ENGAGEMENT

International Service

2023- Counselor, International Neurotoxicology Association

2022 Poster Judge, invited speaker at: Inflammation and Proteinopathy in ALS FTD spectrum Disorder, Joint International Center for Genetic Engineering and Biotechnology (ICGEB) and ALS Society of Canada meeting, Rijeka, Croatia, 06/30/2022 – 07/03-2022.

2022 Oral Presentation Judge, invited speaker at: Inflammation and Proteinopathy in ALS FTD spectrum Disorder, Joint International Center for Genetic Engineering and Biotechnology (ICGEB) and ALS Society of Canada meeting, Rijeka, Croatia, 06/30/2022 – 07/03-2022.

National Service

2022 Panel Member, Interactive Panel - The PI Crash Course, SHARP Training Program (Skills for Health and Research Professionals) at Columbia University, 06/10/2022
2021-2023 Representative Specialty Section Collaboration and Communication Group (SS-CCG), Society of Toxicology
2021- Society of Toxicology Annual Meeting, Chat with an Expert
2021 Society of Toxicology Annual Meeting, Graduate School Virtual Career Fair
2020- President (Presidential Chain), Neurotoxicology Specialty Section, Society of Toxicology
2020 Distinguished Neurotoxicologist Committee, Neurotoxicology Specialty Section, Society of Toxicology
2020 Mentor, Mentor Match, Society of Toxicology

2018-2020 Councilor, Neurotoxicology Specialty Section, Society of Toxicology
2017 External Reviewer, 2016 Neurotoxicology Specialty Section poster judging
2016 External Reviewer, 2016 Neurotoxicology Specialty Section poster judging
2015 External Reviewer, 2015 Neurotoxicology Specialty Section poster judging
2013 Ohio Valley Society of Toxicology, *Postdoctoral Poster Judge*, Annual Meeting
2013 External Reviewer, 2014 Best Postdoctoral Publication Award, The Society of Toxicology

Institutional Service

Purdue University

2023- Member, Graduate Council
2021- Member, Core Strategic Planning Committee, Purdue Animal Behavior
2020- Faculty Advisory Committee for the Bindley Imaging Facility
06/28/2017 Facilitator, Graduate Student and Postdoc Forum at NeuroNetworking, Purdue Institute for integrate Neuroscience
2017 Panel Member, Newly Tenured Professors, Faculty Advancement, Success and Tenure (FAST), ADVANCE Center for Faculty Success

2016- 2017 Member, Subcommittee on animal behavior core, Purdue Institute for Integrative Neuroscience
04/14/2015 Judge, Undergraduate Research Symposium and Poster Session
07/21/14 *Experience Purdue*, Instructor, High ability High School student recruitment/short course, "Environmental exposures and brain damage"
03/2014 Purdue ME Assistance, High-School Recruitment, Featured Laboratory
02/2014 *Ad hoc* Reviewer, Journal of Undergraduate Research
2013-2015 Featured laboratory/tour leader, Neuroscience-Philosophy-Intelligence-Society, Purdue University

College of Health and Human Sciences – Purdue University

2022 - Member, Advisory Board, Center for Research on Brain, Behavior, and NeuroRehabilitation (CEREBBRAL)
2021-2021 Member, Associate Dean for Research Faculty Search Committee, HHS
2020-2021 Member, Faculty Search Committee, Department of Public Health
2019-2020 Member, "Advance Research to Improve Health, Human Functioning, and Quality of Life (including doctoral education)", HHS Strategic Planning Working Group
2017-present Member, Public Health Graduate Program Evaluation Committee
2016 School representative, HHS Fall Welcome
2016-2018 Member, HHS Career Advisory Council
2016-2018 Member, HHS Graduate Education and Curriculum Committee
2014 HHS Scholarship Committee - Presidential Scholarship Selection
2014 HHS Family Day – Faculty Representative

Graduate School – Purdue University

2017- Executive Chair, Executive Committee, Purdue University Interdisciplinary Life Science Program (PULSe)
2017 Judge, 5 Minute Thesis Competition, Purdue University Interdisciplinary Life Science Program (PULSe)
2017 Judge, PULSe Outstanding Teaching Award
2016-2017 Integrative Neuroscience Training Group Representative (training group Chair), Executive Committee, Purdue University Interdisciplinary Life Science Program (PULSe)2012 HSCI Graduate School Admissions, *Ad hoc reviewer*
2014 Presenter, Preliminary Exam Panel (PULSe), "Oral defense of proposal", 02/11/2014
2013 Judge, PULSe Outstanding Graduate Student in Research Award
2012-2014 PRF Research Grant, *Ad hoc reviewer*
2012- Bilsland Dissertation Fellowship, *Ad hoc reviewer*
2012 Faculty representative, Integrative Neuroscience, PULSe Fall Open House

School of Health Sciences and Additional Committees

2022 Member, Compton Travel Award Committee
2022- Chair, Search Committee, Translational and Biomedical Toxicology
2021 Chair, Search Committee, Dual Career Search (Toxicology)
2021- Chair, Search Committee, Computational Toxicology
2019-2020 Chair, Search Committee, Computational or Systems Toxicology
2018- Member, Graduate Committee on Curricula, Admissions and Research policy, School of Health Sciences, Purdue University
2017-2019 Chair, School of Health Sciences Committee to Revise Tenure and Promotion Guidelines
2017-2018 Chair, Search Committee, Exposure Science/Industrial Hygiene Faculty position

2016-2018 Chair, Graduate Committee on Curricula, Admissions and Research policy, School of Health Sciences, Purdue University
2016-present Member, HSCI Primary Committee (Tenure and Promotion)
2015-2016 Chair, HSCI Web Page & Library Committee
2015-2016 Member, Search Committee, Industrial Hygiene/Toxicology Faculty position

2015–present Member, Committee on International Exchange Programs
2014 *Ad hoc* member, PULSe Executive Committee, Integrative Neuroscience
2014 Discussion Leader, Scholarly Excellence, Faculty Retreat, School of Health Sciences, Purdue University
2012-2023 Member, Nominations and Awards, School of Health Sciences, Purdue University
2012-2013 Member, Safety Committee, School of Health Sciences, Purdue University
2012-2016 Member, Graduate Committee on Curricula, Admissions and Research policy, School of Health Sciences, Purdue University
2003-2004 Member, Toxicology Symposium Committee, "Fetal Origins of disease", The 9th Annual Toxicology Research Symposium, The University of Michigan
2002-2003 Chair, Toxicology Symposium Committee, "Toxicants as Tools", The 8th Annual Toxicology Research Symposium, The University of Michigan
2001-2002 Rackham Academic Appeals Panel, The University of Michigan

Other institutional service

2013 Lead effort updating Plans of Study for Toxicology degrees. Created a nonthesis MS plan of study with laboratory-focus and Public Health focus tracks. Gained Graduate Committee and Full Faculty approval.

2012 Faculty representative (School of Health Sciences), August graduation, Purdue University

Service to the Community

2022 Lay presentation "Modifiable Risk Factors in Parkinson's Disease Development", Well-Informed Educational Program, Westminster Village, West Lafayette, IN

2022 Lay presentation "Genetic and Environmental Interactions in the Development and Progression of Parkinson's Disease", Parkinson's Awareness Association of Central Indiana, Inc.

2014 Lay presentation "Etiology and Pathology of Parkinson's disease", Parkinson's disease support group, Westminster Village, West Lafayette, IN

2013 Lay presentation "Role of genes and Environment in Parkinson's Disease", Parkinson's disease support group, Westminster Village, West Lafayette, IN

2012 Faculty representative, College of Health and Human Sciences, Indiana State Fair

2009 Medicine / Health / Microbiology Category Judge - Senior (9th-12th grade), 70th Pittsburgh Regional Science & Engineering Fair. 4/3/2009

2008 Lay presentation; education to outpatient drug addicts; "Effects of drug use on the brain", Night Intensive Outpatient Program at Gateway Rehabilitation Center, Pittsburgh, PA. 5/22/08

EXTERNAL CONSULTING (inclusive of litigation)

11/2024-present	Expert Witness, Plaintiffs' Leadership in the Camp Lejeune litigation in the USDC-EDNC. Services included to date: initial literature review on the role of perchloroethylene (tetrachloroethylene) in Parkinson's disease; general causation report.
11/2024-12/2024	Individual consultation with a farmer reportedly exposed to chlorpyrifos. Provided professional written scientific opinion on the potential adverse health effects in cattle and humans.
08/2024-present	Expert Witness, Johnson and Bell. Services included to date: medical and toxicology file review on alleged medical malpractice. Case No. 2023L006556; Cook County Circuit Court, Chicago, Illinois.
07/2022-05/2023	Expert Witness, BUNGER & ROBERTSON. Services included: discussion on delta-8 tetrahydrocannabinol (THC) – formulation, detection, adverse effects; especially in relation to how contamination and use may relate to assault; expert toxicological analyses of law enforcement, EMS, and hospital records; development and submission of expert witness scientific report. Case No. 53C02-2201-F3-000043; Monroe County Circuit Court II, Indiana.
05/2021-08/2021	Expert Witness, CIYOU & DIXON, P.C.; Analytical toxicology expertise relative to screen results for drugs of abuse. Services included: drug screen results review; literature review; determination of likelihood of use cessation relative to urine, oral fluid, and hair (head and body) screen results; determination of whether video evidence of alleged drug use was supported by screen data; pre-trial conferences with attorneys and clients; expert testimony in court on 08/19/2021 on the above items and also adverse effects during cross-examination. Case No. 53C04-1601-DR-000031; Monroe County Circuit Court VI, Indiana.
11/2020-04/2022	Expert witness. Perkins Coie/Winston & Strawn/Boeing. Services included: complaint review; expertise on neurotoxicology relevant to possible etiology of an amyotrophic lateral sclerosis case; literature review; medical and scientific records review; plaintiff deposition review; plaintiff disclosure review; pre-trial conferences; development and submission of expert witness scientific report; deposition; trial slide development and input; and mock direct and cross examinations. Case settled prior to trial. Case No. 18 L 8347; Circuit Court of Cook County, Illinois.