Firefighters with Parkinson’s Disease
www.FirefightersWithParkinsons.org
The Indiana University Firefighter Health & Safety Research and the Firefighters with Parkinson’s Disease Foundation - Neurotoxin Exposure Trauma Parkinson’s Prevention Treatment Research Initiative and on Carbon Monoxide, Cyanide, and other neurotoxin exposures on Fire Grounds

Dr. Jim Brown, PhD, Director
Firefighter Health & Safety Research Program
Phone: 317-856-2373

Gary Coons, EFO, CFI, Founder
Firefighters with Parkinson’s Disease
Phone: 317-331-7009
Are American firefighters being poisoned unknowingly during the performance of their fire suppression and rescue duties? Recent findings suggest that firefighters may in fact frequently be exposed, sometimes unknowingly, to a dangerous toxins like: Cyanide, low level to high level Carbon Monoxide, N-Hexane, Toluene, and many others.

- Firefighters are at risk for neurodegenerative diseases from occupational exposures to psychological stress, toxic industrial and agricultural chemicals, chemical threat agents, head injury, and even radiofrequency radiation. Parkinson’s Disease (PD), as a particularly relevant disorder induced by a variety of environmental exposures, is a central focus of this legislation.

- Parkinson’s Disease (PD) affects nearly one million Americans. Recent evidence that genetic transmission is a minor component of this disorder confirms the importance of environmental factors, some of which are known, such as exposures to specific toxins. Firefighter occupational exposures are provide risks for neurodegenerative disease, including some Parkinson’s Disease. Fundamental mechanisms underlying the development of PD may also be pertinent to other neurodegenerative diseases.

- Fire Fighters are regularly exposed to burning chemicals and other toxins. There are 70,000 toxic substances on file with the Environmental Protection Agency (EPA) in the United States. In reality, when these substances burn together, there are 70 million possible combinations that are created in a fire. Routinely, exposures to these burning and non-burning toxins occur during the “Overhaul” phase of a fire or Hazardous Materials Incident.

- Parkinson’s Disease normally develops very slowly. Research has shown, however, toxicity is the probable cause when the Parkinson’s symptoms develop rapidly after a probable chemical – toxin exposure.

- A study conducted by the Neurotoxin Institute indicated that Parkinson's Disease was significantly more common in fire fighters than in the general population. This was demonstrated by a finding of 3-4 cases per 1,000 in the general population compared to 30 Parkinson's cases per 1,000 firefighters. Minerbo GM, Jankovic J. Prevalence of Parkinson's disease among firefighters. Presented at the 42nd Annual Meeting of the AAN, Miami, 5/4/90, Neurology (Suppl. 1) 1990;40:348.

- It is fact that even with the best respiratory practices and protective equipment the exposures will continue to occur due to absorption through the skin once a fire fighter’s protective gear has become saturated during fire suppression activities. Furthermore, the concentration of chemicals in today’s normal building contents is much higher than in the past due to the increased use of composite materials.

- In the 70’s materials burnt at 800 BTUs and now in 2009 they burn at 8000 BTUs due to new developments in composite materials and toxins they release.

- In a study by the Swedish National Testing and Research Institute (SNTRI), scientists identified fiberglass-based materials as producing some of the highest levels of cyanide in fire smoke. Use of fiberglass insulation in American building construction is much higher today than 30 years ago. Many fire departments’ tactical overhaul and extinguishing operations have not been updated to address these and other changes that make smoke increasingly dangerous.
Fire services are firmly rooted in the principles and practices of risk management beginning with a thorough risk assessment of the communities they serve. The risk assessment model is also used to determine how, where and when fire department resources will be deployed to minimize community risk and optimize community safety. The risk management approach also drives and shapes all fire department operations, practices and policies. As with the communities they serve fire departments use their risk analysis related to their personnel to minimize risk and optimize safety and health. Fire departments are continually looking for and deploying technologies to improve firefighter performance and safety including the use of a personal protective equipment and enhanced “on-scene” management practices.

Fire departments have been paying considerable attention over the last 30 years to the immediate and long-term impacts of firefighting to health and welfare of firefighters. The evidence continues to point to exposures of firefighters on emergency incidents as a prime cause of early death, chronic illness and increased incidence of health problems such as heart/lung and neurological disease.

<table>
<thead>
<tr>
<th>Carbon Monoxide: Firefighter Risk Review</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Firefighters</strong> routinely exposed to carbon monoxide as part of their firefighting duties.</td>
</tr>
<tr>
<td>Firefighters are at risk during periods the SCBA’s (self-contained breathing apparatus) are not worn.</td>
</tr>
<tr>
<td>Studies have shown low levels of untreated CO lead to central nervous system and cardiovascular disease.</td>
</tr>
<tr>
<td>CO levels are highest during the post-fire or “overhaul”, &amp; search &amp; rescue operations.</td>
</tr>
<tr>
<td>Firefighters typically wear little or no protection from CO at vehicle fires.</td>
</tr>
<tr>
<td>Moderate to high levels are immediate threats to health and safety and cause cardiovascular injury.</td>
</tr>
<tr>
<td>Affinity to hemoglobin 250 X greater than oxygen.</td>
</tr>
<tr>
<td>Interferes with heart and skeletal muscle.</td>
</tr>
<tr>
<td>Oxygen starvation.</td>
</tr>
<tr>
<td>Central nervous system damage.</td>
</tr>
</tbody>
</table>
EXECUTIVE SUMMARY OF PROPOSED RESEARCH

Title: Monitoring blood gas concentrations of carbon monoxide (CO) and hydrogen cyanide (HCN): A comparison of validity and utility of commercially available devices for fire ground application.

Firefighters are routinely exposed to smoke, combustion byproducts, and hazardous materials on the fire ground and other emergency scenes. Many of these compounds are known to be carcinogenic or to cause damage to vascular and neural tissues. Accordingly, firefighters are at great risk for the development of various cancers, heart (or vascular), and neurologic disease.

Two primary toxins, carbon monoxide (CO) and hydrogen cyanide (HCN) have been identified as being of particular concern on the fire ground. Both acute and chronic exposures to these compounds are potentially hazardous or fatal. Therefore, it is important that firefighter exposure to these compounds be minimized (or eliminated) and the ability to detect exposures is easily and readily available. Indeed, firefighters generally wear breathing protection during fire suppression operation. Unfortunately, current standards of practice in the fire service do not sufficiently address the hazardous exposure potential of other phases of fire scene operations. In addition, no currently available device has been shown to be valid or useful on the fire ground for the detection of exposure to these compounds. It is the purpose of the proposed study to evaluate currently available devices for the measurement of blood gas concentrations of CO and HCN and to determine their potential for use on the fire ground.

The study will involve a laboratory-based validation of two commercially available devices, the Massimo® RAD-57 CO detector and the COB® CO breathalyzer. The validation will involve simultaneous, non-invasive measurement of blood gas values by each device and by direct measurement by arterial blood sampling. Using direct blood measurement as the gold standard, the ability of each device to determine real blood gas values. With the validity of each device established, their utility on the fire ground will be evaluated by real-world testing with the Indianapolis Fire Department. During this phase of the study, an embedded scientist will accompany IFD firefighters to fire scenes and evaluate the ease, practicality, and effectiveness of use for each device. As a result of the study, we will establish the validity (accuracy and precision) of each device and supply recommendations for the use of each device on the fire ground.

The Indiana University Firefighter Health & Safety research program relies entirely upon external funding support to conduct its research. Therefore, in order to undertake the study, financial support is essential. Funding is utilized to support researcher salaries, research supplies & equipment. It is estimated that successful completion of the proposed project would require support funding from the Indiana Homeland Security Foundation Grant Program. A detailed operational budget will be made available upon request.

<table>
<thead>
<tr>
<th>DATA Collection</th>
<th>Question</th>
</tr>
</thead>
<tbody>
<tr>
<td>Collect CO/MetHb levels from firefighting/EMS members.</td>
<td>What should be the guide treatment and transport decisions?</td>
</tr>
<tr>
<td>Assist in identifying acute exposure vs. fatigue.</td>
<td>Early Identification of CO levels can drive ongoing patient care.</td>
</tr>
<tr>
<td>Can we reduce length of acute exposure?</td>
<td>What data is being collected to ensure Firefighter REHAB and Medical Monitoring is critical for responder health / safety?</td>
</tr>
<tr>
<td>Development of a policy and procedure for definitive evaluation of members exhibiting exposure.</td>
<td>Does Protective Equipment PREVENT CO toxicity?</td>
</tr>
</tbody>
</table>
RESEARCH PROGRAM PROSPECTUS

FIRE GROUND TOXIC GAS EXPOSURE

Introduction

Each year in the US, approximately 100 firefighters die on the line of duty. Nearly half of those deaths are due to heart attack (myocardial infarction or MI) or some other collapse of the cardiovascular system (Fahey, 2005). Even more disturbing is that the average age of firefighters experiencing these fatal attacks was 42 years. In addition, more than 700 firefighters suffer non-fatal heart attacks per year (NIOSH, 2007). Recently, researchers across the country have taken up this issue in an attempt to determine why so many firefighters experience MI in the line of duty.

Dr. Stephanos Kales of Harvard University completed a study in 2006 (Kales, 2007) that indicated more than 95% of the fatal line of duty heart attacks occurred in firefighters with underlying cardiovascular disease. Dr. Kales and his colleagues postulated that these individuals were predisposed to an MI because of their disease state and that physical and emotional stress of firefighting may simply act as a trigger for a catastrophic event. At the time Dr. Kales published his findings, no comprehensive survey of the cardiovascular stress associated with firefighting was available. In 2007, my research team began a six-month-long study designed to quantify the cardiovascular and respiratory stress associated with structural firefighting. Our findings indicate that structural firefighting, even on non-environmentally heat stressed; relatively non-complex fire scenes can induce extreme cardiac stress in highly trained and physically fit firefighters. Thus, we confirmed Dr. Kales’ postulate that firefighting can act as a trigger for MI, especially in individuals with known or unknown underlying cardiovascular disease.

Problem Statement

With the basic question of whether or not structural firefighting can trigger a heart attack answered, another question has arisen. Is there something about firefighting that causes or promotes the development of cardiovascular disease?

Although we cannot answer definitively yet, the answer is almost assuredly, yes! Career firefighters generally work some multiple of a 24 hour work shift during which they tend not to have time to get proper amounts of sleep. Anecdotally, even in the rare event that they have time to sleep, anticipation of an alarm can prevent the attainment of recuperative non-REM cycle sleep. Deprivations of sleep time and poor quality sleep have been shown to be risk factors for the development of heart disease.

In addition to sleep issues, firefighters are routinely exposed to a multitude of toxic gases and environments known to have cardio- and neuro-toxic effects on the body. Both acute and chronic exposures to compounds like carbon monoxide and hydrogen cyanide have been shown to induce cardiovascular and neural problems and even death in large doses. Therefore, firefighting almost certainly can promote the development of cardiovascular disease in firefighters. Although some exposure to these compounds may be unavoidable. The development of sound operating procedures and guidelines for fire ground toxic gas detection and the use of breathing protective devices is essential to the minimization of exposure.
RESEARCH PROGRAM GOALS

The primary goal of the research program is to establish essential fire ground monitoring practices as well as develop guidelines for the use of the self contained breathing apparatus (SCBA) to prevent fire ground toxic gas exposure. To reach these long term goals, I propose a series of research studies (or phases as outlined below) that will culminate in the development of new standards and guidelines for monitoring the fire ground gas environment as well as firefighters on scene.

Program Phase 1: Pilot Study

This portion of the program is currently underway. We have partnered with local fire service assets as well as federal agencies (NIST) to develop fire ground monitoring protocols. The goal of the pilot study program is to develop protocols necessary to effectively monitor a fire event. Utilizing local assists (Carmel and Indianapolis Fire Departments); we are able to test data collection methodologies during acquired structures burns.

Program Phase 2: Investigation and Validation of fire ground monitoring technology

The goal of this portion of the program is to investigate currently available technologies for the monitoring of fire ground air quality. In addition, we will be looking at technologies currently (and soon to be) available for the detection of carbon monoxide and hydrogen cyanide within the human body. Although it is the goal of the program to help prevent exposure and uptake of toxic gases by firefighters, it is essential that we are able to detect environmental and blood levels to evaluate preventative strategies.

Program Phase 3: Fire Scene gas evolution survey

This tightly controlled program phase will involve the construction of test structures which will be burned to measure the evolution and movement of toxic gases in and around a burning structure. Using the protocols and technologies developed in earlier work, this study phase will involve the quantification of off gassed compounds and the tracking of these combustion products on the fire ground. This portion of the study will form the basis of real world fire scene monitoring guidelines.

Program Phase 4: Real World practices

The final experimental phase of the study will move to the street. By accompanying Indy Fire Department companies to fire scenes, we will test our newly developed protocols for monitoring fire scenes and firefighters.

Program Phase 5: Information Dissemination

An essential element for this work will be the dissemination of study-derived information to the fire service. We will utilize multiple media forms (audio, video, print, web sites etc) to distribute this information to the fire service.
The intent is to help the fire service in general better understand the threat these gases impose and guidelines to minimize exposure.

Identify the NFPA 1584 Standard related to medical rehabilitation guidelines to minimize exposure.

To analyze how much carbon monoxide and hydrogen cyanide firefighters are exposed to under current SCBA standard operation procedure.

Identify the 9 steps of NFPA 1584 rehab.

Track these gases from evolution by pyrolysis through a burning structure, into the firefighter, and finally out of the firefighter.
Understanding of the Disease Mechanisms allows:

1. Determine Risk for Carbon Monoxide Poisoning & Progression of the Disease
2. Exploit Symptom Mechanisms for Early Diagnostic Markers
3. Identify and Validate Neuroprotectants, Therapeutics & Ways to Reduce or Remove Symptom Effects

**Research Application**

**Carbon Monoxide Research**

- Environmental Exposures
  - Determine Risk for Carbon Monoxide Poisoning & Progression of the Disease

- Early Diagnosis and Prognosis Methods
  - Exploit Symptom Mechanisms for Early Diagnostic Markers

- Neuroprotectants and Treatments
  - Identify and Validate Neuroprotectants, Therapeutics & Ways to Reduce or Remove Symptom Effects

**Firefighting Applications**

- Early Detection Methods for Threats to Performance
- Treatments for Neurological Injury and Preventive Methods
PROJECT PLAN DESCRIPTION:

Firefighters may be at risk for neurodegenerative diseases from occupational exposures to psychological stress, toxic industrial and agricultural chemicals, chemical threat agents, head injury, and even radiofrequency radiation. Parkinson’s Disease (PD), as a particularly relevant disorder induced by a variety of environmental exposures, is a central focus of the research program.

Basic research on mechanisms of neurodegeneration will lead to better diagnosis, treatment, and prevention. The NETPPTRI is requesting funding with special congressional appropriations from the NETPR program to study neurodegenerative mechanisms and treatments, with a special emphasis on Parkinson's Disease. There are currently 1 discrete research projects in the program being conducted by the Indiana University Firefighter Safety and Health Research.

Parkinson’s Disease (PD) affects nearly one million Americans. Recent evidence that genetic transmission is a minor component of this disorder confirms the importance of environmental factors, some of which are known, such as exposures to specific toxins. Firefighter occupational exposures create risks for neurodegenerative disease, including some Parkinson’s Disease. Fundamental mechanisms underlying the development of PD may also be pertinent to other neurodegenerative diseases.

In PD there is a loss of brain cells in the substantia nigra that contain the neurotransmitter dopamine. Insufficient dopamine neurotransmission underlies many of the symptoms of PD. New technologies such as neuroimaging and genetic knockout models, and recent advances in neurobiology such as new drugs and growth factors (e.g., GDNF) are being exploited to advance this research. This program directly contributes to common objectives in psychological stress and toxicology research in the core-funded basic research program.

Early Detection

Valid and reliable biological markers of neurodegeneration (e.g., cognitive testing, neuroimaging, specific biochemical markers) are an important technological hurdle. One study tests changes in the ability to manipulate aspects of language to determine if these precede motor dysfunction, as a test for early PD-associated degeneration in the cortical dopaminergic system. A separate study will develop imaging markers diagnostic for early PD and clarify the relationship between striatal dopamine integrity and cortical activation during motor and memory tasks. Several other projects study biomarkers to detect and monitor neuronal degeneration: a marker of dopamine terminal integrity which may directly evaluate the rate of degeneration and will also be used to study the neurotoxicity of L-dopa. Another study creates a genomic database to identify unique molecular markers associated with progressive decrease in striatal dopamine integrity and consequent impaired motor and cognitive performance in inbred and knockout mouse models of PD.

Pathogenesis Oxidative Damage: A large number of studies focus on oxidative damage as a common mechanism of neurotoxicity. The brain is particularly susceptible to oxidative damage for several reasons: in addition to receiving a high percentage of the body's total oxygen, the brain is relatively deficient in catalase which protects against reactive oxygen species (ROS) damage; it's most important defense mechanism against ROS damage, the glutathione system, is easily saturated; it has a high concentration of polyunsaturated lipids, easily peroxidized by free-radicals; and regions such as the substantia nigra have high concentrations of iron which catalyzes the conversion of H2O2 to OH., allowing production of excess quantities of ROS.

Excitotoxic Damage: Increased action of excitatory amino acid transmitters such as glutamate (Glu) and aspartate may initiate or prolong depolarization of neurons, with subsequent clinical signs (e.g. seizures) or neuronal exhaustion and cellular degeneration. The ionotropic glutamate receptors (iGluR) have a primary role in fast neuronal excitation, including the excitotoxic actions of Glu. The prominent role of the NMDA-preferring class of iGluR in the pathophysiology of excitotoxic neurodegeneration is being studied to develop potentially neuroprotective agents.
Apoptosis: Many studies in the program specifically investigate aspects of apoptosis, a process in which developmental cues and environmental stimuli initiate a genetically established cascade that results in cellular degeneration and cell death. Several studies concentrate on interactions of apoptotic cascade events while others focus on mitochondrial intervention points.

Accumulation of Intracellular Aggregates and Other Cell Damage: Mechanisms of cellular disruption are being studied in four cellular protein systems (alpha-synuclein, tissue plasminogen activator, p38 protein kinase family, and neurotoxic esterase). Studies of the alpha-synuclein gene family will investigate expression/cell distribution, mutations, and over-expression on neurodegeneration. Another study tests the hypothesis that gene expression and functional up-regulation of specific neural sodium channels are contributors to neuronal cell death after cerebral ischemia.

ETIOLOGIES

Stress: Early markers of neurodegeneration, such as hippocampal changes and memory impairment, are being studied in a social mouse model of combat stress. Regional susceptibility of the nigrostriatal system to damage after prolonged periods of stress is being studied in 6-OHDA lesioned rats. Another study investigates beneficial short-term and deleterious long-term actions of acetylcholinesterase (AChE) in animals subjected to acute psychological stress or anticholinesterases. Stress, AChE inhibitors (chlorpyrifos), and fuel additives (which may synergistically promote degeneration in structures such as the hippocampus) are being investigated for changes in behavior and CNS excitability.

Other studies include the construction of a tetracycline-inducible vector system in which either an anti-sense message for the AChE or a ribozyme directed against the AChE will be expressed via a plasmid injected into the hippocampus.

Toxins: Insecticides (permethrin and chlorpyrifos), in combination with MPTP, will be studied to clarify whether insecticide exposure can intensify development of PD and will provide a full dose-effect curve useful in extrapolating animal data to man for risk assessment. Mechanisms by which mustard chemical warfare agents induce neuronal cell death (a model for neurotoxic DNA damage) are being studied using mice deficient in DNA repair mechanisms. Another study examines synergistic effects of excitotoxicity, free radicals, and a depleted bioenergetic state in causing dopaminergic cell death. A sample of men who have been well characterized by serial neurologic examinations over a 26-year period, will be used to correlate environmental and occupational risk (including pesticide residues in brain tissue) for development of PD and related neurodegenerative disease.

Trauma: Mechanisms of cell death due to traumatic brain injury will be studied and may lead to identification of optimal brain region targets for calpain inhibitors to prevent further neurodegeneration in already damaged brain tissue.

Viral: Animals infected with Venezuelan equine encephalitis virus (VEE) variants will be studied for gradations of neurotoxicity resulting from glial cell activation, including assessment of alterations in production of cytokines and reactive nitrogen intermediates that influence neuronal degeneration in VEE infection.

Therapeutic Strategies

Interventions Targeting Specific Mechanisms of Damage: Several studies test treatments for excitotoxic damage (including neuroprotective effects provided by a metabotropic GluR (mGluR) agonist); neuroprotectant combinations of vitamin E, Co-enzyme Q, melatonin, FK506, and benzamide; Huperzine A analogs to shield glutamate ionotropic receptors in cultured neuronal cells; and efficacy of NMDA receptor agonist drugs to mitigate excitotoxic neuronal damage (from retinal laser irradiation). The mechanisms by which transforming growth factors protect neurons against excitotoxic damage and induce motor axon growth using gene transfer of GDNF or Bcl-2 into nigral neurons in animal models of PD are also being studied in a comparison of gene delivery methods: encapsulated BHK cells genetically modified to secrete GDNF and intrastriatal infusion of an adenovirus GDNF transgene vector in aged monkeys. One project investigates mechanisms by which some neuronal cell lines resist ROS damage following exposure to the neurotoxins nitrogen mustard and arsenite.
Neuroprotectants: Potential neuroprotective effects of progesterone, based on recent evidence that progesterone decreases excitotoxicity leading to neuronal cell loss, will be explored to determine if the effects are direct or involve neurosteroid metabolites acting on GABA receptors. Another project investigates the effects of glycosphingolipids (GSL) using two unique insect model systems that permit structure-function studies of GSLs on growth and repair of nerve cells. A study investigating liposome delivery systems for a recombinant enzyme (OPAA-2) may greatly increase neuroprotection against alkyl-phosphate chemicals, and the protective actions of various anti-inflammatory drugs, anticonvulsants, and glutaminergic potentiator agents (ampakines) will be studied to identify neuroprotectant drugs which will not block other important receptors or signal transduction systems.

Restoration of Dopaminergic Secretory Capabilities: Replacement graft strategies have suffered from poor survival, insufficient axonal outgrowth from surviving neurons, poor functional recovery provided by the procedure, and disabling side effects such as dyskinesia; however, functional recovery associated with surviving grafts encourages further work. Several studies attempt new approaches to improve transplant survival including use of caspase inhibitors, regulation of Bcl-2 and GDNF to improve survival of transplanted neural cells, and identification of neurotrophic factors in other types of brain cells (O2-A cells). A study of differentiation of mesencephalic progenitor cells will attempt to create new dopaminergic neurons induced by hematopoietic cytokines such as IL-1.
Gary Coons was a career firefighter for 15 years before being medically pensioned with a line-of-duty injury. In 2005, he went through a series of surgeries to repair his shoulder and back damage related to his line-of-duty injuries. During this time, his wife stated that she started noticing symptoms related to an overall slowness in his demeanor, leg tremors while at rest, blank stares, and stooped over walking. At the same time he was aware of an increased stiffness and pain in his shoulder and lower extremities, smaller hand writing, and soft speaking. They both related these symptoms to his injuries and sought medical help from multiple physicians over a two year period. The doctors offered therapies to mitigate the symptoms and did not relate these complications to a more complex neurological disease. Within this two year period, the symptoms were getting worse and his right arm and hand started to tremor, he was stuttering, and developed impaired fine motor dexterity and coordination. They then decided to seek out a neurologist, who conducted several test and medication trials over 6 months resulting in a diagnosis of early onset Parkinson’s Disease at the age of 33.

The average age of onset of Parkinson's Disease is the late fifties. At 33, he soon came to realize that he had developed Parkinson's Disease over 20 years earlier than is normal. The chances of anyone having early onset Parkinson's Disease at this age are less than 1 in 100,000, making it a rare medical disorder, and therefore more likely to be the result of “unusual circumstances.”

A toxic exposure is one of the “unusual circumstances” that can trigger Parkinson’s Disease. So, after a review of his fire department work history, they found an event which provides strong correlations between the onset of his Parkinson’s and a probable significant toxic exposure. In 2005, he was the lead investigator of a large paint warehouse fire. The contents of the fire building included several paint trucks (vehicles), painting materials, paint chemicals, Toluene, Carbon Monoxide, and many other unknown toxins. He was exposed to many toxins during the 3 day investigation with little to no respiratory protective equipment (the scene was deemed “All Clear for SCBA (Self Contained Breathing Apparatus) by the incident commander. It was within a short time frame, he and his wife started noticing the symptoms listed above.

Parkinson's Disease normally develops very slowly. Research has shown, however, toxicity is the probable cause when the Parkinson’s symptoms develop rapidly after a probable chemical exposure.
Fire Fighters are regularly exposed to burning chemicals and other toxins. There are 70,000 toxic substances on file with the Environmental Protection Agency (EPA) in the United States. In reality, when these substances burn together, there are 70 million possible combinations that are created in a fire. Fire Fighters routinely endure exposure to these burning toxins in the course of protecting the lives and property of their fellow citizens. 

It is fact that even with the best respiratory practices and protective equipment the exposures will continue to occur due to absorption through the skin once a fire fighter’s protective gear has become saturated during fire suppression activities. Furthermore, the concentration of chemicals in today’s normal building contents is much higher than in the past due to the increased use of composite materials.

A study by the Neurotoxin Institute revealed an increased rate of disease in the Fire Fighter population versus the general population. The study of fire fighters indicated that Parkinsonism was significantly more common in fire fighters than in the general population. This was demonstrated by a finding of 3-4 cases per 1,000 in the general population compared to 30 Parkinson's cases per 1,000 firefighters. Minerbo GM, Jankovic J. Prevalence of Parkinson's disease among firefighters. Presented at the 42nd Annual Meeting of the AAN, Miami, 5/4/90, Neurology (Suppl. 1) 1990;40:348.

Neurotoxicity can be documented, but perhaps not in the way you might think. A person’s ability to think, perceive, control emotions, plan, and manage his or her life can diminish drastically without anything being visible to a radiologist or neurologist on an MRI or a CT scan.

The most reliable and widely accepted way to assess actual brain function is through neuropsychological evaluation. (This is true for head-injury patients and those suffering from dementia, as well as those affected by exposure to toxic chemicals.)

Researchers have noted that imaging techniques are often of little value in evaluating neurotoxicity. In our and others’ experience, imaging techniques can occasionally pick up abnormalities caused by neurotoxicity and may be helpful for forensic purposes, but they are not cost-beneficial for routine screening. Neuropsychological testing tends to be more sensitive to brain injury than CT and routine MRI scans, which provide only a static and relatively gross view of neural structure. In one study of six head-injury cases, CT and/or MRI scans yielded little or no evidence of neuropathology as detected by neuropsychological testing. Positron emission tomography (PET) scans, however, corroborated the impaired function.4 PET and SPECT (single photon emission computed tomography) scans offer a more dynamic look at brain structure, but both of these tests still need interpretation as to the cause of the abnormality (which could be benign). (Raymond Singer, Ph.D., is a forensic neurobehavioral toxicologist and neuropsychologist in Santa Fe, New Mexico, with additional offices in New York City.)
Governor Mitch Daniels signed SB 376 on May 1, 2009 to enact Parkinson’s Disease to the Firefighters’ and Police Officers’ presumptive law. The bill allows emergency responders afflicted with Parkinson's Disease to become eligible for expanded disability benefits is now become law, thanks to the efforts of State Rep. Mary Ann Sullivan (D–Indianapolis) and State Senator James Merritt (R-Indianapolis).

By a vote of 49-0 in the Senate and a vote of 97–2 in the House Senate Bill (SB) 376 passed, a measure championed by Sullivan and Merritt that will require Parkinson's disease to be treated as a line-of-duty disability under an emergency responder's pension and disability plan. Emergency responders who are disabled in the line of duty are entitled to greater benefits than those who incur other types of disabilities.

Recent studies have shown that toxic fumes, like those inhaled by emergency responders at the site of a burning building, can increase the risk of Parkinson's disease. Sullivan and Merritt said that these studies necessitated the need for the legislation.

"If our community's firefighters, police officers and paramedics put themselves at risk of Parkinson's disease by combating a fire or attending to another dangerous emergency, they should be entitled to the highest level of disability benefits," Sullivan said.

“We had compelling testimony from Medically Pensioned (line-of-duty) Firefighter Gary Coons in the committee hearing for Pension and Labor that illustrated what our heroes face every day as they serve us. There is a website up that provides valuable information and has hits and discussion from around the world,” Merritt said.

The bill was brought to Sullivan's and Merritt’s attention by Indiana firefighter Gary Coons, who was stricken with Parkinson's at age 33. On average, a person doesn't contract Parkinson's until their late fifties. Sullivan and Merritt both explained that toxic exposure from burning chemicals is one of the unusual circumstances that can cause early onset Parkinson's.

Gary realizes that his Parkinson's disease is not a death sentence, but a life sentence and that he must stay active and wants the bell to ring for the next round. Gary exemplifies the fact that he does not look towards lightening his load, but broadening his shoulders. As a result, he and his wife developed a website www.firefighterswithparkinsons.org and a foundation to provide a powerful imperative to get information out about the risk of occupational exposures for firefighters.

The long term affect of not protecting and educating firefighters/public safety personnel could lead to the development of long term neurological degenerative disease, such as Parkinson's disease or Parkinsonism. Toxic exposure can be prevented, by following best practices in safety and wearing full protective gear while in the “Hot Zone”. Gary’s message to those public safety personnel is: “Always remember, it's not how long you have been doing it; it's how well you have been protecting yourself and your fellow firefighters.” He also states: “His Parkinson’s is not his disease, but his family’s disease.” Think of
managing change to protect yourself and your crew as an adventure. It tests your skills and abilities. It brings forth knowledge of best practices that may have been dormant. Change is also a training ground for leadership. When we think of leaders, we remember times of change, innovation and conflict. Leadership is often about shaping a new way of life and paradigm. To do that, you must advance change, accept responsibility for making change to better protect and educate firefighters and other public safety professionals.