



# Addressing Toxic Smoke Particulates in Fire Restoration

**By: Sean M. Scott**

In the restoration industry today, a lot of attention is given to the testing and abatement of microscopic hazardous materials. These include asbestos, lead, mold, bacteria, pathogens, and all sorts of bio-hazards fall into this category. If these contaminants are disturbed, treated, or handled improperly, all of them can cause property damage and serious harm to the health and welfare of those living or working in or near the areas where they're present. However, there are other hazardous toxins that commonly present themselves in restoration projects, that seem to go unnoticed. These are the toxic smoke particulates created during structure fires.

When a building is abated from asbestos, lead, or mold, special care is given to be sure every microscopic fiber, spore, and bacteria is removed. This is done through extensive cleaning, HEPA vacuuming, chemical applications, negative air, and other procedures. Once the remediation is completed, an independent environmental

testing laboratory or industrial hygienist provides an air clearance test to certify that the abatement or remediation process was successful. Upon receipt of the clearance, people can then reenter the remediated area, rooms, or building. However, when the structural repairs are completed after a fire, an air clearance test is rarely ever performed. How then can consumers be assured or restoration companies guarantee that the billions of toxic particulates and volatile organic compounds (VOCs) generated by the fire have been removed? Is there cause for concern or is a simple "sniff" test or wiping a surface with a Chem-sponge sufficient? Why is it so common to hear customers complain of smelling smoke long after the restoration is completed? What measures are being taken to protect workers and their families from toxic particulate matter?

In the following pages, we will be taking a close look at this issue to raise awareness of a segment in the restoration industry that has been long overlooked and in most cases disregarded entirely.

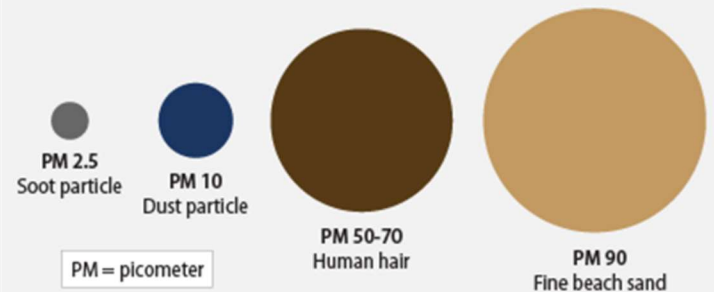
## WHAT IS SMOKE?

Smoke is a complex mixture of toxic gases and particles, which are generated from the vast array of materials that burn during a fire. A typical structure fire may generate literally tens of thousands of toxic chemicals and gasses as a variety of materials and products are burned. Research on cigarette smoke alone has identified over 7,000 chemicals, with 70 identified as cancer causing, even with brief exposure.<sup>(1)</sup> Studies haven't even scratched the surface to determine all the potentially adverse health effects that may result from exposure to the chemicals released from the different products that may burn in a structure fire. These include plastics, foams, textiles, carpets, wood products (treated lumber, plywood, flooring), synthetic fabrics, wool, electronics, household chemicals, and the list goes on. One of the biggest health threats from smoke is from fine and ultra-fine particles. These microscopic particles can penetrate deep into the lungs and can cause a range of health problems, from burning eyes and a runny nose to aggravated chronic heart and lung diseases. Exposure to particle pollution is even linked to premature death.<sup>(2)</sup> Exposure to smoke toxins can be far more hazardous to human health than mold, asbestos, lead, or other contaminants. Respiratory ailments, cardiac hazards, and cancers connected with exposures to an environment affected by a fire are far greater than those from the past, mainly because the materials used today to manufacture products and their chemical composition have changed dramatically.

<b>TOXIC SMOKE ANATOMY</b>	
<p><b>Particulates (soot &amp; dust)</b></p> <ul style="list-style-type: none"> <li>➤ Hydrochloric acid</li> <li>➤ Sulfur dioxide</li> <li>➤ Nitrogen oxides</li> <li>➤ Ammonia</li> <li>➤ Formaldehyde</li> <li>➤ Benzene</li> </ul>	<ul style="list-style-type: none"> <li>• Structure fires often involve polyvinyl chloride (PVC), used to make upholstery, pipe, conduit, blinds, etc.</li> <li>• Burning PVC creates hydrogen chloride &amp; phosgene.</li> <li>• Polyethylene &amp; PVC are more toxic when soldering than during the heat of a working fire. They emit carbon monoxide, hydrogen cyanide, hydrochloric acid, and other toxins.</li> </ul>
<p><b>Asphyxiants/Toxicants</b></p> <ul style="list-style-type: none"> <li>➤ Carbon dioxide</li> <li>➤ Hydrogen cyanide</li> <li>➤ Carbon monoxide</li> <li>➤ Hydrogen sulfide</li> <li>➤ Acrolein Aldehyde</li> <li>➤ Phosgene</li> </ul>	

FIGURE 1

### How big is particle pollution?



Source: U.S. Environmental Protection Agency

## THE HIDDEN HAZARDS OF FIRE SOOT<sup>(3)</sup>

Smoke is the result of incomplete combustion, which produces tiny particles of carbon in the air. When deposited, these particulates are identified as soot. Put simply, the particle size of smoke residue on a surface can present a respiratory hazard. The particle size of soot is approximately 2.5 microns, a size that is associated with deep lung penetration. Particles that are approximately 10 microns or larger get trapped in the upper respiratory tract. Particles that are 5 microns or smaller can make it down to the lower lung where

the gas exchange occurs in the alveoli. In order to offer some perspective on the size of these particles, the dust you see flying in the light coming through a sun lit window is about 40 microns in size. Airborne soot is too small to be seen with the naked eye and can easily be inhaled. As a fire dies down, the smoke it has caused will disperse leaving behind a residue of quickly cooling particles which is generally referred to as soot. Typically, soot is representative of what has burned, but may

include byproducts that at first seem unrelated to the original material.

For example, hydrogen cyanide is a byproduct of burning wool. When wood burns it can produce manganese and benzene. As many products as there are in the world, there are an equal number of byproducts produced in a fire and many are known carcinogens or extremely hazardous when inhaled or absorbed by the skin. A common house fire results in the burning of a wide variety of materials, from wood and paper to plastics and other synthetic items. This results in soot contamination, and poses a serious cleanup problem. Breathing the tiny particles can cause coronary heart disease, asthma, bronchitis, and many other respiratory illnesses.

Research has also shown that many premature deaths are directly related to soot in the environment. **Particle exposure leads to around 20,000 premature deaths in America each year. Many of these deaths were caused by soot-related diseases.** Data also shows that soot annually causes almost 300,000 asthma attacks and 2 million lost workdays due to respiratory problems.<sup>(4)</sup>

While we would not expect most volatile organic compounds to survive a fire and still be present on the soot, there may be metals present as well as some chlorinated compounds. The Phoenix Fire Department examined this phenomenon during a study focused on firefighter exposures after a fire was extinguished and their findings indicated that **some chlorinated products become attached to airborne particulate matter.** This is an important study because it points to the fact that exposure to airborne vapors and residues after a fire are much more complex than our current understanding allows. However, the important point is that soot may be more than just a particulate hazard; it can potentially carry other chemical residues that are potentially harmful to the respiratory system.

## **TOXIC CHEMICALS AND GASES**

The toxic mixture of chemicals and gasses contained in fire smoke is comprised primarily of carbon monoxide, carbon dioxide, hydrogen cyanide, ammonia, hydrogen chloride, sulfur dioxide, hydrogen sulfide, acids, and oxides of

nitrogen. Other toxins may include acetylene, methyl mercaptan, ammonia, nitric oxide, carbon disulfide, creosote, nitrogen dioxide, dimethyl sulfide, phosphine, ethylene, benzene, methylene chloride, lead, chromium, and other metals, trichloroethylene, toluene, trichlorophenol, fluorine, acrolein, mercury, formaldehyde, glutaraldehyde, benzaldehyde, sulfuric acid, hydrochloric acid, arsenic, chromate, phenol, styrene, polycyclic aromatic hydrocarbons, and the list goes on.

Chlorine gas was the first lethal chemical to be used in World War I, resulting in thousands of casualties. Then later phosgene and diphosgene were used. Hydrogen cyanide was also produced,



but its physical properties were found to be unsuitable for use as an effective chemical warfare agent. **(Hydrogen cyanide and phosgene are both commonly found in structure fire smoke)** Phosgene was first used as a Chemical Weapon by the Germans, but was later used by the French, Americans, and British. Initial deployment of the gas was by the Germans at Ypres Salient on December 19, 1915 when they released around 4000 cylinders of phosgene combined with Chlorine against the British. Phosgene was responsible for the majority of deaths that resulted from chemical warfare.<sup>(5)</sup> To illustrate the types of chemicals found in structure fires, here is a list of some of the most common toxic chemicals and gases found after structure fires and their effect on human health:

## Benzene

<https://www.cancer.org/cancer/cancer-causes/benzene.html>

The International Agency for Research on Cancer classifies benzene as carcinogenic to humans, based on sufficient evidence that benzene causes acute myeloid leukemia. Benzene exposure has been linked with acute lymphocytic leukemia, chronic lymphocytic leukemia, multiple myeloma, and non-Hodgkin lymphoma.

## Arsenic

<https://www.globalhealingcenter.com/natural-health/health-dangers-arsenic-toxicity/>

Arsenic is an element that is extremely toxic to human health. Arsenic poisoning occurs when someone is exposed to arsenic, typically by unknowingly ingesting it. Poisoning can occur by inhalation or through dermal absorption.

## Sulfur Dioxide

<https://www.atsdr.cdc.gov/phs/phs.asp?id=251&tid=46>

Sulphur Dioxide reacts with humidity and forms sulphuric and sulphurous aerosol acid. It causes breathing difficulty, airways inflammation, psychic alterations, pulmonary edema, heart failure, and circulatory collapse. Sulphur dioxide is also associated with asthma, chronic bronchitis, morbidity and mortality increase in the elderly and infants.

## Hydrogen Cyanide (HCN)

<https://www.atsdr.cdc.gov/MMG/MMG.asp?id=1141&tid=249>

Breathing small amounts of hydrogen cyanide may cause headache, dizziness, weakness, nausea, and vomiting. Larger amounts may cause irregular heartbeats, seizures, fainting, and rapid death.

## Hydrogen Sulfide

<https://www.atsdr.cdc.gov/MMG/MMG.asp?id=385&tid=67>

Symptoms of acute exposure include nausea, headaches, delirium, disturbed equilibrium, tremors, convulsions, and skin and eye irritation. Inhalation of high concentrations of hydrogen sulfide can produce rapid unconsciousness and death.

## Formaldehyde

<https://www.cancer.org/cancer/cancercauses/formaldehyde.html>

The International Agency for Research on Cancer has concluded that formaldehyde is carcinogenic to humans, based on higher risks of nasopharyngeal cancer and leukemia.

## Phosgene

<http://www.toxipedia.org/display/toxipedia/Phosgene>

During or immediately after exposure to dangerous concentrations of phosgene, the following signs and symptoms may develop: Coughing, burning sensation in the throat and eyes, watery eyes, blurred vision, difficulty breathing or shortness of breath, nausea and vomiting. Skin contact can result in lesions similar to those from frostbite or burns. Exposure to phosgene may cause delayed effects that may not manifest for up to 48 hours after exposure, even if the person feels better or appears well following removal from exposure. Therefore, people who have been exposed to phosgene should be monitored for 48 hours afterward. Delayed effects that can appear for up to 48 hours include the following: Difficulty breathing, coughing up white to pink-tinged fluid (a sign of pulmonary edema) low blood pressure, and heart failure.

## Mercury

<https://www.atsdr.cdc.gov/toxfaqs/TF.asp?id=113&tid=24#bookmark05>

Methylmercury and metallic mercury vapors are more harmful than other forms, because more mercury in these forms reaches the brain. Exposure to mercury can permanently damage the brain, kidneys, and developing fetus. Effects on brain functioning may result in irritability, shyness, tremors, changes in vision or hearing, and memory problems. Very young children are more sensitive to mercury than adults. Mercury in a mother's body passes to the fetus and may accumulate there. It can also pass to a nursing infant through breast milk. Mercury's harmful effects that may be passed from the mother to the fetus include brain damage, mental retardation, incoordination, blindness, seizures, and inability to speak. Children poisoned by mercury may develop problems of their nervous and digestive systems, and kidney damage.

## Styrene

<https://www.osha.gov/SLTC/styrene/hazards.html>

Chronic exposure affects the central nervous system showing symptoms such as depression, headache, fatigue, weakness, and may cause minor effects on kidney function.

## Hydrogen Chloride

<https://www.atsdr.cdc.gov/toxfaqs/TF.asp?id=759&tid=147>

Hydrogen chloride is irritating and corrosive to any tissue it contacts. Brief exposure to low levels causes throat irritation. Exposure to higher levels can result in rapid breathing, narrowing of the bronchioles, blue coloring of the skin, accumulation of fluid in the lungs, and even death. Exposure to even higher levels can cause swelling and spasm of the throat and suffocation. Some people may develop an inflammatory reaction to hydrogen chloride. This condition is called reactive airways dysfunction syndrome (RADS), a type of asthma caused by some irritating or corrosive substances.

## Phenol

<https://www.atsdr.cdc.gov/mmg/mmg.asp?id=144&tid=27>

Exposure to phenol by any route can produce systemic poisoning. Phenol is corrosive and causes chemical burns at the contact site. Coma and seizures can occur within minutes or may be delayed up to 18 hours after exposure. Other symptoms include nausea, vomiting, diarrhea, methemoglobinemia, hemolytic anemia, profuse sweating, hypotension, arrhythmia, pulmonary edema, and tachycardia. As a corrosive substance, phenol denatures proteins and generally acts as a protoplasmic poison. Phenol may also cause peripheral nerve damage (i.e., demyelination of axons). Systemic poisoning can occur after inhalation, skin contact, eye contact, or ingestion. Damage to the nervous system is the primary cause of death from phenol poisoning. However, damage to other organ systems (e.g., acid-base imbalance and acute kidney failure) may complicate the condition. Symptoms may be delayed for up to 18 hours after exposure.

## Acrolein

<https://www.ncbi.nlm.nih.gov/pubmed/19028774>

Acrolein vapor may cause eye, nasal and respiratory tract irritations in low level exposure. A decrease in breathing rate was reported by volunteers acutely exposed to 0.3A ppm of acrolein.

## Methyl Mercaptan

<https://www.atsdr.cdc.gov/MMG/MMG.asp?id=221&tid=40>

Methyl mercaptan is a central nervous system depressant that acts on the respiratory center to produce death by respiratory paralysis. Individuals with pre-existing respiratory, cardiac, nervous system, or liver impairment may be more susceptible to exposure to methyl mercaptan.

## Toluene

<https://www.atsdr.cdc.gov/MMG/MMG.asp?id=157&tid=29>

Toluene is irritating to the skin, eyes, and respiratory tract. It can cause systemic toxicity by ingestion or inhalation and is slowly absorbed through the skin. The most common route of exposure is via inhalation. Symptoms of toluene poisoning include central nervous system effects (headache, dizziness, ataxia, drowsiness, euphoria, hallucinations, tremors, seizures, and coma), ventricular arrhythmias, chemical pneumonitis, respiratory depression, nausea, vomiting, and electrolyte imbalances. The mechanism by which toluene produces systemic toxicity is not known. Toluene crosses the placenta and is excreted in breast milk.

## Trichloroethylene

<https://www.atsdr.cdc.gov/phs/phs.asp?id=171&tid=30>

People who are overexposed to moderate amounts of trichloroethylene may experience headaches, dizziness, and sleepiness; large amounts of trichloroethylene may cause coma and even death. Some people who breathe high levels of trichloroethylene may develop damage

to some of the nerves in the face. Other effects seen in people exposed to high levels of trichloroethylene include evidence of nervous system effects related to hearing, seeing, and balance, changes in the rhythm of the heartbeat, liver damage, and evidence of kidney damage. Relatively short-term exposure of animals to trichloroethylene resulted in harmful effects on the nervous system, liver, respiratory system, kidneys, blood, immune system, heart, and body weight.

## Polycyclic Aromatic Hydrocarbons

<https://www.atsdr.cdc.gov/csem/csem.asp?csem=13&po=11>

Increased incidences of lung, skin, and bladder cancers are associated with occupational exposure to PAHs. Epidemiologic reports of PAH-exposed workers have noted increased incidences of skin, lung, bladder, and gastrointestinal cancers. These reports, however, provide only qualitative evidence of the carcinogenic potential of PAHs in humans because of the presence of multiple PAH compounds and other suspected carcinogens.

**For more detailed information on smoke particulate pollution and their effects on human health visit:**

<https://woodsmokepollution.org/references.html#particulate-references>

## TOXICOKINETICS AND MECHANISM OF TOXICITY<sup>(6)</sup>

**Toxic combustion products are classified as simple asphyxiants, irritant toxins, and chemical asphyxiants.** Simple asphyxiants are space occupying and fill enclosed spaces at the expense of oxygen. In addition to this effect, combustion uses oxygen and creates an oxygen-deprived environment.

Irritant toxins are chemically reactive substances. They produce local effects on the tissue or the respiratory tract. Ammonia is produced by burning wool, silk, nylon, and synthetic resins. Ammonia has high water solubility and dissolves in moist membranes of the upper respiratory tract, resulting in nasopharyngeal, laryngeal, and tracheal inflammation. Acrolein is lipid soluble and



penetrates cell membranes. It denatures nucleic acid and intracellular proteins and results in cell death. Acrolein is a very common irritant gas generated by combustion. Sulfur dioxide is found in more than 50% of smoke from fires. Sulfur dioxide reacts with the moist respiratory membrane mucosa, producing the potent caustic, sulfurous acid. Polyvinyl chloride is ubiquitous in floor coverings, office and home furniture, electrical insulation, and clothing. The resultant combustion products, phosgene, chlorine, and hydrogen chloride are produced in many residential fires. Together with water in the mucosa, chlorine produces hydrogen chloride free oxygen radicals and is very damaging to tissue. Phosgene descends and produces more delayed alveolar injuries. Isocyanates are produced from burning and smoldering upholstery, and intense irritation of both upper and lower respiratory tissue results.

Soot has not only carbon, but also aldehydes, acids, and reactive radicals that adhere to its surface. Inhalation of soot and associated aerosols heightens the effect of other irritant toxins. Soot binds with respiratory mucosal surfaces, allowing other irritant chemicals to adhere and react with adjacent tissue. The penetrance and deposition of these particles within the respiratory tract depend on size. Small particles (1 to 3 microns) reach the alveoli. Lung injury decreases when smoke is filtered to remove particulate matter. Sulfur dioxide shows a high propensity to adhere to soot. In addition, polyvinyl chloride combustion produces a large amount of soot-containing smoke coated with its particular combustion products phosgene, chloride, and hydrogen chloride. Irritant gases, acids, and other combustion products can also adhere to aerosol droplets.

The most important determining factor in predicting the level of respiratory injury is the water solubility of the toxin. Water-soluble chemicals injure the mucosa of upper respiratory airways by releasing the mediators of inflammation and deleterious free radicals. This type of inflammation increases microvascular membrane permeability and results in a net influx of fluid from intravascular spaces into the upper respiratory tissue. The underlying tissue of the supraglottic larynx may become terrifically swollen and edematous. This edematous reaction can result in minutes to hours

post exposure, continue to progress, and close off upper airways completely.

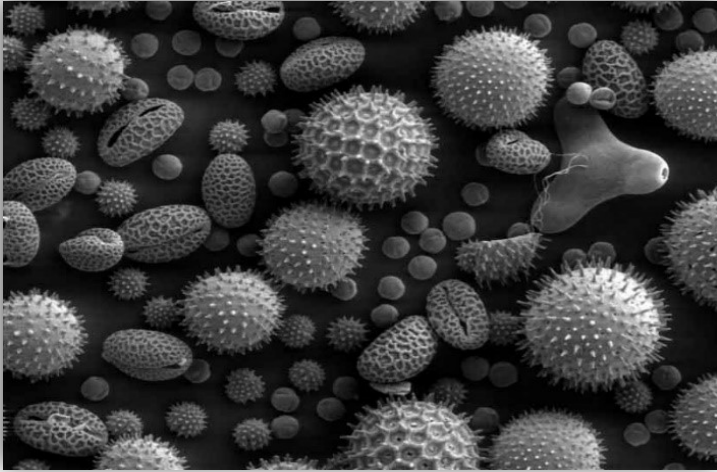
Low water-soluble molecules react with the lung parenchyma. They react more slowly and produce delayed toxic effects. Concentration of the toxic element inhaled, particle size, duration of exposure, respiratory rate, absence of protective reflexes, preexisting disease, and age also contribute to the level and degree of respiratory injury in addition to the water solubility of toxins.

An intense inflammatory reaction develops secondary to the initial injury to respiratory mucosal cells by toxic combustion products. Inhaled soot and toxic gases generate increased airway resistance caused by inspissated secretions, increased mucosal airway edema, and associated bronchospasm. Damaged mucosal cells stimulate copious exudates rich in protein, inflammatory cells, and necrotic debris. If this reaction continues, mucosal sloughing ensues. The degenerative exudates, bronchorrhea, and extensive sloughing produce casts of the airways. In animal victims of smoke inhalation, these casts increase airway resistance by blocking major airways and prevent oxygen passage to the alveoli. Increased vascular permeability of respiratory tissue contributes to airway blockage.

**Nitrogen-containing products, such as wool, silk, nylon, plastics, paper, rubber, pyroxylin, polyurethanes, and polyacrylonitriles, all produce cyanide at combustion.** Cyanide has been detected in samples from many other types of fires as well. Other combustion products can cause systemic and local toxicity. Metal oxides, hydrogen fluoride, hydrogen bromide, and various hydrocarbons can all be retrieved from toxic smoke.

Benzene can be detected in the smoke of plastic and petroleum fires. Antimony, cadmium, chromium, cobalt, gold, iron, lead, and zinc have all been recovered from smoke samples during fires. **In fact, the entire spectrum of potentially toxic combustion products from fires is endless. In addition, short-term exposure to fine particulates has been associated with triggering heart attacks, particularly among people with pre-existing heart disease.**

## FIREFIGHTER EXPOSURE TO SMOKE PARTICULATES (7)



Underwriters Laboratories in collaboration with the Chicago Fire Department and the University of Cincinnati College of Medicine, recently completed a sixteen month study on the smoke and gas exposure firefighters confront during firefighting operations and subsequent contact exposure resulting from residual contamination of personal protective equipment.

This study investigated and analyzed the combustion gases and particulates generated from three scales of fires: residential structure and automobile fires, simulated real-scale fire tests, and material based small-scale fire tests. Material-level tests were conducted to investigate the combustion of forty-three commonly used residential building construction materials, residential room contents and furnishings, and automobile components under consistent, well-controlled radiant heating conditions. In these tests, material based combustion properties including weight loss rate, heat and smoke release rates, smoke particle size and count distribution, and effluent gas and smoke composition were characterized for a variety of natural, synthetic, and multi-component materials under flaming. The results from these tests were used to assess the smoke contribution of individual materials.

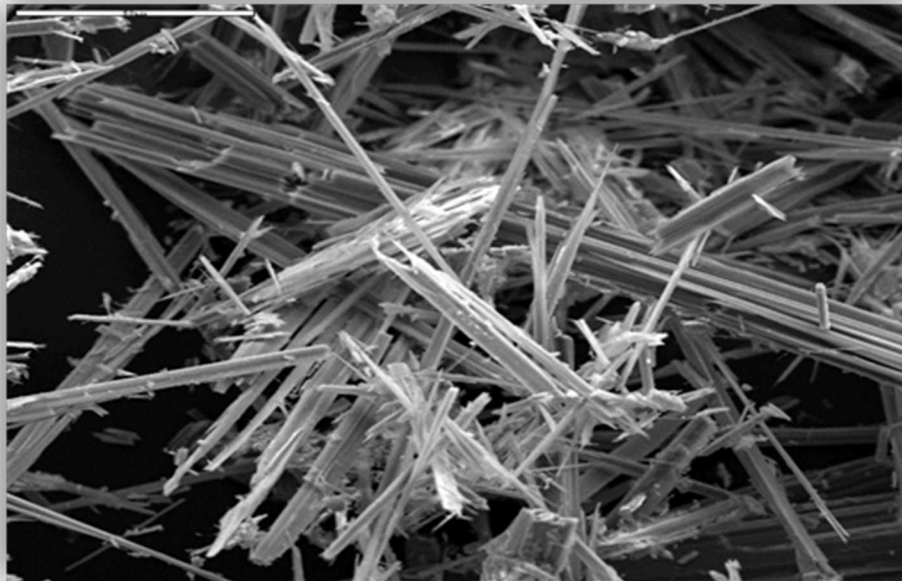
Nine real-scale fire tests representing individual room fires, an attic fire, deck and automobile fires were conducted at UL's large-scale fire test laboratory to collect and analyze the gas effluents, smoke particulates, and condensed residues produced during fire growth, suppression and overhaul under controlled, reproducible laboratory conditions.

### KEY FINDINGS

- The type and quantity of combustion products (smoke particles and gases) generated depended on the chemistry and physical form of the materials being burned.
- The most prolific smoke production was observed for styrene based materials commonly found in residential households and automobiles. These materials may be used in commodity form (e.g. disposable plastic glasses and dishes), expanded form for insulation, impact modified form such as HIPS (e.g. appliances and electronics housing), copolymerized with other plastics such as ABS (e.g. toys), or copolymerized with elastomers such as styrene-butadiene rubber (e.g. tires).
- Vinyl polymers also produced considerable amounts of smoke. Again, these materials are used in commodity form (e.g. PVC pipe) or plasticized form (e.g. wiring, siding, resin chairs and tables).
- Average particle sizes ranged from 0.04 to 0.15 microns with wood and insulation generating the smallest particles.
- For a given particle size, synthetic materials will generate approximately 12.5X more particles per mass of consumed material than wood based materials.
- Combustion of the materials generated asphyxiants, irritants, and airborne carcinogenic species that could be potentially debilitating. The combination and concentrations of gases produced depended on the base chemistry of the material:
- Styrene based materials formed benzene, phenols, and styrene.
- Vinyl compounds formed acid gases (HCl and HCN) and benzene.
- Wood based products formed formaldehyde, formic acid, HCN, and phenols.
- Roofing materials formed sulfur gas compounds such as sulfur dioxide and hydrogen sulfide.
- Collected smoke particulates contained multiple heavy metals including arsenic, cobalt, chromium, lead, and phosphorous, lead, mercury, phthalates and PAHs.

## HEALTH IMPLICATIONS

- Multiple asphyxiants (e.g. carbon monoxide, carbon dioxide and hydrogen sulfide), irritants (e.g. ammonia, hydrogen chloride, nitrogen oxides, phenol and sulfur dioxide), allergens (e.g. isocyanates), and chemicals carcinogenic for various tissues (e.g. benzene, chromium, formaldehyde and polycyclic aromatic hydrocarbons) were found in smoke during both suppression and overhaul phases. Carcinogenic chemicals may act topically, following inhalation or following dermal absorption, including from contaminated gear.
  - Concentrations of several of these toxicants exceeded OSHA regulatory exposure limits and/or recommended exposure limits from NIOSH or ACGIH.
  - Exposures to specific toxicants can produce acute respiratory effects that may result in chronic respiratory disease.
- High levels of ultrafine particles (relative to background levels) were found during both suppression and overhaul phases.
  - Exposure to particulate matter has been found to show a positive correlation with increased cardiovascular morbidity and mortality for general population studies.
  - The high efficiency of ultrafine particle deposition deep into the lung tissue can result in release of inflammatory mediators into the circulation, causing toxic effects on internal tissues such as the heart. Airborne toxics, such as metals and polycyclic aromatic hydrocarbons, can also be carried by the particles to the pulmonary interstitium, vasculature, and potentially other body tissues, including the cardiovascular and nervous systems and liver.
  - Interactions between individual exposure agents could lead to additive or synergistic effects exacerbating adverse health effects.
  - Long-term repeated exposure may accelerate cardiovascular mortality and the initiation and progression of atherosclerosis.



*Asbestos fibers magnified with an electron microscope*

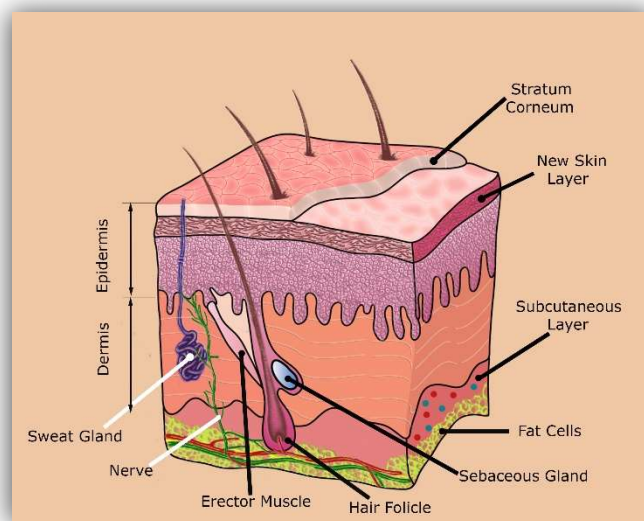
## THE TOXIC ASSAULT ON THE HUMAN BODY<sup>(8)</sup>

Toxins enter the human body by several routes including, ingestion, inhalation, injection and absorption. The total toxic load encountered by a body is the sum of all possible routes of entry.

With an exchange surface area approximately that of a tennis court and a very small diffusion distance, the lung is designed for the exchange of gases between inhaled gas and the blood stream. Although the lung is set up this way to facilitate the exchange of oxygen and carbon dioxide as part of normal respiration, it also provides an effective pathway for toxic gases to enter the blood stream. The two most important fire ground toxic gases that utilize this pathway are CO and HCN. CO works as an asphyxiant by binding hemoglobin 200 times more effectively than oxygen. It eliminates the blood's ability to deliver oxygen throughout the body. HCN is also an asphyxiant. It attacks the cell's ability to utilize oxygen and generate energy. Significant exposure to HCN generally results in penalization of respiratory muscle and asphyxiation. More importantly, both HCN and CO are produced in a structural fire. They work synergistically to hurry death by attacking respiration from two sides, oxygen delivery and oxygen use.

Skin absorption of a toxic substance is far more complicated than inhalation. Many factors affect the rate or even whether or not a substance is absorbed through the skin. The skin can be pictured as a two-layer system. The outer layer, the stratum corneum or epidermis, is a thin layer of dead cells that act as a primary barrier to absorption. Below the epidermis is a much thicker layer of living tissue that contains blood vessels, sweat glands, hair follicles and nerves. Absorption through this system is driven by diffusion alone. When a substance is deposited on, or in contact with skin surface, a concentration gradient is established that drives diffusion. This relationship is described by Fick's law of diffusion which in essence says, how much of the material reaches

the blood stream and contributes to a toxic load is determined by the characteristics of both the compound and the tissue. Fick's law indicates that the rate of diffusion is determined by several factors including the surface area for diffusion (area of skin contaminated) and the concentration of the contaminant on the skin. In addition, the chemical characteristics of the contaminant are also important. The epidermis is a hydrophobic layer meaning it repels water. Therefore, compounds similar to water will have a difficult time getting through. Organic compounds, like solvents, cross the epidermis more easily. Gases, like HCN and hydrogen sulfide (H<sub>2</sub>S) move easily across the dermis and, in appropriate concentration, can contribute substantially to a toxic load. Following the movement of a compound from the skin surface to the perfusion rich area of the skin, the amount of blood flow through the skin is another factor which contributes to toxic load.



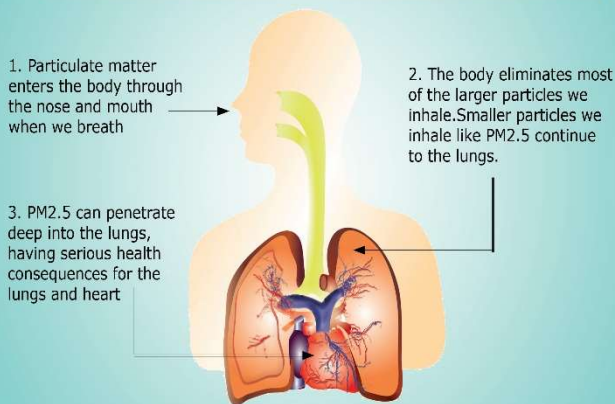
## PARTICULATE MATTER AND AIR POLLUTION (9)

Particulate pollution, also called particle pollution or particulate matter (PM), is a mixture of solid particles and liquid droplets in the air. Particles come in many sizes and shapes and can be made up of hundreds of different chemicals. Particulate matter is classified by size. "Fine particles," or PM<sub>2.5</sub>, are those smaller than 2.5µm (micrometers, or microns), in diameter, which is about 30 times smaller than the diameter of an average human hair. Coarse particles, or PM<sub>10</sub>, are between 2.5 and 10µm in diameter. Smaller than PM<sub>2.5</sub> are ultrafine particles, PM<sub>0.1</sub>.

### In the Lungs and Bloodstream

Particles larger than PM<sub>10</sub> do not go into the deepest parts of the lungs, and they can be coughed out. Particles smaller than PM<sub>10</sub>, however, are inhaled into the deepest parts of the lungs, where they become embedded and can cause disease. The smallest ultrafine particles, which are numerous in wood smoke, are so minute they behave like gases, passing through the lungs and directly into the bloodstream. Once in the bloodstream, these ultrafine particles carry toxins around the body and promote inflammation.

#### HOW PARTICULATE MATTER ENTERS THE BODY



### Bypassing the Blood/Brain Barrier

Ultrafine particles also travel up through the nose and, rather than passing down into the lungs, are delivered directly into the brain and central nervous system via the olfactory nerve, bypassing the body's protective blood/brain barrier.

### Adverse Health Effects and Raised Death Rate

Numerous peer-reviewed studies have described the adverse health effects, including premature death, that are associated with particulate pollution. Effects have been observed at levels as low as 10–20 µg/m<sup>3</sup> of PM<sub>10</sub>, which are levels equal to or below background in many parts of the world. Many studies have shown no evidence of a threshold — an amount under which exposure does not harm health.

### Bad for the Heart and Lungs

Increases in particulate pollution levels can trigger heart attacks, strokes and irregular heart rhythms, especially in those with preexisting heart or lung diseases, and aggravate other lung diseases such as asthma and COPD. Particulate air pollution levels have been significantly associated with hospitalization rates for pneumonia in the elderly. Associations have also been found between small increases in PM<sub>2.5</sub> levels and sudden cardiac arrest. A 2013 review in the *Lancet* found a close and consistent association between short-term exposure to ambient air pollution levels and acute decompensated heart failure and heart failure deaths. The authors estimated that, in the US alone, a mean reduction in PM<sub>2.5</sub> of only



3.9  $\mu\text{g}/\text{m}^3$  would prevent 7,978 heart failure hospitalizations and save a third of a billion dollars a year.

### Even Low Levels Increase the Death Rate

A 2015 study of New England Medicare recipients over age 65 determined that the death rate rises for each 10  $\mu\text{g}/\text{m}^3$  increase in PM<sub>2.5</sub>, both from short-term and long-term exposure, and even when pollution levels do not exceed US EPA or World Health Organization thresholds. These findings were consistent with those of several other studies that have also found an association between particulate pollution levels and an increase in mortality rate, even at levels that are considered low by regulatory standards.

### Like Lead Pollution, There Is No Safe Level

According to Joel Schwartz, senior author of the New England study and professor of environmental epidemiology at Harvard T.H. Chan School of Public Health, this was the first study to examine the effect of airborne soot particles over an entire region.

"The harmful effects from the particles were observed even in areas where concentrations were less than a third of the current standard set by the EPA." According to Schwartz, "Particulate pollution is like lead pollution; there is no evidence of a safe threshold even at levels far below current standards, including in the rural areas we investigated."

More recently, Schwartz and other researchers at Harvard looked at data covering 60 million U.S. seniors. This much larger study, which covered approximately 97% of U.S. residents aged 65 and older, similarly found that long-term exposure to PM<sub>2.5</sub> increases the risk of premature death, even at levels below current regulatory standards.

### Fine Particle Matter Is Officially Recognized as Carcinogenic Linked to Brain Shrinkage and Strokes

Research has shown that long-term exposure to even slightly elevated PM<sub>2.5</sub> levels is associated with a .32% smaller total brain volume and a 46% higher risk of silent strokes, even in people who haven't yet developed dementia or had obvious strokes. It has also been shown that older women living in areas with higher levels of particulate pollution have smaller white matter volumes. Other research has also shown an association between strokes and particulate pollution, especially from "air pollutants related to combustion." Risk of death from an ischemic stroke is higher when levels of PM<sub>2.5</sub> are higher. Even five years after an initial stroke, survival rates are reduced significantly with increased exposure to PM<sub>2.5</sub>.

### Particulate Pollution and Parkinson's and Alzheimer's Diseases

Evidence shows that particulate pollution is also a risk factor both in the development and progression of neurodegenerative diseases such as Alzheimer's and Parkinson's.

Short-term increases in PM<sub>2.5</sub> are associated with an increased risk of hospitalization and an increased risk of death for people with diabetes and, even more so, for people with Parkinson's disease.

Long-term exposure to elevations in particulate levels is significantly associated with increased first-time hospital admissions for Parkinson's disease, Alzheimer's disease and dementia. The risk was seen even at relatively low levels of pollution.

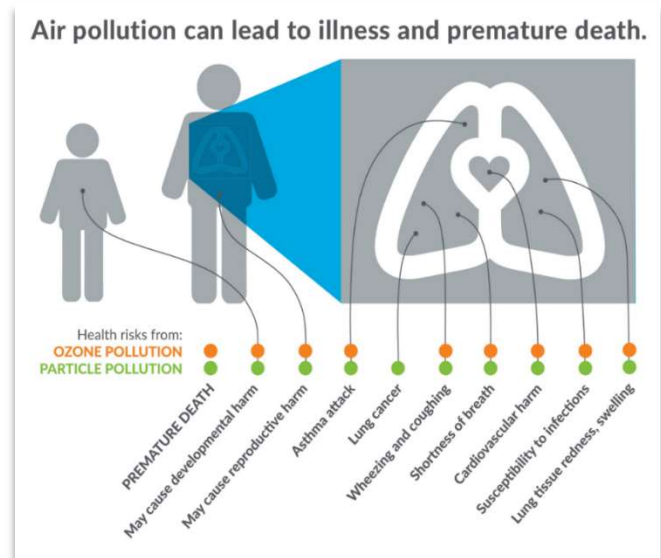
A recent study found that older women residing in places with fine particulate matter exceeding EPA standards had an 81% increased risk of global cognitive decline, and were 92% more likely to develop dementia from any cause, including Alzheimer's. The risk was even stronger in women who had the APOE4 gene, a genetic variation that increases the risk for Alzheimer's.

Researchers who study the neuroinflammatory effects of air pollution in young people have declared that exposure to air pollution should be considered a risk factor for Alzheimer's and Parkinson's diseases.

### More Links to Cognitive Impairment

Another study has also linked fine particulate pollution with worsened cognitive function. It was found that episodic memory is most impaired by exposure to PM<sub>2.5</sub>. "Episodic memory impairment is one of the core diagnostic criteria used to determine mild cognitive impairment and dementia in older adults, and it has been suggested that impairments in this aspect of memory represent some of the earliest signs of dementia."

It was noted that particulates can affect the respiratory and cardiovascular systems, which can in turn affect the vascular pathology in the brain. Particulates may even directly damage the brain itself. "Studies of both humans and animals have confirmed that exposure to PM is linked to harmful changes in brain health and functioning."



Graphic provided by The American Lung Association: <http://www.lung.org/our-initiatives/healthy-air/sota/health-risks/>

Other studies have also confirmed an association between cognitive decline and long-term exposure to particulate pollution. For example, it was found that exposure to particulate pollution at levels "typically experienced by many individuals in the United States is associated with significantly worse cognitive decline in older women."

## OZONE - REASONS YOU SHOULD NEVER USE AN OZONE GENERATOR TO CLEAN INDOOR AIR (10)

The use of ozone in fire restoration is not a cure-all and it can be dangerous. After a fire, it is all too common to hear "industry experts" advocate for the use of ozone as the silver bullet to completely sanitize the air and remove all particulate matter. Unfortunately, much of the material that supports the use of ozone makes claims and draws conclusions without substantiation or sound science. There are even some vendors which suggest that their devices have been approved by the federal government, despite the fact that there is not one agency within the federal government which has approved ozone generators for use in occupied spaces. The EPA published several documents which highlight the risks and dangers of ozone and why ozone generators should be avoided.

The reliance on ozone to remove all particulate matter is similar to someone who has cancer and is only given narcotics to stop the pain. This treats the symptoms, not the cause and may make the condition much worse.

### Why is Ozone Dangerous?

Ozone is a tiny molecule composed of three oxygen atoms. It is highly reactive which makes it an unstable and potentially toxic gas. Ground-level ozone is considered to be a major component of smog which plagues larger cities during the summertime and has been tied to a variety of potential health risks.

## Health Effects of Ozone & Particle Pollution

The EPA has reported there is a variety of health effects associated with high levels of ozone. Ozone is the most widespread pollutant in the U.S. It is also one of the most dangerous pollutants—and it's invisible.

Exposure to ozone may include decreased lung function, throat irritation, severe asthma symptoms, cough, chest pain, shortness of breath, irritation of lung tissue, and the higher sensitivity to respiratory infection. They went on to note that there were additional risk factors which can perpetuate the side effects of ozone such as undertaking activities which raise breathing rates (such as exercising indoors), certain pre-existing lung diseases, and greater duration of exposure.

## 4 Reasons Why You Should Avoid Air Purifiers that Produce Ozone

Not only is ozone potentially dangerous to your health, it may not work at all. Here are four reasons why you should never use an air purifier that produces ozone.

### 1. **Ozone Generators May Not Work at All**

Some manufacturers suggest that ozone will render nearly every chemical contaminant in the home harmless by producing a chemical reaction. **This is incredibly misleading because a thorough review of scientific research has shown that in order for many of the dangerous chemicals found indoors to be eliminated the chemical reaction process may take months or even years.** Other studies have also noted that ozone cannot effectively remove carbon monoxide. If used at concentrations that do not exceed public health standards, ozone applied to indoor air pollution does not effectively remove viruses, mold, bacteria, or other biological pollutants.

### 2. **The Chemical Reaction Can Be Dangerous**

Even if ozone generators were proven to be effective at eliminating these chemicals, there are certain side effects everyone must be aware of. Many of the chemicals ozone reacts to results in a variety of harmful by-products. For example, when ozone was mixed with chemicals from new carpet in a laboratory setting, the ozone created a variety of dangerous organic chemicals in the air.

### 3. **Ozone Generators Do Not Remove Particulates**

A third factor to consider when looking at ozone generators is that **they do not remove particulates such as dust or pollen from the air. This includes the particles which are primarily responsible for allergic reactions.** To combat this, some ozone generators include an ionizer which disperses negatively charged ions into the air. In recent analysis's, this process was found to be less competent in the removal of air-borne molecules of dust, smoke, pollen, and mold spores than HEPA filters and electrostatic precipitators.

### 4. **It Is Impossible to Predict Exposure Levels**

The EPA notes that it is increasingly difficult to determine the actual concentration of ozone produced by an ozone generator because so many different factors come into play. Concentrations will be higher if more powerful devices are used in smaller spaces. Additional factors which affect concentration levels include how many materials and furnishings are in the room to react with ozone, the level of outdoor air ventilation, and the proximity of a person to the ozone generating device.

### **HEPA Filters: The Safer & More Effective Solution**

Choosing a HEPA filter is a significantly safer and effective solution than an ozone generator. Instead of releasing dangerous ozone, a HEPA filter traps indoor air pollutants rather than trying to create a chemical reaction with them. In many cases, this type of filter is often combined with other technologies, such as activated carbon, to provide even better results. Utilizing a pre-filter and activated carbon, air purifiers can remove solid, chemical, and gaseous pollutants from the air and dramatically improve indoor air quality.

## CONCLUSION:

The information compiled in this paper provides compelling evidence that restoration practitioners need to consider when addressing smoke and particulate matter generated by structure fires. As an industry, we can no longer overlook the fact that people who have their properties damaged by fire and smoke will be exposed to toxic elements long after the repairs are completed if proper measures aren't taken to remove toxic particulate matter and restore the indoor air quality. The following points summarize a few of the key findings of the research compiled in this paper:

- 1) Residual smoke, soot, chemicals, and microscopic particulates generated from structure fires are hazardous to human health and can cause severe injury and even death.
- 2) Toxic particulate matter can enter the body through the lungs or by contact with the skin.
- 3) Toxic particulates can adversely affect the health of everyone. Those at the greatest risk of injury are infants and children, the elderly, and those with weakened respiratory systems or cardiac ailments.
- 4) Particulate pollution is like lead pollution; there is no evidence of a 100% safe threshold of exposure.
- 5) Ozone and Hydroxyl can produce hazardous byproducts and are not proven to be 100% effective to remove all particulate matter from the air.
- 6) Sealing/encapsulating exposed surfaces or applying liquid deodorizers does not address particulate matter suspended in the air or soot. Relying on these methods alone will most likely leave occupants exposed to the hazards associated with toxic particulate exposure and odor reoccurrence.
- 7) Toxic smoke particulate matter must be properly remediated prior to the return of occupants who may live or work in or around the affected areas.

## STEPS TO ENSURE A RESTORED INDOOR AIR ENVIRONMENT

Exposure to toxic smoke particulates can pose a greater health risk than that of toxic mold, asbestos, or lead-based paint dust exposure. In order to restore the indoor air quality after a structure fire or wildfire, similar procedures and protocols used in mold, asbestos, and lead abatement should be employed. These steps should be taken to properly clean and restore the indoor air quality, primarily by removing the particulates rather than relying solely on treating, deodorizing, or masking them. Steps would include, but are not limited to: Having a preliminary air sample test performed to determine what types and concentrations of chemicals and particulates are present. (this is important, so chemicals used in the cleaning process don't accidentally interact with the chemicals or gases in the air, which may produce more toxic byproducts) setting up containments, wearing proper personal protective equipment, running negative air filtration with proper HEPA filters, HEPA vacuuming surfaces, deodorizing, and encapsulation of exposed surfaces, etc. Once the cleaning process is completed, an independent environmental testing company should be hired to provide testing that includes, but is not limited to:

- 1) Surface dust wipe samples to test surfaces for combustion byproduct particulate matter including soot, char & ash.
- 2) Surface dust wipe samples to test for the presence of metals including lithium, chromium, lead, mercury, etc.
- 3) Air samples to test for Volatile Organic Compounds (VOC's) and smoke particulate matter associated with burned materials, paints, glues, plastic, synthetic materials, etc.
- 4) Air clearance sampling to verify that the air has been properly cleaned of toxic soot, char and ash particulate matter. An air clearance should be achieved prior to project completion and reentry of unprotected occupants.

**Without these steps, occupants could be left exposed to unseen toxins, which could lead to adverse health conditions or even death.**

For more information on testing for toxic smoke particulates, gases, and VOC's, and air quality clearance testing, contact [Bruce Rosenblatt with Rarefied Air Environmental at 619-888-4840](mailto:Bruce.Rosenblatt@rarefiedair.com).

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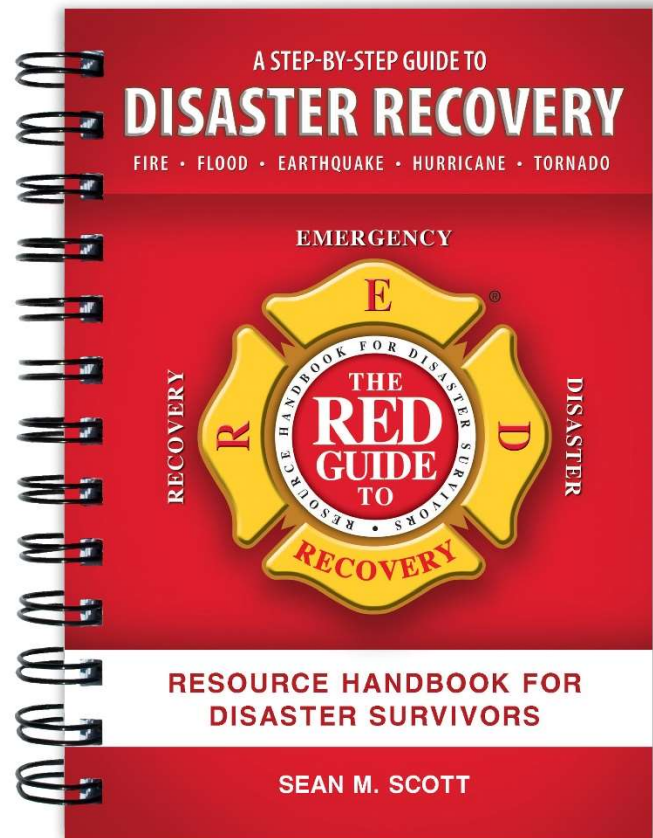


Mr. Scott is a licensed general contractor in the State of California who has spent over 39 years in the construction and restoration industry. As a second-generation restoration contractor, he has been involved with literally thousands of property damage claims of all types and sizes, ranging from commercial and residential floods, high-rise fires, smoke claims, mold contaminations, subsidence and earthquake claims, explosions, vehicular collisions, and many other types of property damaging incidents. Throughout his career, Sean has worked with all the major insurance carriers that underwrite residential and commercial policies and has worked directly with claims adjusters, independent adjusters, third party administrators, public adjusters, and attorneys. Sean is also the author of two books, ***The Red Guide to Recovery - Resource Handbook for Disaster Survivors*** and ***Secrets of the Insurance Game*** and the co-author of ***The Native Family Disaster Preparedness Handbook***.

Sean has devoted his life assisting individuals and families rebuild their homes, businesses, and lives and has witnessed first-hand the physical, emotional, and financial challenges people face once the first responders leave the scene. Since 2009, Sean's award-winning book ***The Red Guide to Recovery*** has been adopted by fire departments, emergency management agencies, and relief organizations across the U.S.

Sean now uses his time and expertise to help people navigate the recovery process, speak on recovery and restoration topics, and consult with those in the restoration industry who need guidance to be more successful. He also provides expert witness testimony, estimating services on property damage claims, insurance claim appraisal services, construction defect investigation, and training.

For more information on disaster recovery, **The Red Guide to Recovery – Resource Handbook for Disaster Survivors** and **Secrets of The Insurance Game** both provide a wealth of insight and useful information. Visit [www.TheRedGuideToRecovery.com](http://www.TheRedGuideToRecovery.com) for details on the content of these books and other disaster preparedness and recovery resources.





*Building Disaster Resilient Communities*

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