Review Article

Review of Toxic Gases and Their Impact on Human Health

Abstract

The likelihood of toxic gases occurring within a specific timeframe and location determines the likelihood of the event. The aim of this study is to review the literature on the effects of hazardous gases on human health. Hazardous gas releases can tragically occur as a result of industrial accidents, natural disasters, or terrorist attacks in inhabited regions. The literature introduces several modeling methods and approaches to both prevent and assess the effects of these disasters.These tools are invaluable resources for risk managers evaluating the risks associated with vulnerable areas. Although risk assessments for toxic gas dispersion have significantly improved, they often do not account for people's movements and behavior during emergencies.To enhance the accuracy of risk assessments for disasters involving toxic gases, this study proposes a method that considers both gas dispersion modeling and evacuation dynamics. Identifying effective methods for reducing toxic gas pollution and its adverse effects on the environment and human health is crucial. Toxic gases can harm nearly every organ and system in the body. Additionally, we discuss various corrective measures to mitigate their toxicity.

Keywords:toxic gasses, sources, effects, controls, human health.

1. Introduction

Natural or industrial disasters, as well as terrorist acts, can disperse toxic gases [1, 2]. Hazardous compounds known as toxic gases pose significant risks to human health when released into the environment. Exposure to these harmful gases can result from various sources, including natural disasters, industrial processes, transportation, and agricultural activities. Understanding how these gases impact human health is essential for taking preventative action and addressing potential health repercussions [3].Three main processes can release these gases into the atmosphere: the dispersal of toxic gases, the dispersion of combustion products after a fire involving hazardous chemicals, and the dispersion of toxic gases following an explosion [4]. Toxic gas dispersion can pose risks to humans, as it may affect people far from the original source, depending on atmospheric conditions [4]. Toxic substances can enter the human body through various pathways, including oral ingestion, skin contact, parenteral administration, or inhalation [5].To assess risks and understand their effects on human health, it is essential to identify and quantify the toxic substances involved[6]. The risk of poisoning is associated with the physicochemical properties of the substance and the conditions of exposure, such as dosage, route of entry, and duration of exposure. Additionally, environmental factors like weather and chemical agents, as well as human factors such as gender, age, body weight, diet, and physical fitness, contribute to the risk [7]. Although poisoning cases do not appear to be declining, deaths from poisoning have decreased due to more effective and accurate treatment methods [6].Polycyclic aromatic hydrocarbons (PAHs) can result from incomplete combustion processes, both man-made (e.g., vehicle emissions, cigarette smoke) and natural (e.g., forest and brush

fires). As a result, PAHs are commonly found in soil, water, and air, leading to the belief that these compounds are ubiquitous in the environment [8, 9]. The pyrolysis and burning of materials can produce several harmful combustion products that cause irritation, incapacitation, systemic toxicity, asphyxiation, and even fatal exposures after brief encounters. Common harmful substances in fire effluent include complex molecules like polycyclic aromatic hydrocarbons, nitrogen oxides (NOx), asphyxiant gases like carbon monoxide (CO) and hydrogen cyanide (HCN), and irritants such as acrolein and phosgene. These issues affect many people and are primarily caused by industrial activities.

2. The sources of the poisonous gas

2.1 Human made sources of toxic gas

Automobile Exhaust: Toxic chemicals such as sulfur dioxide (SO□), nitrogen dioxide (NO□), and carbon monoxide (CO) are released when vehicles emit exhaust. These pollutants can degrade air quality and harm human health [10].

Industrial Processes: Industries such as chemical manufacturing can release toxic gases like sulfur dioxide (SO□), hydrogen sulfide (H□S), ammonia (NH□), and volatile organic compounds (VOCs) [11].

Home products: Items like paints, cleaning supplies, and pesticides may contain volatile organic compounds (VOCs) that can release hazardous gases when misused or improperly stored [12].

Sewage and Landfills: The decomposition of organic waste produces methane (CH□), a potent greenhouse gas that, at higher concentrations, can be extremely toxic and dangerous [13, 14].

2.2 Natural sources of toxic gases:

Volcanic Eruptions: The production of hazardous gases such as sulfur dioxide (SO□), hydrogen sulfide (H□S), and carbon dioxide (CO□) can pose health risks to nearby humans and animals [15].

Geothermal Activity: Natural geothermal sources can release toxic gases like carbon dioxide (CO□) and hydrogen sulfide (H□S). These gases can accumulate in low-lying areas, posing dangers to people in the vicinity [16].

Wildfires: When vegetation burns, harmful chemicals such as carbon monoxide (CO), nitrogen oxides (NO_x), and volatile organic compounds (VOCs) are released. These pollutants can damage respiratory systems and reduce air quality [17].

3. Toxic gasses and human health effects

3.1 Volatile organic compounds (VOCs) [18]:

Effects on the respiratory system:VOCs can irritate the respiratory system, causing symptoms such as wheezing, coughing, and shortness of breath. Prolonged exposure to high levels of volatile organic compounds (VOCs) can exacerbate respiratory conditions like asthma.

Neurotoxic effects: Certain volatile organic compounds (VOCs), such as benzene and toluene, have been shown to have neurological effects. These effects can manifest as headaches, dizziness, and, in severe cases, neurological disorders and cognitive impairment.

Irritation of the eyes and throat: Exposure to volatile organic compounds (VOCs) can cause irritation of the eyes, nose, and throat, leading to discomfort, swelling, and redness.

Damage to the liver and kidneys: Prolonged exposure to some volatile organic compounds (VOCs), such as solvents and chlorinated chemicals, has been linked to potential liver and kidney damage.

Risk of cancer: Certain volatile organic compounds (VOCs), such as benzene and formaldehyde, are classified as carcinogens and have been associated with an increased risk of cancer, particularly in occupational settings with high exposure levels.

3.2 Hydrogen sulfide (H_2S) [19]

Effects on the respiratory system: Inhaling hydrogen sulfide can irritate the respiratory system, leading to symptoms such as coughing, shortness of breath, and chest tightness. Excessive exposure can result in pulmonary edema and respiratory failure, which may be fatal.

Neurological effects: Hydrogen sulfide is known to affect the central nervous system, causing symptoms such as headaches, nausea, dizziness, and loss of consciousness. High-level or prolonged exposure can lead to long-term cognitive impairment and neurological damage.

Irritation of the eyes and skin: Prolonged exposure to hydrogen sulfide can irritate the eyes and skin, causing redness, inflammation, and, in severe cases, chemical burns.

Effects on the heart: Research suggests that exposure to hydrogen sulfide may adversely affect the heart, potentially leading to decreased heart rate variability and impaired cardiac function.

Olfactory weariness: At high concentrations, hydrogen sulfide can overwhelm the sense of smell, leading to olfactory fatigue, which can make it difficult for individuals to detect the gas at potentially lethal levels.

3.3 Polycyclic aromatic hydrocarbons (PAHs)

Chronic or long-term exposure to polycyclic aromatic hydrocarbons (PAHs) can have various negative health effects, including impaired immune function, cataract formation, liver and kidney damage (including jaundice), respiratory issues, asthma-like symptoms, and lung function abnormalities. Additionally, frequent skin contact with PAHs can cause inflammation and redness. Inhalation or ingestion of high amounts of certain PAHs, such as naphthalene, can lead to the breakdown of red blood cells. The potential adverse effects of human exposure to PAHs primarily depend on the route of exposure [20, 21].

3.4 Carbon Nanomaterials(CNMs)

Carbon nanomaterials (CNMs) are tiny particles that can directly penetrate cells, leading to an increased inflammatory response, particularly in immune cells [22]. Additionally, the introduction of CNMs causes toxicity and compromises cellular integrity. Some research suggests that carbon nanotubes (CNTs) may directly damage bacterial cell membranes, resulting in decreased metabolism and leakage of nucleic acids. Graphene (GRA) has different toxicity mechanisms in cells; the jagged edges of GRA sheets can damage the cell walls of algae upon contact [23].Carbon nanotubes (CNTs) are also hazardous to benthic organisms in rivers, as they are deposited in sediment, which is insoluble in water and can alter the behavior of other coexisting contaminants [24]. Determining the precise environmental impact of CNTs is challenging because they do not exist in isolation in the real world; instead, they adsorb other contaminants to some extent. Direct contact with CNTs may pose health risks to humans as well [25]. The physical and chemical properties of CNTs, particularly their small size, are likely related to their damaging effects. Due to their small size, carbon nanotubes can enter the body through the respiratory system and reach the lungs, where they may cause pulmonary granulomas, fibrosis, or inflammation [26].As CNTs enter the lungs, they are removed by alveolar macrophages. However, after macrophages ingest the CNTs, granulomatous inflammation can develop as the majority of the CNTs become deposited in the alveolar cavity and septum. This deposit can easily and deeply penetrate lung tissue [27].

3.5 Carbon monoxide (CO)

Carbon monoxide (CO) toxicity is caused by hypoxia, a decrease in the blood's ability to carry oxygen [28, 29]. Hypoxia occurs after CO exposure due to the competition between oxygen (O_2) and carbon monoxide (CO) for hemoglobin-binding sites, leading to the formation of carboxyhemoglobin (COHb) [29]. Hemoglobin's affinity for CO is widely recognized to be 200–250 times greater than its affinity for O_2, which is the primary reason for CO's toxicity [28-30]. Because carbon monoxide behaves chemically like oxygen in the blood, it is extremely toxic to humans and can lead to suffocation [31]. Acute health effects of CO-induced hypoxia, even at concentrations below lethal levels, include neurological symptoms such as headache, dizziness, confusion, disorientation, loss of coordination, memory loss, fainting, cerebral edema, and coma [28, 32].CO exposure can also cause heart-related issues, including decreased myocardial function, hypotension, vasodilation, cyanosis, cardiac arrhythmias, shock, circulatory failure, and cardiac arrest, as the heart is particularly sensitive to its effects [16, 17, 33]. Newborns, pregnant women, and fetuses are especially vulnerable to CO exposure. CO binds more strongly to fetal hemoglobin than to maternal hemoglobin and crosses the placenta easily. Additionally, CO is cleared from fetal blood much more slowly than from maternal blood, resulting in a 10-15% higher production of COHb in the fetus.

3.6 Hydrogen cyanide (HCN)

After exposure and systemic absorption, hydrogen cyanide (HCN) dissociates in the blood to produce the cyanide ion. This cyanide ion is highly toxic because it disrupts cellular respiration—the process by which cells use oxygen. The cyanide ion is widely distributed

throughout the body and exerts its toxicity by inhibiting cytochrome oxidase, a key enzyme involved in cellular oxygen utilization. When the cyanide ion binds to cytochrome oxidase, it forms a cyanide-oxidase complex that inhibits the enzyme's function. The inhibition of cytochrome oxidase leads to cytotoxic hypoxia and a rapid loss of cellular function. The effects of cyanide on cellular respiration are particularly harmful to the tissues of the heart and brain. The cytotoxic hypoxic effect of cyanide ions on the central nervous system is the most common cause of respiratory system depression, which is often the primary cause of death in HCN overdose cases. However, the cardiovascular system can also be affected [29].Acute exposure to sub-lethal levels of hydrogen cyanide can cause a range of symptoms, including bradycardia, cardiac arrhythmia, headache, nausea, dizziness, disorientation, muscle weakness, loss of coordination, hyperventilation, and rapid loss of consciousness or coma [29, 34].

Table 1: Acute emergency guideline levels (AEGL) and Worker exposure limits (WEL) for products resulting from the combustion of asphyxiate gas [35, 36].

	Well (ppm)		**AEGL-2/AEGL-3 (ppm)**				
	LTEL	STEL	10 min	30 min	60 min	4hour	8hour
CO	30	200	420/1700	150/600	83/330	33/150	27/130
HCN	-	10	17/27	10/21	7.1/15	3.5/8.6	2.5/6.6

3.7 Hydrogen bromide (HBr)

Hydrogen bromide (HBr) is known for its corrosive properties and its ability to irritate and damage the skin, eyes, and respiratory system. Contact with HBr can cause chemical burns and tissue damage, while inhalation of the gas may lead to acute respiratory distress and lung injury [37]. Like other gases, HBr exposure at a concentration of 5 parts per million can cause nasal irritation, and one participant reported throat irritation [36]. Fatalities have occurred with exposure levels between 1300 and 2000 ppm [37]. Some theories suggest that sensory irritation from exposure to 200 parts per million of HBr could be sufficient to impede a person's ability to escape from a hazardous environment [38].

3.8 Hydrogen fluoride (HF)

Hydrogen fluoride, a strong sensory irritant and corrosive gas, has been shown to irritate and damage the mucous membranes of the nose, mouth, and throat when inhaled for one hour at concentrations as low as 0.5ppm. Higher concentrations can cause lung damage, with pulmonary edema potentially developing 24 to 48 hours after exposure [39, 40]. Volunteer studies have reported mild sensory and lower airway irritancy symptoms, such as coughing, expectoration, wheezing, chest tightness, and pain, at concentrations ranging from 0.2 to 2.9 ppm. More severe effects were observed at concentrations between 3.0 and 6.3 ppm [41].

3.9 Sulphur dioxide (SO_2)

When healthy individuals inhale 1 ppm of sulfur dioxide (SO□), their forced expiratory flow and volume decrease, while airway resistance increases over the course of one to six hours [39]. Because SO□ is readily absorbed by the mucosa of the upper respiratory tract, the most common side effect of inhalation is irritation of the mouth and nose [37, 38]. Exposure to approximately 10 parts per million of SO□ can cause moderate to severe eye irritation, leading to lachrymation [39]. SO□ reacts with the mucous membranes of the upper respiratory tract and eyes to form sulfurous acid, which further irritates these mucosal structures. Additionally, sulfur dioxide stimulates the pain receptors in the epithelial fibers [42]. In cases of excessive SO□ exposure, severe irritation leading to upper respiratory tract obstruction can cause asphyxiation and potentially be fatal [43].

3.10 Oxides of nitrogen (NOx)

Due to their lower solubility compared to most irritating gases, nitrogen oxides have a higher likelihood of reaching the bronchioles and alveoli after inhalation, which can lead to respiratory harm [44]. Nitric oxide irritates the upper respiratory tract and eyes. Physical exertion may exacerbate the delayed onset of pulmonary edema, which can occur several hours after exposure [44]. In healthy human volunteers, exposure to nitric oxide at concentrations higher than approximately 20 ppm (24.6 mg/m³) results in a noticeable increase in overall airway resistance (~10%) [44].

Nitrogen dioxide (NO□) has an irritating effect because it reacts with water in the mucous membranes of the respiratory tract to form nitric acid (HNO□) and nitrous acid (HNO□) [28]. Low concentrations of NO□ can cause symptoms such as fatigue, nausea, dizziness, coughing, headaches, and breathing difficulties [28]. Exposure to NO□ at concentrations as low as 2.5 ppm has been shown to significantly increase airway resistance in healthy individuals [44]. High concentrations of NO□ can cause significant airway and laryngeal closure, potentially leading to sudden death [45]. Severe pulmonary edema may develop within a few hours of removal from NO□ exposure [37, 42]. A decrease in the partial pressure of arterial oxygen may also be detected due to reduced diffusion capacity during pulmonary edema [45]. The presence of carbon dioxide (CO□) can enhance the toxicity of NO□ compared to NO□ alone. Research on healthy subjects has indicated a 2 ppm impact threshold, while individuals with asthma have a lower threshold of approximately 0.2 ppm [3].

Table 2: Acute emergency guideline levels (AEGL) and Worker exposure limits (WEL) for inorganic acid combustion products [43,44].

	WEL (ppm)		**AEGL-2/AEGL-3 (ppm)**				
	LTEL	STEL	10 min	30 min	60 min	4hour	8hour
HBr	-	3	100/740	43/250	22/120	11/31	11/31
HF	1.8	3	95/170	34/62	24/44	12/22	12/22

SO_2	-	-	0.75/30	0.75/30	0.75/30	0.75/19	0.75/9.6
NO	NR						
NO_2	-	-	20/34	15/25	12/20	8.2/14	6.7/11

4. Prevention and Control

4.1 Interventional Strategies

The methods for administering first aid when signs of poisoning are observed include:

- Remove the individual from the contaminated area.
- Provide artificial respiration if the individual is not breathing.
- Contact a physician or take the victim to the hospital for further treatment.
- The poisoned person should remain in the hospital for monitoring and supportive care.
- Serious cases may result in hypothermia.
- Continue to closely monitor the patient after recovery from the poisoning.

4.2 Poison control centers

Medical professionals experienced in treating poisoning patients should work in these centers. These doctors should have access to a comprehensive database and a wealth of reference materials. Additionally, they must have prompt access to laboratory toxicological studies when further investigation is required [6]. Poison control centers are medical facilities that provide immediate, professional, and free treatment guidance and support over the phone for individuals exposed to toxic or dangerous substances. In addition to offering management advice for issues related to household items, medications, pesticides, plants, bites and stings, food poisoning, and odors, poison control centers also respond to inquiries about potential poisons [46].

4.3 Prevention of pharmaceutical poisonings with dangerous gases

The problem of poisonings can be greatly reduced through preventative measures and actions. Effective prevention relies heavily on knowledge and information. Informing professional groups and the most vulnerable populations is crucial [5]. Poisoning can be caused by a wide range of chemicals and highly toxic gases. Among these substances are household chemicals, which are commonly used for various purposes, including personal hygiene. Because these products can cause severe illness or even death, it is essential to take precautions to prevent them from entering the human body. To address these issues effectively, individuals must adhere to the following guidelines to protect themselves from chemical poisoning and exposure to highly toxic gases [47].

4.4 Some ways to avoid chemical gas poisonings [6]:

- Wear gloves and appropriate clothing to prevent direct contact with chemicals.
- Carefully wash contaminated clothing and undergarments.
- Practice basic hygiene, such as washing your hands and taking a shower after work.

- Medical professionals can mitigate the adverse effects of hazardous gases through various methods.

4.5 Limitation of toxic gas

- **Controlling the Release and Exposure:** Limiting the use of toxic gases involves controlling their release, exposure, and potential negative effects on human health and the environment. Effective management of toxic gases is essential for environmental protection, worker safety, and public health.
- **Engineering Controls:** Toxic gas releases in industrial environments can be minimized by implementing engineering controls such as ventilation systems, containment measures, and process adjustments. The National Institute for Occupational Safety and Health (NIOSH) provides comprehensive guidance, including safety guidelines for various hazardous compounds [48].
- **Monitoring and Exposure Limits:** Establishing and strictly enforcing exposure limits for toxic gases in occupational settings is crucial for worker protection. Regular monitoring of gas concentrations and air quality ensures that exposure levels remain within acceptable limits. The Occupational Safety and Health Administration (OSHA) sets permissible exposure limits (PELs) for various hazardous compounds, including toxic gases [49].
- **Personal Protective Equipment (PPE):** Providing employees with appropriate PPE, such as respirators and protective clothing, helps reduce the risk of contact with harmful gases. The selection and use of PPE should be based on thorough risk assessments and compliance with relevant regulations and guidelines [50].
- **Emergency Response Planning:** Developing and practicing emergency response plans are essential for minimizing potential damage to the environment and public health in the event of an accidental release of hazardous gases. The Environmental Protection Agency (EPA) offers tools and guidelines for emergency response planning and preparedness [51].
- **Compliance with Regulations:** Preventing emissions and ensuring safe management of toxic gases depend heavily on adherence to relevant laws and guidelines related to their handling, storage, and transportation. Government agencies such as the EPA and OSHA provide regulatory frameworks and compliance support for the management of hazardous substances, including toxic gases [52].

Conclusion

In this critical assessment, we have discussed the general impact of toxic gases on human health, highlighting the dangers these hazardous substances and their ions pose to both human health and the environment. We addressed concerns related to exposure from significant pollutants associated with the rapid and intense industrialization of the gas industry, as well as toxins directly related to this sector. The effectiveness of exposure controls is questionable, as evidenced by the significant increase in hospitalizations for acute respiratory and circulatory disorders corresponding with rising levels of harmful pollutants in the air.

The growing body of published research supports decisions made by various jurisdictions regarding emissions from the industry and their adverse effects on health. Particulate matter plays a continuous and significant role in atmospheric chemistry and is critically important for human health. Regular exposure to particulate matter has been linked to increased risks of respiratory disorders, chronic asthma, early mortality, and hospital admissions.

Investigating and developing effective countermeasures against these substances is justified by their potential for illicit use or unintentional exposure. Variations in responses could offer genetic explanations for differences in symptom diagnosis and treatment among groups exposed to toxic environments.

A thorough analysis of the health effects of the unconventional gas sector is essential. Governments should prioritize efforts to prevent contamination of air, water, and food by enforcing stricter regulations, ensuring proper hazardous waste disposal, and promoting chemical-free processes and procedures. Public education on harmful chemical hygiene by NGOs and the media is also crucial. The general population should be informed about the risks of consuming toxic-laden foods, medications, cosmetics, and other daily-use items. Additionally, scientists must actively collaborate to raise awareness. If implemented, these initiatives will significantly reduce the risk of exposure to harmful gases.

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