

relevance of low-dose phenomena is extremely difficult to establish not only because the cumulative average exposure for people is approximately 8-10 cGy per lifetime.

1.4. AIR QUALITY

Air is a mixture of gases. It contains different percentages of nitrogen (78%), oxygen (21%), argon (0.9%), carbon dioxide (0.033%).

Oxygen is a colorless, odorless, and tasteless gas that is only slightly soluble in water. It is the only gas used by the human body and it is essential to life. The other gases breathed from the atmosphere serve only as a vehicles and diluents for oxygen.

Health hazards: At 15-19% oxygen impaired coordination and decrease ability to work and at 10-14% creates respiratory problems. Few recorder after more than 5 minutes in less oxygen.

Exposure limits:

Deficiency: 18%

Enrichment: 23%

Nitrogen is a colorless, odorless and tasteless gas. It is chemically inert and is incapable of supporting life. At normal pressure nitrogen does not influence the human organism and an amount of about 1 liter is dissolved in the blood and the tissues.

Health hazards: Acts as asphyxiant through displacement of normal air.

Carbon dioxide is a gas produced by various natural processes such as animal metabolism, combustion, and fermentation. It is colorless, odorless, and tasteless. A person should not breathe air containing more than 0.1% CO₂ by volume.

Health hazards: Causes headache, dizziness, increases the heart rate and blood pressure and coma.

Exposure limits:

5000 ppm -15000 ppm

Argon, neon, and hydrogen have been used experimentally as diluents for oxygen in breathing gas mixtures.

Water vapors. The normal weight of water vapors in the air is considered 1-1.5%. They have a harmful effect on the human organism if a large amount is present.

1.5. OUTDOOR AIR POLLUTION

The dramatic air pollution episodes that occurred in the early part of the twentieth century in the Meuse Valley of Belgium, Donora, Pennsylvania and London, England are not likely to occur in the world today.

These episodes were due to the large scale burning of coal in the presence of "ideal" meteorologic conditions-atmospheric inversion leading to a stagnant air mass.

A clearly evident excess mortality was observed during and after these episodes.

However, certain environmental air pollutants, such as ozone and respirable particles, do reach levels that may cause acute and chronic respiratory effects.

Furthermore, in some Eastern European countries where sulfur-containing fuels are burned without adequate air-quality regulations, air pollution levels may be attained similar to those that were associated with excess mortality.

1.5.1. REGULATION OF OUTDOOR AIR POLLUTANTS

The Environmental Protection Agency (EPA) lists those pollutants for which there is sufficient scientific evidence documenting the risk to public health from unregulated exposure.

The EPA has identified children, people with chronic respiratory disease such as asthma, and people with ischemic heart disease as constituting sensitive groups (ie, that demonstrate a response to a pollutant at a lower level or to a greater degree than the average response of the general population).

1.5.2. TYPES AND SOURCES OF EXPOSURE

Outdoor air contains an array of naturally occurring pollutants including soil, dust, pollens, and fungi.

In addition, human activity generates complex mixtures of pollutants.

The sources of outdoor air pollution are usually categorised as stationary or mobile.

Stationary sources are primarily power or manufacturing plants and are responsible for most sulfur dioxide (SO_2) emissions as well as considerable amounts of nitrogen oxides (NO_x) and particulate matter.

Atmospheric acidity is largely due to the oxidation of SO_2 to sulfuric acid (H_2SO_4) and other acid sulfate species.

The combustion of fossil fuel is the most important cause of stationary sources emissions, although release of *volatile organic compounds* (VOC_s) by various industrial facilities can contribute to the generation of ozone (O_3) in the atmosphere.

In contrast to the pollution from stationary sources "smog" is primarily derived from automotive, or **mobile sources**, emissions.

A large fraction of ambient O_3 is the product of complex photochemical reactions involving NO_x and VOC_s emitted from automotive tailpipes.

Nitric acid (HNO_3) is a more important contributor to atmospheric acidity than H_2SO_4 , and is formed in the atmosphere from the reaction of NO_x with the hydroxyl radical (OH^\cdot).

Motor vehicle emissions are also responsible for much carbon monoxide and particulate pollution.

1.5.3. PERSONAL EXPOSURE

Most people spend most of their time indoors where the concentrations of pollutants are generally lower than in outdoor air.

The concentration of NO_2 , however, may be higher in indoor air, largely as a result of natural gas-burning stoves.

Individuals who spend a lot of time outdoors, especially if they are increasing their effective dose by means of increased minute ventilation from exercise, may sustain relatively high exposures to pollutants such as O_3 and particulate matter.

Therefore, total personal exposure should be considered, this is estimated by the summation of the products of the concentrations of the pollutant in various microenvironments with the duration spent in each.

1.5.4. PRINCIPLES OF INHALATIONAL INJURY

Pollutants in inhaled air are **gases as aerosols** -droplets of liquid or **particles suspended** in gas- and this site of deposition after inhalation is largely determined by their water solubility.

Gases that are extremely water-soluble, such as SO_2 and HNO_3 vapor, will be deposited and removed primarily by the upper respiratory tract.

Therefore, these water-soluble gases will mainly induce toxic effects on the proximal airways and will only damage the distal lung when inhaled in high concentrations.

In contrast, gases that are of relatively lowwater solubility, such as NO_x and O_3 , may predominantly injure the distal lung.

The less soluble the gas, the greater the potential for damage at the level of the terminal respiratory unit.

The deposition of aerosols is determined by a number of factors, including the site and chemical characteristics of the aerosol, the anatomy of the respiratory tract, and the breathing pattern of the exposed person.

The size of the droplet or **particle** is usually the primary factor affecting deposition, although the chemical nature of the inhaled pollutant can be important, especially if it is a water-soluble acid aerosol that can be neutralised by oral ammonia such as a H_2SO_4 mist.

The majority of inhaled particles with a mass median aerodynamic diameter (MMAD) $\geq 10 \mu\text{m}$ are deposited in the nasopharynx and will not penetrate below the larynx.

Particles in the range of $2.5\text{--}6 \mu\text{m}$ will deposit primarily in the conducting airways below the larynx and particles in the range of $0.5\text{--}2.5 \mu\text{m}$ will deposit primarily in the distal airways and alveoli.

Particles with a $\text{MMAD} < 0.5 \mu\text{m}$ are exhaled without significant deposition.

The site of particle deposition is also influenced by hygroscopic growth in the humidified environment of the airways, the shape and dimensions of the respiratory tree, ventilatory pattern (respiratory rate and tidal volume), oral versus nasal breathing, and the amount and nature of respiratory tract secretions.

Clearance of inhaled pollutants occurs by several mechanisms.

In general, highly water-soluble particles and gases, are absorbed through the epithelial layer into the blood stream near where they have been deposited.

The clearance of insoluble particles is dependent on where they impact. Those deposited in the anterior nasal cavity are expelled by sneezing or rhinorrhea, while the remainder of particles deposited in the nose are cleared posteriorly to the pharynx. Particles deposited in the trachea, bronchi, or bronchioles, where there is ciliated epithelium and a layer of mucus, are transported up the mucociliary escalator to be expelled by coughing or swallowing.

Particles deposited distal to the terminal bronchioles are cleared by alveolar macrophages and/or dissolution.

Alveolar macrophages will ingest particles and migrate to the mucociliary escalator or into lymphatics.

A small fraction of particles deposited in the alveoli will migrate through the alveolar epithelial layer directly into the lymphatic circulation.

1.5.5. TOXIC EFFECTS OF GASES AND VAPOURS

Grantham (1992) has classified gases and vapours into three groups, for health and safety purposes:

- irritants
- asphyxiants
- miscellaneous

IRRITANTS

Irritant gases and vapours cause inflammation of tissues to which they are exposed. Symptoms of exposure can range from mild irritation of the mucous membranes (eyes, nose and throat) to severe lung damage. Examples of irritant gases include chlorine, phosgene, formaldehyde and nitrogen dioxide.

ASPHYXIAN GASES

Asphyxiant gases fall into two groups:

- **Simple asphyxiants**

Simple asphyxiants act by displacing oxygen from the inspired air. This is a particular hazard for workers entering confined spaces, which may be oxygen deficient due to presence of high concentrations of simple asphyxiants as: methane, carbon dioxide or nitrogen.

- **Chemical asphyxiants**

Chemical asphyxiants are gases that interfere with the transport of oxygen by the blood or with the uptake of oxygen by tissues. Low concentrations may be toxic and examples include carbon monoxide and hydrogen cyanide.

MISCELLANEOUS GASES

Many gases and nearly all solvent vapours fall into the category of miscellaneous gases. Effects caused by these gases and vapours include acute effects (on the Central Nervous System-CNS) and chronic, toxic effects in many different organs of the body.

The following tables show the miscellaneous effects of gases:

MISCELLANEOUS EFFECTS OF GASES	
GAS	EFFECTS
Helium	Vocal changes
Nitrous oxide	Analgesia
Oxygen-excess	Pulmonary inflammation and lung oedema
Oxygen-deficiency	CNS effects and brain disturbance

MISCELLANEOUS EFFECTS OF VAPOURS	
Vapour	Effects
n-Hexane	Peripheral neural neuropathy
Nitroglycerine	Vasodilatation decreasing blood pressure
Toluene	Loss of memory
Ethylene glycol monoethyl ether	Foetotoxic effects
Trichloroethylene	Psychoactive effects
Fluorocarbons	Cardiac arrhythmia
Benzene	Leukaemia
Methyl bromide	Cardiac effects
Carbon disulphide	Cardiac disease
Coal tar pitch volatiles	Skin sensitisation and lung cancer
Vinyl chloride	Angiosarcoma of the liver

1.5.6. SPECIFIC OUTDOOR AIR POLLUTANTS

The health effects of the outdoor air pollutants have been compiled by the interpretation of toxicologic studies (ie, animal studies, in vitro studies, and controlled human exposure studies), and epidemiologic studies (ie, ecologic, cross-sectional, and case control designs).

OZONE

It is a pale blue, relatively unstable molecule made up of three oxygen atoms. It is formed from molecular oxygen (O_2) by ultraviolet and extreme ultraviolet photolysis followed by recombination of atomic oxygen ($O\cdot$) with O_2 .

It may also be formed by passing an electrical discharge through gaseous oxygen. In fact, the term ozone is derived from the Greek word ozein which means "to smell".

Excess oxygen atoms, also known as free radicals, oxidize materials that they contact and are associated with the aging process.

Formation of the Ozone Layer

High in the atmosphere, some oxygen (O_2) molecules absorbed energy from the Sun's ultraviolet (UV) rays and split to form single oxygen atoms. These

atoms combined with remaining oxygen (O_2) to form ozone (O_3) molecules, which are very effective at absorbing UV rays. The thin layer of ozone that surrounds Earth acts as a shield, protecting the planet from irradiation by UV light.

The amount of ozone required to shield Earth from biologically lethal UV radiation, wavelengths from 200 to 300 nanometers (nm), is believed to have been in existence 600 million years ago. At this time, the oxygen level was approximately 10% of its present atmospheric concentration. Prior to this period, life was restricted to the ocean. The presence of ozone enabled organisms to develop and live on the land. Ozone played a significant role in the evolution of life on Earth.

Ozone production and destruction

Stratospheric ozone is created and destroyed by ultraviolet radiation. The air in the stratosphere is bombarded continuously with ultraviolet radiation from the Sun. When high energy ultraviolet rays strike molecules of ordinary oxygen (O_2), they split the molecule into two single oxygen atoms. The free oxygen atoms can combine with oxygen molecules (O_2) to form ozone (O_3) molecules.

Ozone is a highly unstable molecule that readily donates its extra oxygen molecule to free radical species such as nitrogen, hydrogen, bromine, and chlorine. These compounds naturally occur in the stratosphere, released from sources such as oil, water vapor, and the oceans.

Anthropogenic destruction

Manufactured compounds are also capable of altering atmospheric ozone levels. Chlorine, released from CFCs and bromine (Br), released from halons, are two of the most important chemicals associated with ozone depletion. Halons are primarily used in fire extinguishers. CFCs are used extensively in aerosols, air conditioners, refrigerators, and cleaning solvents. Two major types of CFCs are trichlorofluorocarbon (CFC13) or CFC-11, and dichlorodifluoromethane (CF₂Cl₂), or CFC-12.

Trichlorofluorocarbon is used in aerosols, while dichlorodifluoromethane is typically used as a coolant.

Near Earth's surface, chlorofluorocarbons are relatively harmless and do not react with any material, including human skin. While CFCs remain in the troposphere they are virtually indestructible. They are not water soluble and cannot even be washed out of the atmosphere by rain. We now understand that the very quality that made them seem to be safe, their stability, is what makes them so dangerous. CFCs remain in the troposphere for more than 40 years before their slow migration to the stratosphere is complete. Even if we were to end their production and use at this very moment, they would continue to contribute to ozone destruction far into the future.

In the stratosphere, high energy ultraviolet radiation causes the CFC molecules to break down through photodissociation. Atomic chlorine, a true catalyst for ozone destruction, is released in the process. Chlorine initiates and takes part in a series of ozone destroying chemical reactions and emerges from the process unchanged. The free chlorine atom initially reacts with an unstable oxygen containing compound, such as ozone, to form chlorine monoxide, that then reacts with molecular oxygen to produce atomic chlorine. The regenerated chlorine atom is then free to initiate a new cycle. This destructive chain of reactions

will continue over and over again, limited only by the amount of chlorine available to fuel the process.

The ozone balance

Scientists are finding that ozone levels change periodically as part of regular natural cycles such as seasons, period of solar activity, and changes in wind direction. Concentrations are also affected by isolated events that inject materials into the stratosphere, such as volcanic eruptions.

Polar regions reflect the greatest changes in ozone concentrations, especially the South Pole. The topography of Antarctica is such that a stagnant whirlpool of extremely cold air forms over the region during the long polar night. The air stays within this polar vortex all winter, becoming cold enough to allow formation of polar stratospheric clouds.

We are losing ozone in both hemispheres. Ozone levels in the atmosphere have been monitored from the ground since the 1950s and by satellite since 1970s. Regional total ozone levels measured from satellites over Antarctica have decreased 30-50% since their monitoring began.

Since ozone is created and destroyed by solar UV radiation, there is some correlation of ozone concentration with 11-year sunspot cycles. Sunspot emit high levels of electromagnetic radiation. The increased UV radiation contributes to ozone production. Sunspot variations only account for 2 to 4% of total variation in ozone concentrations. Natural cycles in ozone variation are also associated with the quasi-biennial oscillation in which tropical winds switch from easterly to westerly every 26 months. This cyclic change in wind direction accounts for approximately 3% of the natural variation in ozone concentration.

International Agreements

In 1973, two scientists from the University of California at Irvine, Mario Molina and F. Sherwood Rowland, first discovered that manmade substances called chlorofluorocarbons (CFCs) could play a major role in the destruction of the stratospheric ozone.

Nations from all over the world have come together and agreed to establish international industrial regulations in hope of protecting the ozone layer.

Through the 1985 Vienna Convention for the Protection of the Ozone Layer, the 1987 Montreal Protocol on substances that deplete the ozone layer, and the 1990 London Amendments to the protocol, members from nations around the world have committed to phasing out the production and consumption of CFCs, and a number of related chemicals, by the year 2000.

A United Nations Environmental Program to protect the ozone layer was signed in Vienna in 1985, and a protocol outlining proposed protective actions followed. The Vienna convention of 1985 embodied an international environmental consensus that ozone depletion was a serious environmental problem. However, there was no consensus on the specific steps that each nation should take. The Montreal Protocol, signed in September 1987, stated that there would be a 50% cut back in CFC production by 2000. The 1990 London Amendments to the protocol state that the production of CFCs, CC14, and halons will be completely halted by the year 2000. The phaseout schedule for others compounds was accelerated by 4 years by the 1992 Copenhagen agreement.

Destructive ozone

Depending on where ozone resides, it can protect or harm life on Earth. When it is close to the planet's surface, in the air we breathe, ozone is a harmful pollutant that causes damage to lung tissue and plants, and is considered to be "bad ozone". It is powerful photochemical oxidant that damages rubber, plastic, and all plant and animal life. It also reacts with hydrocarbons from automobile exhaust and evaporated gasoline to form secondary organic pollutants such as aldehydes and ketones. The peroxyacyl nitrates are especially damaging photochemical oxidants that are very irritating to the eyes and throat.

Ozone pollution originating in urban areas can extend into surrounding rural and forested areas that are hundreds of kilometers downwind. Episodes of elevated ozone concentrations are associated with warm, slow moving high pressure system and contain between 30 and 50 parts per billion by volume. Concentrations 3 to 8 times greater than natural background levels have been observed.

"Destructive ozone" (O_3) is a colorless, pungent, relatively water insoluble gas that occurs with other photochemical oxidants and fine particles to form "smog".

Peak concentrations of O_3 typically occur in midafternoon, after both the morning rush hour and several hours of bright sunlight.

Ozone is a potent oxidant and is capable of reacting with a variety of extracellular and intracellular molecules.

When the molecules are unsaturated lipids, free radicals and toxic intermediate products, such as hydrogen peroxide and aldehydes, are generated and can lead to cellular damage or cell death.

Although direct cytotoxicity is clearly a necessary mechanism of O_3 induced-tissue injury, secondary damage from the inflammatory response may also play a role.

O_3 inhalation by healthy subjects causes mean decrements in forced expiratory volume in 1 second (FEV_1) and forced vital capacity (FVC) that correlate with concentration exposure, duration and minute ventilation.

Another adverse effect of short-term exposure to O_3 is enhanced airway responsiveness to nonspecific stimuli such as methacholine and histamine.

This effect may persist longer than the acute decrements in lung function.

The effect of chronic O_3 exposure in humans have not been adequately defined. It has been hypothesized that chronic exposure would lead to emphysematous or fibrotic parenchymal changes.

Ozone toxicity may be exchanged by exposure to other pollutants such as other oxidants, particulates and atmospheric acidity commonly seen in urban smog.

Because O_3 inhalation can induce both airway inflammation and enhanced air-way responsiveness, it is reasonable to expect persons with asthma to have greater susceptibility to this pollutant.

Ozone is rarely the sole pollutant of concern in urban smog, and it is likely that environmental cofactors enhance its toxicity.

NITROGEN DIOXIDE

Most ambient nitrogen dioxide (NO_2) is generated by the burning of fossil-derived fuels, during which oxygen and nitrogen react to form nitrogen oxide (NO), which further reacts to form NO_2 and other NO_x .

The principal source of NO_2 in outdoor air is motor vehicle emissions, but power plants and fossil fuel-burning industrial facilities also contribute.

In contrast to other criteria pollutants, NO_2 is a common contaminant of indoor air, and indoor levels often exceed those found outdoors.

Indoor sources of NO_2 include gas cooking stoves, gas furnaces, and kerosene space heaters.

Nitrogen dioxide, like O_3 , is an oxidant, but it is less chemically reactant and is therefore usually considered less potent.

Although both pollutants are relatively insoluble in water, the solubility of NO_2 is somewhat higher.

When NO_2 is absorbed into the moist surfaces of the respiratory tract, it can be hydrolysed to evolve surfaces of the respiratory tract, it can be hydrolysed to evolve acidic species such as HONO and HNO_3 . The potential for NO_2 to cause the local generation of hydrogen ions in the airways may be an important feature of its toxicity.

In contrast to what is seen with O_3 , short-term exposure to NO_2 at concentrations in the ambient range does not induce airways inflammation.

Chronic exposure of experimental animals to high concentrations of NO_2 has caused emphysema-like changes and decreased resistance to bacterial infection.

NO_2 remains an important air pollutant because of its role in the generation of tropospheric O_3 .

PARTICLES, SULFUR DIOXIDE AND ACID AEROSOLS

Particles, sulfur dioxide and acid aerosols are discussed as a group, as they usually occur together as components of a complex pollutant mixture.

Their production is primarily a result of fossil fuel combustion. Particles and sulfur dioxide (SO_2) are the primary products of combustion, and acid aerosols are formed by subsequent atmospheric chemical reactions.

This mixture of solid and liquid particles suspended in the air is termed "particulate air pollution"; the constituent particles differ in size and composition.

Particles with an aerodynamic diameter of $\leq 10 \mu\text{m}$ (PM_{10}) are the focus of regulatory interest because particles of these diameter may penetrate into and deposited in the airways of the lower respiratory tract and the gas exchanging portions of the lung. Acid aerosols are usually a complex and variable mixture and included dissolved gaseous pollutants.

Sulfur dioxide is a major air pollutant in many urban areas. The gas is emitted by coal and oil-fired power plants and by industrial processes involving fossil fuel combustion. It leads to the secondary formation of acid aerosols.

Particle exposure has been associated with increased emergency room visits for respiratory illness, such as asthma and pneumonia, and higher rates of hospital admissions for respiratory and cardiovascular illnesses.

Studies in adults have also shown associations between exposure to particles and reports of respiratory symptoms severe enough to restrict activity.

Sulfur dioxide is highly soluble in water and is mostly absorbed in the upper airways. Although the nose effectively removes much of the inhaled gas, significant

amounts may penetrate to the large airways. Here, the irritant molecules may cause reflex bronhoconstriction.

In asthmatics, low-level exposure has been shown to cause bronhoconstriction.

Particle toxicity is often complicated by the presence of their air pollutants that may cause interactive effects.

Particle size is thought to be a critical determinant of toxicity.

Acid aerosols consist predominantly of sulfate (SO_4^{2-}) and bisulfate (HSO_4^-) ions that coexist with ammonium (NH_4^+) ions.

The toxicity of the acid aerosols is related to their acidity, which can cause airway irritation.

LEAD

Lead continues to be recognized as a significant toxicant and is known to have adverse health effects on humans of all ages.

The phase-out of the additive tetra ethyl lead has been associated with declines in ambient level concentrations and blood lead levels in the population.

CARBON MONOXIDE

Carbon monoxide (CO) is a colorless, odorless, nonirritating gas that is generated by the incomplete combustion of carbon-containing fuels, such as oils, gasoline, coal and wood.

Because of these described properties, exposure to high levels of CO is the leading cause of poisoning death, although low-dose exposure may be associated with adverse health effects.

The most common source of exposure in nonsmoking individuals is from vehicle emissions.

Emissions from nonvehicular sources such as lawn mowers, chain saws, space heaters, and charcoal briquettes also contribute to ambient CO exposure.

The toxicity of CO lies in its ability to strongly bind to hemoglobin and interfere with the transport of oxygen from the alveoli to tissues. The gas rapidly diffuses across the alveolar-capillary membrane after inhalation. Here, it binds with an affinity greater than 200 times that of oxygen to hemoglobin to form carboxyhemoglobin (COHb). This COHb complex interferes with oxygen delivery to the tissues by two major mechanisms.

Because CO has no direct effect on the lungs, its principal adverse health effects are through its ability to cause or exacerbate diseases associated with impaired oxygen delivery.

Effects on fetal development, cardiovascular disease, chronic respiratory diseases, and nervous system diseases have been described.

In individuals with ischemic heart disease, a shorter duration to onset and an increased duration of angina, as well as earlier ST-T changes, have been observed with low-level CO exposure.

1.5.7. THE AMBIENT AIR QUALITY STANDARDS (AAQS)

Environmental Protection Agency (EPA) has established Ambient Air Quality Standards for six pollutants: ozone, lead, carbon monoxide, sulfur dioxide, nitrogen dioxide, and respirable particulate matter. The standards were established to protect the public from exposure to harmful amounts of pollutants. When the

pollutant levels in an area have caused a violation of a particular standard, the area is classified as "nonattainment" for that pollutant.

Air pollution concentrations required:

Pollutant	Averaging period	Standard	Primary AAQS	Secondary AAQS
Ozone	1-h	Not to be at or above this level on more than three days over three years.	125 ppb	125 ppb
	8-h	The average of the annual fourth highest daily eight-hour maximum over a three-year period is not to be at or above this level	85 ppb	85 ppb
Carbon Monoxide	1-h	Not to be at or above this level more than once per calendar year	35.5 ppm	35.5 ppm
	8-h	Not to be at or above this level more than once per calendar year	9.5 ppm	9.5 ppm
Sulfur Dioxide	3-h	Not to be at or above this level more than once per calendar year	-	550 ppb
	24-h	Not to be at or above this level more than once per calendar year	145 ppb	-
	Annual	Not to be at or above this level	35 ppb	-
Nitrogen Dioxide	Annual	Not to be at or above this level	54 ppb	54 ppb
Respirable Particulate Matter (10 microns or less) (PM ₁₀)	24-h	The three-year average of the annual 99 th percentile for each monitor within an area is not to be at or above this level	155 µg/m ³	155 µg/m ³
	Annual	The three-year average of annual arithmetic mean concentrations at each monitor within an area is not to be at or above this level	51 µg/m ³	51 µg/m ³
Respirable Particulate matter (2.5 microns or less) (PM _{2.5})	24-h	The three-year average of the annual 99 th percentile for each monitor within an area is not to be at or above this level	66 µg/m ³	66 µg/m ³
	Annual	The three-year average of annual arithmetic mean concentrations at each monitor within an area is not to be at or above this level	15.1 µg/m ³	15.1 µg/m ³
Lead	Quarter	Not to be at or above this level	1.55 µg/m ³	1.55 µg/m ³

Primary AAQS: the level of air quality that the EPA judges necessary, with an adequate margin of safety, to protect the public health.

Secondary AAQS: the level of air quality that the EPA judges necessary to protect the public welfare from any known or anticipated adverse effects.