

# **Health Effects of Air Pollution**

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**Air pollution** has been distinguished on the basis of the chemical redox nature of its primary components.

was characterized by **SO<sub>2</sub> and smoke** from incomplete combustion of coal accumulated during an inversion as a chilled,

Always had a characteristically “**oxidant-type**” pollution consisting of **NO<sub>x</sub>** and many secondary photochemical oxidants, such as O<sub>3</sub>, aldehydes, and **hydrocarbon radicals**.

# Criteria Air Pollutants

- **Gases:**  $O_3$ , CO,  $NO_x$ ,  $SO_x$
- **Particles:** PM<sub>2.5</sub>, Pb

# Exposure to Air Pollutants

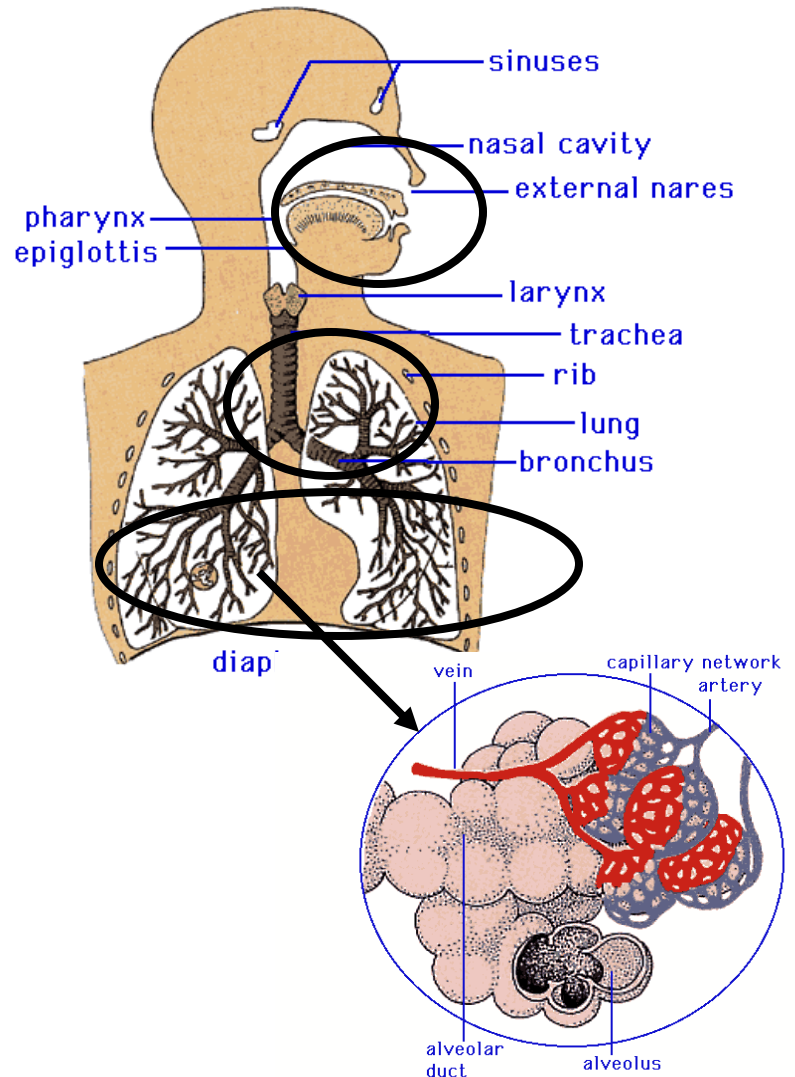
- **Chronic**
- **Acute**
  - **Air Pollution Episode – short term increase concentration**
- **Dependent on local conditions**
- **Epidemiological studies**
  - **Latency period**
    - **Lung cancer – up to 30 years**
- **Toxicological studies**
  - **Determine effects of toxic substances**
- **Pollutant interactions**



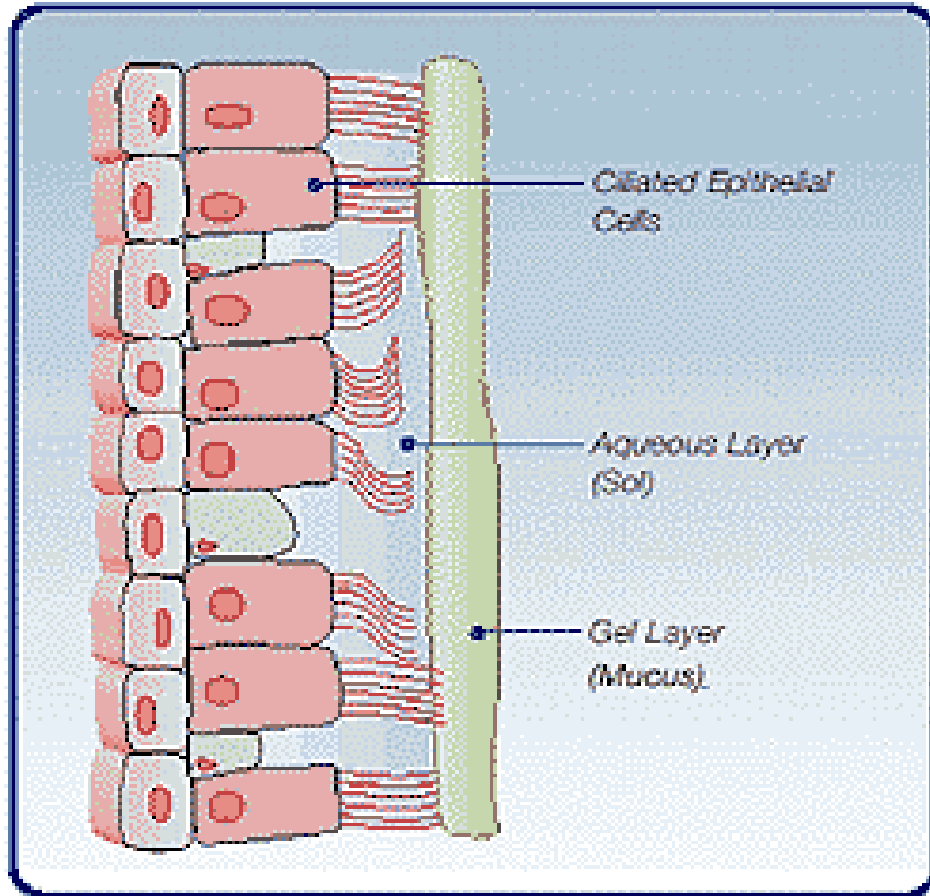
**Smog Episode in New York City, 1963**

# Respiratory System

- Pollutants transported in via inhalation-respiratory tracts
- Person at rest breathes 12 to 15 times a minute (10 liters/min)
- 3 parts of respiratory system
  - Naso-pharyngeal (NAR)
  - Tracheo-bronchial (TBR)
  - Pulmonary-Alveolar Lungs serve as portal of entry
  - Highly permeable and lots of blood flow
  - Pulmonary-Alveolar Surface Area > 75 m<sup>2</sup>



# Respiratory System



- Natural protection mechanisms (for particles)
- Naso-pharyngeal (HAR)
  - Nose hairs (filter particles)
  - Cough, Sneeze
  - Mouth breathing vs nasal breathing
- Tracheo-bronchial (TBR)
  - Mucociliary “escalator”
  - Bronchial constriction
- Pulmonary-Alveolar Macrophages (phagocytosis)
  - No cilia in Alveoli



***Question: Do the natural protection mechanisms protect against toxic gases such as CO, O<sub>3</sub>, SO<sub>2</sub>?***

# **Criteria Air Pollutants:**

## **Particulate Matter PM**

- **Small solid/liquid aerosol particles that remain suspended in air .**
- **Causes: materials handling, combustion processes, gas conversion reactions.**
- **Main sources: industrial processes, coal and oil burning, diesel motor vehicles**

# **Criteria Air Pollutants:**

## **Particulate Matter**

- **Following inhalation: two possible fates**
  - **Deposition or Exhalation**
- **Particle Fate depends upon:**
  - **Aerodynamic & physiological behavior (human being)**



# Criteria Air Pollutants: Particulate Matter

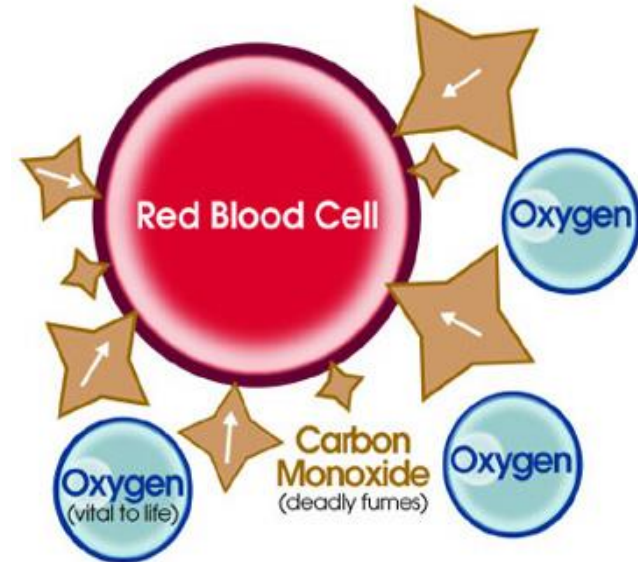
- **Wheezing & coughing to heart attacks and death**
- **TSP (Total Suspended Particles)**
  - **In presence of SO<sub>2</sub>, direct correlation between TSP and hospital visits for bronchitis, asthma, emphysema, pneumonia, and cardiac disease**
  - **Studies suggest ~60,000 deaths from PM**
  - **1% increase in mortality for 10 μg/m<sup>3</sup> increase in PM**
    - **Respiratory mortality up 3.4% for the same**
    - **Cardiovascular mortality up 1.4% for the same**

# **Criteria Air Pollutants: Carbon Monoxide CO**

- **Colorless, odorless, tasteless gas → “Silent Killer”**
  - **Cause: incomplete combustion of carbon based fuels**
  - **Source: transportation sector, residential heating units**
  - **NAAQS regulates CO in outdoor air**
- **50 ppm CO averaged over 8-hour period)**

# Criteria Air Pollutants: Carbon Monoxide CO

- Reacts with blood hemoglobin
  - Forms **carboxyhemoglobin** (HbCO) rather than **oxyhemoglobin** (HbO<sub>2</sub>)
  - Prevents oxygen transfer
- Toxic effects on humans
  - Low-level: cardiovascular & neurobehavior
  - Headaches/nausea/fatigue/ **death**
  - Oxygen deficient to vulnerable people (anemia, chronic heart or lung disease, high altitude residents, smokers)
    - Cigarette smoke: 400-450 ppm; smoker's blood 5-10% HbCO vs 2% for non-smoker



# Criteria Air Pollutants: Carbon Monoxide CO

- Concern in homes
- Install CO monitor
- No indoor CO regulations
  - >70 ppm → flu-like symptoms (w/out fever)
  - 150-200 ppm → disorientation, drowsiness, vomiting
  - >300 ppm → unconsciousness, brain damage, death
    - 500 Americans die/year from unintentional CO poisoning
- **Treatment:** fresh air, oxygen therapy, hyperbaric chamber

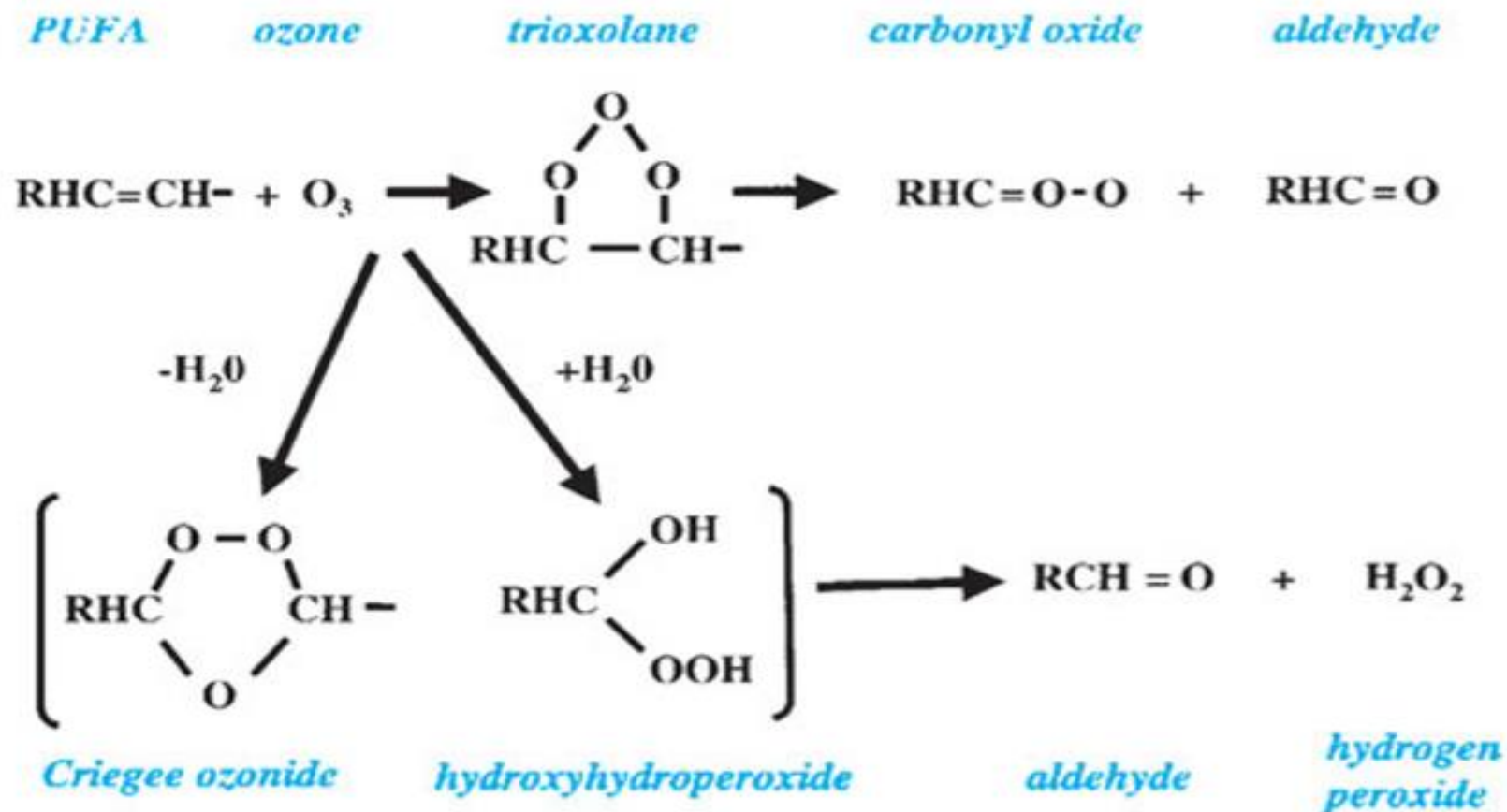


# Criteria Air Pollutants:

## Ozone O<sub>3</sub>

- **Cause: atmospheric photochemical reaction**
- **Reactants: Hydrocarbons & Nitrogen Oxides**
- **NAAQS . 80 ppb 8 hr average**
  - **. 120 ppb 1 hr average**
- **Acute Health effects**
  - Severe ear/nose/throat irritation
  - Eye irritation at 100 ppb ozone
  - Interferes with lung functions
    - Coughing at 2 ppm ozone
- **Chronic Health Effects**
  - Irreversible, accelerated lung damage
  - **Why do we use ozone as disinfectant for water and wastewater treatment?**





*Figure 28-12. Major reactions pathways of O<sub>3</sub> with lipids in lung lining fluid and cell membranes. (Adapted with permission from the Air Quality Criteria Document for Ozone and Photochemical Oxidants. 600/P-93/004cF, NCEA Research Triangle Park, NC: U.S. EPA, 1996).*

The mechanisms by which O<sub>3</sub> causes injury have been studied using cellular as well as cell-free systems. As a powerful **oxidant, O<sub>3</sub>** seeks to extract electrons from other molecules.

The surface fluid lining the respiratory tract and cell membranes that underlie the lining fluid contain a **significant quantity of polyunsaturated fatty acids** (PUFA), either free or as part of the lipoprotein structures of the cell. The double bonds within these fatty acids have an, **unpaired electron** which is easily attacked by O<sub>3</sub> to form **ozonides** that progress through a less stable zwitter ion or **trioxolane**(depending on the presence of water); these ultimately recombine or decompose to lipohydroperoxides, **aldehydes, and hydrogen peroxide**. These pathways are thought to initiate propagation of **lipid radicals and autooxidation** of cell membranes and macromolecule

Damage to the air-blood interface disrupts its barrier function and promotes inflammation. **Inflammatory cytokines**(e.g., interleukins 6, 8 and others, TNF, etc.) are released from epithelial cells and macrophages that mediate early responses and initiate repair. This inflammatory process is generally transient, but it may also **interact with neural irritant responses** to affect lung function acutely. This latter response may have implications for those with preexistent inflammation or disease



# Criteria Air Pollutants: Nitrogen Oxides NO<sub>x</sub>

- Cause: Fuel combustion at high temps
- Source: mobile & stationary combustion sources
- **Prolonged exposure** → pulmonary fibrosis, emphysema, and higher lower respiratory tract illness in children
- NAAQS = Annual Average 0.053 ppm as NO<sub>2</sub>
- **Toxic effects at 10-30 ppm NO<sub>x</sub>**
  - Nose and eye irritation
  - Lung tissue damage
    - Pulmonary edema (swelling)
    - Bronchitis
  - Pneumonia
  - Aggravate existing heart disease

.**Potential life-threatening exposure** is a world-wide problem for farmers, as near-lethal high levels of NO<sub>2</sub> can be liberated from fermenting fresh silage.

Being heavier than air, the generated NO<sub>2</sub> and CO<sub>2</sub> displace air and oxygen and diffuse into closed spaces where workers can be exposed to very high concentrations perhaps with **depleted oxygen**.

Typically, shortness of breath rapidly ensues with exposures nearing 75 to 100 ppm NO<sub>2</sub>, with delayed **edema and symptoms of pulmonary damage**.

**Nitrogen dioxide** is also an important indoor pollutant, especially in homes with unventilated gas stoves or kerosene heaters

Under such circumstances, very **young children and their mothers** who spend considerable time indoors may be especially at risk.

studies indicate that **NO<sub>2</sub>** is deposited along the length of the respiratory tree, with preferential deposition being in the distal airways.

the pattern of damage to the respiratory tract reflects this profile: **damage is most apparent in the terminal bronchioles, more proximal in the airway than is seen with O<sub>3</sub>.**

At high concentrations, the alveolar ducts and alveoli are also affected, showing their sensitivity to **oxidant** challenge. In the airways of these animals there is also damage to **epithelial cells in the bronchioles**, notably with loss of ciliated cells, as well as a loss of secretory granules in cells. The pattern of injury of **NO<sub>2</sub>** is quite similar to that of O<sub>3</sub>, but its **potency** is about an order of magnitude lower

# Criteria Air Pollutants: Sulfur Oxides $\text{SO}_x$

- Cause: Burning fuel that contains sulfur
- Source: Electric power generation, diesel trucks
- Soluble and absorbed by respiratory system
- NAAQS = 0.14 ppm 24 hr average
- **Short-term intermittent exposures**
  - Broncho-constriction (temporary breathing difficulty)
  - Ear/Nose/Throat irritation
  - Mucus secretion
- **Long-term exposures**
  - Respiratory illness
  - Aggravates existing heart disease

# Criteria Air Pollutants: Lead (Pb)

- Source: burning **fuels** that contain lead (phased out), metal processing, waste incinerators, lead smelters, lead paint
- Absorbed into blood; similar to calcium
- NAAQS =  $1.5 \mu\text{g}/\text{m}^3$  Pb Quarterly Average
- **Accumulates** in blood, bones, muscles, fat
  - Damages organs – kidneys, liver, brain, reproductive system, bones (osteoporosis)
  - **Brain and nervous system** – seizures, mental retardation, behavioral disorders, memory problems, mood changes,
    - **Young children** - lower IQ, learning disabilities
  - Heart and blood – high blood pressure and increased heart disease
  - Chronic poisoning possible



# Criteria Air Pollutants: Air Quality Index (AQI)

- EPA (AQI ) is for reporting daily air quality. The AQI focuses on short term health effects (1-48 hr after exposure). AQI is calculated from concentrations of SO<sub>2</sub>, CO, O<sub>3</sub>, and particles.
- AQI values in the **0-50** indicates Good air quality.
- AQI in the **51-100** range indicates Moderate air quality and exposures will cause short term health effects to some sensitive people (*and unhealthy effects for long-term exposure for most people*).
- Pilat opinion is that **“Moderate”** air quality is not very healthy.

# Criteria Air Pollutants: Air Quality Index (AQI)

- **AQI is the highest magnitude of the PM, SO<sub>2</sub>, CO, and O<sub>3</sub> individual Index values**

<b>AQI Value</b>	<b>Air Quality</b>	<b>24 hr PM<sub>2.5</sub> (µg/m<sup>3</sup>)</b>	<b>24 hr SO<sub>2</sub> (ppm)</b>	<b>8 hr CO (ppm)</b>	<b>8 hr O<sub>3</sub> (ppm)</b>
<b>0-50</b>	<b>Good</b>	<b>0-15.4</b>	<b>0.0 - .034</b>	<b>0.0-4.4</b>	<b>.000-.064</b>
<b>51-100</b>	<b>Moderate</b>	<b>15.5-40.4</b>	<b>.035-.144</b>	<b>4.5-9.4</b>	<b>.065-.084</b>
<b>101-150</b>	<b>Unhealthy to Sensitive</b>	<b>40.5-65.4</b>	<b>.145-.224</b>	<b>9.5-12.4</b>	<b>.085-.104</b>
<b>151-200</b>	<b>Unhealthy</b>	<b>65.5-150.4</b>	<b>.225-.304</b>	<b>12.5-15.4</b>	<b>.105-.124</b>
<b>201-300</b>	<b>Very Unhealthy</b>	<b>150.5-250.4</b>	<b>.305-.604</b>	<b>15.5-30.4</b>	<b>.125-.374</b>
<b>NAAQS</b>		<b>35 µg/m<sup>3</sup></b>	<b>0.14 ppm</b>	<b>9 ppm</b>	<b>.08 ppm</b>



# Hazardous Air Pollutant HAP:

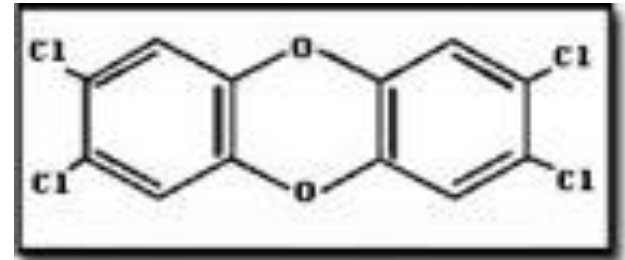
## Mercury Hg

- **Elemental** Hg inhaled as a vapor, absorbed by lungs
- **Cause:** vaporized mercury
- **Sources:** coal combustion, accidental spill, mining (*teeth silver fillings*)
- **Effects:** Nervous system (acute, high), respiratory system (chronic, low), kidneys, skin, eyes, immune system; Mutagenic properties
- Symptoms
  - **Acute:** chills, nausea, chest pains/tightness, cough, gingivitis, general malaise
  - **Chronic:** weakness, fatigue, weight loss, tremor, behavioral changes

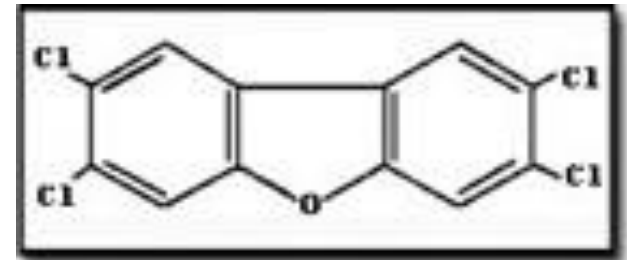


# Hazardous Air Pollutant **Dioxins**

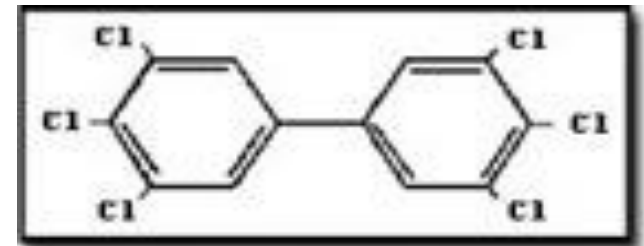
- Generic term for several chemicals that are highly persistent in the environment
  - chlorinated dibenzo-p-dioxins (CDDs)
  - chlorinated dibenzofurans (CDFs)
  - certain polychlorinated biphenyls (PCBs)
- Cause: burning hydrocarbons in presence of chlorine or chlorides
- Sources: waste incinerators
- Notice the **Chlorine** atoms on the benzene ring type molecules (probably all these type compounds are **carcinogenic**)
- **Does using Chlorine to treat drinking water result in the formation of carcinogenic compounds?**



2,3,7,8-Tetrachlorodibenzo-p-dioxin



2,3,7,8-Tetrachlorodibenzofuran



3,3',4,4',5,5'-Hexachlorobiphenyl

# Hazardous Air Pollutant: Dioxins



## Comparative Photos Showing Immediately Prior To And Immediately Following Dioxin Poisoning

(Note: this is an extreme case of dioxin poisoning)

- Varying toxicity
  - Problems with high exposures
  - Exact effects of low exposures not known
- Health Effects
  - **Carcinogenic**
    - Some are “known human carcinogen” (2,3,7,8 tetrachlordibenzo-p-dioxin, TCDD)
    - Other dioxins are “reasonably anticipated to be a Human Carcinogen”
  - **Reproductive and developmental effects**
  - **Chloracne**

**Aldehydes** Carbonyl compounds, notably short-chained (2–4 C) aldehydes, are common photo-oxidation products of unsaturated hydrocarbons.

Two aldehydes are of major interest by virtue of their concentrations and irritancy:

formaldehyde (HCHO) and acrolein (H<sub>2</sub>C=CHCHO). They contribute to the odor as well as eye and sensory effects of smog.

Formaldehyde accounts for about 50% of the estimated total aldehydes in polluted air, while acrolein, accounts for about 5% of the total

**Formaldehyde** and particularly a crolein are also found in main stream **tobacco smoke** .Formaldehyde is also an important **indoor air** pollutant and can often achieve higher concentrations indoors than outdoors due to out-gassing by new upholstery or other furnishing

**Formaldehyde** is a primary sensory irritant. Because it is very soluble in water, it is absorbed in mucous membranes in the nose, upper respiratory tract, and eyes

Along time concern regarding formaldehyde has been its potential **carcinogenicity by the detection of DNA adducts**

**Nasal cancer** had been induced empirically with formaldehyde vapor in a 2-year study where rats were exposed to 2, 6, or 14 ppm 6 hours per day, 5 days per week.

# Other Aerosols: Bioaerosols

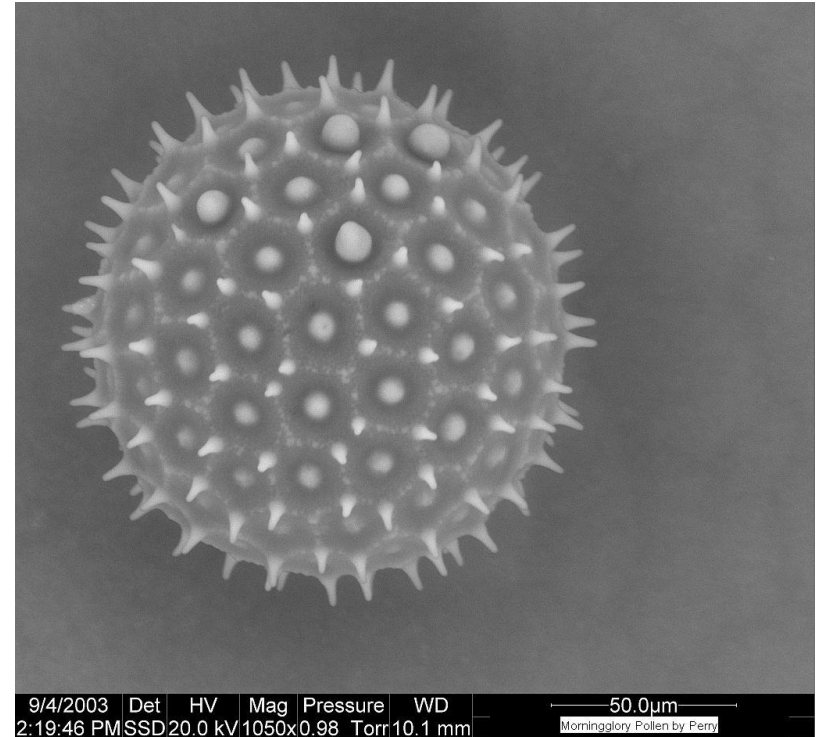


**Mechanical aeration at  
Waste water treatment plant**

- **Aerosols with organic origin**
  - **Non-viable:** pollen, dander, insect excreta, sea salt
  - **Viable:** microorganisms
- **Cause:** aerosolization of organic materials
- **Sources:**
  - Human: sneezing, coughing
  - wind, waves, Waste water treatment plants, cooling towers
- **Health Effects:** allergies (pollen) to death (pathogenic organisms)
  - Pathogenic – Minimum Infectious Dose

# Other Aerosols: Bioaerosols

- Allergies
  - Pollen, dander, fungi (spores)
- Airborne transmission of disease
  - Bird flu,, Legionnella (pneumonia)
  - Indoor Air Quality
    - Ventilation Systems – moist ductwork, protection, recycled air
    - Office Buildings – Sick Building Syndrome
      - Hospital (nosocomial)
    - Biological Warfare
      - Anthrax, Ebola virus



Morning Glory Pollen SEM