Environmental Factors in Oral Health

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Oral health is an essential component of general health as poor oral health effects growth, development and learning for children, communication, nutrition, self-esteem and various systemic conditions. Because of its effects on daily living, oral health is considered a determinant of quality of life.

Impact of environmental change on the lives of natives of a particular geographical region influences the overall health status of an individual. Environmental pollution is now recognized as a global threat and actions of mankind are largely accountable for this.

Various oral diseases like dental caries, fluorosis are influenced by the food and water quality in a particular topographic area. Chemicals in water can be both naturally occurring or introduced by human interference and can have a huge impact on teeth and oral mucosa. Arsenic occurs naturally or by phosphorus from fertilizers. High concentrations of arsenic in water can have an adverse effect on health particularly skin and other tissues of the body including tongue, gingiva and buccal mucosa.

Without access to clean water, people living in developing and underdeveloped countries may suffer oral health deterioration well into their adulthood. This leads to tooth loss, gum disease and even oral cancer.

Air pollution influences the development of oral clefts in animals. There is evidence from the epidemiologic data on the relation of prenatal air pollution exposure and the risk of oral clefts.

Oral precancer (oral leukoplakia & oral submucous fibrosis) and cancer are complex multi-factorial diseases arising from the interplay between the genetic components and the environmental determinants. Environmental exposures in farmers can be explained by solar (ultra violet) exposure. Climate change induced oxidative stress, dietary transition, contamination; impacts on food chain ecosystem and food security have increased the vulnerability to oral cancer.

Exposure to solvents and possibly to pesticides, fertilizers, engine exhaust, textile dust and leather dust also increase the risk of oral cancer. Indoor air pollution contributes to oral cancer, with other significant inflammatory respiratory diseases and infections. Estimated exposure to wood smoke and biomass smoke released from cooking in households in developing countries has led to increased risk of oral cancer, particularly in women and children.

In India, environmental pollution related disorders may further weaken the existent inadequate public health infrastructure. Research should be targeted to understand the complex interplay of gene-environment interactions and oxidant mediated oral diseases. Epidemiological assessment and willingness to tackle health burden arising due to impact of environmental factors needs to be strengthened. This would facilitate a harmonious balance to enable sustainable development of optimum oral and in turn overall health.
What is oral health & why consider oral diseases as a serious public health threat?
The World Health Organization (WHO) defines oral health as ‘a state of being free from mouth and facial pain, oral and throat cancer, oral infection and sores, periodontal disease, tooth decay, tooth loss, and other diseases and disorders that limit an individual’s capacity in biting, chewing, smiling, speaking, and psychosocial wellbeing.’
Oral health is an essential component of general health as poor oral health effects growth, development and learning for children, communication, nutrition, self-esteem and various systemic conditions.
Because of its effects on daily living,

“oral health is considered a determinant of quality of life”
• Many general conditions increase the risk of oral diseases, such as an increased risk of periodontal disease in patients with diabetes.

• Equally, poor oral health can adversely affect a number of general health conditions and their management.

• The close bi-directional relationship between oral and general health, and its impact on an individual’s health and quality of life, provides a strong conceptual basis for the integration of oral healthcare into general healthcare approaches.
Yet, the high burden of oral diseases represents a widely underestimated public health challenge for almost all countries worldwide.

**Estimated Number of People Affected by Common Diseases 2010**

- **3,054m** untreated decay of primary and permanent teeth
- **1,013m** migraine
- **743m** severe periodontitis
- **549m** diabetes
- **334m** asthma

Tooth decay is the most prevalent of conditions, affecting almost half (44%) of the world population in 2010, followed by tension-type headache (21%), migraine (15%), severe periodontitis (11%), diabetes (8%) and asthma (5%).

Humans interact with the environment constantly.

These interactions affect quality of life, years of healthy life lived, and health disparities.

The World Health Organization (WHO) defines environment, as it relates to health, as

“all the physical, chemical, and biological factors external to a person, and all the related behaviors.”

Environmental health consists of preventing or controlling disease, injury, and disability related to the interactions between people and their environment.
Environmental Factors

**Physical Environment**
- Geography
- Soils
- Climate
- Shelter etc

**Biological Environment**
- Humans
- Animals
- Plants
- Other micro-organisms

**Socio-economic Environment**
- Socio-political
- Cultural
- Economic
- Demographic
Effects of environmental alterations on human health are both direct and indirect.

People are exposed directly to changing weather pattern (temperature & sea-level rise) and more frequent extreme events.

Indirectly, it affects changes in the quality of water, air, food, changes in ecosystem, agriculture, industry, human settlements & the economy.

Health problems increase vulnerability and reduce the capacity of groups to adapt to these alterations.

Therefore, the socio-economic, physical & biological environmental determinants of health have a vital role to play.
**Health** is ‘a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity’ (WHO) it represents a balanced relationship of the body and mind and complete adjustment to the total environment.

**Disease**, on the other hand, is maladjustment or maladaptation in an environment, a reaction for the worse between man and hazards or adverse influences in his external environment.

The response of the individual to these influences is conditioned by his genetic make-up or internal environment.
The Healthy People 2020 Environmental Health objectives focus on 6 themes, each of which highlights an element of environmental health:

- Outdoor air quality
- Surface and ground water quality
- Toxic substances and hazardous wastes
- Homes and communities
- Infrastructure and surveillance
- Global environmental health

Creating healthy environments can be complex and relies on continuing research to better understand the effects of exposure to environmental hazards on people's health.
• Oral diseases, like all other diseases, share a wide range of risk factors.

• Some, such as age, sex and hereditary conditions, are intrinsic to the individual and cannot be changed or modified.

• Others, which are subject to behaviours and lifestyle, are considered to be modifiable risk factors, because individual action and modification of a particular habit or behaviour is possible.

• The modifiable risk factors of oral diseases include an unhealthy diet, particularly one high in sugar, tobacco use, and unhealthy alcohol consumption. These key risk factors are also shared with most of the other major NCDs.
• A range of external factors that can be mitigated to only a small extent by individual behaviours also determine oral health.

• These determinants include
• poor living conditions
• low education
• Unemployment
• limited access to safe water
• sanitary facilities,
• limited access to oral healthcare.
Environmental factors are a root cause of a significant disease burden, particularly in developing countries. An estimated 25% of death and disease globally, and nearly 35% in regions such as sub-Saharan Africa, is linked to environmental hazards. Some key areas of risk include the following:

- **Unsafe water, poor sanitation and hygiene** kill an estimated 1.7 million people annually, particularly as a result of diarrhoeal disease.

- **Indoor smoke** from solid fuels kills an estimated 1.6 million people annually due to respiratory diseases.

- **Noma** kills over 1.2 million people annually, mostly African children under the age of five. Poorly designed irrigation and water systems, inadequate housing, poor waste disposal and water storage all may be contributing factors.

- **Urban air pollution** generated by vehicles, industries and energy production kills approximately 800 000 people annually.

- **Unintentional acute poisonings** kill 355 000 people globally each year. In developing countries, where two-thirds of these deaths occur, such poisonings are associated strongly with excessive exposure to, and inappropriate use of, toxic chemicals and pesticides present in occupational and/or domestic environments.

- **Climate change** impacts including more extreme weather events, changed patterns of disease and effects on agricultural production, are estimated to cause over 150 000 deaths annually.
General socioeconomic, cultural and environmental conditions also affect individuals’ oral health, but these are beyond the influence of any given individual.

- Tobacco control legislation and water fluoridation programmes are examples of so called ‘upstream’ measures to address such factors.
- Across the whole social gradient, from the richest to the poorest, those in lower positions suffer worse health and poorer access to appropriate care than those immediately above them.
- In all societies the poorest have the worst health, the worst access to care and the worst health outcomes. These inequalities can be observed both between and within regions and countries.
Noma (Cancrum Oris)

Noma, a **debilitating orofacial gangrene**, is an important contributor to the disease burden on many developing countries, particularly in Asia and Africa.

Noma primarily starts as a localised gingival ulceration and spreads rapidly through the orofacial tissues, establishing itself with a blackened necrotic centre.

About 70-90% of cases are fatal in the absence of care.

Fresh noma is seen predominantly in the age group of 1-4 years although late stages of disease occur in adolescents and adults.
WHO has suggested a global incidence of 140 000 cases with a prevalence in 1997 of 770 000 victims.

Poverty is the key risk condition for the development of noma.

**Environment that induces noma is characterised by:**

- severe malnutrition and growth retardation
- unsafe drinking water
- deplorable sanitary practices
- residential proximity to unkempt animals
- and a high prevalence of infectious disease such as measles, malaria, diarrhoea, pneumonia, tuberculosis and HIV/AIDS.
• Impact of environmental change on the lives of natives of a particular geographical region influences the overall health status of an individual.

• Environmental pollution is now recognized as a global threat and actions of mankind are largely accountable for this.
Surface and ground water quality concerns apply to both drinking water and recreational waters.

Contamination by infectious agents or chemicals can cause mild to severe illness.

Water quality has been seen to directly affect oral health based on various studies.

Indonesian case study proved that without access to clean and safe drinking water, almost all of the children aged 5-7 years who were involved in the study had some level of tooth decay. The water in their area was found to have no fluoride, high levels of manganese, and high acidity level which all contributed to the poor oral health of the residents.
• **Long-Term Effects of Poor Water Quality**

Without access to clean water, people living in developing and underdeveloped countries may suffer oral health deterioration well into their adulthood.

This leads to tooth loss

gum disease

oral cancer.
• Various oral diseases like dental caries, fluorosis are influenced by the food and water quality in a particular topographic area.

• Chemicals in water can be both naturally occurring or introduced by human interference and can have a huge impact on teeth and oral mucosa.

• Arsenic occurs naturally or by phosphorus from fertilizers. High concentrations of arsenic in water can have an adverse effect on health particularly skin and other tissues of the body including tongue, gingiva and buccal mucosa.
Water pollution

• The contamination of water by harmful or undesirable substances making it unfit for use

• Chemicals in water can be both naturally occurring or introduced by human interference and can have serious health effects

• Without access to clean water, people living in developing and underdeveloped countries may suffer oral health deterioration well into their adulthood.

• This leads to tooth loss, gum disease and even oral cancer.
Flourides

• Although fluoride is introduced into some water supplies for its protective effect on tooth enamel, too much fluoride can lead to tooth and bone damage. Fluoride exposure most often comes through drinking water, but some coal contains high levels of fluoride, which can lead to additional exposure.

• Fluoride. Fluoride in the water is essential for protection against dental caries and weakening of the bones, but higher levels can have an adverse effect on health. In India, high fluoride content is found naturally in the waters in Rajasthan.

• Children are the most susceptible to fluoride poisoning. It can lead to opaque white lesions on their teeth at low levels, and at higher levels it can lead to discolored and damaged teeth.
Severe effects of fluoridated water: discoloration, pitting, cracking and chipping
Heavy metals

• These may be airborne, or may be found in water, food, or other sources of exposure.
• These can also cause damage to your teeth.
• Metal amalgam is a common source of heavy metal exposure. Although the heavy metals in amalgams are not damaging to teeth, they can damage the body if they are mobilized through vigorous chewing, tooth grinding, or with time.
• Studies have shown that people in areas with high levels of heavy metal pollution have significant damage to their teeth as a result. The metals result in roughening of the tooth surface, which makes them more susceptible to decay from bacteria.
• **Arsenic** - Arsenic occurs naturally or by phosphorus from fertilizers. High concentrations of arsenic in water can have an adverse effect on health particularly skin.

• **Lead** - Pipes, fittings, solder, and the service connections of some household plumbing systems contain lead that contaminates the drinking water source.

• **Petrochemicals** - Petrochemicals contaminate the groundwater from underground petroleum storage tanks.

• **Other heavy metals** - These contaminants come from mining waste and tailings, landfills, or hazardous waste dumps.

• **Chlorinated solvents** - Metal and plastic effluents, fabric cleaning, electronic and aircraft manufacturing are often discharged and contaminate groundwater.
Arsenic

Occupational hazard /Can cause acute or chronic poisoning

Dermatological alterations:

- Diffuse macular pigmentation
- Palmar and plantar hyperkeratosis
- Premalignant lesion - arsenical keratosis that can transform to basal cell carcinoma and cutaneous squamous cell carcinoma
- Arsenic keratosis on the palms of a patient who ingested arsenic from contaminated well over a prolonged period
ORAL MANIFESTATIONS • Intense inflammation of oral mucosa • Severe gingivitis • Tissue becomes painful • Local contact with arsenic trioxide produces ulceration • Systemic poisoning can cause excessive salivation
Occupational Hazard

An occupational hazard is a hazard experienced in the workplace. Occupational hazards can encompass many types of hazards, including chemical hazards.

Chemical hazards are a subtype of occupational hazards that involve dangerous chemicals.

Exposure to chemicals in the workplace can cause acute or long-term detrimental health effects.
<table>
<thead>
<tr>
<th>Aetiological agent</th>
<th>Occupation</th>
<th>Oral manifestation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Physical agent</strong></td>
<td><strong>Principal action</strong></td>
<td><strong>Specific factor</strong></td>
</tr>
<tr>
<td>Solid</td>
<td>Physical</td>
<td>Instrument</td>
</tr>
<tr>
<td>Organic bone, celluloid, sawdust, flour, tobacco</td>
<td>Bone, celluloid, flour, sawmill, textile and tobacco workers</td>
<td>Bone, celluloid, flour, sawmill, textile and tobacco workers</td>
</tr>
<tr>
<td>Inorganic arsenic</td>
<td>Chemical</td>
<td>Chemical workers, electroplaters, metal refiners, rubber mixer, lead smelters, insecticide makers</td>
</tr>
<tr>
<td>Bismuth</td>
<td>Bismuth handlers, dusting powder makers</td>
<td>Blue pigmentation of gingiva, oral mucosa, gingivostomatitis</td>
</tr>
<tr>
<td>Chromium</td>
<td>Aniline compounds, chrome, photographic and steel workers, blue printers, rubber mixers</td>
<td>Necrosis of bone, ulceration of oral tissues</td>
</tr>
<tr>
<td>Chemical</td>
<td>Flourine</td>
<td>Cryolite workers</td>
</tr>
<tr>
<td>Lead</td>
<td>Electrotypers, insecticides and storage battery makers, lead refiners printers, rubber compounders</td>
<td>Blue black pigmentation of gingiva, gingivostomatitis</td>
</tr>
<tr>
<td>Mercury</td>
<td>Bronzers (gun barrels), battery and paint makers, dentists, detonators, explosives and mercury salt workers</td>
<td>Gingivostomatitis, osteomyelitis, pytalism</td>
</tr>
<tr>
<td>Phosphorus</td>
<td>Brass founders, match factory, phosphor bronze workers, fertilizer and firework makers</td>
<td>Gingivostomatitis, ulceration of oral tissues, osteomyelitis</td>
</tr>
<tr>
<td>Organic sugar</td>
<td>Refiners, bakers, candy makers</td>
<td>Caries</td>
</tr>
</tbody>
</table>
Bismuth stomatitis

Long term treatment with bismuth compounds

Diffuse dark bluish discoloration on gingival sulcus, buccal mucosa and tongue. Metallic taste and burning sensation in the mouth may also be present.
ORAL MANIFESTATIONS •

• “BISMUTH LINE”: a thin blue black line in the marginal gingiva sometimes confined to gingival papillae.

• Also seen in buccal mucosa and ventral surface of tongue

• Pigmentation shows precipitated granules of bismuth sulfide produced by action of hydrogen sulfide on bismuth.

• Hydrogen sulfide is produced by bacterial degradation of organic material or food debris.

• Burning sensation

• Metallic taste
ORAL MANIFESTATIONS

Gray or bluish black line of sulfide pigmentation in gingiva – “lead line” or “Burtons line”.

Found in other areas of oral cavity. Diffuse in nature.

Excessive salivation • Metallic taste
<table>
<thead>
<tr>
<th>Aetiologic agent</th>
<th>Occupation</th>
<th>Possible oral manifestations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Physical states</strong></td>
<td><strong>Principal action</strong></td>
<td><strong>Specific factor</strong></td>
</tr>
<tr>
<td>Liquid</td>
<td>Hot food</td>
<td>Tasters</td>
</tr>
<tr>
<td>Physical</td>
<td>Aniline</td>
<td>Coal tar, explosive workers, painters</td>
</tr>
<tr>
<td>Benzene</td>
<td>Dry cleaners, vulcanizers, smokeless powder makers</td>
<td></td>
</tr>
<tr>
<td>Chemical</td>
<td>Cresol</td>
<td>Coal tar, rubber, surgical dressing workers</td>
</tr>
<tr>
<td></td>
<td>wine and liquor atmosphere</td>
<td>Tasters</td>
</tr>
<tr>
<td></td>
<td>Increased pressure</td>
<td>Divers, caisson workers</td>
</tr>
<tr>
<td></td>
<td>Decreased pressure</td>
<td>Aviators</td>
</tr>
<tr>
<td>Physical</td>
<td>Acids</td>
<td>Acid cartridges and dippers, explosives and gun cotton workers</td>
</tr>
<tr>
<td></td>
<td>Amyl acetate</td>
<td>Alcohol distillery, explosives, shoe factory workers</td>
</tr>
<tr>
<td>Aetiologic agent</td>
<td>Occupation</td>
<td>Possible oral manifestations</td>
</tr>
<tr>
<td>------------------------</td>
<td>------------------------------------------------</td>
<td>----------------------------------------</td>
</tr>
<tr>
<td><strong>Physical states</strong></td>
<td><strong>Principal action</strong></td>
<td><strong>Specific factor</strong></td>
</tr>
<tr>
<td>Gas</td>
<td>Acrolein</td>
<td>Bone grinders, soap, linoleum markers</td>
</tr>
<tr>
<td>Chemical, CO, CO₂</td>
<td>Miners, smelters, gasoline motor workers</td>
<td>Coloration of lips</td>
</tr>
<tr>
<td>Radiations</td>
<td>Physico-chemical</td>
<td>Technicians, watch dial painters</td>
</tr>
<tr>
<td>Actinic</td>
<td>Sailors, fishermen</td>
<td><strong>Carcinoma of lip</strong></td>
</tr>
</tbody>
</table>


Air Pollution

• Poor air quality is linked to premature death, cancer, and long-term damage to respiratory and cardiovascular systems.

• Exposure to solvents and possibly to pesticides, fertilizers, engine exhaust, textile dust and leather dust also increase the risk of oral cancer. Indoor air pollution contributes to oral cancer, with other significant inflammatory respiratory diseases and infections. Estimated exposure to wood smoke and biomass smoke released from cooking in households in developing countries has led to increased risk of oral cancer, particularly in women and children.

• There is evidence from the epidemiologic data on the relation of prenatal air pollution exposure and the risk of oral clefts.
Particulate matter

• Primary particulate matter: Combustion processes emit particulate matter less than 1um size. Large quantities of NO₂ and SO₂ are also emitted.
• Secondary particulate matter: gas to particle conversion.
• Gaseous molecules transformed to liquid and solid particles
• Gases: O₃, CO, NOₓ, Sox
• Particles: Pb, Bioaerosols
<table>
<thead>
<tr>
<th>Pollutant</th>
<th>Mechanism</th>
<th>Potential health effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Particles (small particles less than 10 microns, and particularly less 2.5 microns aerodynamic diameter)</td>
<td>Acute: bronchial irritation, inflammation and increased reactivity. Reduced mucociliary clearance. Reduced macrophage response and reduced local immunity. Fibrotic reaction</td>
<td>Wheezing, exacerbation of asthma. Respiratory infections. Chronic bronchitis and chronic obstructive pulmonary disease. Exacerbation of chronic obstructive pulmonary disease</td>
</tr>
<tr>
<td>Carbon monoxide</td>
<td>Binding with haemoglobin to produce carboxy haemoglobin, which reduces oxygen delivery to key organs and the developing fetus.</td>
<td>Low birth weight (fetal carboxyhaemoglobin 2–10% or higher). Increase in perinatal deaths</td>
</tr>
<tr>
<td>Polycyclic aromatic hydrocarbons, e.g. benzo[a]pyrene</td>
<td>Carcinogenic</td>
<td>Lung cancer. <strong>Cancer of mouth, nasopharynx and larynx</strong></td>
</tr>
<tr>
<td>Nitrogen dioxide</td>
<td>Acute exposure increases bronchial reactivity. Longer term exposure increases susceptibility to bacterial and viral lung infections</td>
<td>Wheezing and exacerbation of asthma. Respiratory infections. Reduced lung function in children</td>
</tr>
<tr>
<td>Sulphur dioxide</td>
<td>Acute exposure increases bronchial reactivity. Longer term: difficult to dissociate from effects of particles</td>
<td>Wheezing and exacerbation of asthma. Exacerbation of chronic obstructive pulmonary disease, cardiovascular disease</td>
</tr>
<tr>
<td>Biomass smoke condensates including polycyclic aromatics and metal ions</td>
<td>Absorption of toxins into lens, leading to oxidative changes</td>
<td>Cataract</td>
</tr>
</tbody>
</table>
Mercury

• Elemental Hg inhaled as a vapor, absorbed by lungs
• Cause: vaporized mercury
• Sources: coal combustion, accidental spill, mining, teeth-silver fillings
Spontaneous exfoliation of teeth following severe elemental mercury poisoning: case report and histological investigation for mechanism

Michael D Martin DMD, MPH, MA, MSD, PhD; Bryan J Williams DDS, MSD (Director); Jay D Charleston PhD; Dolphine Oda BDS, MSc

Abstract

**Background.** Although the spontaneous exfoliation of teeth and breakdown of oral tissues from severe mercury intoxication have been noted for over a century, there are no published reports investigating the mechanisms of these phenomena. Severe mercury poisoning is rare in modern times, but it does occur. We present a case report and a histopathologic investigation into the mechanism of the associated tooth loss.

**Methods.** An exfoliated tooth and periodontal and gingival tissues were obtained from a 15-month-old patient who had been severely intoxicated with elemental mercury over a period of months and hospitalized for severe neurologic and renal effects. The tissues were
### Common Symptoms of Chronic Mercury Poisoning

<table>
<thead>
<tr>
<th>System</th>
<th>Symptoms</th>
</tr>
</thead>
<tbody>
<tr>
<td>Digestive System</td>
<td>Colitis, Diarrhea/constipation, Loss of appetite, Weight loss, Nausea/vomiting</td>
</tr>
<tr>
<td>Emotions</td>
<td>Aggressiveness, Anger, Anxiety, Confusion, Depression, Fear and nervousness, Hallucination, Lethargy, Manic depression, Mood swings, Shyness</td>
</tr>
<tr>
<td>Energy Levels</td>
<td>Apathy, Chronic tiredness, Restlessness</td>
</tr>
<tr>
<td>Head</td>
<td>Dizziness, Faintness, Headaches (frequent), Ringing in ears</td>
</tr>
<tr>
<td>Heart</td>
<td>Anemia, Chest pain, Heartbeat rapid or irregular</td>
</tr>
<tr>
<td>Lungs</td>
<td>Asthma/bronchitis, Chest congestion, Shallow respiration, Shortness of breath,</td>
</tr>
<tr>
<td>Muscles &amp; Joints</td>
<td>Cramping, Joint aches, Muscle aches, Muscle weakness, Stiffness</td>
</tr>
<tr>
<td>Neurological/Mental</td>
<td>Fine tremor, Lack of concentration, Learning disorders, Memory loss, short and long term Numbness, Slurred speech</td>
</tr>
<tr>
<td>Nose</td>
<td>Inflammation of the nose, Sinusitis, Excessive mucus formation, Stuffy nose,</td>
</tr>
<tr>
<td>Oral/Throat</td>
<td>Bad breath (halitosis), Bone loss, Burning sensation, Chronic coughing, Gingivitis/bleeding gums, Inflammation of the gums, Leukoplakia (white patches), Metallic taste, Mouth inflammation, Sore throats, Ulcers of oral cavity</td>
</tr>
<tr>
<td>Other</td>
<td>Allergies, Anorexia, Excessive blushing, Genital discharge, Gland swelling, Hair loss, Hypoxia, Illnesses (frequent), Insomnia, Loss of sense of smell, Perspiration excessive, Renal failure, Skin cold and clammy, Skin problems, Vision problems (tunnel vision), Water retention (edema)</td>
</tr>
</tbody>
</table>
Oral Manifestations:

- Bad breath (halitosis)
- Bone loss
- Burning sensation
- Chronic coughing
- Gingivitis/bleeding gums
- Gingivitis
- Leukoplakia (white patches)
- Metallic taste
- Mucositis
- Sore throat
- Ulcers of oral cavity
Health Effects of Indoor Fluoride Pollution from Coal Burning in China

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The combustion of high fluoride-content coal as an energy resource for heating, cooking, and food drying is a major exhaust emission source of suspended particulate matter and fluoride. High concentrations of these pollutants have been observed in indoor air of coal-burning families in some rural areas in China. Because airborne fluoride has serious toxicological properties, fluoride pollution in indoor air and the prevalence of fluorosis have been analyzed in a fluorosis area and a healthy nonfluorosis area in China and in a rural area in Japan. For human health, fluoride in indoor air has not only been directly inhaled by residents but also has been absorbed in stored food such as corn, chilies, and potatoes. In the fluorosis area in China, concentrations of urinary fluoride in the residents have been much higher than in the nonfluorosis area in China and in the rural area in Japan. In the fluorosis area, almost all elementary and junior high school students 10–15 years of age had dental fluorosis. Osteosclerosis in the skeletal fluorosis patients was very serious. Urinary deoxypyridinoline in rural residents in China was much higher than in rural residents in Japan. Data suggest that bone resorption was extremely stimulated in the residents in China and that fluoride may stimulate both bone resorption and bone formation. Because indoor fluoride from combustion of coal is easily absorbed in stored food and because food consumption is the main source of fluoride exposure, it is necessary to reduce airborne fluoride and food contamination of fluorosis have been observed in some rural areas in China, the potential exposure to fluoride has been a matter of great concern in China. In the present study, changes in the concentrations of SPM and fluoride in indoor/outdoor air and fluoride contamination in stored food were measured in rural areas in China.

Many reports have confirmed that fluoride stimulates bone formation (18–20). On the other hand, almost no confirmed or reliable relationships have been observed between fluoride exposure and the prevalence of bone fractures (21–25). It is generally accepted that the hydroxyapatite structure is a prototype for the structure of bone mineral, and fluoride ions substitute for the
Health Effects of Indoor Fluoride Pollution from Coal Burning in China

• The combustion of high fluoride-content coal as an energy resource for heating, cooking, and food drying is a major exhaust emission source of suspended particulate matter and fluoride. High concentrations of these pollutants have been observed in indoor air of coal-burning families in some rural areas in China.

• Airborne fluoride has serious toxicological properties. Fluoride pollution in indoor air and the prevalence of fluorosis have been analyzed in a fluorosis area and a healthy non-fluorosis area in China and in a rural area in Japan.

• For human health, fluoride in indoor air has not only been directly inhaled by residents but also has been absorbed in stored food such as corn, chilies, and potatoes.
Indoor air pollution in developing countries: a major environmental and public health challenge

Nigel Bruce,¹ Rogelio Perez-Padilla,² & Rachel Albalak³

Around 50% of people, almost all in developing countries, rely on coal and biomass in the form of wood, dung and crop residues for domestic energy. These materials are typically burnt in simple stoves with very incomplete combustion. Consequently, women and young children are exposed to high levels of indoor air pollution every day.

There is consistent evidence that indoor air pollution increases the risk of chronic obstructive pulmonary disease and of acute respiratory infections in childhood, the most important cause of death among children under 5 years of age in developing countries. Evidence also exists of associations with low birth weight, increased infant and perinatal mortality, pulmonary tuberculosis, nasopharyngeal and laryngeal cancer, cataract, and, specifically in respect of the use of coal, with lung cancer. Conflicting evidence exists with regard to asthma. All studies are observational and very few have measured exposure directly, while a substantial proportion have not dealt with confounding. As a result, risk estimates are poorly quantified and may be biased. Exposure to indoor air pollution may be responsible for nearly 2 million excess deaths in developing countries and for some 4% of the global burden of disease.

Indoor air pollution is a major global public health threat requiring greatly increased efforts in the areas of research and policy-making. Research on its health effects should be strengthened, particularly in relation to tuberculosis and acute lower respiratory infections. A more systematic approach to the development and evaluation of interventions is desirable, with clearer recognition of the interrelationships between poverty and dependence on
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DNA Damage in Buccal Mucosa Cells of Pre-School Children Exposed to High Levels of Urban Air Pollutants

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Abstract

Air pollution has been recognized as a human carcinogen. Children living in urban areas are a high-risk group, because genetic damage occurring early in life is considered able to increase the risk of carcinogenesis in adulthood. This study aimed to investigate micronuclei (MN) frequency, as a biomarker of DNA damage, in exfoliated buccal cells of pre-school children living in a town with high levels of air pollution. A sample of healthy 3-6-year-old children living in Brescia, Northern Italy, was investigated. A sample of the children’s buccal mucosa cells was collected during the winter months in 2012 and 2013. DNA damage was investigated using the MN test. Children’s exposure to urban air pollution was evaluated by means of a questionnaire filled in by their parents that included items on various possible sources of indoor and outdoor pollution, and the concentration of fine particulate matter (PM10, PM2.5) and NO2 in the 1–3 weeks preceding biological sample collection. 181 children (mean age±SD: 4.3±0.9 years) were investigated. The mean±SD MN frequency was 0.29±0.13%. A weak, though statistically significant, association of MN with concentration of air pollutants (PM10, PM2.5 and NO2) was found, whereas no association was apparent between MN frequency and the indoor and outdoor exposure variables investigated via the questionnaire. This study showed a high MN frequency in children living in a town with heavy air pollution in winter, higher than usually found among children living in areas with low or medium-high levels of air pollution.
Ozone and Other Air Pollutants and the Risk of Oral Clefts

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BACKGROUND: Air pollution influences the development of oral clefts in animals. There are few epidemiologic data on the relation of prenatal air pollution exposure and the risk of oral clefts.

OBJECTIVES: Our goal in this study was to assess the relations between exposure to ambient air pollution and the risk of cleft lip with or without cleft palate (CL/P).

METHODS: We conducted a population-based case–control study of all 653 cases of CL/P and a random sample of 6,530 control subjects from 721,289 Taiwanese newborns in 2001–2003. We used geographic information systems to form exposure parameters for sulfur dioxide, nitrogen oxides, ozone, carbon monoxide, and particulate matter with an aerodynamic diameter ≤ 10 μm (PM₁₀) during the first 3 months of pregnancy using inverse distance weighting method. We present the effect estimates as odds ratios (ORs) per 10-ppb change for SO₂, NO₂, and O₃, 100-ppb change for CO, and 10-μg/m³ change for PM₁₀.

RESULTS: The risk of CL/P was increased in relation to O₃ levels in the first gestational month [adjusted OR = 1.20; 95% confidence interval (CI), 1.02–1.39] and second gestational month [adjusted OR = 1.25; 95% CI, 1.03–1.52] in the range from 16.7 ppb to 45.1 ppb, but was not related to CO, NO₂, SO₂, or PM₁₀.

CONCLUSIONS: The study provides new evidence that exposure to outdoor air O₃ during the first and second month of pregnancy may increase the risk of CL/P. Similar levels of O₃ are encountered globally by large numbers of pregnant women.

• Air pollution influences the development of oral clefts in animals.

• There are few epidemiologic data on the relation of prenatal air pollution exposure and the risk of oral clefts.

• The study was done to assess the relations between exposure to ambient air pollution and the risk of cleft lip with or without cleft palate (CL/P).

• The study provides new evidence that exposure to outdoor air O3 during the first and second month of pregnancy may increase the risk of CL/P.

• Similar levels of O3 are encountered globally by large numbers of pregnant women.
Agrochemicals: Health And Environment Linkages

• In developing countries, deaths by unintentional poisoning may be strongly associated with inappropriate use and poor environmental management of toxic chemicals, including pesticides.

• Good management, use, and disposal of agrochemicals – particularly pesticides – is an important health and environment issue in developing countries – where economies may be heavily reliant on agriculture.

• Chronic pesticide exposure is most often a problem in the occupational setting, particularly among poor rural populations where men, women, and children all work and live in close proximity to fields and orchards where chemicals are applied and stored.

• Long-term exposure to pesticides can increase the risk of developmental and reproductive disorders, immune-system disruption, endocrine disruption, impaired nervous-system function, and development of certain cancers.

• Pesticides, as well as fertilizers, can infiltrate water sources – contaminating drinking water and animal species, e.g. fish, upon which humans rely for nutrition. Such contamination can lead to a range of secondary public health impacts.
Pesticides

A study of pesticides sales different parts of Brazil and cancer mortality rates a decade later finds pesticide sales show statistically significant correlation with the mortality rates for several cancers, including cancer of the lip.

A Swedish study based on a cancer registry of agricultural workers finds an increased risk of cancer of the lip by a factor of greater than 2.51.

These chemicals damage to DNA in cells can lead to cancer. However, cells can often repair DNA damage. If the damage is extreme, the cells may die. Unrepaired DNA damage can lead to mutations or changes, in genes and mutations in certain genes can cause cancer.
Developmental Dental Aberrations After the Dioxin Accident in Seveso

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Children’s developing teeth may be sensitive to environmental dioxins, and in animal studies developing teeth are one of the most sensitive targets of toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Twenty-five years after the dioxin accident in Seveso, Italy, 48 subjects from the contaminated areas (zones A and B) and in patches lightly contaminated (zone R) were recruited for the examination of dental and oral aberrations. Subjects were randomly invited from those exposed in their childhood and for whom frozen serum samples were available. The subjects were frequency matched with 65 subjects from the surrounding non-ABR zone for age, sex, and education. Concentrations of TCDD in previously analyzed plasma samples (zone ABR subjects only) ranged from 23 to 26,000 ng/kg in serum lipid. Ninety-three percent (25 of 27) of the subjects who had developmental enamel defects had been <5 years of age at the time of the accident. The prevalence of defects in this effects in laboratory rodents include cleft palate, disturbed development of the mandible, various ureteric and kidney malformations in mice (Abbott and Birnbaum 1989; Abbott et al. 1987; Allen and Leam 2001; Peters et al. 1999), and alterations in the reproductive tract development and function in mice and rats (Hurst et al. 2000; Theobald and Peterson 1997). Impaired mammary gland development and differentiation were found in female mice after gestational and lac-
In two episodes of epidemic poisoning in Japan and Taiwan (so-called Yusho and Yucheng accidents, respectively), severe developmental effects were observed in infants and children born to mothers who had been exposed to polychlorinated dibenzofurans/ biphenyls (PCDFs/PCBs) (Rogan et al. 1988; Yamashita and Hayashi 1985; Yao et al. 2002).
A variety of **dental and oral changes** were also reported in children exposed to PCB/PCDF in the Yusho and Yucheng accidents.

- **Birth** – natal teeth
  - oral pigmentation
- **Childhood** – missing permanent teeth
  - delayed eruption of permanent teeth,
  - disturbed root development (Akamine et al. 1985; Fukuyama et al. 1979; Rogan et al. 1988).
- **Adulthood** - periodontal disease was common (Hashiguchi et al. 2003).
Tobacco and Oral Diseases


It is well known that smoking contributes to the development of lung cancer and cardiovascular disease, and there is weighty evidence that it has a considerable influence on oral health.

**Negative effects on Oral Health**

- staining of teeth and dental restorations,
- reduction of the ability to smell and taste
- smoker’s palate
- **smoker’s melanosis**
- coated tongue
- oral candidosis
- dental caries
- **periodontal disease**
- implant failure
- **oral precancer and cancer.**
localized gingivitis

severe gingival inflammation overlying chronic periodontitis

moderate periodontitis

acute advanced periodontitis
• Smoking interferes with basic functions that fight disease and promote healing. Thus affects the way gum tissue responds to all types of treatment.

• The chemicals contained in tobacco interfere with the flow of blood to the gums that leads to a slowdown in the healing process. It makes the treatment results less predictable and often unfavorable.

• It is not just cigarette smoke that contributes to periodontal disease. All tobacco products can affect gum health. This includes pipe tobacco, smokeless tobacco and cigars. Labels on smokeless products such as chewing tobacco or snuff include warnings that the products can cause oral cancer, gum disease or tooth loss.

• A study conducted at Temple University showed this risk. Researchers reported that 18% of former cigar or pipe smokers had moderate to severe gum disease. This is three times the amount found in non-smokers.
• Half of periodontal (gum) disease in smokers is caused by smoking. Chronic (long-term) gum disease can lead to the loss of teeth.

• Studies have shown that smokers have more calculus (tartar) than non-smokers. This may be the result of a decreased flow of saliva. Calculus is the hardened form of plaque.

• Smoking tobacco products can make gum disease get worse faster. Smokers have more severe bone loss and more deep pockets between their teeth and gums than non-smokers.

• In studies, smokers were three to six times more likely to have gum destruction than non-smokers.

• Severe bone loss was five times greater among current or former heavy smokers than among people who never smoked.
Oral precancer (oral leukoplakia & oral submucous fibrosis) and cancer are complex multifactorial diseases arising from the interplay between the genetic components and the environmental determinants.

• Environmental exposures in farmers can be explained by solar (ultra violet) exposure. Climate change induced oxidative stress, dietary transition, contamination; impacts on food chain ecosystem and food security have increased the vulnerability to oral cancer.
Fig. 1. Squamous cell carcinoma of the mouth in a heavy smoker.

Fig. 2. Leukoplakia characterized by whitish changes, erythematous areas, and nodules in the right buccal commissure in a heavy smoker.

Fig. 3. White changes in right buccal commissure in a heavy smoker.

Fig. 4. Same lesion as in figure 3 after 3 months’ of tobacco abstinence.

Fig. 5. Smokers’ palate in pipe smoker.

Fig. 6. Smokers’ melanosis in the floor of the mouth in a heavy smoker.
Tobacco use is a leading cause of cancer and of death from cancer. People who use tobacco products or who are regularly around environmental tobacco smoke (also called second hand smoke) have an increased risk of cancer because tobacco products and second hand smoke have many chemicals that damage DNA.
Oncogenesis is often multifactorial, involving genetic and environmental factors.

In addition to infectious agents, there are a host of other influences from the environment that may be carcinogenic alone, or in combination with one or more predisposing factors.

Stress, physical and mental abuse, nutrition and diet, exposure to toxins, pathogens, radiation and chemical as environmental factors and determinants of growth and body composition can contribute to the risk of some human cancers such as oral cancer.
Oral Cancer

Tobacco's greatest threat to your health may be its link to oral cancer. The American Cancer Society reports that:

• About 90% of people with mouth cancer and some types of throat cancer have used tobacco. The risk of developing these cancers increases as people smoke or chew more often or for a longer time.
• Smokers are six times more likely than non-smokers to develop these cancers.
• Tobacco smoke from cigarettes, cigars or pipes can cause cancers anywhere in the mouth or the part of the throat just behind the mouth.
• It also can cause cancers of the larynx, lungs, esophagus, kidneys, bladder and several other organs. Pipe smoking also can cause cancer in the area of the lips that contacts the pipe stem.
• Smokeless tobacco has been linked to cancers of the cheek, gums and inner surface of the lips. Smokeless tobacco increases the risk of these cancers by nearly 50 times.
OVERVIEW OF CARCINOGENESIS

ENVIRONMENTAL AGENTS
that damage DNA
• Chemicals
• Radiation
• Viruses

INHERITED MUTATIONS IN GENES affecting:
• DNA repair
• Cell growth
• Apoptosis

DNA damage

Mutations in somatic cells

Activation of growth promoting oncogenes

Impaired apoptosis

Inactivation of tumor suppressor genes

Altered gene products (proteins); abnormal structural & regulatory proteins

Malignant tumor
a) Incidence of oral cavity cancer among males (age-standardized rate (ASR) per 100,000 world population), December 2004

Source: ref. 75.
Many environmental factors cluster in specific geographical regions.

For example, oral cancers are highly prevalent in regions such as India and Southeast Asia as a result of widespread betel nut and tobacco chewing practices.

Higher incidences of lung cancer occur where there is abundant indoor air pollution or radon, such as in India and China, as well as smoking predicted changes in the mortality of cancer by the year 2020.
Incidence of oral cavity cancer among females (age-standardized rate (ASR) per 100 000 world population), December 2004

Source: ref. 15.
Environmental Factors in Oral Health

GEOGRAPHY OF CANCER

1. North-east
   What Highest cancer rate, especially of oesophagus
   Why Tobacco, household burning of firewood

2. West Bengal
   What Lung, urinary bladder cancer
   Why Air and water pollution

3. South and coastal India
   What Leads in stomach cancer
   Why Diet rich in spice, salt

4. Goa
   What Leads in colon cancer
   Why Red meat, alcohol and tobacco

5. Gujarat and Rajasthan
   What Head and neck cancer
   Why Tobacco and pan masala

6. Punjab, Malwa belt
   What All cancers higher than average, especially kidney, urinary bladder, breast cancer
   Why Pollution, pesticide, toxins in food

7. Gangetic plain (UP, Bihar, West Bengal)
   What Gall bladder, head and neck cancer
   Why Polluted water, sediments in the river, diet rich in animal protein or fish

8. Madhya Pradesh
   What Oral cancers highest
   Why Tobacco and pan masala
• Biological Hazards Natural and anthropogenic environmental factors account for most of the diseases, and this is particularly relevant for cancer, because of the cell mediated genome affected nature of the disease, which in fact defeats the earliest level of cancer detection.

• Microbial causes for carcinogenesis is one of the important areas.

• Human papilloma viruses, hepatitis virus, and helicobacter pylori etc. are connected with various cancers.
Bioindicators

The rate of environmental pollution and human exposure deriving from the effects of dangerous toxic chemicals in the environment is usually difficult to assess.

One possible alternative method is the use of bio-indicators to demonstrate environmental pollution.

In recent years there has been an increased interest on the use of human bio-indicators such as teeth, bone, blood, nail and hair to monitor environment pollution with toxic heavy metals.

The content of trace elements in human teeth is a more suitable indicator to demonstrate environmental pollution.

• Therefore the determination of heavy metals content in teeth is understood to play an important role for monitoring the impact of environmental pollution.
Teeth as Indicators of Environmental Pollution with Lead

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Abstract

Environment pollution can be determined using physical and chemical methods and with bio-indicators. In recent years there has been an increased interest on the use of human bio-indicators such as teeth, bone, blood, nail and hair to monitor environment pollution with toxic heavy metals. Therefore the determination of heavy metals content in teeth is understood to play an important role for monitoring the impact of environmental pollution. The aim of this review of literature is to illustrate the current status of teeth used as indicators in environmental pollution with lead.

Keywords: Environment; Pollution; Lead; Heavy Metals; Bio-Indicator; Bio-Marker

Introduction

Human civilization and concomitant increase in industrial activity have gradually influenced in redistribution of many toxic metals and the lead is one of the most studied. Most published data on tooth lead have been based on whole tooth analysis, with no attempt to distinguish among tooth types as different teeth are formed at different ages or to differentiate the lead concentration in enamel from that in dentin [12].

Several studies in humans have demonstrated that lead level in teeth has been used as an index of accumulation of lead and environmental
• Teeth have an **advantage over bones as biopsy tissues**: they are easy to collect and are physically stable.

• There is evidence that teeth are superior to bone as an indicator of cumulative lead exposure because the losses from teeth are much slower as there is no turnover of apatite in teeth, as in bone, hence teeth are the most useful material for studying total past lead exposure.

• Human teeth, both deciduous and permanent, are useful indicators of lead exposure of **recent and historical populations**.

• The use of permanent teeth is limited because the extraction of healthy permanent teeth just for this purpose is hardly acceptable.

• Analysis of lead levels in deciduous teeth is a relatively simple and non-invasive method for determining a child’s lead burden at a very young age.

• Since the mid 1970’s, lead levels in deciduous teeth have been used as an exposure indicator in a number of studies.
A wide range of perceptible effects provide substantial evidence to conclude that the environment may have and effect on human health.

However, our knowledge is very far from exhaustive and for the sake of conciseness many hazards or their effects have not been mentioned.

These include the distinction between mere association, and causation, or the quantitative implications of understanding the difference between hazard and risk.

In epidemiological studies, using reliable biomarkers of dose and of early effects yields better assessments of exposure and outcome.
• In addition, markers of genetic susceptibility to environmental agents allow the identification of individuals that are at particularly high risk.

• While tobacco and other environmental toxins are the causes of cancer, all smokers or those exposed to environmental hazards do not get cancer, indicating the importance of genetic alterations that occur in the DNA.

• Alterations in the sequences of certain genes, which are inherited, are equally responsible for carcinogenesis.
There is a huge burden of oral diseases that afflict humankind which require population-wide prevention and access to appropriate care.

The many links between general and oral health, particularly in terms of shared risk factors and other determinants, provide the basis for closer integration of oral and general health for the benefit of overall human health and wellbeing.
In India, environmental pollution related disorders may further weaken the existent inadequate public health infrastructure.

Research should be targeted to understand the complex interplay of gene-environment interactions and oxidant mediated oral diseases.

Epidemiological assessment and willingness to tackle health burden arising due to impact of environmental factors needs to be strengthened.

This would facilitate a harmonious balance to enable sustainable development of optimum oral and in turn overall health.
Thanks a lot for a patient hearing...!!!