

Environmental Tobacco Smoke (ETS) General Information and Health Effects Fact Sheet



WHAT IS ENVIRONMENTAL TOBACCO SMOKE (ETS)?

Environmental tobacco smoke (ETS) refers to exposure to tobacco smoke – not from your smoking, but from being exposed to someone else’s cigarette, cigar, or pipe smoke. ETS can also be described as the material in indoor air that originates from tobacco smoke. Breathing in ETS is known as passive smoking, second-hand smoke, or involuntary smoking.

What information is covered in this document?

This document will cover the basic issues of what environmental tobacco smoke is and what are the health effects of passive smoking. Second-hand exposure to vapours from electronic cigarettes (e-cigarettes) will also be covered. Examples from workplace exposure situations are used wherever possible.

What is the general composition of tobacco smoke?

Tobacco smoke consists of solid particles and gases. More than 7,000 different chemicals have been identified in tobacco smoke. The number of these chemicals that are known to cause cancer in animals, humans, or both are reported to be about 70.

The solid particles make up about 10 percent of tobacco smoke and include “tar” and nicotine. The gases or vapours make up about 90 percent of tobacco smoke. The major gas present is carbon monoxide. Others include formaldehyde, acrolein, ammonia, nitrogen oxides, pyridine, hydrogen cyanide, vinyl chloride, N-nitrosodimethylamine, and acrylonitrile. Of these, formaldehyde, N-nitrosodimethylamine and vinyl chloride are suspected or known carcinogens in humans. Acrylonitrile has been shown to cause cancer in animals.

What is the general composition of electronic cigarette (e-cigarette) vapour?

The e-cigarette is the most widely used form of non-tobacco nicotine (NTN) which consists of a device containing a heating element that atomizes a solution containing water, nicotine, propylene glycol, vegetable glycerine, and usually some flavouring. The devices have evolved over time and those containing nicotine deliver nicotine more effectively. The puffing (vaping) technique and puffing regimen also affect the nicotine delivery to the user. Compared with traditional cigarettes, e-cigarettes emit lower levels of many of the chemicals found in tobacco smoke. The concentration of nicotine, propylene glycol, glycerine, and flavouring in solution (e-liquid) can vary significantly; particularly in relation to nicotine concentration. The e-cigarette solution may be nicotine-free.

In Canada, nicotine-containing e-cigarettes have not been approved for sale.

Similar to tobacco smoke, vapour generated by e-cigarettes consists of potentially harmful chemicals including volatile organic compounds, aldehydes, tobacco-specific nitrosamines (TSNAs) carbonyls, and metal particles, but at much lower levels than in cigarette smoke. Vaporisation at high temperatures can produce relatively higher levels of formaldehyde and other aldehydes. The long-term adverse effects for e-cigarette users and from passive exposure to e-cigarettes vapour are less well understood than long-term effects from ETS because e-cigarettes have only been in use since around 2003. Passive exposure varies according to device, e-liquid constituents, and vaping technique. One study found that nicotine from exhaled vapour can be deposited on surfaces but at levels so low that is unlikely that nicotine could enter the body at a dose capable of causing health effects.

What is meant by “mainstream” and “sidestream” smoke?

The smoke that is inhaled and then exhaled from the smoker’s lungs is called mainstream smoke (MS).

Sidestream smoke (SS) is the smoke that enters the air directly from the burning end of a cigarette, cigar, or pipe. The burning end of a cigarette is not usually hot enough for complete combustion of the tobacco to occur. Since some chemicals are favoured by this incomplete burning, undiluted sidestream smoke contains higher concentrations of several chemicals than the mainstream smoke inhaled by the smoker. These chemicals include 2-naphthylamine, N-nitrosodimethylamine, 4-aminobiphenyl, and carbon monoxide.

Environmental tobacco smoke (ETS) is composed of both mainstream and sidestream smoke. ETS is diluted by the air in the room before it is inhaled and is therefore less concentrated than mainstream or sidestream smoke.

Every person – both smokers and non-smokers – in a room with ETS will have similar exposure because nearly 85 percent of ETS in a room comes from sidestream smoke. The smoker is also exposed to mainstream smoke, but this exposure is limited to the time it takes to smoke a cigarette. However, exposure to ETS remains constant for the entire time spent in that room.

Vaping does not generate sidestream vapour between puffs although some mainstream vapour is emitted when an e-cigarette user exhales.

Can exposure to ETS be measured?

It is hard to measure the exposure of a passive smoker to environmental tobacco smoke. The exposure varies according to the type and number of cigarettes or other tobacco products burned, the number of smokers present, the rate and manner of smoking, the room volume, the room ventilation rate, and the percentage of fresh (or makeup) air supplied.

Exposure to ETS has been estimated in terms of “cigarette equivalents”. Cigarette equivalents can be measured by determining carboxyhemoglobin levels in blood. Carboxyhemoglobin is formed in the blood when someone inhales carbon monoxide. The hemoglobin in the blood that has oxygen bound to it is called oxyhemoglobin. It is the oxyhemoglobin that carries oxygen to the tissues. However, carbon monoxide has a much stronger attraction to hemoglobin than oxygen. Thus, inhaled carbon monoxide quickly replaces the oxygen in the oxyhemoglobin and binds to the hemoglobin to form carboxyhemoglobin which can be measured.

Some studies use a urine test that measures the amount of cotinine in the body. Most of the nicotine absorbed by the body is broken down (metabolized) rapidly to form cotinine as the major by-product (metabolite). Cotinine stays in the blood about 30 hours and reaches high concentrations in blood and urine.

Other studies can test for the level of nicotine in hair. Hair nicotine levels are a more accurate biomarker than urine cotinine.

Briefly, what would some “exposure equivalents” be?

Various studies suggest that passive exposure to ETS over an eight-hour day is comparable to directly smoking one to three cigarettes.

Such estimates depend greatly on the particular tobacco smoke contaminant chosen for the active/passive smoking comparisons. For example, it is thought that someone exposed to ETS will breathe in the same amount of the following contaminants as if they actively smoked one cigarette:

- same amount of carbon monoxide in one or two hours,
- same number of smoke particles in eleven hours,
- same amount of acrolein in seven hours, and
- same amounts of nicotine and hydrogen cyanide in fifty hours.

What should I know about the research into the health effects of ETS?

There is a consistent finding of a relative risk* greater than 1.0 for non-smokers who are exposed to ETS. This finding shows that there is an association between certain diseases and exposure to ETS. While no single study can say that there is a 100% chance of health problems as a result of exposure to ETS, an association between ETS and various health conditions is considered very likely because there is:

- the proven link between heart diseases and lung cancer to active smoking,
- the presence of several known carcinogens in environmental tobacco smoke, and
- the general acceptance that the risks of certain diseases are directly related to the amount of tobacco smoke inhaled.

*Briefly, a relative risk of 3.0 or greater would indicate a very positive association (what could be reasonably termed a “cause” of the disease) while a relative risk of 1.0 is considered neutral. In more general terms, a risk of 1.3 translates into about seven excess deaths in 1000 persons over a lifetime, or an extra 30% chance of developing the disease.

What are the health effects from exposure to ETS?

Lung Cancer

When evidence from various studies is combined, they indicate that exposure to ETS increases the number of lung cancers detected in non-smokers. Non-smoking co-workers of smokers have a relative risk of approximately 1.39.

Cancers Other than Lung Cancer

Traditionally, studies focused on finding the effects of ETS on the respiratory system. More recently, studies show that exposure to ETS may increase the risk of cancer at sites other than the lung. While there have been fewer studies conducted, associations have been found with cancers such as cervical, bladder, nasal-sinus, and brain.

In addition to the cancers mentioned for passive smokers, studies of active smokers have also recorded a risk of cancer to:

- the renal pelvis (part of the ureter that receives urine from the kidney),
- the adrenal glands (renal adenocarcinoma),
- parts of the mouth and throat such as the lip, oropharynx (the back of the mouth), larynx (voice box), and hypopharynx (area below the pharynx or throat),
- the esophagus (tube from the pharynx to the stomach),
- stomach, liver, bladder, and pancreatic cancers.

In addition, animal studies have seen cancers of the liver, pancreas, and aerodigestive tract (head and neck, esophagus, and lungs).

Heart Disease

It is known that active smoking is a cause of heart disease. Some studies have found an association between ETS exposure and an increase of heart disease among persons who never smoked. Among non-smokers exposed to ETS, there is an estimated 20 to 30% increase (relative risk of 1.2 to 1.3) in the risk of death from myocardial infarctions (heart attacks) or ischemic heart disease (group of diseases caused by inadequate oxygen supply to the heart caused by constricted blood vessels and resulting decreased blood supply).

In addition, it was found in experimental studies on the effects of ETS on the heart, that ETS exposure has damaging effects on blood platelets (needed for clotting) and the endothelium (tissues lining the heart, blood vessels, lymph vessels, etc.).

Effects on Persons with Pre-Existing Diseases

Non-smokers with heart disease (angina pectoris) exposed to ETS in ventilated and unventilated rooms had increased heart rates, elevated blood pressures and increased carbon monoxide in the blood.

ETS aggravates allergy symptoms. It is generally more irritating to the respiratory tract of asthmatics and it can aggravate some asthmatic symptoms such as wheezing.

Pregnant Workers (Effects on Fetus)

While only a limited number of studies have looked at the possibility that exposure to ETS in the workplace may have effects on the fetus, there is some indication that non-smoking women exposed to passive smoke are 2-4 times more likely to have a baby born with a low birth weight (less than 2,500 grams or 5.5 pounds – premature birth or small for their gestational age). Generally speaking, babies with low birth weight have a smaller chance of survival.

Other studies have listed effects such as congenital anomalies, longer body lengths, and smaller head circumferences. These babies were also found to have an increased risk of developing asthma and allergy related symptoms and behavioural problems in childhood.

Note: Regularos defines a congenital anomaly as “are abnormalities that are present at birth, even if not diagnosed until months or years later. They are usually structural in nature and can be present from the time of conception (e.g., Down syndrome), but largely occur in the embryonic period (up to the end of the seventh week of gestation e.g., spina bifida), or in the early fetal period (eight to sixteenth week).”

Irritant Effects

Many of the substances in cigarette smoke are very irritating to the eyes, throat and respiratory mucous membranes. A high proportion of non-smokers report eye irritation, headache, nasal discomfort, cough, sore throat, or sneezing when exposed to cigarette smoke. Eye irritation seems the main symptom during passive exposure to cigarette smoke.

Why is there still some debate about the extent of these health effects?

Sometimes finding answers in scientific studies is not clear cut. When studying ETS, it is often difficult to determine what exact level of smoke a person was exposed to as most studies rely on a person's memory about events that happened a long time ago. In these situations, small increases in risk are hard to detect.

However, ETS is considered to increase a person's risk of certain health effects for the following reasons:

- an elevated risk is seen when all of the studies of ETS are pooled together,
- the chemicals in ETS and in the mainstream smoke of an active smoker are very similar,
- it makes biological sense (biological plausibility) that these chemicals will cause the health effects observed, and
- the evidence that there is a dose-related risk of lung cancer in active smokers.

Does exposure to ETS have other health implications in the workplace?

Yes. While most of the studies have looked at the health effect of active smokers, it has been shown that tobacco smoke can interact with other materials and chemicals in the workplace.

Cigarette smoke can:

- transform existing chemicals into more harmful ones
- increase exposure to existing toxic chemicals
- add to the biological effects caused by certain chemicals, and
- interact synergistically with existing chemicals (the effects will be more than the sum of the effects from the exposure to each chemical or material alone). (Also known as multiplicative effects)

What are examples of smoke interacting with other occupational hazards?

Examples of these situations (as studied with active smokers) is as follows:

Occupation	Exposure	Smoking / Occupation Interaction +: Additive X: Multiplicative X?: Probably Multiplicative	Disease
Asbestos workers, construction workers and others in contact with asbestos	Asbestos	+ , X	Lung cancer Chronic lung disease
Aluminum smelter workers	Polynuclear aromatic hydrocarbons (PAHs)	+ or X	Bladder cancer
Aircraft and mining industry and many others	Noise	+	Loss of hearing and hearing acuity
Cement workers	Cement dust	+	Chronic bronchitis Obstructive lung disease
Chlorine manufacturing	Chlorine	+	Chronic obstructive lung disease
Coal miners	Coal dust	+	Chronic obstructive lung disease
Copper smelter workers	Sulphur dioxide	+	Chronic obstructive lung disease
	Arsenic	+ or X	Lung cancer
Grain workers	Grain dust	+	Chronic bronchitis Obstructive lung disease
Organic chemicals	Carcinogens	+ or X	Cancer of various organs and tissues
Rock cutters Foundry workers	Silica dust	+	Chronic obstructive lung disease
Textile workers	Cotton, hemp, flax, dust	X?	Acute airway obstruction (byssinosis) Chronic bronchitis

Uranium miners and many other workers in radioactive environments	Alpha radiation (Radon)	X?	Lung cancer
Welders	Irritant gases, metal fumes, dusts, (Radon)	+	Chronic bronchitis Obstructive lung disease

What is third-hand smoke?

Third hand smoke is cigarette smoke particles that become re-suspended in air after being on the furniture or clothes. For example, when smoking indoors, smoke particles can become adsorbed into indoor surfaces such as clothes, curtains, carpets or cushions and remain on these surfaces even after the room has been ventilated. One study found ETS exposure in women whose husbands smoke outside the house to be similar with those women whose husbands smoke inside the house. This finding implies that cigarette smoke that becomes adsorbed onto the clothing of smokers (third hand smoke) pose a similar risk to second hand smoke. Smoker's breath may also contribute to third hand smoke. A relatively high level of benzene can be detected in the breath of a smoker immediately after smoking. Currently, little is known about what chemicals contribute to third hand smoke, and whether third hand smoke poses a health risk and to what extent.

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