

# Passive Smoking and Oral Cancer Risk: A Case Report

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## ABSTRACT

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**Objective:** Defining association and perceived risks of Passive Smoking with oral cancer occurrence, the same being considered a risk factor of many cancer occurrences with its public health impacts.

The smoke exhaled and emitted by the tip of a burning cigarette is called second hand tobacco smoke (SHS) / environmental tobacco smoke (ETS) and the inhalation of this smoke is known as passive smoking. It is the inhalation of smoke from tobacco products used by others, from the side-stream and exhaled mainstream smoke, by persons other than the “active” smoker. It has been considered as a risk factor of many cancers

Concerns around second-hand smoke have played a central role in the debate over the harms and regulation of tobacco products, especially to children and women in pregnancy

**Keywords:** Second hand smoke, Third hand smoke, Tobacco industry, Epidemiology, Blue Ribbon campaign

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## PREAMBLE

The smoke exhaled and emitted by the tip of a burning cigarette is called second hand tobacco smoke (SHS) / environmental tobacco smoke (ETS) and the inhalation of this smoke is known as passive smoking. It is the inhalation of smoke from tobacco products used by others, from the side-stream and exhaled mainstream smoke, by persons other than the “active” smoker. It has been considered as a risk factor of many cancers - lungs, breast, oral cavity, gastrointestinal tract, renal, cervical cancers etc. Evidences show that at least 17 carcinogenic chemicals contained in tobacco smoke are emitted at higher levels in sidestream smoke than mainstream smoke (Mohtashamipur et al., 1990). Benzo (a) pyrenediol epoxide, one of the chemicals of tobacco smoke is found in both mainstream and sidestream smoke shows a direct aetiological association with lung cancer (Denissenko et al., 1996).

Tobacco in any form- smoking or smokeless is an important and established risk factor for pre-malignant lesions in the oral cavity and oral cancer. It could be associated with oral cancer in non smokers. The smoke stays in the air upto 2 ½ hours in enclosed places. Second hand smoke contains many chemicals which are irritants and toxins - formaldehyde irritates the eyes, nose and throat; hydrogen cyanide, carbon monoxide and ammonia weaken the natural cleaning mechanism that clear the airways of toxins; polycyclic

aromatic hydrocarbons and nitrosamines are DNA-damaging chemicals. Arsenic, Benzene, Cadmium and Tar are some of the other chemicals in tobacco smoke. A healthy person living with a smoker has a 30% increased risk of developing lung cancer than non smokers living with partners who are also non smokers.

## CAUSALITY

Exposure to second-hand tobacco smoke causes disease, disability, and death. The health risks of second-hand smoke are a matter of established scientific consensus. These risks have been a major motivation for smoke-free laws in indoor public places, like offices, restaurants, workplaces, as well as open public spaces. The most common site which has the highest relative risk for cancer due to smoking is the lungs. Next highest relative risk is for the larynx and the oral cavity. Close associates and family members of smokers are exposed to the environmental tobacco smoke. They become passive smokers and are subjected to many health hazards including oral cancers. The poisonous chemicals in tobacco smoke can damage the DNA, causing in cell nuclear aberrations facilitating neoplastic growths. Smoke is exhaled by the smoker and also released from the burning tip of the cigarette or *Beedies*, the latter accounted for 48% of Indian tobacco consumption in 2008. It is possible for a heavy smoker to live a cancer free life while a non smoker living with him could develop lung cancer as per research from the

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U.K. The same theory could be applied in the case of oral cancer also.

### Tobacco Industry Concerns

Concerns around second-hand smoke have played a central role in the debate over the harms and regulation of tobacco products, especially to children and women in pregnancy. Since the early 1970s, the tobacco industry has viewed public concern over second-hand smoke as a serious threat to its business interests, when it was perceived as a motivator for stricter regulation of tobacco products. Several well-established carcinogens have been shown by the tobacco companies' own research, to be present at higher concentrations in side stream smoke than in mainstream smoke. This fact has been known to the tobacco industry since the 1980s, which it had chosen to keep its findings secret. Despite the industry's awareness of these harms as early as the 1980s, the tobacco industry coordinated for sustained scientific controversy with the aim of forestalling regulation of their products, not to ignore the socio-economic fact that this industry issues employment to a large section of people giving them economic sustenance partly directly and mostly indirectly by way of its distribution network. Over 3 million Indians are employed in the manufacture of *beedis*, a cottage industry that is typically done by women in their homes.

'*Beedi*' smoking is the most common form of tobacco smoking in India and is predominantly a habit of men in South Asia. It is hand made with a small amount of tobacco wrapped in the leaf of another plant, - Tendupatta, (Coromandel Ebony or East Indian Ebony - *Diospyros melanoxylon* leaves are used for wrapping the tobacco and making "*beedis*" or Indian cigar. These leaves make excellent wrappers, and the success of the *beedi* is due, in part, to this leaf) Although *beedis* are smaller than cigarettes and contain much less tobacco, they deliver higher amounts of nicotine per gram of tobacco and comparable or greater amounts of tar as it has to be puffed in deeper and more often to keep it lighted. It can cause cancers of respiratory and digestive sites, including mouth, oropharynx, larynx, lung, esophagus, and stomach. In almost all such case-control studies a dose-response relationship was found.

### "Third-Hand Smoke"

This term is also recently coined to identify the residual tobacco smoke contamination that remains after the cigarette is extinguished and second-hand smoke has cleared from the air. Preliminary research suggests that by-products of third-hand smoke may pose a health

risk, though the magnitude of risk, if any, remains unknown. The WHO - Framework Convention on Tobacco Control (FCTC), states that "Parties recognize that scientific evidence has unequivocally established that exposure to tobacco smoke causes death, disease and disability.

### Tobacco Industry Corporate Denialism

A 2003 study by Enstrom and Kabat, activated by the tobacco industry and published in the *British Medical Journal*, argued that the harms of passive smoking had been overstated. Their analysis reported no statistically significant relationship between passive smoking and lung cancer. This paper was widely promoted by the tobacco industry as evidence that the harms of passive smoking were unproven and even over-enthusiastic. The *New Scientist* and the *European Journal of Public Health* have identified these industry coordinated activities as one of the earliest expressions of corporate denialism.

The American Cancer Society (ACS), whose database Enstrom and Kabat used to compile their data criticized the paper as "neither reliable nor independent", stating that scientists at the ACS had repeatedly pointed out serious flaws in Enstrom and Kabat's methodology prior to their publication. Notably, the study had failed to identify a comparison group of "unexposed" persons. In a US racketeering lawsuit against tobacco companies, the Enstrom and Kabat paper was cited by the US District Court as "a prime example of how nine tobacco companies engaged in criminal racketeering and fraud to hide the dangers of tobacco smoke." The Court found that the study had been funded and managed by the Center for Indoor Air Research, a tobacco industry front group tasked with "offsetting" damaging studies on passive smoking. When all the evidence, is assessed, the scientific conclusion is that ETS is a low-level lung carcinogen - WHO asserted. Measures to tackle second-hand smoke pose a serious economic threat to the tobacco industry, having broadened the definition of smoking beyond a personal addictive habituation to the larger extent of an undesirable public health concern.

## DISCUSSION

This is the case of a young married female who have presumably developed squamous cell carcinoma of the maxillary alveolus that could possibly be associated with six years of living with a spouse who is a heavy smoker. Incidence and prevalence of oral cancer in smokers is well documented. Oral cancer has known etiologic factors namely tobacco, betel nut, alcohol,

oral sepsis, ill-fitting dentures and premalignant lesions such as leukoplakia, erythroplakia, erosive lichen planus and oral submucous fibrosis. In this case none of these were present, suggesting passive smoking as the risk factor. Spouses of smokers are constantly exposed to the second hand smoke but not many cases of oral cancer are reported in persons who live with partners who smoke. Acute irritation in the upper and lower respiratory tract, worsening of the existing breathing problems and increase in the risk for lung cancer are reported in passive smokers.

## CASE REPORT

A 30 years old female undergoing treatment for oral cancer in the Regional Cancer Centre (RCC), Trivandrum, Kerala, reported for dental treatment at Noorul Islam Centre for Dental Sciences, Neyyattinkara, Thiruvananthapuram. She gave a history of toothache and difficulty in opening the mouth about a year ago. She reported to the local government hospital. The complaints did not subside with extraction of the maxillary third molar and supportive medications. She was referred to the RCC, on a clinical suspicion. C.T scan was suggestive of malignancy of the left maxillary alveolus with destruction of the inferior wall of the maxillary sinus. Sequential clinical protocols were followed and it proved to the suspicion. On the diagnosis of squamous cell carcinoma of the maxillary alveolus she underwent two cycles of pre-adjuvant chemotherapy followed by wide excision, partial maxillectomy and selective block dissection of neck lymph nodes. Plastic repair followed with split thickness skin grafting in the RCC. After recovery she was referred for further dental treatment.

A detailed history revealed that she is a thirty year old female with a healthy childhood and adolescence, with no smokers in her family. She stays with her husband, who smokes two packets – twenty cigarettes and many packets of *beedi* and chews *pan masala* daily. He is habituated to alcohol as well. Intraoral examination revealed healthy dentition with no dental caries or periodontal disease. Her oral hygiene was satisfactory. There was no evidence of any premalignant lesion, oral sepsis or any other established risk factor for oral cancer.

Oral prophylaxis was advised and done. Periodic reviews were recommend.

## REVIEW OF LITERATURE

In 1986, the International Agency for Research on



Figure 1. Oral lesions

Cancer (IARC ) Working Group found that there was sufficient evidence that active tobacco smoking was carcinogenic in humans, and concluded that tobacco smoking caused cancers not only of the lung, but also of the lower urinary tract including the renal pelvis and bladder; upper aero-digestive tract including oral cavity, pharynx, larynx, and esophagus; and pancreas. Reviewing the evidence accumulated on a worldwide basis, the IARC concluded in 2004 that “Involuntary smoking - exposure to secondhand or ‘environmental’ tobacco smoke - is carcinogenic to humans. Second-hand smoke causes many of the same diseases caused by direct smoking, like cardiovascular diseases, respiratory diseases, lung cancer and other types of cancer as well.

The association between passive smoking and lung cancer has been extensively studied. The assessment of the evidence was based on well-established principles for evidence and evaluation with application of criteria of causality. These principles included consideration

of lack of any bias or plausible confounding factors that could explain the observed associations, strength of association, dose–response relationships, biologic plausibility, and the consistency. It increases the risk of breast cancer in younger, primarily premenopausal women by 70%. The evidence is “suggestive,” but still insufficient to assert such a causal relationship of findings across investigations, study designs, and countries. When histologic data were available, the relative risk was increased more clearly for squamous-cell carcinoma of the nasal sinuses than for adenocarcinoma. The 2002 IARC Working Group, found that an increased risk of sinonasal cancer and nasopharynx cancer among cigarette smokers has been consistently reported in several case–control studies, with a positive dose–response trend associated with the amount and duration of smoking.

Many meta-analyses then showed that there is a statistically significant and consistent association between lung cancer risk and in other cancer risks in general as well, in spouses of smokers exposed to second-hand tobacco smoke. The excess risk is in the order of 20% for women and 30% for men and remains after controlling for some potential sources of bias and confounding. In 2004, the IARC and the World Health Organization (WHO), re-published that there was sufficient evidence that second-hand smoke caused cancer in humans. The overall risk depends on the effective dose received over time. The risk level is higher if non-smokers spend many hours in an environment where cigarette smoke is widespread, such as workplaces, or a residential care facility where other residents smoke freely.

**SHS Epidemiology**—Many epidemiological studies show that non-smokers exposed to second-hand smoke are at risk for many of the similar health problems associated with direct smoking. Most of the research has come from studies of nonsmokers whose partners were heavy smokers. Those conclusions are also backed up by further studies of workplace exposure to smoke. Side-stream smoke contains more than 4,000 chemicals, including 69 known carcinogens. Evidence also shows that inhaled side stream smoke, the main component of second-hand smoke, is about four times more toxic than mainstream smoke, as it is ‘unfiltered’ to that of the direct smoke, ‘compared’ to the tobacco industry hype of propagating that ‘filtered’ cigarettes takeaway much of the intended harm of smoking. The particles in the smoke that drifts from burning cigarette tips can be finer and more concentrated, meaning that they can be inhaled deeper into the lungs and stay longer in the body of the ‘second-hand smoker’, than in the person who is smoking. Obviously, the more

time people spend in close company with smokers, the more they are exposed to second hand tobacco smoke and the worse the threat to their health. Naturally, this often means those most at risk are the people, smokers care most about—their loved ones, spouse and children.

### **Epidemiological Evidence**

Considerable epidemiologic evidence of the carcinogenicity of tobacco smoke has become available since the review by IARC in 1986, suggested to conclude that tobacco is a potent multisite carcinogen with a substantial worldwide impact, causing cancers of the lung, upper aero- digestive tract (oral cavity, nasal cavity, nasal sinuses, pharynx, larynx, esophagus), pancreas, stomach, liver, lower urinary tract (renal pelvis and bladder), kidney, and uterine cervix, and causing myeloid leukemia. Both cigarette smoking and smoking other forms of tobacco, including *bidi*, pipe, and cigars, can cause cancers in multiple organs. There is high coherence for causality between the epidemiologic evidence and the mechanistic or biologic evidence involving measurements of carcinogenic metabolites of tobacco compounds, the formation of DNA or protein adducts, and the spectrum of gene mutations in cancers developed by smokers. Strength of evidence for an increased risk of cancer due to tobacco consumption in ETS is now held as sufficient for lung cancer and is possible for larynx and oropharynx.

### **Supportive Evidence – Mechanistic**

The causal nature of the associations reported above, and of those already recognized as causal in the 1986 IARC Monograph, is supported by mechanistic evidence. Developments in biochemistry and molecular biology have allowed researchers to measure metabolites of tobacco smoke in different body fluids and organs, to measure carcinogen– protein and carcinogen–DNA adducts, and to identify genetic damage (mutations or chromosome aberrations) related to smoking. These investigations have confirmed the multistage nature of tobacco carcinogenesis, which is already suggested by epidemiologic evidence. The high level of coherence between the results of mechanistic and epidemiologic studies adds strength to the causal interpretation of the associations between tobacco smoking and carcinogenesis repeatedly observed in the large body of epidemiologic evidence.

### **Public Health Concerns**

Measures that substantially prevent young individuals from starting smoking could avoid much of the future disease burden. Although 1 billion people worldwide



already smoke and more will start, individuals who stop smoking reduce their smoking-related cancer risks effectively. A balanced public health strategy is therefore needed that not only prevents young individuals from starting to smoke, but also helps adults to stop smoking. Though the beneficial effects of smoking cessation were first observed for lung cancer, evidence is now available that smoking cessation has similar effects of reducing risk for the other main tobacco-related cancers and for the main non-neoplastic diseases caused by smoking. Worldwide tobacco consumption has caused an estimated 100 million deaths in the last century and if the current trends continue it will kill 1,000 million deaths in the 21<sup>st</sup> century.

The Blue Ribbon Campaign: The World Health Organization (WHO) launched the Blue Ribbon Campaign on 26 March 2012 to mobilise support for 100 per cent smoke free indoor environments in the Western Pacific Region and combat harm from second hand smoke. WHO estimates that at least two people die each minute from a tobacco related disease in the Western Pacific Region and that more than 600,000 people die each year globally from exposure to second hand smoke. Of these deaths, it is estimated that 28 per cent are children.

## END NOTE

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