

The Exigency Of Third Eye For Third Hand Smoke Exposure In Cardiovascular System

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ABSTRACT

Environmental Smoke or Third hand smoke [THS] can affect heart and blood vessels that increase the risk of coronary heart diseases by 30%. Everyday dentists meet patients with tobacco smoking habit and some with precancerous lesions. Dentists are people who are most aware of the hazards caused by smoking. It is a need and responsibility of dental professionals to also be aware of various levels of smoke exposure and currently of Third Hand Smoke exposure. This article reviews on the nature and effects of THS, particularly on the cardiovascular system and ways to deal with THS exposure in our day to day lives.

Key Words: Second hand smoke, Third hand smoke, Hazards, Cardiovascular system

INTRODUCTION

More than a billion smokers exist around the world and about 12% of them are in India. Mainstream smoke [MSS] or First Hand Smoke is the primary level of smoke exposure, inhaled completely by the smoker. The exhaled smoke and smoke from the burning end of a cigarette (also called Side Stream Smoke or SSS) form the Second Hand Smoke. Third hand smoke [THS] is a relatively new term described by Jonathan Winickoff, a paediatrician at Dana-Farber/Harvard Cancer Centre, which refers to the tobacco smoke contamination that persists long after the cigarette has been extinguished. The authors emphasized on the potential hazards to children¹. The article stated that children are particularly susceptible to THS exposure because they breathe and play near contaminated surfaces. However there are no conclusive studies linking THS with cancer, except for one study conducted on mice exposed to third hand smoke causing lung cancer².

TYPES OF SMOKE EXPOSURES

A cigarette is basically a cylindrical object made of tobacco scraps, fillers, and glue and sprayed with nicotine that is wrapped in a paper and lighted at one end to produce smoke. After lighting the

cigarette, the smoke that is directly inhaled by the smoker through the opposite end is termed as 'First-Hand Smoke". The exhaled smoke and smoke released through the burning end is referred to as 'Second-Hand Smoke'. Third hand smoke is essentially second hand smoke that is left on surfaces and progressively becomes toxic over time.

THIRD HAND SMOKE

Third Hand Smoke or THS is the residual second hand smoke that remains in surrounding area for months¹. THS can cling to fabric from the clothes of the smoker, walls, carpets, furniture, hair, toys and vehicle surfaces. THS is a growing concern because unlike first and second hand smoke, THS affects unsuspecting common people, especially children.

Cigarette smoke contains a number of chemical components, particularly the notorious nicotine among many others. National Toxicology Program demonstrated the toxicity of low levels of cigarette constituents which included 250 poisonous gases, chemicals and metals, such as, hydrogen cyanide, carbon monoxide, ammonia, butane, arsenic, chromium, cadmium, polonium and toluene³. These compounds are well known carcinogens. A study conducted by the researchers of Lawrence Berkeley National Laboratory revealed Nicotine as a potential health hazard due to its ability to cling to surfaces and react with nitrous acid to produce harmful carcinogens⁴. It is exactly this ability of nicotine that forms the crux of THS. Nicotine reacts with ambient gases to form carcinogenic tobacco-specific nitrosamines or TSNAs, 4-(methylnitrosamino)-1-(3-pyridinyl)-4-butanal) or NNK, cotinine and formaldehyde. An experiment conducted on humans by exposing them to THS revealed changes in human nasal epithelium, THS induced cell survival responses, including up regulation of DNA repair genes, increased mitochondrial activity and inhibition of cell death⁵. Furthermore, THS components are suspended into air again and again over time, even after many days of the cessation of smoking activity¹.

The most dangerous part about THS exposure is that it affects non-smokers. Matt GE et al, in a study, concluded that dust and surfaces in homes of smokers are contaminated with environmental smoke or ETS. Not just smokers` homes but second hand homes of smokers, cars and even smokers` clothes retain harmful THS components that are inhaled, ingested or absorbed by innocent bystanders⁶.

DIFFERENCE BETWEEN FIRST HAND SMOKE AND SECOND HAND SMOKE

First hand and second hand tobacco smoking are the two greatest causes of many illnesses and even death. First hand smoke refers to the smoke inhaled directly by the smoker and causes cancer, heart disease, stroke, lung diseases, diabetes and chronic obstructive pulmonary disease. On the other hand second hand smoke is the inhalation of exhaled smoke or smoke from burning end of a cigarette by non-smokers. SHS can cause heart disease, lung cancer and stroke in adult. Children and babies, because of their small bodies are more vulnerable to SHS. Babies who breathe SHS can die unexpectedly of sudden infant death syndrome (SIDS). ⁷ Second hand smoke can affect an individual much similar to MSS but on the other hand THS persists long after burning a cigarette, for months the toxins get accumulated, react with ambient gases and reemit in a toxic cycle.

IMPACT ON CARDIOVASCULAR SYSTEM

Cigarette smoking is a major risk for coronary heart disease. Both active and passive smoking seem to increase the risk of acute coronary thrombosis and myocardial infarctions. The chemicals in smoke alter the haemostatic mechanisms by alteration of endothelial cells, platelets, fibrinogen and clotting factors. In a study on mice exposed to THS it was found to cause increased haemostasis and a consequential increase in blood clots. The researches at Western University of Health Sciences in Pomona, California concluded that similar reactions in humans could lead to acute coronary thrombosis, which can obstruct blood flow to heart and lead to a heart attack⁸.

EFFECT OF SMOKING: ATHEROTHROMBOTIC EVENTS

Atherosclerosis is the build-up of arterial wall plaques and it begins due to certain molecular changes in the innermost layer of arterial vessel wall called the tunica intima. The intima is lined with endothelial cells . Endothelial injury promote deposition of lipoproteins into the vessel wall. Monocytes infiltrate the site and endocytosis of lymphocytes occur leading to the formation of foam cells. Inflammatory mediators and smooth muscle cells migrate into the intima to promote plaque formation. Subsequently blood clot is formed which occludes the vessel leading to thrombosis⁹.

This process of thrombosis is influenced by nicotine and other toxins present in cigarette smoke. Several studies¹⁰ show that cigarette smoke increased vascular smooth muscle proliferation and migration due to the activation of the platelet-derived growth factor-protein kinase C signalling cascade. This causes hypercoagulability of blood and hemostasis¹¹.

Nitrous oxide production as a result of endothelial dysfunction decreases the flow mediated dilation [FMD]. This was shown in a study where the participants were exposed to THS⁹.

HYPERTENSION

Nicotine can influence the release of catecholamines by stimulation of the sympathetic nervous system via nicotine acetylcholine receptors present on peripheral postganglionic sympathetic nerve endings and the adrenal medulla¹².

Furthermore, nicotine acts on nicotinic cholinergic receptors present on endothelial cells to cause hypertension. The nicotinic acetylcholine receptors [nAChRs] are arranged in a barrel-like configuration to form a channel in the cell membrane. On activation of nAChRs the permeability to cations is increased leading to hypertension¹³.

OTHER POTENTIAL HAZARDS

MOLECULAR LEVEL

Genotoxicity is the ability of chemicals to produce damage to genetic information in a cell causing mutations and eventually cancer. THS and its residual components are such chemicals. Hang B et al conducted an experiment which used two different assays to evaluate the in vitro genotoxic nature of THS and its component NNA. Both the assays revealed that THS causes significant levels of DNA damage in human cells. It suggested that THS exposure is related to increased oxidative stress and could be a contributing factor in THS mediated toxicity².

DISEASES IN ADULTS

CANCER

Cigarette smoking is a common cause of cancer. It can cause cancer of mouth, throat, oesophagus, stomach, colon, rectum, liver, pancreas, larynx, trachea, bronchus, kidney, urinary bladder and acute myeloid leukaemia. Second hand smoke also causes lung cancer. Researchers have identified THS to increase lung cancer risk in mice. Antoine Snijders, Jian-Hua Mao and Bo Hang of Berkeley Lab reported in 2017 that brief exposure to THS is associated with low body weight and immune changes in juvenile mice².

These studies indicated that THS exposure induced molecular level damage, breakage of DNA double strands, increased cell proliferation and colony formation.

FATTY LIVER DISEASE

Non-alcoholic fatty liver disease [NAFLD] is one of the most common chronic liver diseases. Tobacco smoking is significantly associated with causation of NAFLD¹⁴. Animal studies have shown that THS stimulated the accumulation of fat in liver cells which caused non-alcoholic fatty liver disease¹⁵. NAFLD is known to worsen into cirrhosis and liver cancer.

POOR WOUND HEALING

Smoking in general is associated with interference in wound elasticity and poor wound healing which was also found to be true in the case of THS exposure. THS interferes with scar tissue development and wound contraction¹⁴.

INSULIN RESISTANCE

Smoking and insulin resistance have a dose-dependent association¹⁶. Nicotine may be the potential cause; it indirectly causes insulin resistance mainly via hormone activation. Studies in mice have found that there is oxidative damage to insulin receptors caused by THS leading to reduction of insulin receptors on pancreatic cells and insulin resistance. As a result there is risk of diabetes in prediabetic or non-diabetic individuals¹⁵.

PULMONARY FIBROSIS

Pulmonary fibrosis [PF] is a chronic illness affecting the respiratory system, is characterized by thickening of lung tissue and scar tissue formation. Smoking is a main risk for PF. Animal studies suggest that THS affects collagen production in bronchioles leading to scar tissue formation¹⁵. This leads to complications such as asthma, chronic obstructive pulmonary disease or cystic fibrosis¹⁷.

PREVENTION AND MANAGEMENT

Residual smoke is hard to manage as it is immune to traditional cleaning methods. Prevention is the best cure even in this situation.

• Banning cigarette smoking in public places is an effective method along with an additional waiting period of 10 minutes before entering indoor to prevent THS exposure³.

- Opening windows do not offer sufficient protection against second and third hand smoke exposure, hence it is vital for smokers to move far away from children when they are smoking.
- Smokers must consistently get rid of their clothes that they used during smoking. People who have quit smoking must replace the rags and furniture at their houses, repaint the walls of their homes¹⁸.
- Education of public is another effective method to prevent THS exposure.
- Last but not the least is 'quitting the habit'. Adults who smoke have to be counselled and given enough motivation to quit smoking. Nicotine replacement therapies can be used to aid the process³.

CONCLUSION

It is very difficult to bring this review to a conclusion as THS is a phenomenon that is not completely understood and requires a lot of research. It is established that THS accumulates in households, cars and even on clothes. It can stay in surrounding for months and slowly remits into atmosphere producing harmful carcinogens. It affects particularly non-smokers and children. The influence seen on the cardiovascular system is dangerous and potentially life threatening. Preventive modalities are very little and have scope for newer concepts and innovations. Many studies have been made showing molecular effects of THS compounds and potential for causing cancer however there is scope for further research in this topic.

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