Knowledge and Attitude of Bangladeshi People Regarding Passive Smoking

A Dissertation submitted to the Department of Pharmacy, East West University, Bangladesh, in partial fulfillment of the requirements for the Degree of Bachelor of Pharmacy

Submitted by

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Declaration by the Research Candidate

I, Md. Faysal, ID: 2012-1-70-042, hereby declare that the dissertation entitled "Knowledge and Attitude of Bangladeshi People Regarding Passive Smoking" submitted by me to the Department of Pharmacy, East West University and in the partial fulfillment of the requirement for the award of the degree Bachelor of Pharmacy, under the supervision and guidance of Nishat Nasrin, Senior Lecturer, Department of Pharmacy, East West University, Dhaka.

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Certificate by the Supervisors

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Dedication

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Table of Content

List of Table

List of Figure

List of Abbreviation

nAChRs-Nicotinic Acetylcholine Receptors

CNS- Central Nervous System

VTA- Ventral Tegmental Area

NAc- Nucleus Accumbens

PFC- Prefrontal Cortex

GABA- Gamma-Aminobutyric acid

GERD- Gastro Esophageal Reflux Disorder

PUD - Peptic Ulcer Disease

PAH- Polycyclic Aromatic Hydrocarbons

NF-kB - Nuclear Factor Kappa B

ARb1 - Androgen Receptor Beta 1

CHRNA- cholinergic receptor nicotinic alpha

Abstract

Passive smoking is injurious to health and it causes severe problems, even death. People are exposed to passive smoking in their daily life in different places. The aim of this study was to find out the knowledge level and attitude of general people regarding passive smoking. This was a survey based study conducted with 709 people, who are from both rural and urban area. This study was conducted on both smokers and non-smokers. People above the age of 18 were included as the study population. Most of the people (71%) had a good knowledge about passive smoking. Most (48.24%) people believed that it causes severe problems on the other hand a minor (5.92%) number of people believed that it had no effect on the health. A huge number (96.47%) of people were exposed to passive smoking and in most cases (84.36%) there were no restriction on those areas. More over 87% people supported the ban of smoking in public places. Most (86%) people felt the need of awareness program to make people aware of the consequences. Most (94.08%) of the respondents knew about passive smoking in this study. It is not possible to establish a general theory which will be accepted undoubtedly, because the respondent numbers were very low due to the time limitation. So there are chances of doing further studies on this topic.

Key Words

Passive Smoking, Knowledge of Passive Smoking, Attitude Towards Passive Smoking, Exposure, Restriction, Law Enforcement.

Chapter One
Introduction

1.1 Overview

More than 1 billion people smoke around the world. Tobacco kills around 6 million people each year. More than 5 million of those deaths are the result of direct tobacco use while more than 600 000 are the result of non-smokers being exposed to passive smoking (WHO, 2015).

Smoking is a process of inhaling smoke from a device and then exhaling it into the environment. The person who is smoking directly from the device is an active smoker and the person who is inhaling the smoke indirectly is a passive smoker (Cancer Institute NSW, 2015).

Active smoking is dangerous for health so as the passive smoking. Tobacco is the compound that is mainly smoked all over the world. The active ingredient in the tobacco is nicotine which is responsible for the immediate effects that a smoker gets after smoking and in the long run it creates dangerous diseases (Encyclopedia Britannica, 2014).

Nicotine acts on the brain rapidly within 10 seconds and produce stimulation or sedation depending on the amount taken. It stimulates the adrenal gland and increase secretion of dopamine that affects the mood. Tobacco smoking can produce bronchospasm, destroy cilia that fails the system to trap toxins from entering into the lungs. These toxins produce different kind of bronchial diseases. Nicotine constrict the blood vessel, increases low density lipoprotein, increase thrombin level, decrease coronary artery elasticity all these jointly can produce cardiovascular disease. Smoking disturbs the gastric balance and produce gastric acid reflux, heart burn. It decreases the mucus secretion and that make the stomach susceptible to gastric acid which can result in ulceration. Smoking weakens the immune system of the body that allows many hidden pathogens to show their activity. It decreases the absorption of many essential nutrients trough intestinal wall. It can also interact with the drug metabolism and thus activity.

Each puff of a cigarette contains a mixture of thousands of compounds, including more than 60 well-established carcinogens. Long term exposure to tobacco smoke can produce diseases. Smoke damage the cell lining of epithelium in the lung and produce lung cancer, it also produce oral cancer, stain teeth, erectile dysfunction and abnormal sperm, premature birth, hearing loss and many other diseases (ACS, 2015).

1.2 Epidemiology

There are 7.2 billion people all over the world (Schlesinger, 2014) and more than 1 billion people smoke (WHO, 2015).

According to the recent study conducted on Bangladeshi people, 46.4% male adults smoke and 1.96% of female adults smoke tobacco (Trading economics, 2015). Available evidence suggests high rates of any tobacco use, particularly among men (men in rural area – 52%, men in urban area 41%; women in rural area – 29%, women in urban area – 17%) (Bleich, *etal*., 2011).

Tobacco consumption alone accounts for nearly 5.4 million deaths per year and one billion people may die in this century if global tobacco consumption remained at the current levels. An international treaty spearheaded by WHO in 2003 and signed by 170 countries, aims to encourage governments to reduce the production, sales, distribution advertisement and promotion of tobacco products (WHO, 2015).

On average, 435,000 people in the United States die prematurely from smoking-related diseases each year; overall, smoking causes 1 in 5 deaths. The chance that a lifelong smoker will die prematurely from a complication of smoking is approximately 50%. Currently, about 45 million Americans smoke tobacco. Seventy percent of smokers say they would like to quit, and every year, 40% do quit for at least 1 day. Some highly addicted smokers make serious attempts to quit but are able to stop only for a few hours. Moreover, the 80% who attempt to quit on their own return to smoking within a month, and each year, only 3% of smokers quit successfully. Unfortunately, the rate at which persons, primarily children and adolescents become daily smokers nearly matches the quit rate, so the prevalence of cigarette smoking has declined only very slowly in recent years (Schwartz and Benowitz, 2010).

1.3 Smoking

Smoking is the inhalation of the smoke of burning tobacco and exhalation of the smoke in the environment. Tobacco is mainly burned and the smoke is inhaled through a device (American Cancer Society, 2015).

1.4 Categories of Smoking

Smoking can be divided into two categories, active smoking and passive smoking. Active smoking is inhalation of smoke directly from cigarette, pipe or cigar. Passive smoking is when smoke is breathed in by someone other than the active smoker. It is also called second-hand smoking. Second-hand smoke is sometimes referred to as environmental tobacco smoke (Cancer Institute NSW, 2015).

1.5 Passive Smoking

Passive smoking is the act of breathing in tobacco smoke produced by the others. Any person around an active smoker is a passive smoker. Most of the time passive smokers do not even notice that they are smoking passively (American Cancer Society, 2015).

1.5.1 Categories of Passive Smoke

A Passive smoker breath in two types of smoke, the mainstream smoke and the side stream smoke. The mainstream smoke is the smoke that is inhaled directly by a smoker from the tobacco smoking device and exhaled in the environment. The side stream smoke is the smoke released from the lighted end of the cigarette, pipe or cigar (American Cancer Society, 2015).

1.5.2 Passive Smoking is more dangerous than Active Smoking

Passive smoking is actually more dangerous than active smoking because a passive smoker smokes the total smoke. Total smoke means the combination of mainstream and side stream smoke. The general concept is that active smoking is more dangerous because a person directly smoking the tobacco. In reality passive smoking could cause exactly the same complications and diseases that active smoking cause. Side stream smoke has higher concentrations of cancer-causing agents and is more toxic than mainstream smoke. It has smaller particles than mainstream smoke. These smaller particles make their way into the lungs and the body's cells more easily. There are more than 4000 chemicals in tobacco smoke, of which at least 250 are known to be harmful and more than 50 are known to cause cancer. There is no safe level of exposure to second-hand tobacco smoke.

Effects of passive smoking are almost same as the active smoking. It can create many complications in short exposure and in long time exposure it could produce cancer. It can inspire a non-smoking person to smoke. Passive smoking can affect the brain, the respiratory system, the cardiovascular system, the ocular system, the renal system, the reproductive system, the gastrointestinal system, the metabolic system etc. in long term exposure it could create lung cancer, oral cancer, gastrointestinal cancer, breast cancer etc. (ACS, 2015).

1.6 Devices of Smoking

1.6.1 Roll-Your Own Cigarettes

Roll-your-own cigarettes are hand-filled cigarettes made from loose tobacco and rolling papers. RYO cigarettes can be hand-rolled by the user or made with a hand-held rolling machine. A common misconception is that RYO cigarettes are more natural and therefore "safer" than manufactured cigarettes; however, both contain the same ingredients. Additionally, in all combustible tobacco products, it is the actual burning of the tobacco that produces many of the toxic chemical components in tobacco smoke (WHO, 2015).

1.6.2 Cigars

Cigars consist of tightly rolled dried and fermented tobaccos wrapped in tobacco leaf. The user draws the smoke into his or her mouth but typically does not inhale it. However, cigar smokers who also smoke cigarettes or are ex-smokers of cigarettes are significantly more likely to inhale the smoke than are users of cigars only. Cigars come in a variety of shapes and sizes, and they can also be "reverse smoked," which means that the ignited end of the cigar is placed inside the mouth. Cigars have regained some popularity with both men and women in some parts of the world. In the United States, cigar smoking among women increased fivefold in a six-year period in the 1990s (WHO, 2015).

1.6.3Pipes and Water Pipes

Pipes are made of a variety of substances, including wood, briar, slate, and clay. Tobacco is placed in the bowl of the pipe, and the smoke is inhaled through the stem. Clay pipes are used throughout South-East Asia. The water pipe (also known as narghile, shisha, hookah, or hubble-bubble) is widely used to smoke tobacco in the Middle East, Northern Africa, and some parts of Asia, and it

has gained popularity in some Western countries. In some regions, use of the water pipe is more prevalent than use of cigarettes, and in some Arab countries, there is less stigma associated with women's use of the water pipe than with cigarette smoking (WHO, 2015).

1.6.4 Bidis

Bidis are thin, hand-rolled, filterless cigarettes consisting of flavoured or unflavoured tobacco wrapped in a tendu or temburni leaf. They may be tied with a coloured string at either end, and they come in a wide variety of flavours (e.g. vanilla, strawberry, mango). Bidis may be perceived as less harmful or more natural than conventional cigarettes; however, bidi smoke contains higher concentrations of nicotine, tar, and carbon monoxide than conventional cigarettes sold in the United States. Tar and carbon monoxide levels of bidi smoke can be higher than those of manufactured cigarettes because the user needs to puff harder to keep a bidi lit. Bidis are India's most used type of tobacco. Jha and colleagues examined prevalence data from India and Sri Lanka and estimate that about half of the male smokers and roughly 80% of the female smokers smoke bidis (WHO, 2015).

1.7 Nicotine

Nicotine is the main ingredient released from tobacco. The chemical formula is $C_{10}H_{14}N_2$It is the principal alkaloid of tobacco. Nicotine occurs throughout the tobacco plant and especially in the leaves. The compound constitutes about 5% of the plant by weight (Encyclopedia Britannica, 2014).

Nicotine was first extracted from tobacco by German physicians Wilhelm Heinrich Posselt and Karl Ludwig Reimann. Nicotine, a strong alkaloid, in its pure form is a clear liquid with a characteristic odor. It turns brown on exposure to air. It is water soluble and separates preferentially from organic solvents. It is an amine composed of pyridine and pyrrolidine rings. Nicotine is a dibasic compound and the availability and absorption in human body depends upon the pH of the solution. The absorption can occur through oral mucosa, lungs, skin or gut. The increase in pH of a solution causes an increase in concentrations of uncharged lipophilic nicotine, in this form it can actively pass through all biological membranes (Chaturvedi *et al.*, 2015).

Use of nicotine sustains tobacco addiction, which in turn causes devastating health problems, including heart disease, lung disease, and cancer, and increased susceptibility to a variety of infectious diseases. Smoking harms almost every organ of the body.

1.7.1 Mechanism of Action

Nicotine acts via 3 major mechanisms, producing physiological and pathological effects on a variety of organ systems.

1. Ganglionic transmission.
2. Nicotinic acetylcholine receptors (nAChRs) on chromaffin cells via catecholamines.
3. Central nervous system (CNS) stimulation of nAChRs.

Brain imaging studies demonstrate that nicotine acutely increases activity in the prefrontal cortex and visual systems. There is release of a variety of neurotransmitters important in drug-induced reward. Nicotine also causes an increased oxidative stress and neuronal apoptosis, DNA damage, reactive oxygen species and lipid peroxide increase. nAChRs were originally thought to be limited to neuronal cells, however, studies have identified functional nAChRs in tissues outside the nervous system. Actions on nicotinic receptors produce a wide variety of acute and long-term effects on organ systems, cell multiplication and apoptosis, throughout the body (Hammond, 2008; Committee of Smoking Cessation in Military and Veteran populations, 2009).

1.7.2 Nicotine and Dependency

Although most of the toxicity of smoking is related to other components of cigarette smoke, it is primarily the pharmacologic effects of nicotine that produce the addiction to tobacco. Quitting smoking at any age leads to significant reductions in the risks associated with it, and the vast majority of smokers throughout the world indicate an interest in quitting. Despite these facts, however, approximately 80% of smokers who attempt to quit on their own relapse within the first month of abstinence, and only approximately 3% remain abstinent at six months. This illustrates the powerful force of tobacco addiction and the chronic nature of the disorder (Benowitz and Neal, 2009).

Nicotine dependence is characterized by three phases:

Phase 1 (Acquisition and maintenance of nicotine-taking behavior): The administration of nicotine through tobacco smoking produces a mild pleasurable rush, mild euphoria, increased arousal, decreased fatigue, and relaxation (Henningfield *et al*., 1985). These reinforcing effects play an important role in the initiation and maintenance of tobacco smoking (Committee on Reducing Tobacco, 2007).

Phase 2 (Withdrawal symptoms upon cessation of nicotine intake): Chronic nicotine use induces neuroadaptations in the brain's reward system that result in the development of nicotine dependence. Thus, nicotine-dependent smokers must continue nicotine intake to avoid distressing somatic and affective withdrawal symptoms. Newly abstinent smokers experience symptoms such as depressed mood, anxiety, irritability, difficulty concentrating, craving, bradycardia, insomnia, gastrointestinal discomfort, and weight gain (Progress in Respiratory Research, 2015).

Phase 3 (Vulnerability to relapse): Abstinent smokers remain prone to relapse for weeks, months, or even years after cessation of tobacco smoking. Resumption of smoking, like relapse to other drugs of abuse, often occurs upon exposure to people, places, objects, or other stimuli that individuals have learned to associate with the positive rewarding effects of the drug. Stress and cigarette smoking itself can also precipitate resumption of habitual smoking (Progress in Respiratory Research, 2015).

1.7.3 The Role of Nicotinic Receptors

Nicotine influences mood, cognition, and body function by binding to and activating nicotinic acetylcholine receptors (nAChRs) located on neurons in the brain. When activated by either nicotine or the endogenous neurotransmitter acetylcholine, the nAChR opens a channel that allows ions to pass through the neuron's membrane from the exterior to the interior of the cell and trigger changes that activate the cell.

Nicotine produces rewarding effects by interacting with nAChRs on neurons in the brain's mesolimbic reward system. This system comprises dopaminergic neurons that originate in the ventral tegmental area (VTA) and release the neurotransmitter dopamine in regions involved in information processing, memory, and emotions, such as the nucleus accumbens (NAc),

hippocampus, amygdala, and prefrontal cortex (PFC). Increases in dopamine levels within the mesolimbic system give rise to rewarding effects. Nicotine directly enhances dopamine levels in the mesolimbic system by interacting with nAChRs on the dopaminergic neurons and causing them to release more of the neurotransmitter (Balfour, 2009; Barrett *et al.*, 2004).

Nicotine also modulates dopamine release indirectly by binding to nAChRs located on excitatory glutamatergic and inhibitory gamma aminobutyricacid neurons in the ventral tegmental area. These glutamatergic and GABAergic neurons originate from a number of brain areas, such as the NAc, hippocampus, PFC, amygdala, ventral pallidum, and pedunculopontine tegmental nucleus, and regulate the activity of dopaminergic neurons (Koob and Volkow, 2010).

1.8Effect of Smoking

Smoking kills a person in the long run but it has some acute effects too. There are so many effects that a person experience immediately after inhaling tobacco smoke. The main ingredient a smoker gets from tobacco is nicotine which is inhaled into the lungs and most of it stays. The rest passes into the blood stream, reaching the brain and throughout the body.

1.8.1 Immediate Effects

Nicotine on direct application in humans causes irritation and burning sensation in the mouth and throat, increased salivation, nausea, abdominal pain, vomiting and diarrhea. Gastrointestinal effects are less severe but can occur even after cutaneous and respiratory exposure. Pulse rate and blood pressure is increased. Nicotine also causes an increase in plasma free fatty acids, hyperglycemia, and an increase in the level of catecholamines in the blood. There is reduced coronary blood flow but an increased skeletal muscle blood flow. The increased rate of respiration causes hypothermia, a hypercoagulable state, decreases skin temperature, and increases the blood viscosity. Nicotine is one of the most toxic of all poisons and has a rapid onset of action. Apart from local actions, the target organs are the peripheral and central nervous systems. In severe poisoning, there are tremors, prostration, cyanosis, dypnoea, convulsion, progression to collapse and coma. Even death may occur from paralysis of respiratory muscles and/or central respiratory failure with a LD50 in adults of around 30-60 mg of nicotine. In children the LD50 is around 10 mg (Mishra *et al*., 2015).

1.8.2 Acute and Chronic Effects of Smoking on Different Organs and Systems

1.8.2.1 The Brain

Nicotine is one of more than 4,000 chemicals found in the smoke from tobacco products, it is the primary component that acts on the brain. Smokeless tobacco products also contain many toxins as well as high levels of nicotine. It has a number of complex and sometimes unpredictable effects on the brain and the body. Nicotine is absorbed through the skin and mucosal lining of the nose and mouth or in the lungs. Nicotine can reach peak levels in the bloodstream and brain rapidly, depending on how it is taken. Inhalation of nicotine in the form of smoke provides the quickest delivery with nicotine reaching the brain in approximately 7 seconds (WHO, 2015).

Brain imaging studies demonstrate that nicotine acutely increases activity in the prefrontal cortex, thalamus, and visual system, consistent with activation of corticobasal ganglia-thalamic brain circuits. Stimulation of central nAChRs by nicotine results in the release of a variety of neurotransmitters in the brain, most importantly dopamine. Nicotine causes the release of dopamine in the mesolimbic area, the corpus striatum, and the frontal cortex. Of particular importance are the dopaminergic neurons in the ventral tegmental area of the midbrain, and the release of dopamine in the shell of the nucleus accumbens, as this pathway appears to be critical in drug-induced reward. Other neurotransmitters, including norepinephrine, acetylcholine, serotonin, γ-aminobutyric acid (GABA), glutamate, and endorphins, are released as well, mediating various behaviors of nicotine (Sharma and Brody, 2009).

Most of the nicotine-mediated release of neurotransmitters occurs via modulation by presynaptic nAChRs, although direct release of neurotransmitters also occurs. Dopamine release is facilitated by nicotine-mediated augmentation of glutamate release and, with long-term treatment, by inhibition of GABA release. In addition to direct and indirect stimulation of neurotransmitter release, chronic cigarette smoking (but not nicotine administration) reduces brain monoamine oxidase A and B (MAOA and MAOB) activity, which would be expected to increase monoaminergic neurotransmitter levels such as dopamine and norepinephrine in synapses, thus augmenting the effects of nicotine and contributing to addiction (Jiloha, 2010).

- **Dopamine**

Dopamine has been strongly implicated in the reinforcing and withdrawal effects of nicotine. The key evidence includes experiments in laboratory animals that show:

- Administering nicotine increases dopamine transmission within the mesolimbic reward system and
- Administering compounds that block dopamine binding to its receptors (D1, D2, D3, D4, and D5 receptors) decreases the reinforcing effects of nicotine (D'Souza and Markou, 2011).

- **Gamma-Aminobutyric Acid**

In nicotine-naïve animals, acute nicotine exposure increases GABA release by activating excitatory 4 2-containing nAChRs that are located on GABAergic neurons in the VTA. Thus initially, nicotine-induced GABA release limits the rewarding effects of nicotine. By contrast, chronic nicotine exposure desensitizes 4 2-containing nAChRs on GABAergic receptors (Mansvelder and McGehee, 2002). Hypothetically, this desensitization will decrease nicotine-induced GABA release, leading to decreased inhibition of VTA dopaminergic neurons and increased dopamine release in the nucleus accumbens (NAc) and so facilitate the reinforcing effects of nicotine (D'Souza and Markou, 2011).

- **Glutamate**

Glutamate, the brain's primary excitatory neurotransmitter, also plays a critical role in the development of nicotine dependence (Liechti and Markou, 2008). Nicotine increases the release of glutamate by binding to excitatory α7-containing nAChRs located on presynaptic terminals of glutamatergic neurons in the VTA, NAc, amygdala, hippocampus, and PFC (Mansvelder and McGehee, 2002). The released glutamate binds to ionotropic and metabotropic glutamate receptors located on neurons in these areas. Glutamate released into the VTA after nicotine administration binds to glutamate receptors on dopaminergic neurons. The resulting increased firing of VTA dopaminergic neurons leads to dopamine release in the NAc and, consequently, nicotine reward (D'Souza and Markou 2011).

1.8.2.2 The Respiratory System

The effects of nicotine on respiratory system are twofold. First one is directly by a local exposure of lungs to nicotine through smoking or inhaled nicotine, and second via a central nervous system mechanism. Nicotine plays a role in the development of emphysema in smokers, by decreasing elastin in the lung parenchyma and increasing the alveolar volume (Mishra *et al.*, 2015).

Nicotine stimulates vagal reflex and parasympathetic ganglia and causes an increased airway resistance by causing bronchoconstriction. Nicotine alters respiration through its effects on the CNS. The simultaneous effect of bronchoconstriction and apnea increases the tracheal tension and causes several respiratory disorders. Smoke produced from the burning tobacco irritates the airways of the lungs that tighten it to produce bronchospasm. Bronchospasm makes airways smaller and leads to wheezing similar to that experienced by someone with asthma during an asthma attack. While smokers may not have asthma, they are susceptible to this type of reaction to tobacco smoke. An asthmatic that starts smoking can severely worsen the condition. Bronchospasm makes breathing more difficult, as the body tries to get more air into irritated lungs (Mishra *et al.*, 2015).

The lungs have finger like projection called cilia that combining with the mucas, clear the toxic chemical and dirt from the lungs. Tobacco smoke paralyzes the cilia, allowing mucus to collect in the lungs of the smoker. Cigarette smoke also promotes goblet cell growth resulting in an increase in mucus. More mucus is made with each breath of irritating tobacco and the smoker cannot easily clear the increased mucus. A smoker will likely have a persistent, annoying cough from the time they start smoking. A smoker who is not coughing is probably not doing an effective job of clearing the lungs of the harmful irritants found in tobacco smoke. The combination of bronchospasm and increased phlegm production result in airway obstruction and decreased lung function, leading to poor physical performance. In addition, smoking has been shown to stunt lung development in adolescents, limiting adult breathing capacity. Smoking not only limits one’s current state of fitness, but can also restrict future physical potential (Olson 2012).

1.8.2.3 The Cardiovascular System

- **The Acute Hemodynamic Effects**

These effects of cigarette smoking or smokeless tobacco are mediated primarily by the sympathomimetic action. The intensity of its hemodynamic effect is greater with rapid nicotine delivery. Nicotine causes catecholamine release both locally and systemically leading to an increase in heart rate, blood pressure and cardiac contractility. It reduces blood flow in cutaneous and coronary vessels; and increases blood flow in the skeletal muscles. Due to restricted myocardial oxygen delivery there is reduced cardiac work. Persistent stimulation by nicotine can contribute to Coronary Vascular Disease by producing acute myocardial ischemia. In the presence of coronary disease, myocardial dysfunction can be worsened (Nicotine addiction, 2000).

- **Fluctuation in HDL and LDL Balance**

Nicotine increases the low density lipoprotein and decrease the high density lipoprotein in the circulating blood. LDL can be harmful to the body and produce its greatest effect on blood vessels. If produced in excess or accumulated over time, they can stick to blood vessel walls and cause narrowing. Such narrowing can impair blood flow to the heart, brain and other organs, causing them to fail. Most bodies have a balance of good and bad fats. However, that is not the case for smokers (Benowitz, 1998).

In addition, a recent study in Japan showed a measurable decrease in the elasticity of the coronary arteries of nonsmokers after just 30 minutes of exposure to second hand smoke. Sudden death is four times more likely to occur in young male cigarette smokers than in nonsmokers due to formation of clot in the blood vessel (Siegel, 2007).

Smokers have elevated levels of thrombin, an enzyme that causes the blood to clot, after fasting, as well as a spike immediately after smoking. This process may result in blockage of blood vessels, stopping blood flow to vital organs. Even light smoking, causes the body’s blood vessels to constrict. Smoking does this by decreasing the nitric oxide which dilates blood vessels, and increasing the endothelin-1 which causes constriction of blood vessels. The net effect is Nicotine consumption increases a resting heart rate, as soon as 30 minutes after puffing and the higher the nicotine consumption the higher the heart rate (CDC, 2010).

Nicotine alters the structural and functional characteristics of vascular smooth muscle and endothelial cells. It enhances release of the basic fibroblast growth factor and inhibits production of transforming growth factor-β1. These effects lead to increased DNA synthesis, mitogenic activity, endothelial proliferation and increases atherosclerotic plaque formation. Neovascularization stimulated by nicotine can help progression of atherosclerotic plaques. These effects lead to myointimal thickening and atherogenic and ischemic changes, increasing the incidence of hypertension and cardiovascular disorders (CDC, 2010).

Nicotinic acetylcholine receptor's actions on vascular smooth muscle proliferation and plaque neovascularization increases the risk of peripheral arterial disorders.

1.8.2.4 Ocular System

In a clinical study, the most virulent form of age-related maculopathy was associated with retinal neovascularization that contributed to visual deterioration. Tobacco smokers are known to be at greater risk of age-related macular degeneration than are nonsmokers (CDC, 2010).

1.8.2.5 Renal System

Risk of chronic kidney disease in smokers is high. Cigarette smoking has been found to increase albumin excretion in urine, decrease glomerular filtration rate, causes increased incidence of renal artery stenosis and is associated with an increased mortality in patients with end-stage renal disease. The pathogenesis of renal effects is due to the action of nicotine via COX-2 isoform induction. The COX-2 isoforms causes increased glomerular inflammation, acute glomerulonephritis and ureteral obstruction. There is impaired response of kidneys to the increased systemic blood pressure in smokers. This loss of renoprotective mechanism in smokers also leads to pathogenetic effects of nicotine on the renal system (Hua, 2010).

1.8.2.6 Reproductive System

- **Male**

Nitrous oxide liberated from parasympathetico-nergic nerves plays a pivotal role in generating immediate penile vasodilatation and corpus cavernosum relaxation, and NO derived from endothelial cells contributes to maintaining penile erection. Nicotine causes impairment of NO synthesis. This may lead to loss of penile erections and erectile dysfunction (Mishra *et al*., 2015).

- **Female**

Menstrual Cycle

Nicotine by inhibiting the 21 hydoxylase causes hypoestrogenic state. It shunts the metabolites to formation of androgen. This leads to chronic anovulation and irregular menstrual cycles. Nicotine can predispose the endometrium to inappropriate cytokine production and irregular bleeding. There is consistent evidence that increase in follicle-stimulating hormone levels and decreases in estrogen and progesterone that are associated with cigarette smoking in women, is atleast in part due to effects of nicotine on the endocrine system (Jin and Roomans, 1997).

Effect on Oocytes

Nicotine affects the ovaries and alters the production of oocytes in various animal studies. Nicotine-treated oocytes appeared nonspherical with rough surface and torn and irregular zona-pellucida. Nicotine also caused disturbed oocyte maturation. There is a decreased blood flow to the oviducts and thus impaired fertilization (Hammer *et al.*, 1981).

Peri-Natal Effects

Maternal smoking has always been known to have deleterious effects on the fetal outcome. There is an increased incidence of intrauterine growth restriction, still birth, miscarriages and mental retardation. Maternal as well as grand maternal smoking has been found to increase risk of pediatric asthma. Another serious and important effect is the transgenic transmission of the addictive pattern (Hammer *et al.*, 1981).

1.8.2.7 Gastrointestinal System

Mucus is produced in the stomach to provide a protective barrier between stomach acid and cells of the stomach. Unlike in the lungs where mucus production is stimulated by cigarette smoke, mucous production in the stomach is inhibited. Peptic ulcers usually result from a failure of wound-healing due to outside factors, including tobacco smoke. Smoking also decreases blood flow to the inner layer of the esophagus, stomach and small intestine. In these ways, cigarette smoking immediately hinders gastrointestinal wound healing, which has been shown to result in peptic ulcer formation, when not treated (Adak, 2014).

Nicotine use has been associated with Gastro Esophageal Reflux Disorder (GERD) and peptic ulcer disease (PUD). This effect is mediated by increased gastric acid, pepsinogen secretion and stimulatory effects on vasopressin. The action on the cyclo-oxygenase pathway also increases the risk of GERD and PUD. Nicotine causes smooth muscle relaxation by action of endogenous nitric oxide as a nonadrenergicnoncholinergic neurotransmitter.The decrease in tone of the colon and gastric motility and reduced lower esophageal sphincteric pressure might be the reason of increased incidence of GERD (Chu *et al.*, 2013).

There is an increased incidence of treatment resistant *Helicobacter pylori* infection in smokers. It potentiates the effects of toxins of *H. pylori* by its action on the gastric parietal cells. This effect could be due to histamine mediated response of nicotine (Graham and Shiotani, 2008).

1.8.2.8 Immune System

Nicotine has been known to be immunosuppressive through central and peripheral mechanisms. It impairs antigen and receptor mediated signal transduction in the lymphoid system leading to decreased immunological response. The T-cell population is reduced due to arrest of cell cycle. Even the macrophage response, which forms the first line defense against tuberculosis becomes dysfunctional and causes increased incidence of tuberculosis (Shirazi and Huinet, 2003).

The migration of fibroblasts and inflammatory cells to the inflamed site is reduced. There is decreased epithelialization and cell adhesion and thus there is a delayed wound healing as well as increased risk of infection in nicotine exposed individuals.

The action on the hypothalamo-pituitary adrenal axis and autonomic nervous system stimulation via sympathetic and parasympathetic pathways affects the immune system. The adrenocorticotropic hormone (ACTH) secretion pathway and corticotrophin release is affected and this causes immunosuppression (Sopori *et al.*, 1998).

1.8.2.9 Metabolic System

Micronutrients are dietary components necessary to maintain good health. These include vitamins, minerals, enzymes and other elements that are critical to normal function. They must be consumed and absorbed in sufficient quantities to meet the body's needs. The daily requirement of these micronutrients changes naturally with age and can also be affected by environmental factors, including tobacco smoke. Smoking interferes with the absorption of a number of micronutrients, especially vitamins C, E, and folic acid that can result in deficiencies of these vitamins. A deficiency in Vitamin C can lead to scurvy which is a disease characterized by weakness, depression, inflamed gums, poor wound healing, and uncontrolled bleeding. Vitamin E deficiency may cause blood breakdown, eye disease, and irreversible nerve problems of the hands, feet, and spinal cord. Folic acid deficiency may result in long-lasting anemia, diarrhea, and tongue swelling.

Smoking increases the number of circulating oxidants, it also increases the consumption of existing antioxidants. This increase in antioxidant consumption reduces the levels of antioxidants such as alpha-tocopherol, the active form of vitamin E. Oxidants are active particles that are byproducts of normal chemical processes that are constantly underway inside the body. Their formation is called oxidation. These particles are usually found and destroyed by antioxidants, including vitamins A, C, and E. The balance of oxidation and anti-oxidation is critical to health. When oxidation overwhelms anti-oxidation, harmful consequences occur. Oxidants directly damage cells and change genetic material, likely contributing to the development of cancer, heart disease, and cataracts. Oxidants also speed up blood vessel damage due to atherosclerosis which is a known risk factor for heart disease (Myoclinic, 2015).

Table 1.1 Major Effects of Smoking on Human Organ

Organ/ System on the body	Major effects
Brain	Smoking increases the risk of stroke by at least 50%, which can cause brain damage and death.
Heart	Smoking damages the heart and blood circulation, increasing the risk of conditions such as coronary heart disease, heart attack, stroke, peripheral vascular disease and cerebrovascular disease.
Lungs	Smoking can cause fatal diseases such as pneumonia, emphysema and lung cancer. Smoking causes 84% of deaths from lung cancer and 83% of deaths from chronic obstructive lung disease.
Stomach	Smokers have an increased chance of getting stomach cancer or ulcers.
Skin	Smoking reduces the amount of oxygen supply to the skin, causes premature ages of skin and makes it three times more likely facial wrinkling, particularly around the eyes and mouth, gives yellow-grey complexion, hollow cheeks and dull.
Mouth and throat	Smoking causes bad breath, stained teeth, gum disease and damage sense of taste. More than 93% of oropharangeal cancers (cancer in part of the throat) are caused by smoking.
Bones	Smoking can cause the bones to become weak and brittle. Women are more likely to suffer from brittle bones (osteoporosis) than non-smokers.
Respiratory System	Lung diseases caused by smoking include COPD, which includes emphysema and chronic bronchitis.

Reproduction and fertility	Smoking can cause male impotence, as it damages the blood vessels that supply blood to the genital organ. It can also damage sperm, reduce sperm count and cause testicular cancer. One study found that the fertility of smoking women was 72% that of nonsmokers.
Cardiovascular System	Smoking damages blood vessels and can make them thicken and grow narrower resulting stroke and coronary heart disease as a leading cause of death in the globe.

Effects of smoking on the body (Adak, 2014.p:2)

1.9Miscellaneous Effect

1.9.1 Smoking and Teeth Staining

Teeth stain is of two types, extrinsic and internalized. The causes of extrinsic staining can be divided into two categories, those compounds which are incorporated into the pellicle and produce a stain as a result of their basic colour, and those which lead to staining caused by chemical interaction at the tooth surface. Direct staining has a multi-factorial etiology with chromogens derived from dietary sources or habitually placed in the mouth. These organic chromogens are taken up by the pellicle and the colour imparted is determined by the natural color of the chromogen. Tobacco smoking and chewing are known to cause staining. The stains taken up into the body of enamel or dentine are the same as those causing extrinsic tooth discoloration, including in particular dietary chromogens and the by-products of tobacco smoking (Miller, 2015).

1.9.2 Smoking Developmental Defects and Premature Birth

Mothers who smoke during pregnancy are more likely to deliver babies with low birth weight, even if the babies are full term. Mothers who smoke during pregnancy are also more likely to deliver their babies early. Low birth weight and preterm delivery are leading causes of infant disability and death. Smoking during pregnancy can cause tissue damage in the fetus, especially in the lungs and brain. Carbon monoxide in tobacco smoke is a dangerous toxin that can harm the central nervous system and impair fetal growth. Damage from maternal smoking can last

throughout childhood and into the teenage years. Babies whose mothers smoked during pregnancy or who are exposed to secondhand smoke after birth are more likely to die of SIDS than are babies who are not exposed (CDC, 2015).

1.10 Smoking and Asthma

When a person has asthma, the tubes that carry air into and out of the lungs tend to be swollen and easily irritated. When something is breathed irritates the swollen airways, they swell up even more and this makes it hard for air to get through to the lungs. This swelling and irritation in the airways causes asthma symptoms such as coughing, wheezing tightness in the chest, and trouble breathing. These symptoms can lead to an asthma attack. Smoking can make the asthmatic condition worse. The extra irritation caused by smoking makes a person more likely to have an asthma attack.

In asthmatic patients who smoke, disease control is poorer than in asthmatic nonsmokers. Of all forms of SHS, maternal exposure seems to have the largest impact on asthma by increasing the frequency and severity of the disease and decreasing lung function. Asthmatic children exposed to multiple household smokers face an increased risk for respiratory illness-related absences from school, and these effects persist during adolescence but weaken during adulthood. Airway mucosal permeability is increased in smokers, which could lead to increased clearance of inhaled corticosteroids from the airways. Smokers also have decreased histone deacetylase activity, which is necessary for corticosteroids to fully suppress cytokine production, and can lead to corticosteroid resistance (Stapleton *et. al.*, 2011).

1.11 Smoking and Tuberculosis

Smoking impairs the host immune defenses right from the beginning. Smoking impairs the clearance of secretions present on tracheobronchial mucosa. This is the first line of defense which aids in clearance of inhaled particles. Thus, by impairing the clearance, it allows the tubercle bacilli to escape the defense and propels it to reach alveoli. Pulmonary alveolar macrophages constitute early defense mechanism against the tubercle bacilli. Studies have shown that smoking alters the function of these macrophages and thus impairing their ability to clear the bacilli from airways. The macrophages from smokers were bigger in size, had abnormal surface morphology which led to impaired antigen presenting function. The alveolar macrophages of smokers had reduced phagocytic activity and low levels of pro-inflammatory cytokines (Indian J Tuberc, 2012).

1.12 Smoking and Diabetes

There is a growing body of evidence to show that smoking is a risk factor for Type 2 Diabetes. Several hypotheses have been proposed to explain this link. Smoking has been identified as a possible risk factor for insulin resistance, a precursor for diabetes. Smoking has also been shown to deteriorate glucose metabolism which may lead to the onset of Type 2 diabetes. There is also some evidence which suggests that smoking increases diabetes risk through a body mass index independent mechanism. Smoking has further been associated with a risk of chronic pancreatitis and pancreatic cancer, suggesting that tobacco smoke may be toxic to the pancreas. Cigarette smoking and nicotine intake increase the circulating levels of hormones, such as catecholamines, glucagon and growth hormone, which impair the action of insulin. Interestingly, it was recently shown that nicotine administered intravenously in non-smokers caused a marked reduction (about 30%) in insulin sensitivity in people with type 2 diabetes but not in those without the condition. These results suggest that nicotine may be particularly damaging to people who, due to a health condition such as diabetes, are already vulnerable (Eliasson, 2005).

1.13 Drug Interaction

Drug breakdown, or metabolism, is important to drug effectiveness and safety. Medicines are naturally broken down into their components by enzymes. Factors that affect drug metabolism effect drug function. Factors that speed up drug metabolism decrease drug exposure time and reduce the circulating concentrations of the drug, which compromises the effectiveness of the prescription. Conversely, factors that slow down drug metabolism increase the circulating time and concentration of the drug, allowing the drug to be present at harmful levels. Tobacco smoke interferes with many medications by both of these mechanisms. For example, the components of tobacco smoke hasten the breakdown of some blood-thinners, antidepressants, and anti-seizure medications; and tobacco smoke also decreases the effectiveness of certain sedatives, painkillers, heart, ulcer, and asthma medicines (Fathima, 2012).

1.14 Deadly Compounds from Tobacco

Long-time exposure of tobacco smoke produces disease some of which can cause death. Each puff of each cigarette contains a mixture of thousands of compounds, including more than 60 well-established carcinogens. The carcinogens in cigarette smoke belong to multiple chemical classes, including polycyclic aromatic hydrocarbons, *N*-nitrosamines, aromatic amines, aldehydes, volatile organic hydrocarbons, and metals. In addition to these well-established carcinogens, others have been less thoroughly investigated. These include alkylated PAHs, oxidants, free radicals, and ethylating agents. Considerable evidence indicates that in human cancers caused by cigarette smoking, PAHs, *N*-nitrosamines, aromatic amines, and certain volatile organic agents play a major role.

1.14.1 Polycyclic Aromatic Hydrocarbons

PAHs are incomplete combustion products first identified as carcinogenic constituents of coal tar. These products occur as mixtures in tar, soot, broiled foods, automobile engine exhaust, and other materials generated by incomplete combustion. Generally, PAHs are carcinogens that act locally. Some PAHs, such as benzopyrene, have powerful carcinogenic activity.

1.14.2 Heterocyclic Compounds

These include analogs of PAHs containing nitrogen, as well as simpler compounds such as furan, which is a liver carcinogen. N-nitrosamines are a large class of carcinogens with demonstrated activity in at least 30 animal species. They are potent and systemic carcinogens that affect different tissues depending on their structure. Two of the most important N-nitrosamines in cigarette smoke are the tobacco-specific 4(methylnitrosamino)-1-(3-pyridyl)-1-butanone and N'-nitrosonornicotine.

1.14.3 Aromatic Amines

These are combustion products that include the well-known human bladder carcinogens 2-naphthylamine and 4 aminobiphenyl, which were first characterized as human carcinogens attributable to industrial exposures in the dye industry. Heterocyclic aromatic amines are also combustion products and are best known for their occurrence in broiled foods, but they also occur in cigarette smoke.

1.14.4 Aldehydes

Formaldehyde and acetaldehyde occur widely in the human environment and are endogenous metabolites found in human blood. The phenolic compounds cat-echol and caffeic acid are common dietary constituents. High doses of catechol cause glandular stomach tumors when administered in the diet. Catechol can also act as a cocarcinogen, enhancing the activity of carcinogens such as Benzo alpha pyrine.

1.14.5 Oxidants

Cigarette smoke also contains oxidants such as nitric oxide and related species. Free radicals have been detected by electron spin resonance and spin trapping. Researchers postulate that the major species of free radicals are a quinine- hydroquinone complex. Other compounds may also be involved in the oxidative damage produced by cigarette smoke. In addition, several studies demonstrate the presence in cigarette smoke of an uncharacterized ethylating agent, which ethylates both DNA and hemoglobin.

Most carcinogens in cigarette smoke require a metabolic activation process, generally catalyzed by cyto-chrome P-450 enzymes, to convert the carcinogens to forms that can covalently bind to DNA and form DNA adducts. P-450s 1A1 and 1B1, which are inducible by cigarette smoke through interactions with the aryl hydrocarbon receptor, are particularly important in the metabolic activation of PAHs. The inducibility of these P-450s may be a critical aspect of cancer susceptibility in smokers. Competing with the activation process is metabolic detoxification, which excretes carcinogen metabolites in generally harmless forms and is catalyzed by a variety of enzymes, including Glutathione-*S*-transferases, uridine-5′-disphosphate-glucuronosyltransferases, epoxide hydrolases, and sulfatases. The balance between metabolic activation and detoxification of carcinogens varies among persons and likely affects cancer susceptibility. Persons with a higher activation and lower detoxifica-tion capacity are at the highest risk for smoking-related cancers.

Other carcinogenic organic compounds in cigarette smoke include the human carcinogens vinyl chloride in low amounts and ethylene oxide in substantial quantities. Ethylene oxide is associated with malignancies of the lymphatic and hematopoietic systems in both humans and laboratory

animals. Diverse metals such as the human carcinogen cadmium are also present in cigarette smoke, as is the radioisotope polonium 210, which is carcinogenic to humans (CDC, 2010).

1.15Tobacco Smoking and Cancer

The stimulation of nAChRs by nicotine has biologic effects on cells important for initiation and progression of cancer. It activates signal transduction pathways directly through receptor-mediated events, allowing the survival of damaged epithelial cells. In addition, nicotine is a precursor of tobacco specific nitrosamines (TSNAs), through nitrosation in the oral cavity. It is shown that nitrosation of nicotine could lead to formation of N'-nitrcssonornicotine(NNN) and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK). This effect of nicotine may be important because of its high concentration in tobacco and nicotine replacement products. NNN and NNK are strongly carcinogenic.

Nicotine forms arachidonic acid metabolites which cause increased cell division. Binding to B cell lymphoma-2 and action on vascular endothelial growth factor and cyclooxygenase-2 (COX-2) causes increased cancer proliferation and survival. Promotion of tumor angiogenesis accelerates tumor growth which is mediated by β-adrenergic activation and stimulation of nAChRs. Nicotine also suppresses apoptosis by phosphorylation mediated extracellular signal regulated kinases of Bcl-2. Recent studies show that nicotine, activates nuclear factor kappa B (NF-kB)-dependent survival of cancer cell and proliferation.

In normal cells, nicotine can stimulate properties consistent with cell transformation and the early stages of cancer formation, such as increased cell proliferation, decreased cellular dependence on the extracellular matrix for survival, and decreased contact inhibition. Thus, the induced activation of nAChRs in lung and other tissues by nicotine can promote carcinogenesis by causing DNA mutations. Through its tumor promoter effects, it acts synergistically with other carcinogens from automobile exhausts or wood burning and potentially shortenthe induction period of cancers (Mishra *et al.*, 2015).

1.15.1 Smoking and Lung Cancer

Smoking causes lung cancer by damaging the cells that line the lungs. When someone inhales cigarette smoke, which is full of cancer-causing substances, changes in the lung tissue begin almost immediately. At first the body may be able to repair this damage. But with each repeated exposure, normal cells that line your lungs are increasingly damaged. Over time, the damage causes cells to act abnormally and eventually cancer may develop (Warren and Singh, 2014).

The two general types of lung cancer include:

- **Small cell lung cancer**
  Small cell lung cancer occurs almost exclusively in heavy smokers and is less common than non-small cell lung cancer.

- **Non-small cell lung cancer**
  Non-small cell lung cancer is an umbrella term for several types of lung cancers that behave in a similar way. Non-small cell lung cancers include squamous cell carcinoma, adenocarcinoma and large cell carcinoma (ACS, 2015).

A study relates lung carcinogenesis by nicotine due to genetic variation in Cytochrome P2B6. Cotinine has been found to promote lung tumorigenesis by inhibiting anti-apoptotic pathway.Nuclear translocation of ARb1 - Androgen receptor beta 1gene by nicotine has found in proliferation and progression of nonsmall-cell lung cancer. Several Studies have shown that nicotine has significant role in tumor progression and metastasis via C-X-C chemokine receptor type 4 and increased angiogenesis. Carriers of the lung-cancer-susceptibility loci in their DNA extract more nicotine. Smokers carrying the gene cholinergic receptor nicotinic alpha 3(CHRNA3) and CHRNA5 were found to extract more nicotine and cells were thus exposed to a higher internal dose of carcinogenic nicotine-derived nitrosamines. Additionally, modulation of the mitochondrial signaling pathway leads to resistance to the chemotherapeutic agents. (Nakada *et al.*, 2013)

Cotinine is an alkaloid found in tobacco and is also the predominant metabolite of nicotine. Cotinine has been found to promote lung tumorigenesis by inhibiting anti-apoptotic pathway. Nuclear translocation of ARB1 gene by nicotine has found in proliferation and progression of nonsmall-cell lung cancer. Several Studies have shown that nicotine has significant

role in tumor progression and metastasis via CXCR4 and increased angiogenesis. Carriers of the lung-cancer-susceptibility loci in their DNA extract more nicotine. Smokers carrying the gene CHRNA3 and CHRNA5 were found to extract more nicotine and cells were thus exposed to a higher internal dose of carcinogenic nicotine-derived nitrosamines. Additionally, modulation of the mitochondrial signaling pathway leads to resistance to the chemotherapeutic agents (Improgo *et al*., 2013).

Nicotine may have a broad spectrum of tumor-promoting activities in lung cancer. Nicotine and its metabolites increase proliferation, migration, invasion, EMT, and angiogenesis with a concomitant decrease in sensitivity to chemotherapy and/or radiotherapy. The effects of nicotine occur through activation of nAChRs and β-AdrRs leading to common downstream activation of Src, Ras-Raf-MAPK-MEK-ERK pathways, and PI3K-Akt pathways that further drive several parallel oncogenic pathways. Nicotine and its metabolites can promote tumor progression through modulation of oncogenic signals in both cancerous and non-cancerous tissues. Substantial work is required to definitively test the effects of nicotine on clinical outcomes in cancer patients, but current data suggest that nicotine is not a benign substance in cancer progression and therapy (Warren and Singh, 2013).

1.15.2 Smoking and Oral Cancer

Oral cancer represents about 4 percent of all cancers and 2.2 percent of all cancer deaths in the U.S. In the western world in general, oral cancer accounts for 2-6 percent of all malignancies. In Asia, oropharyngeal cancer is the leading cancer in men, and in Africa and Asia it is the third most frequent cancer site in women. The average five-year survival rate of patients with oral cancer is about 50 percent. This is primarily due to late detection of the disease. Early detection and prevention are the key to fighting this deadly disease. Squamous cell carcinoma is the most common cancer of the oral cavity. In men, most oral cancers are found on the floor of the mouth and tongue; in women, the most common sites are the tongue and gums (Oral Cancer Foundation, 2014).

1.15.3 Gastrointestinal Cancer

The carcinogenic role may be mediated by the MAPK/COX-2 pathways, α-7 nAchR and β-adrenergic receptor expression, and mi RNAs α-BTX antagonist. Nicotine forms adducts with liver DNA which enhances its mutagenic potential. activation of cell-surface receptors by nicotine stimulates downstream kinases that can mediate resistance to chemotherapy. It has been shown by the finding that smokers who continue to smoke during chemotherapy have a worse prognosis. Moreover, they also have increased toxicity and lower efficacy of chemo therapeutic drugs. Nicotine affects the periostin gene, α-7-nAChR and e-cadherin suppression which explains the mechanism of gastric cancer growth, invasion and metastasis. Nicotine negatively impacts tumor biology by promoting angiogenesis, tumor invasion and increased risk of metastasis (Jensen *et.al.*, 2012; Liu, 2011).

1.15.4 Breast Cancer

Nicotine causes α9-nAChR-mediated cyclin D3 overexpression which might cause transformation of normal breast epithelial cells and induce cancer. Nicotine and cotinine has been found to be present in the breast fluid of lactating women. α9-nAChR mediated mechanism leads to increased tumor growth, metastasis and tumor cells resistant to chemotherapeutic drugs in breast cancer (Nishioka *et al.*, 2011).

Chapter Two
Literature Review

2.1 Knowledge and Attitude toward Smoke-free Legislation and Second-Hand Smoking Exposure among Workers in Indoor Bars, Beer Parlors and Discotheques in Osun State of Nigeria.

One of the requirements of the Osun State smoke-free legislation is to ensure smoke-free enclosed and partially enclosed workplaces. This survey was conducted to assess the knowledge and attitude of workers in indoor bars, beer parlors and discotheques to smoke-free legislation in general and the Osun State smoke-free law in particular.

A convenience sampling of 36 hospitality centers was conducted. Interviewer-administered questionnaires were used to elicit responses about the objectives from non-smoking workers. The questionnaires had sections on knowledge of the Osun State smoke-free law, attitude toward the law and smoke-free legislation in general and exposure to second-hand tobacco smoke by the workers. Questions were also asked about the second-hand tobacco smoking status of these workers.

154 participants were recruited into the study. There were 75 males (48.0%) and 79 females (52.0%). On the overall, respondents had a good knowledge of the effects of second-hand smoke on health (70.2%) with 75.0% of them being aware of the general smoke-free law and 67.3% being aware of the Osun State smoke-free law although none of them had ever seen a copy of the law. A high proportion (60.0%) was in support of the Osun smoke-free law although all of them think that the implementation of the law could reduce patronage and jeopardize their income. Attitude toward second-hand smoking was generally positive with 72.0% of them having no tolerance for second-hand tobacco smoke in their homes. Most participants (95.5%) had been exposed to tobacco smoke in the workplace within the past week.

Despite the high level of awareness of the respondents about the dangers of second hand smoke and their positive attitude to smoke-free laws, nearly all were constantly being exposed to second hand smoke at work. This calls for policy level interventions to improve the implementation of the smoke-free law (Onigbogi *et al*., 2015).

2.2 Public Knowledge and Attitude towards Passive Smoking in Bahrain.

A sample of 506 Bahraini adults who accompanies patients during their visits to the four major health centers in Bahrain in the summer of 2001 was surveyed. Simple random sampling was used and individuals ask to fill an anonymous questionnaire which contained questions on knowledge and attitude towards passive smoking. Among the 506 people 58.5% were males of whom 27.5 % current smokers. The age range was between 18-60 years and the mean was 29.5 years. More than 80% of the population knew that smoke from cigarette or sheesha is harmful to other people around them and it causes eye irritation and cough. More than 77% also knew that smoking pregnant women can increase their chance of delivering low birth weight babies and passive smoking is harmful to mother's unborn child. Around 30% were not aware that living with a smoker person can increase the risk of lung cancer. Knowledge about passive smoking was generally higher among the educated, high professional job, married, older people groups, ex- smokers and non-smokers.

The result of this survey was relatively good level of awareness about hazards of passive smoking but they think sheesha smoking is safe. Public health efforts to reduce the harmful effects of passive smoking should be focused particularly on males, lower educated professional persons (Al-Haddad, Hamadeh and Bahram, 2006).

2.3 Public Knowledge about Passive Smoking: Results from a Survey in the Australian Capital Territory.

Using data from a random sample of Australian Capital Territory residents conducted in November-December 1991, this paper examined the level of general and specific knowledge that individuals possess about passive smoking. While 87% of the sample gave an accurate definition of passive smoking, the 13% who did not were distinguished by lack of educational qualifications and by employment outside the government sector. A similar proportion did not see any illnesses being associated with passive smoking, and this group was significantly more likely to consist of current smokers. Employment status emerged as a factor influencing specific knowledge of passive smoking, highlighting the importance of continued moves to restrict smoking within the workplace (Makkai *et al.*, 1994).

2.4 Evaluation of Nurses' Knowledge About Health Effects of Active and Passive Smoking and Their Attitude Towards Providing Smoking Cessation Activities.

The aim of the study was to evaluate the nurses' knowledge about health effects of active and passive smoking and their attitude towards providing smoking cessation activities. The study population consisted of 299 nurses. About 57% of the women thought that some kind of cigarettes can be less dangerous than other. Almost all of the nurses indicated that tobacco smoking may lead to addiction. Unfortunately, only 67% of study population thought that sidestream smoke contains substances dangerous for health. All nurses indicated that tobacco smoking causes lung cancer and 77% of them that it can cause cardio-vascular diseases and only 26% that it increases the risk of bladder cancer. Almost all women indicated that environmental tobacco smoke exposure is dangerous for pregnant women and the faetus. The nurses were educated about health effects of active and passive smoking but they have limited knowledge about the methods used in antismoking counseling (Zaborszczyk *et al.*, 2009).

2.5 Smoking Behavior and Views On the Smoking Topic in The Syrian Population—Results of A Survey at The Graz Autumn Convention.

Cigarette smoking has been identified as a major risk factor for cardiovascular diseases apart from hypercholesterolemia and arterial hypertension. Diseases of the respiratory system and several neoplasms as well as complications of pregnancy are considered to be "smoking-related", too. In order to survey the smoking habits and views on smoking in the Styrian population, a questionnaire was issued to 1034 visitors of the Graz Autumn Fair 1992. Pleasure proved to be the main motivation for smoking both in men and in women, habit, addiction, stress and boredom being further motivations. Not only non-smokers, but also smokers were assessed to be aware of smoking being a health hazard. Prohibition of tobacco advertising was accepted most widely as a mean of anti-smoking campaign. About half the non-smokers are exposed to environmental tobacco smoke, at place of work more frequently than at home. Percentage of smokers differs with age and job classes, however, no geographical variations were found. Further anti-smoking measures should be considered. Smoking restrictions at place of work and in public could prevent passive smoking. Ban on advertising seems to be the most effective anti-smoking campaign (Maier, 1996).

2.6 Environmental Tobacco Smoke Exposure in the Home and Worksite and Health Effects in Adults: Results from The 1991 National Health Interview Survey.

The main objective of this study was to determine the effect of environmental tobacco smoke exposure in the home and worksite on the health of adults in the United States. It is a Cross-sectional survey. 43,732 adults who completed the Health Promotion and Disease Prevention supplement in the 1991 National Health Interview Survey. This study demonstrates that never-smoking adults exposed to ETS report more acute health effects than unexposed, never-smoking adults, and suggests similar findings in current and former smoking adults (Mannino, *et al*., 1997).

2.7 Tobacco Related Knowledge and Support for Smoke-Free Policies Among Community Pharmacists in Lagos State, Nigeria.

The objective of this study was to determine the tobacco-related knowledge of community pharmacists and assess their support for smoke-free policies in Lagos state, Nigeria.

A cross-sectional descriptive study design using both quantitative and qualitative methods was employed. Two hundred and twelve randomly selected community pharmacists were surveyed using a pre-tested self-administered questionnaire. In addition, one focus group discussion was conducted with ten members of the Lagos state branch of the Association of Community Pharmacists of Nigeria.

The quantitative survey revealed that the majority (72.1%) of the respondents were aged between 20 and 40 years, predominantly male (60.8%), Yoruba (50.2%) or Igbo (40.3%) ethnicity and had been practicing pharmacy for ten years or less (72.2%). A majority (90.1%) of respondents were aware that tobacco is harmful to health. Slightly less (75.8%) were aware that second hand smoke is harmful to health. Among the listed diseases, and esophageal (68.9%) cancers were the most common diseases associated with tobacco use. Less than half of those surveyed associated tobacco use with heart disease (46.9%), chronic obstructive pulmonary disease (27.8%), bladder cancer (47.2%), peripheral vascular disease (35.8%) and sudden death (31.1%). Only 51.9% had heard of the World Health Organization Framework Convention on Tobacco Control (WHO FCTC). A little over half of the respondents (53.8%) were aware of any law in Nigeria controlling tobacco use. The majority of respondents supported a ban on smoking in homes (83.5%), in public places (79.2%), and in restaurants, nightclubs and bars (73.6%). Current smokers were 1.3 times less

likely to support smoke-free policies compared with non-smokers. The findings emanating from the focus group discussion reinforced the fact that the pharmacists were in support of smoke-free policies particularly in homes and public places. It also demonstrated that most of them were aware of the health risks associated with tobacco use and second hand smoke however some misconceptions seemed to exist.

The pharmacists surveyed expressed support of smoke-free policies and most of them were aware of the health risks associated with tobacco use. However, awareness of WHO FCTC and country-level tobacco legislation was low. Current smokers were less likely to support smoke-free policies. Community pharmacists should therefore be considered worth engaging for the promotion of smoke-free policies. Efforts should also be made to educate pharmacists about country level smoke-free laws (Poluyi *et al.,* 2015).

2.8 Attitudes and Beliefs About Secondhand Smoke and Smoke-Free Policies in Four Countries: Findings from the International Tobacco Control Four Country Survey.

This paper describes the varying levels of smoking policies in nationally representative samples of smokers in four countries and examines how these policies are associated with changes in attitudes and beliefs about secondhand smoke over time.

Data was reported on 5,788 respondents to Wave 1 of the International Tobacco Control Four Country Survey who were employed at the time of the survey. A cohort of these respondents was followed up with two additional survey waves approximately 12 months apart. Respondents' attitudes and beliefs about secondhand smoke as well as self-reported policies in their workplace and in bars and restaurants in their community were assessed at all waves.

The level of comprehensive smoke-free policies in workplaces, restaurants, and bars increased over the study period for all countries combined and was highest in Canada (30%) and lowest in the United Kingdom (0%) in 2004. In both cross-sectional and longitudinal analyses, stronger secondhand smoke policies were associated with more favorable attitudes and support for comprehensive regulations. The associations were the strongest for smokers who reported comprehensive policies in restaurants, bars, and their workplace for all three survey waves.

The conclusion of this study was comprehensive smoke-free policies are increasing over time, and stronger policies and the public education opportunities surrounding their passage are associated with more favorable attitudes toward secondhand smoke regulations. The implication for policy makers is that, although the initial debate over smoke-free policies may be tumultuous, once people understand the rationale for implementing smoke-free policies and experience their benefits, public support increases even among smokers, and compliance with smoke-free regulations increases over time (Hyland *et al.*, 2009).

Significance of the Study

Tobacco smoking is dangerous for both the active and passive smokers. Active smokers directly inhale the smoke and passive smokers indirectly. The indirect inhalation of the smoke is sometimes more dangerous than the direct smoking because the smoke exhaled by the smoker contains chemical that is converted into more deadly compound inside the smoker's system (ACS, 2015).

Nicotine is the main ingredient that is inhaled by both the active and passive smokers. It is said that if a person is injected the nicotine contained in two cigarettes then the person is going to die. Ingestion of five cigarettes or 10 ml of a dilute nicotine-containing solution could kill an adult. The inhalation route allows a person to take this much of nicotine without causing any immediate severe complications. So a cigarette is not killing immediately but slowly (Mayer, 2014).

In a developing country like Bangladesh the issue of passive smoking is not very popular. There are rules to stop public place smoking which is not implemented properly. So people smoke freely not caring about the others. People who smoke usually enjoy smoking but people who do not smoke face the consequences of passive smoking even though they are not enjoying anything. Advertisement about the dangerous health effects of active smoking is common. In news paper, television radio this kind of advertisement is always found, even on the packet of cigarette it is written that 'smoking is injurious to health'. There is no advertisement about passive smoking, about the consequences of passive smoking. Parents seem really concern about their children either they are smoking or not but they are not really concerned about either they are being exposed to passive smoke or not. Passive smoking can harm a child as well as inspire them to smoke. Children and adolescents are more likely to start smoking for the first time. So it is very much important to know about the knowledge as well as the attitude of general people of Bangladesh towards passive smoking.

Aims and Objectives of the Study

The aims of this study were

- To find out if people know about what passive smoking is.
- To see if people are aware enough to save them from passive smoking.
- To identify what are the places people get exposed in.
- To find out which classes of people are more exposed and what their approach towards being exposed.
- To gather information about general people's knowledge of the dangerous effects of passive smoking.
- To know either general people support any activity to stop passive smoking.

Chapter Three
Methodology

3.1 Type of the Study

It was a survey based study.

3.2 Study Population

The general people, both smokers and non-smokers were the study population. The study was carried out in Dhaka, Gazipur and Noakhali. The total number of study population is 709. The rickshaw puller, tea stall manager, saloon worker and labor working in different sectors was targeted as the people who are not higher educated or illiterate. University students and job holders in different sectors were targeted as higher educated people.

3.3 Inclusion Criteria

- Both males and females
- Anyone over the age of 18 years

3.4 Exclusion Criteria

- Anyone under the age of 18

3.5 Data Collection Method

The data was collected through questionnaire that is formed in English language. It is a questionnaire consists of multiple choice type questions. The data was collected by both face to face interview and by questionnaire supply.

3.6 Development of the Questionnaire

The questionnaire was developed based on different findings in available journal and research paper. Also from the observation of different behavior of Bangladeshi people.

3.7 Sampling Technique

In this study random sampling was followed.

3.8 Data collecting period

The duration of the study was about three months that started from August, 2015 up to October, 2015.

3.9 Data Analysis

After collecting, all the data were checked and analyzed with the help of Microsoft Excel 2007.

Chapter Four
Result

4.1 Personal Information

4.1.1 Age Distribution

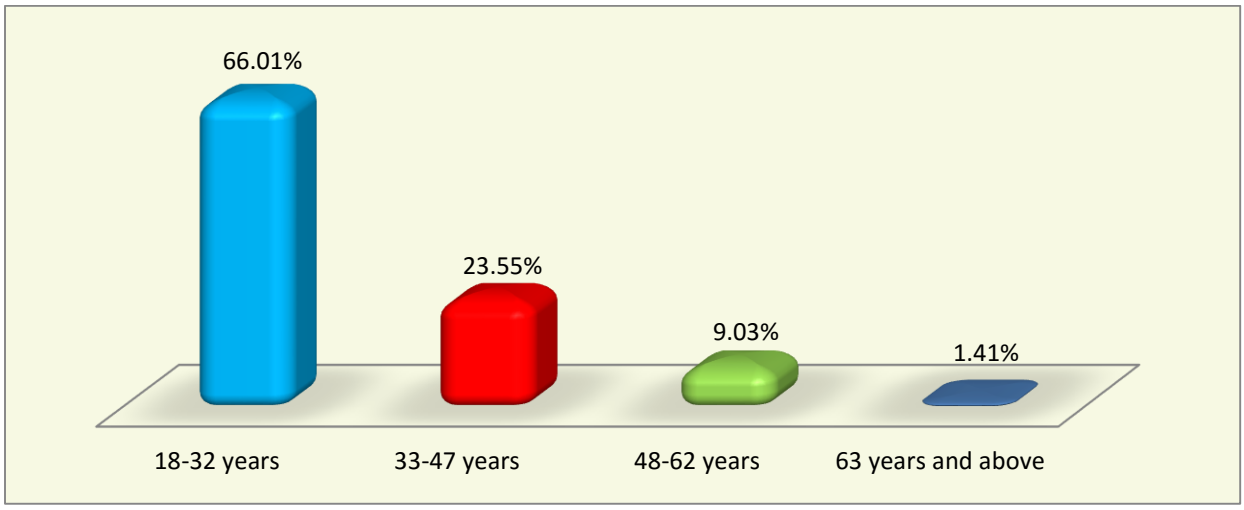

Fig 4.1.1: Age Distribution

Most (66.01%) of the respondents were between the ages of 18-32 years. Only 1.41% of the respondents were above 63 years old.

4.1.2 Gender Distribution

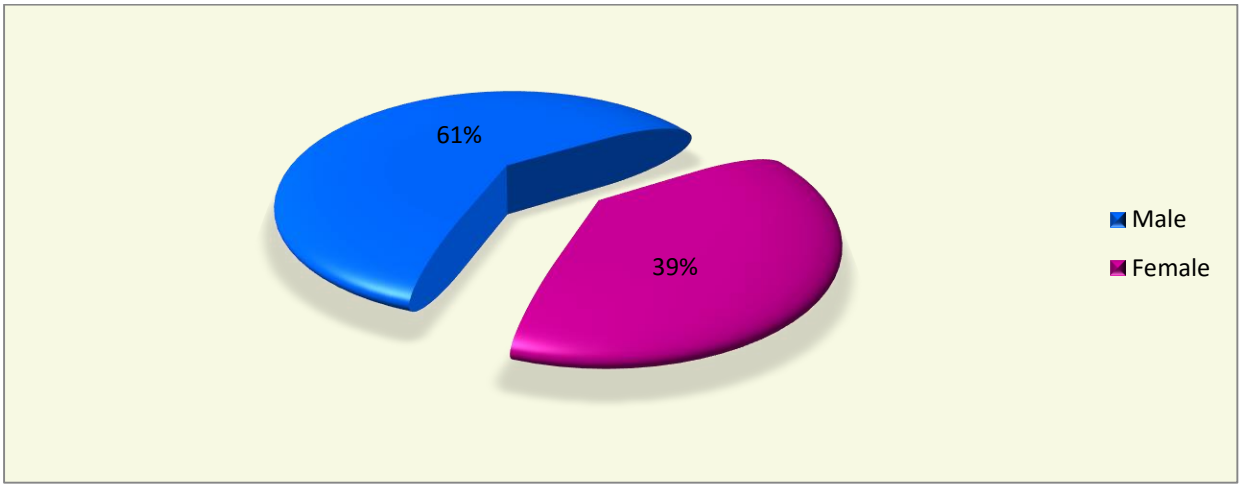

Fig 4.1.2: Gender Distribution

Most of the respondents were male. Among the respondents 61% are male and 39% Female.

4.1.3 Educational Qualification

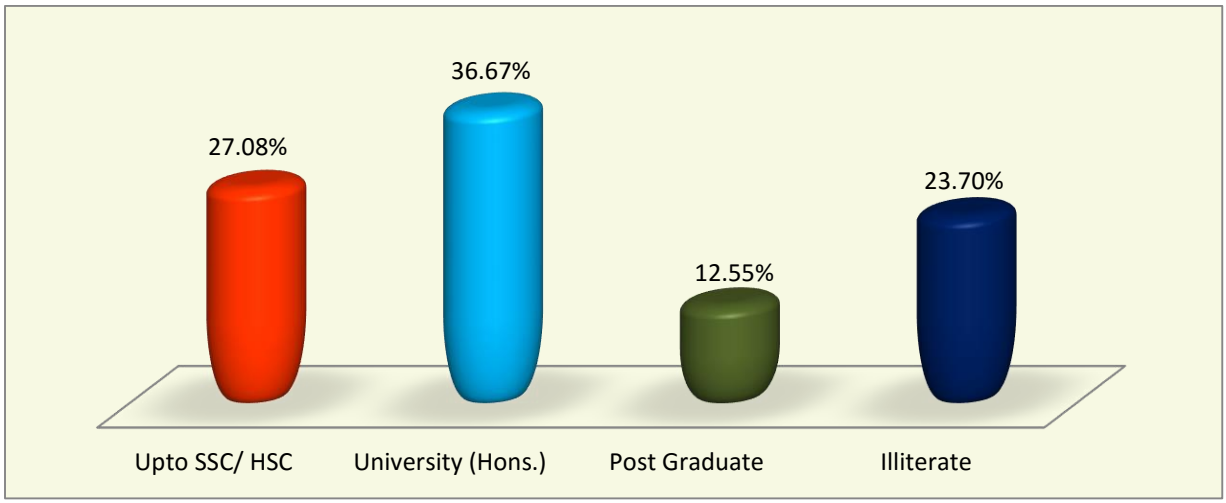

Fig 4.1.3: Educational Qualifications

Most (36.67%) of the respondents were either current university students or completed their graduation.

4.1.4 Occupation Status

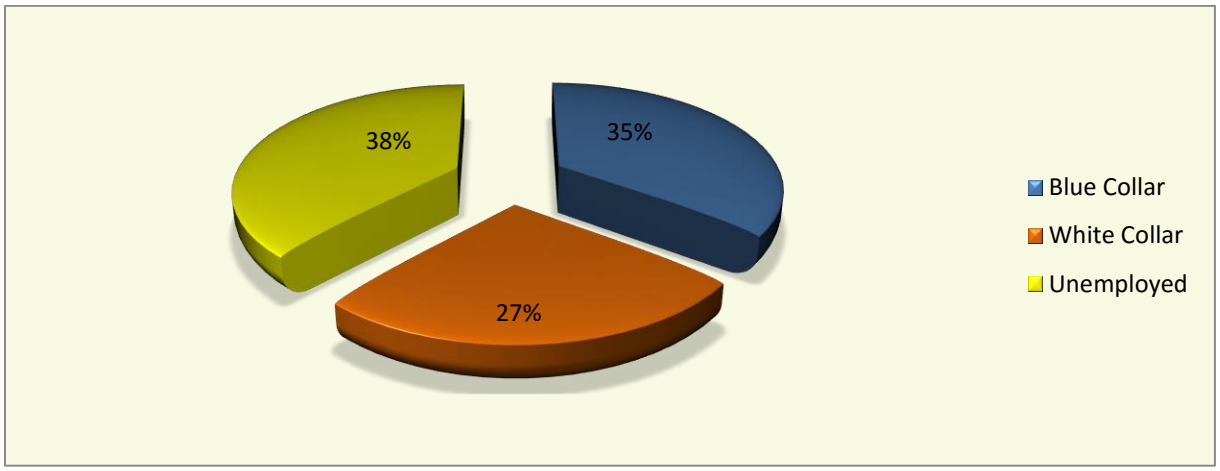

Fig 4.1.4: Occupation Status

People who are not professionally involved in manual labor are in the white collar group. People who need to put their manual labor in the working field are in the blue collar group. Most (38%) of the people were unemployed and 35% belonged in the blue collar group (Dictionary.com, 2015).

4.1.5 Monthly Income Distribution

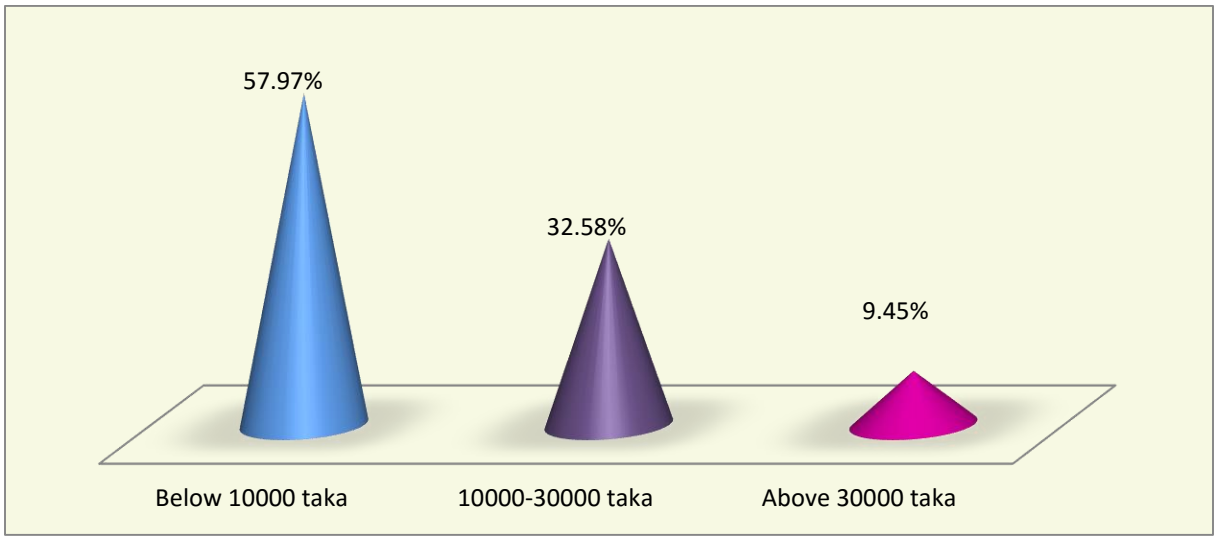

Fig 4.1.5: Monthly Income Distribution

Most (57.97%) of the respondent's monthly income were below 10000tk. The pocket money of the unemployed respondents was included in this class. Only 9.45% of the respondents used to earn above 30000tk monthly.

4.1.6 Living Area

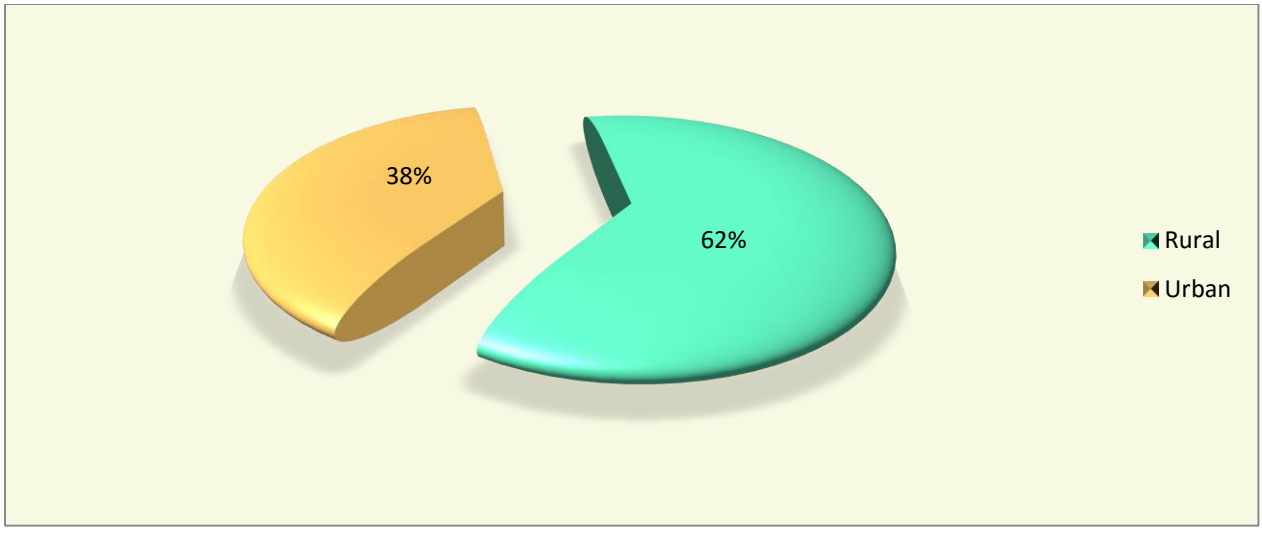

Fig 4.1.6: Living Area

Most (62%) of the respondents were rural and only 38% were urban.

4.1.7 Smoking Status

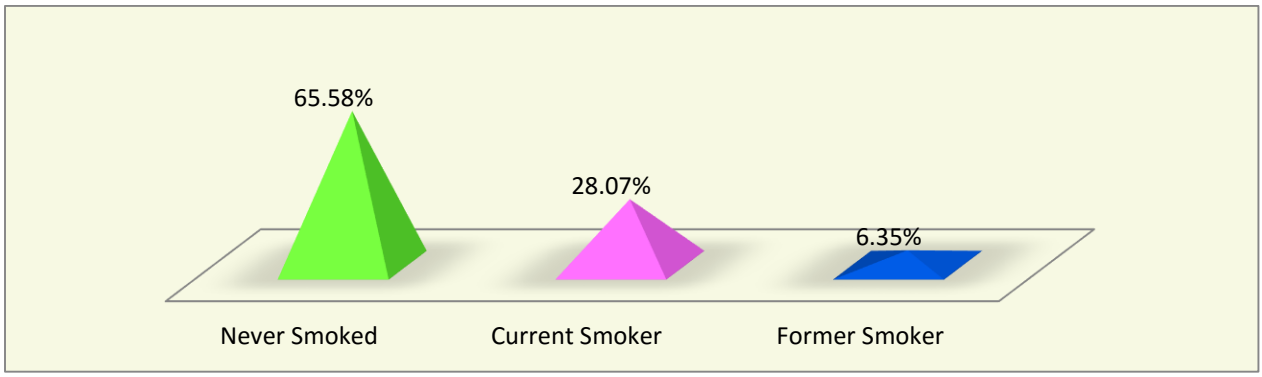

Fig 4.1.7: Smoking Status

Most (65.58%) of the respondents were no-smokers. Only 28.07% respondents were smokers.Respondents were defined as current smokers if they were smoking at the time of the survey and had smoked more than 100 cigarettes in their lifetime; they were defined as former smokers if they had smoked more than 100 cigarettes in their lifetime but no longer smoked;and they were defined as neversmokers if they had never smoked or had smoked fewer than 100 cigarettes in their lifetime (Menon *et al.*, 2000).

4.2 Knowledge and Attitude

4.2.1 Knowledge about Passive Smoking

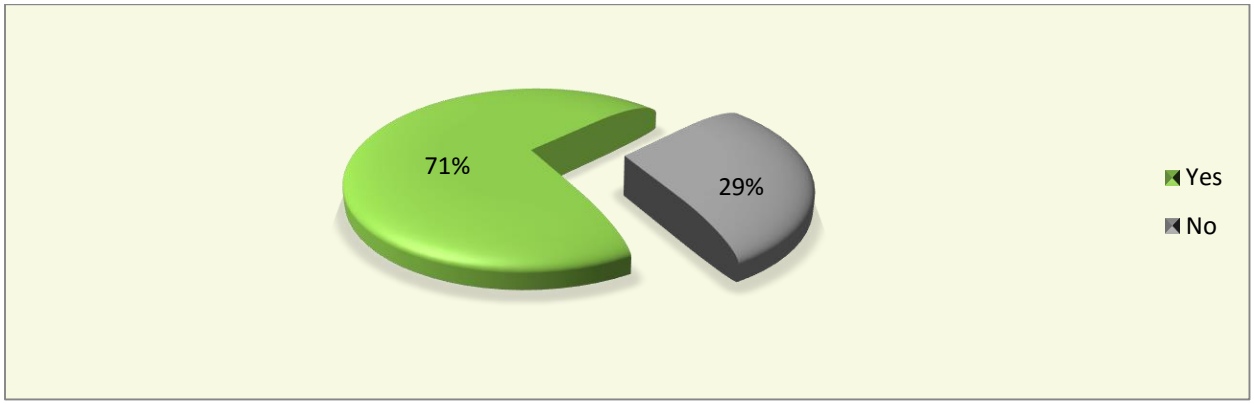

Fig 4.2.1: Knowledge about passive smoking

Most (71%) of the respondents knew about passive smoking and only 29% of the respondents did not know about passive smoking.

Passive smoking is the act of breathing in tobacco smoke produced by the others. Any person around an active smoker is a passive smoker (American Cancer Society, 2015).

4.2.2 Knowledge about Harms of Passive Smoking

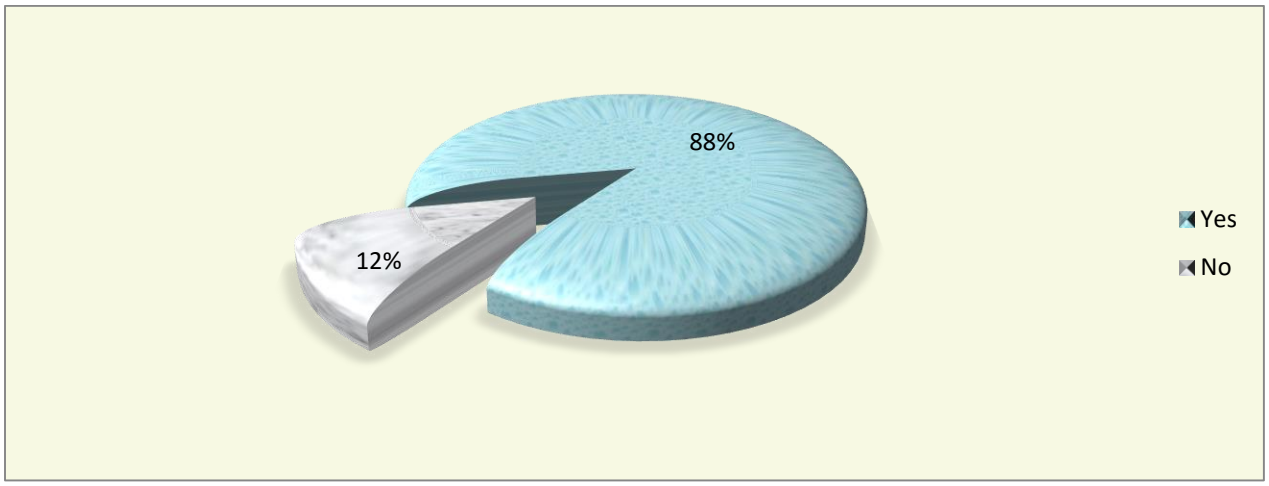

Fig 4.2.2: Knowledge about Harms of Passive Smoking

Most (88%) of the people knew that passive smoking is harmful, only 12% did not know this.

4.2.3 Effect of Passive Smoking

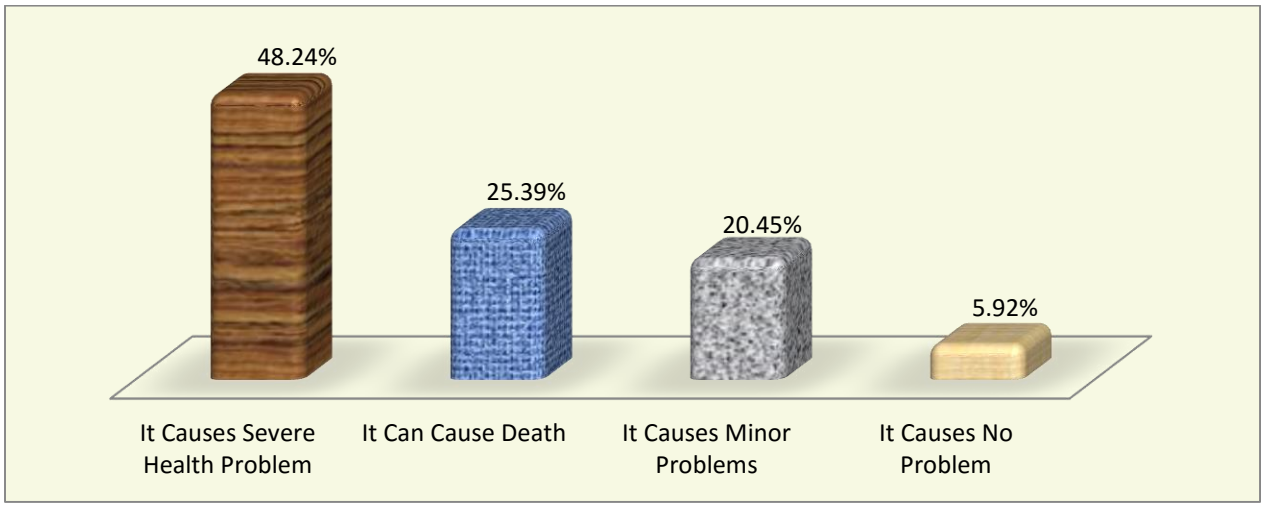

Fig 4.2.3: Effect of Passive Smoking

Majority (48.24%) of the people said that it can cause severe health problem, only 5.92 % respondents said it has no effect on health.

4.2.4 Breathing Problems

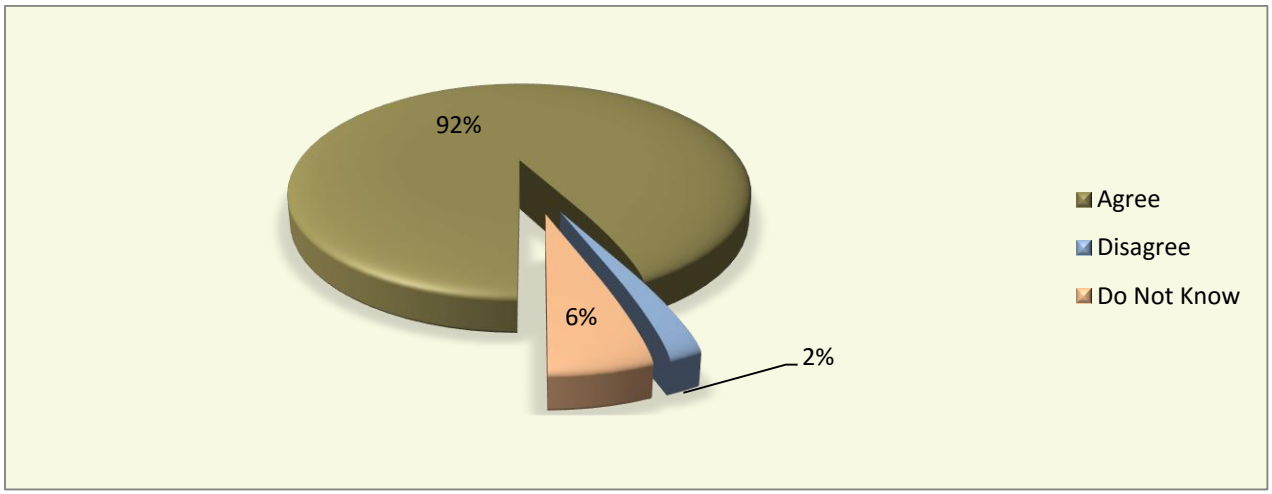

Fig 4.2.4: Breathing Problems

Most (92%) of the people agreed on the matter that breathing problem is associated with passive smoking. Only 2% of the respondents disagreed.

4.2.5 Lung Cancer

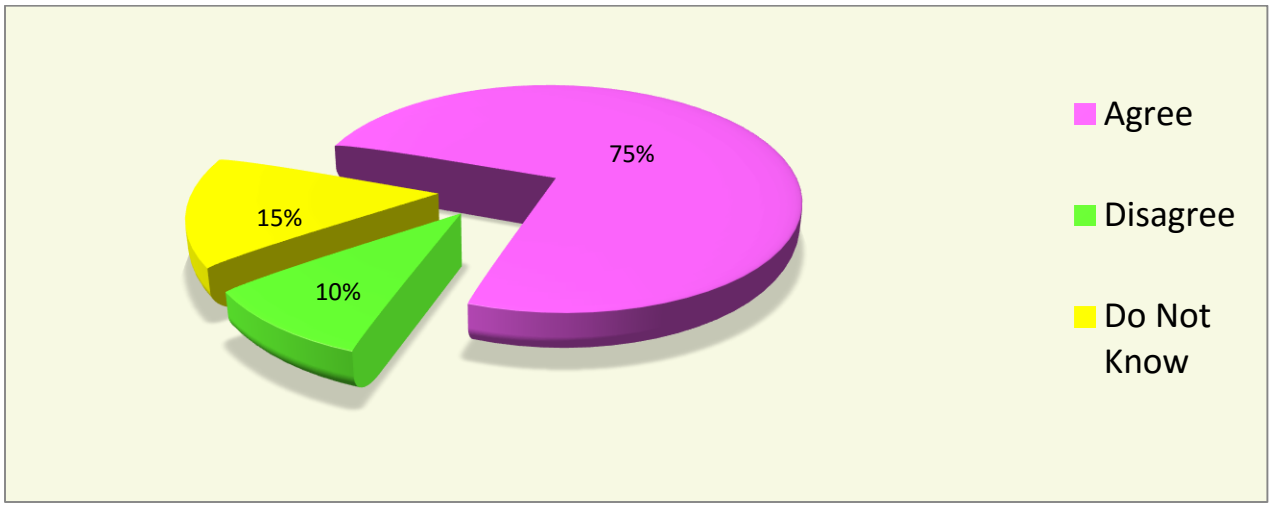

Fig 4.2.5: Lung Cancer

Most (75%) of the respondents believed that lung cancer is associated with passive smoking. Only 10% disagreed on this matter.

4.2.6 Oral Cancer

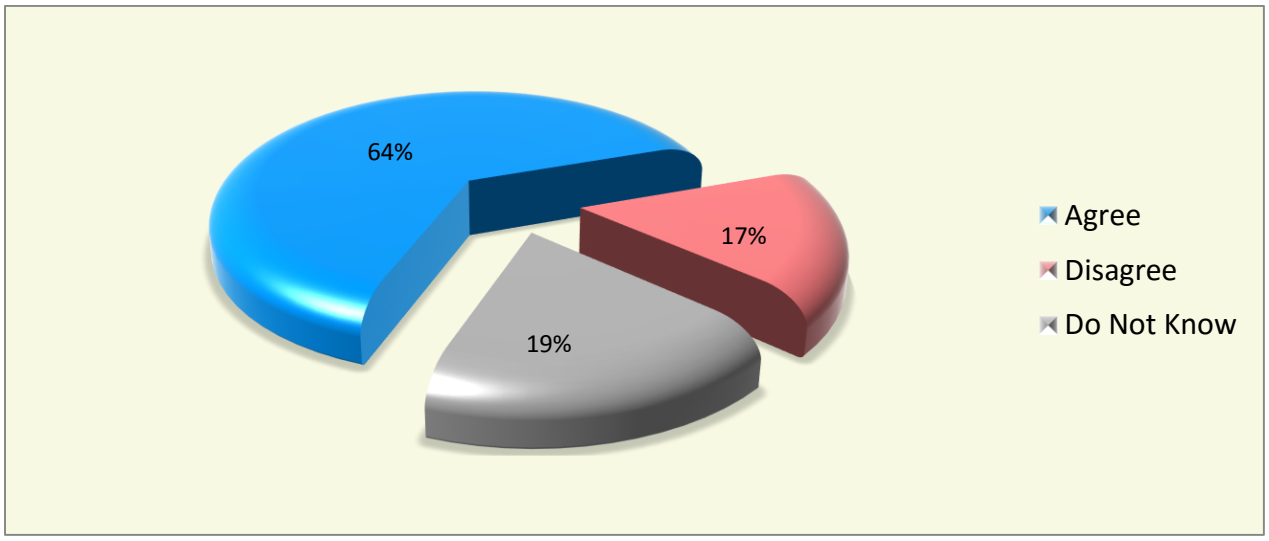

Fig 4.2.6: Oral Cancer

Most (64%) of the respondents agreed that lung cancer is associated with passive smoking. Only 17% disagreed on this matter.

4.2.7 Stained Teeth

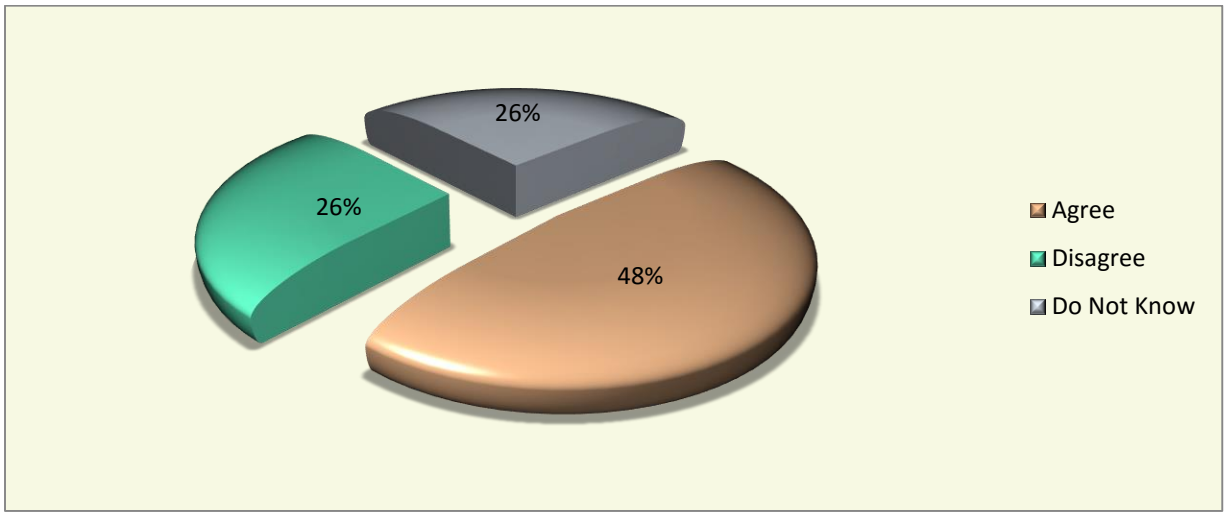

Fig 4.2.7: Stained Teeth

Most (48%) of the respondents said that passive smoking is associated with stained teeth. A good (26%) number of respondents disagreed on this matter.

4.2.8 Impotence

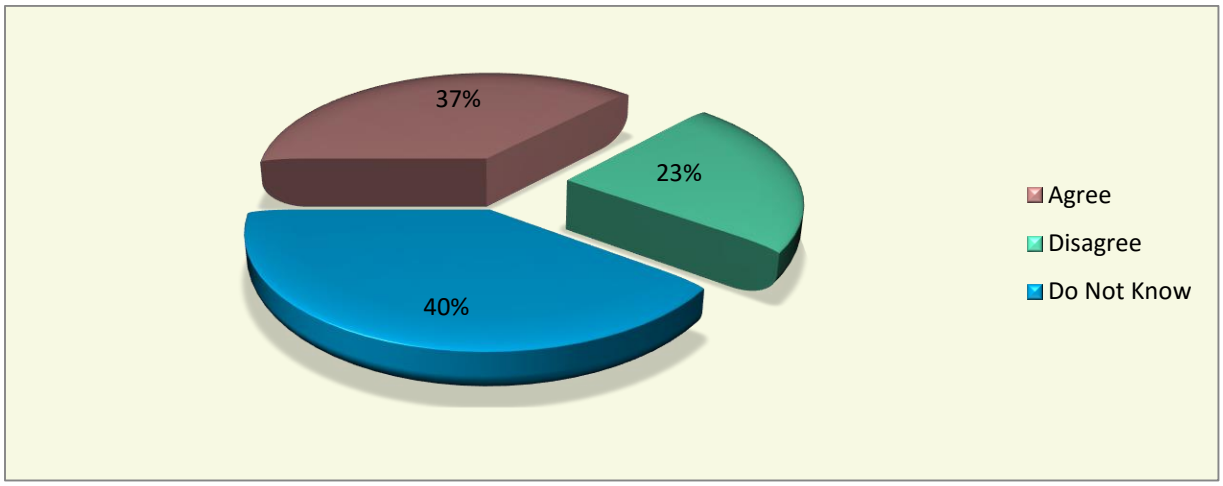

Fig 4.2.8: Impotence

Most (40%) of the respondents did not know that impotence is associated with passive smoking and 23% respondents disagreed.

4.2.9 High Blood Pressure

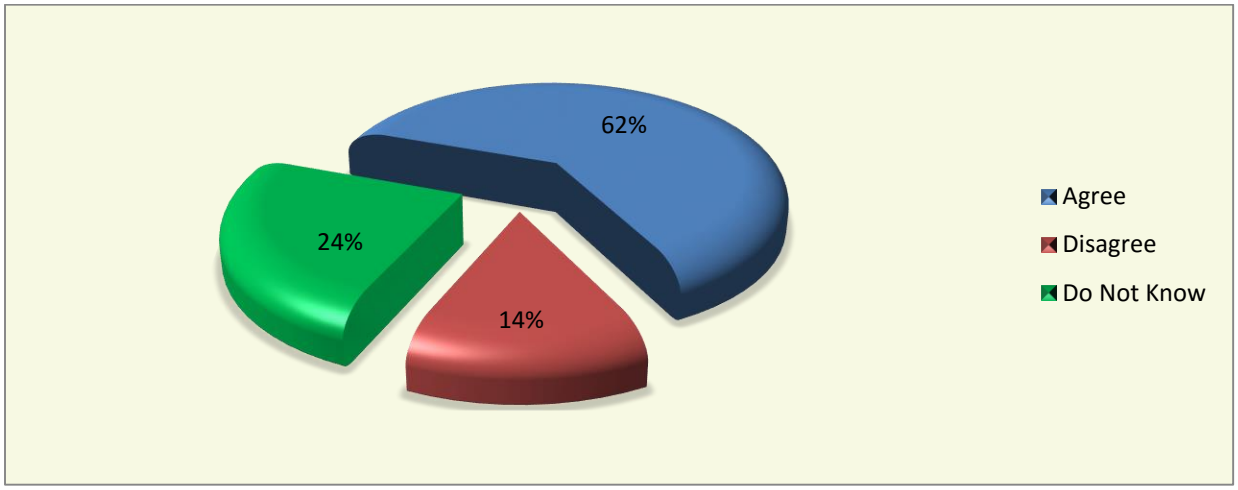

Fig 4.2.9: High Blood Pressure

Most (62%) of the respondents agreed that high blood pressure is associated with passive smoking. Only 14% respondents disagreed on this.

4.2.10 Heart Disease

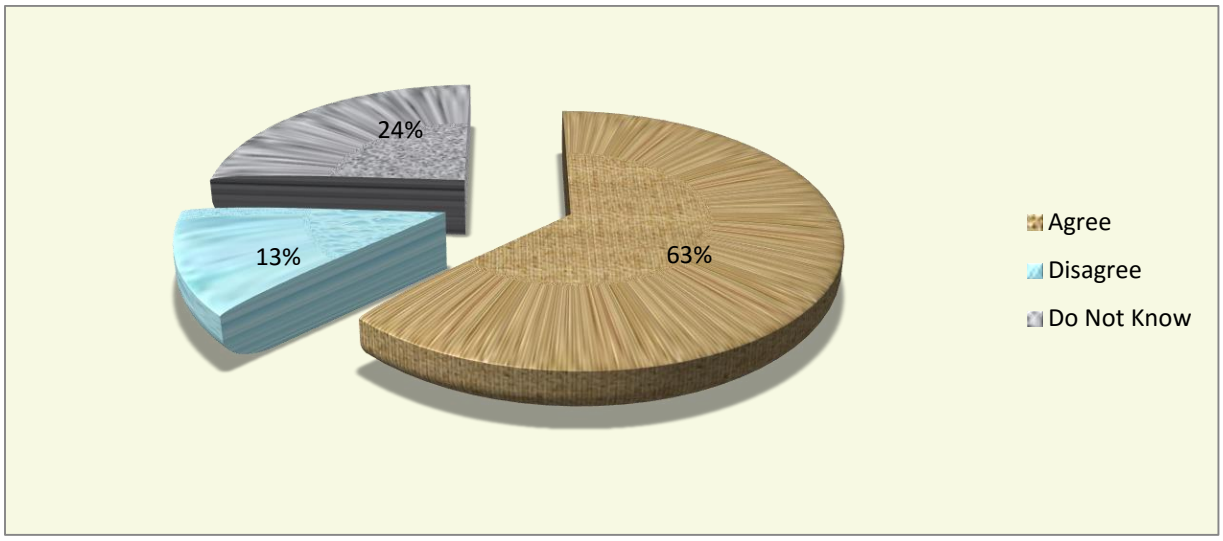

Fig 4.2.10: Heart Disease

Most (63%) of the respondents said that passive smoking is associated with heart disease. Minor (13%) number of respondents disagreed on this matter.

4.2.11 Eye Irritation

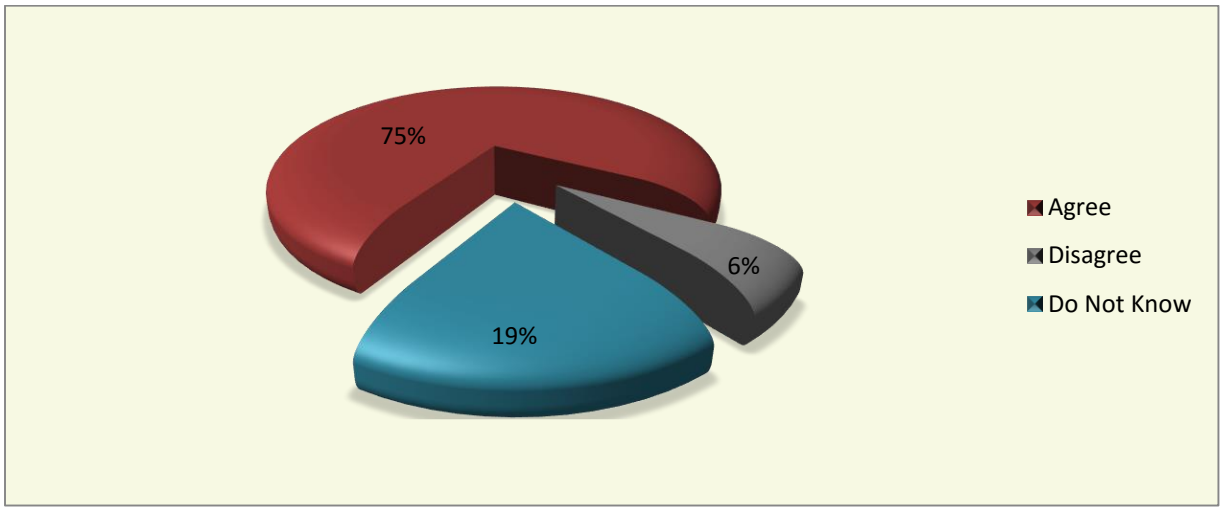

Fig 4.2.11: Eye Irritation

Most (75%) of the respondents agreed on the matter that eye irritation is associated with passive smoking.

4.2.12 Hearing Loss

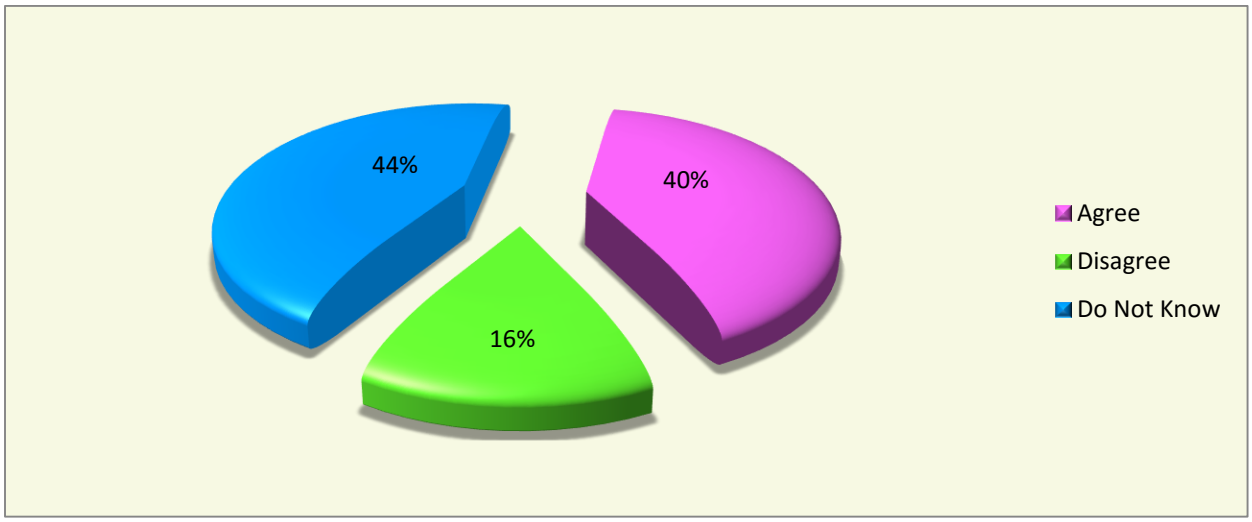

Fig 4.2.12: Hearing loss

Most (44%) of the respondents agreed that hearing loss is associated with passive smoking. Only 16% disagreed on this matter.

4.2.13Developmental Defects in Children

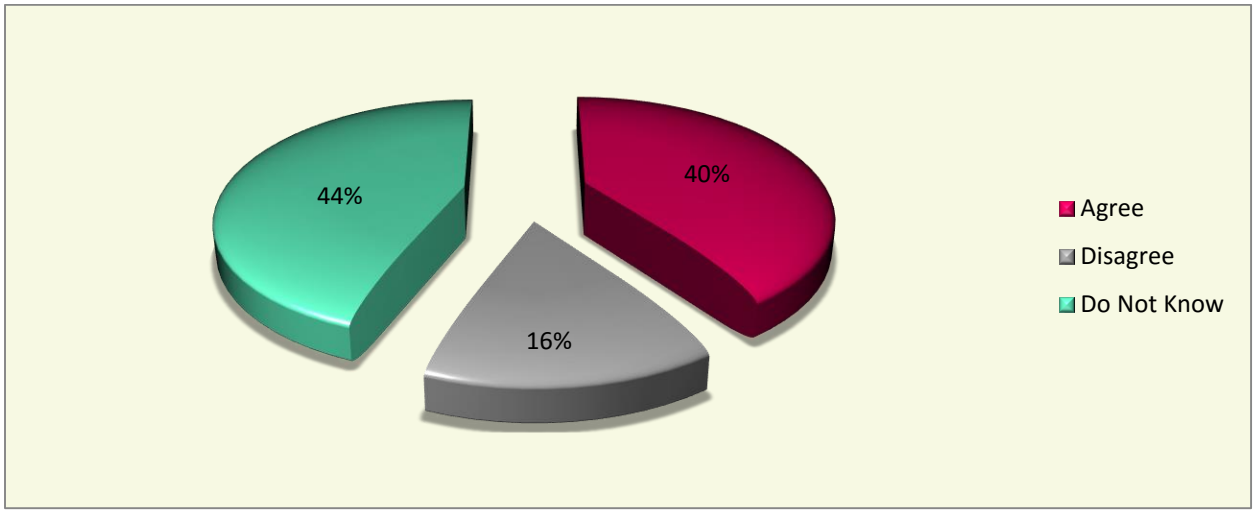

Fig 4.2.13: Developmental Defects in Children

Most (44%) of the respondents did not know either there is an association of passive smoking with developmental defects in children and 40% agreed on the association.

4.2.14Premature Birth

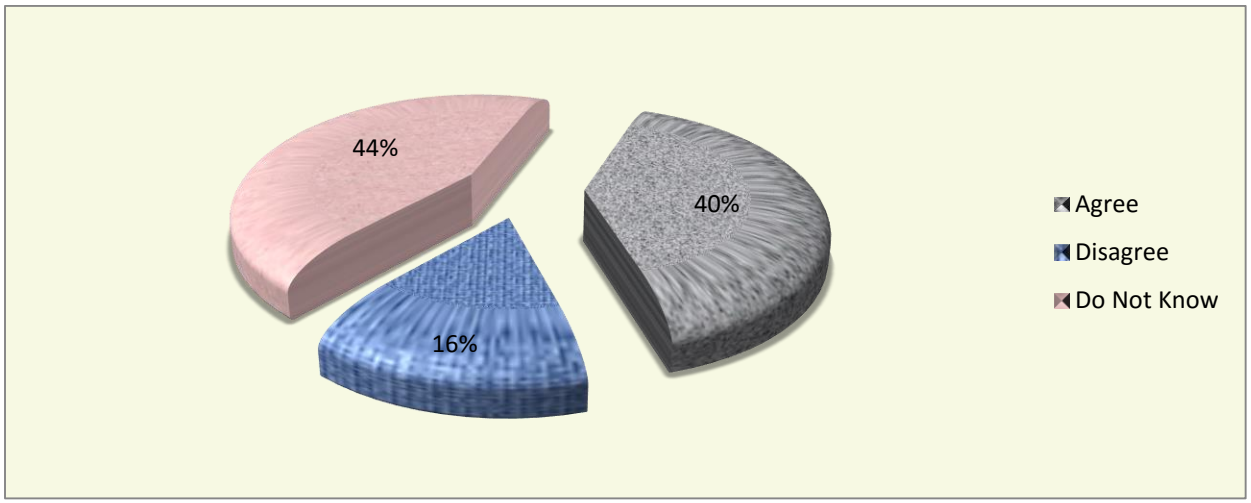

Fig 4.2.14: Premature Birth

Most (44%) of the respondents did not know either there is an association of passive smoking with premature birth and 40% agreed on the association.

4.2.15 Allergy

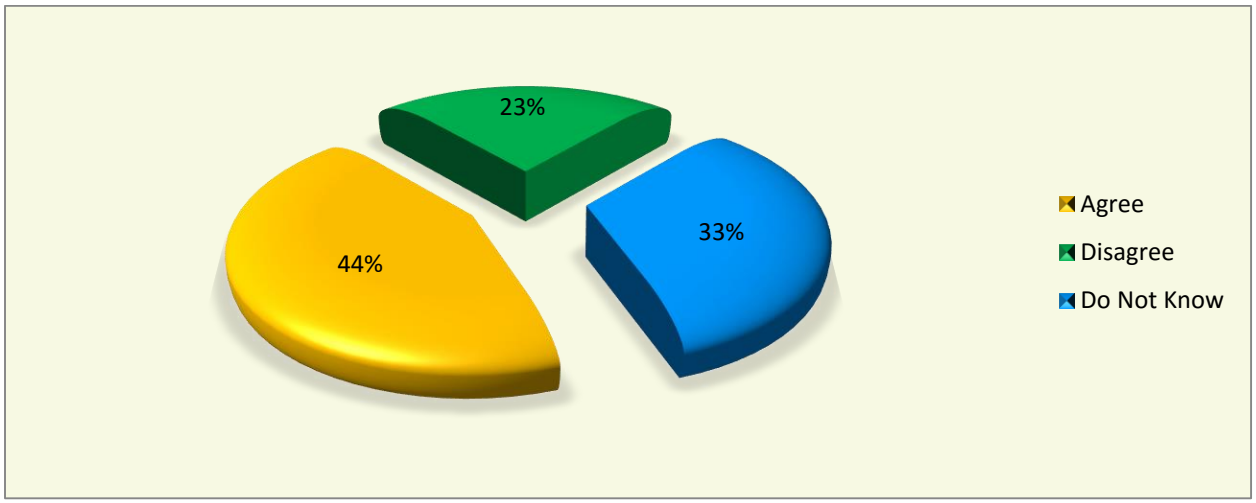

Fig 4.2.15: Allergy

Most (44%) of the respondents agreed that allergy is associated with passive smoking. Only 23% disagreed on this.

4.2.16 Asthma

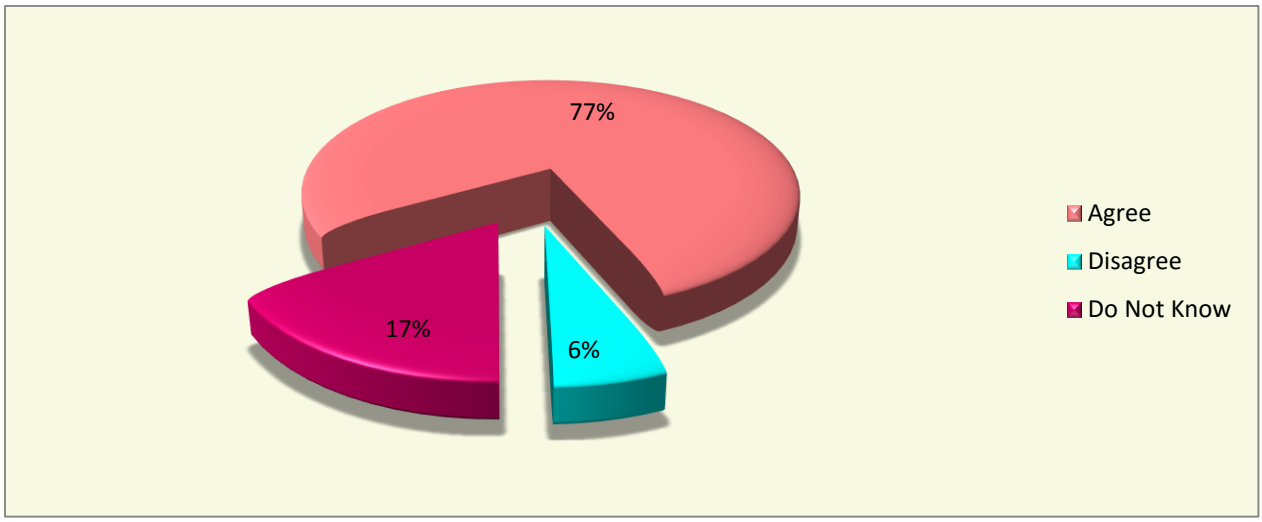

Fig 4.2.16: Asthma

Most (77%) of the respondents agreed that allergy is associated with passive smoking. Only 6% disagreed on this.

4.2.17 Tuberculosis

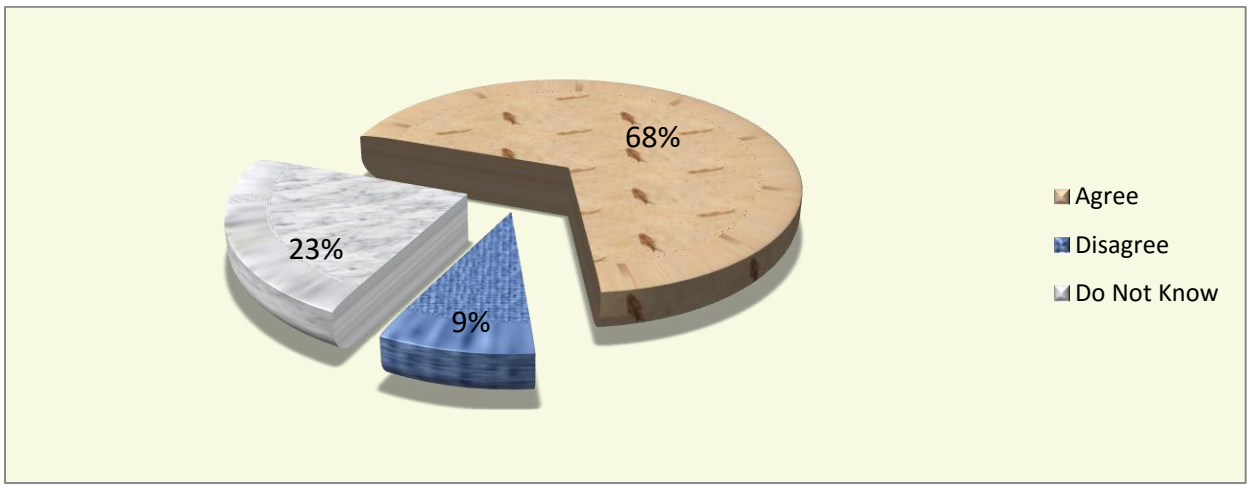

Fig 4.2.17: Tuberculosis

Most (68%) of the respondents agreed that allergy is associated with passive smoking. Only 9% disagreed on this.

4.2.18 Other Problems associated with Passive Smoking

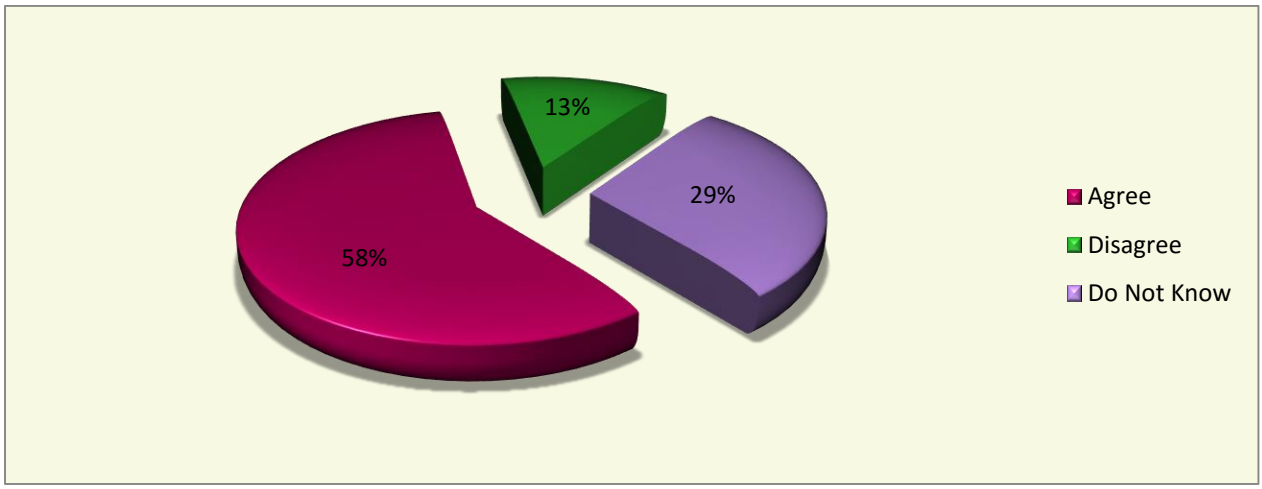

Fig 4.2.18: Other Problems Associated with Passive Smoking

Most (58%) respondents agreed that passive smoking is associated with other problems such as diabetes, ulcer etc. Only13% respondents disagreed with the concept that passive smoking is associated with other problems such as diabetes, ulcer etc.

4.2.19Exposure to Passive Smoking

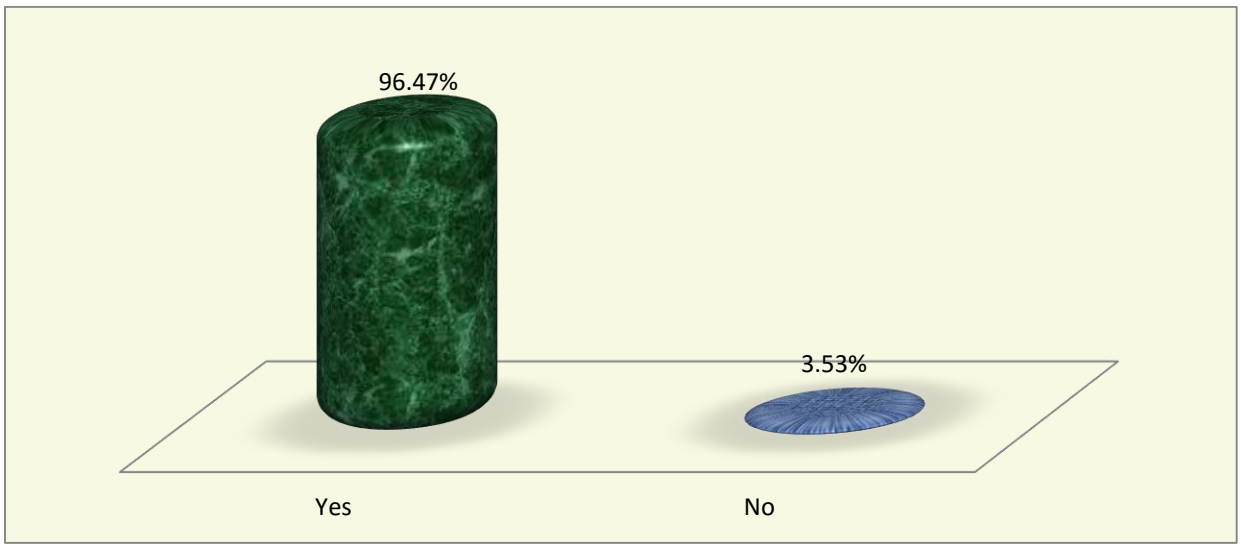

Fig 4.2.19: Exposure to Passive Smoking

Most (96.47%) of the respondents were exposed to passive smoking. Only 3.53% of the respondents did not get exposed to passive smoking.

4.2.20Area of Exposure

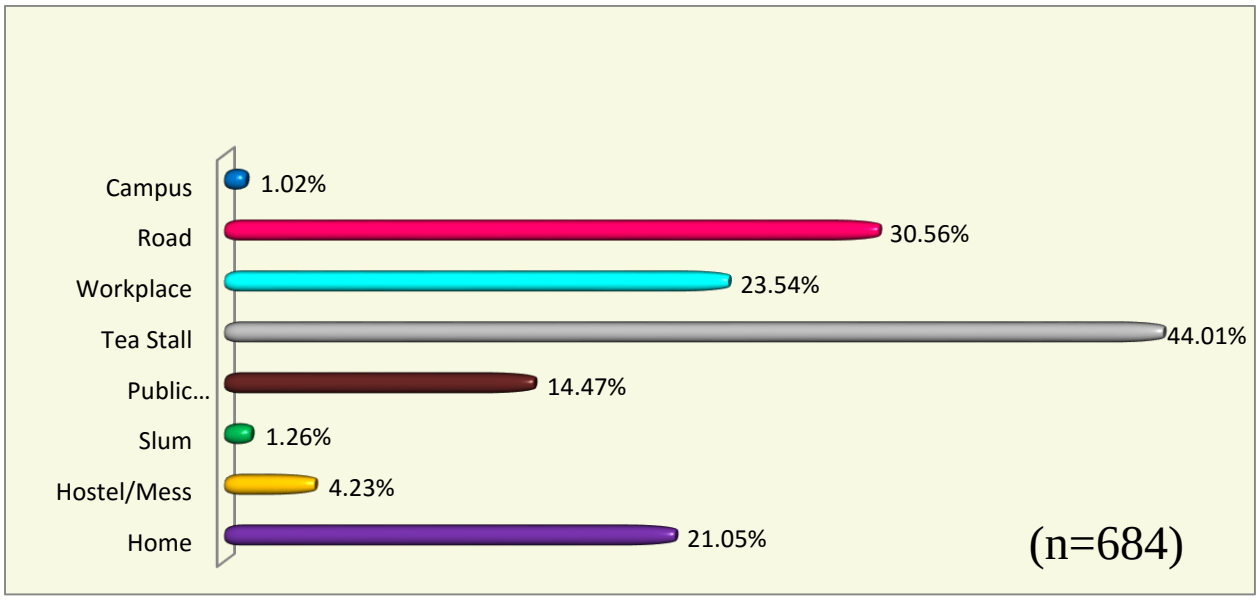

Fig 4.2.20: Area of Exposure

Most (44.01%) of the respondents get exposed to passive smoking at tea stall.Only 1.02% of the respondents got exposed to passive smoking in campus.

4.2.21Restriction in Area of Exposure

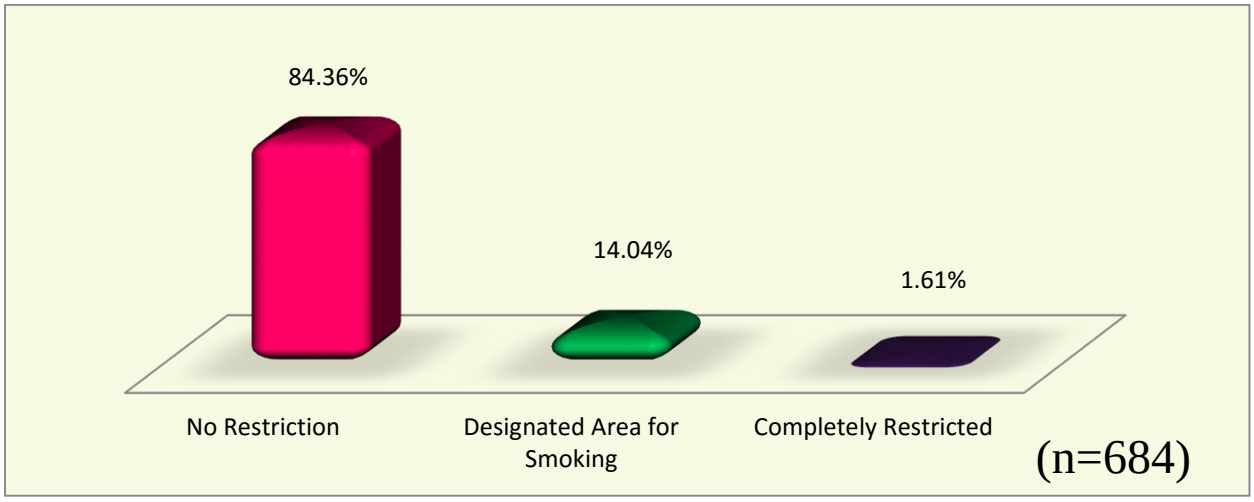

Fig 4.2.21: Restriction in Area of Exposure

In most (84.36%) cases there were no restriction in the area of exposure. In only 1.61% cases the areas were completely restricted.

4.2.22People Compliance with Smoking Restriction

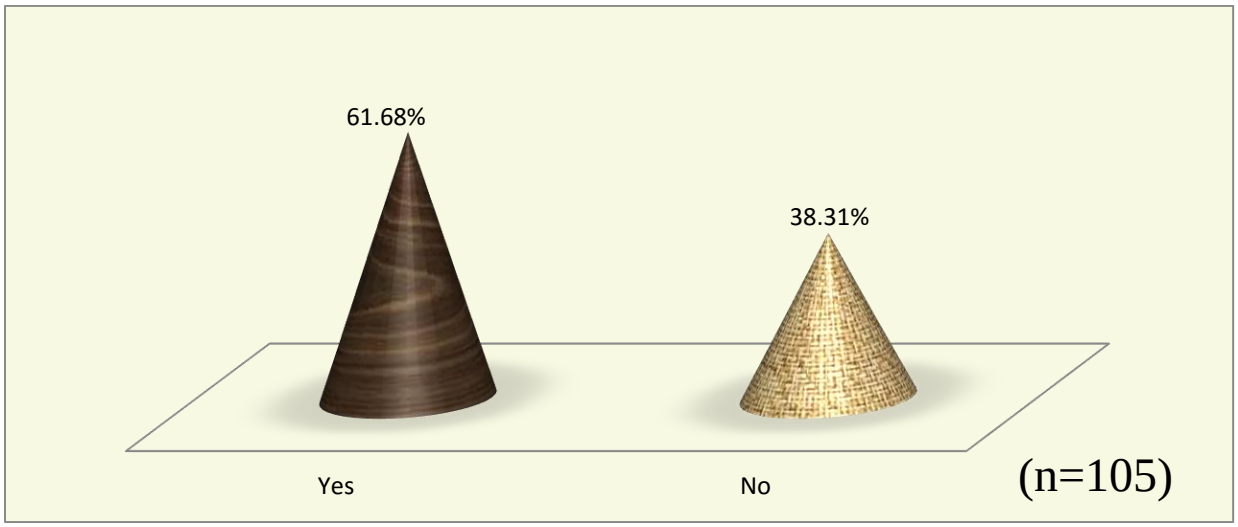

Fig 4.2.22: People Compliance with Smoking Restriction

In most (61.68%) cases smokers did comply with the restriction and in 38.31% cases they did not comply.

4.2.23Comfortableness in No-Smoking Zone

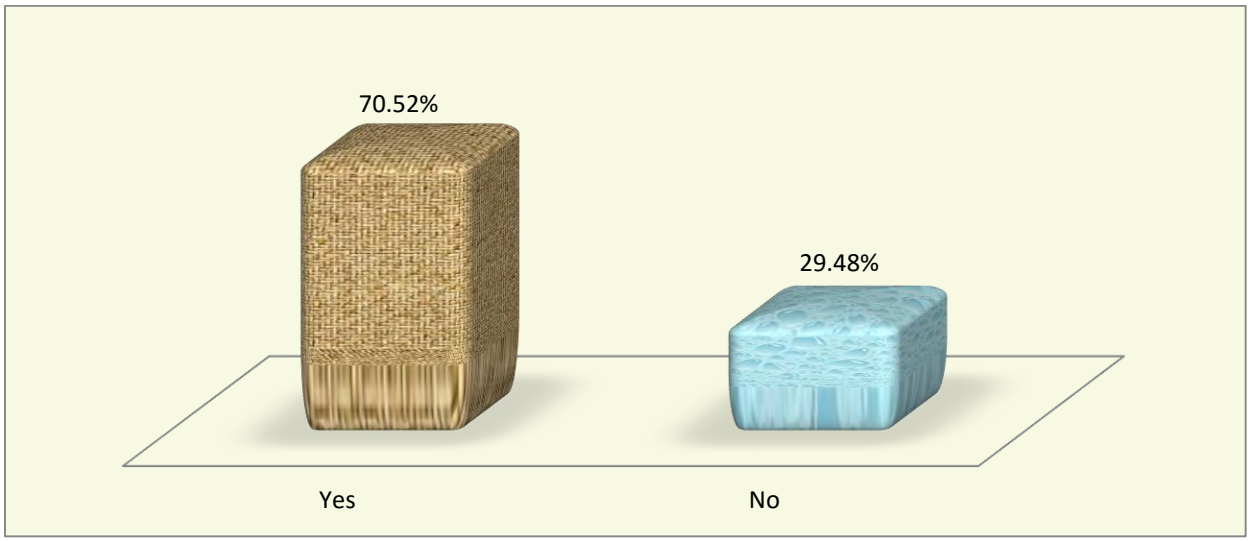

Fig 4.2.23: Comfortableness in No-Smoking Zone

Most (70.52%) of the respondents said that they felt comfortable in a no-smoking zone. Only 29.48% respondents said that they did not feel comfortable in a no-smoking zone.

4.2.24Botheration in Passive Smoking

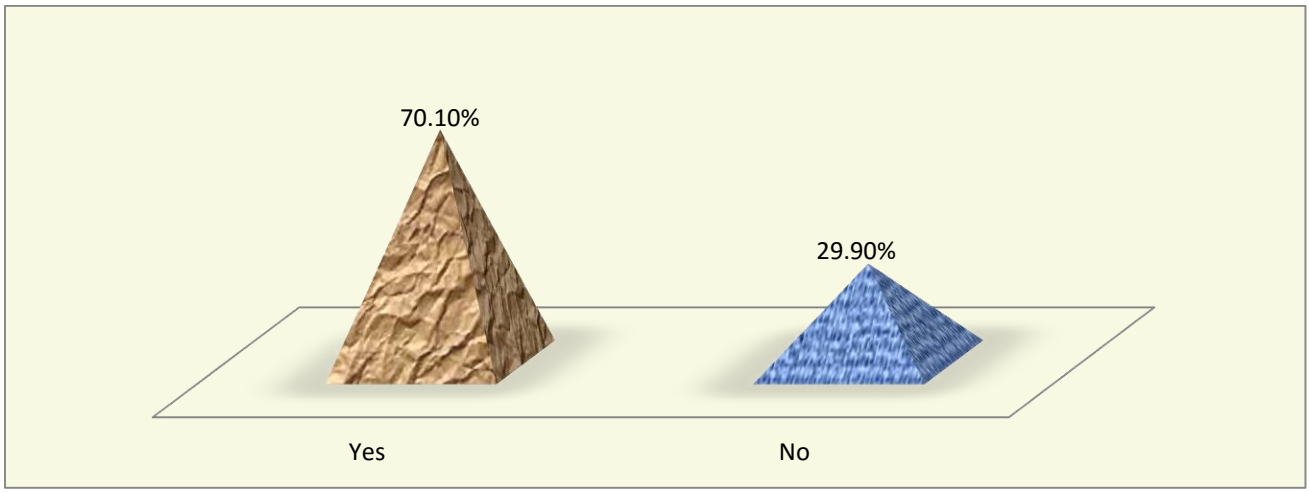

Fig 4.2.24: Botheration in Passive Smoking

Most (70.10%) people said that they felt bother when exposed to passive smoking.

4.2.25Way of Dealing with Passive Smoking

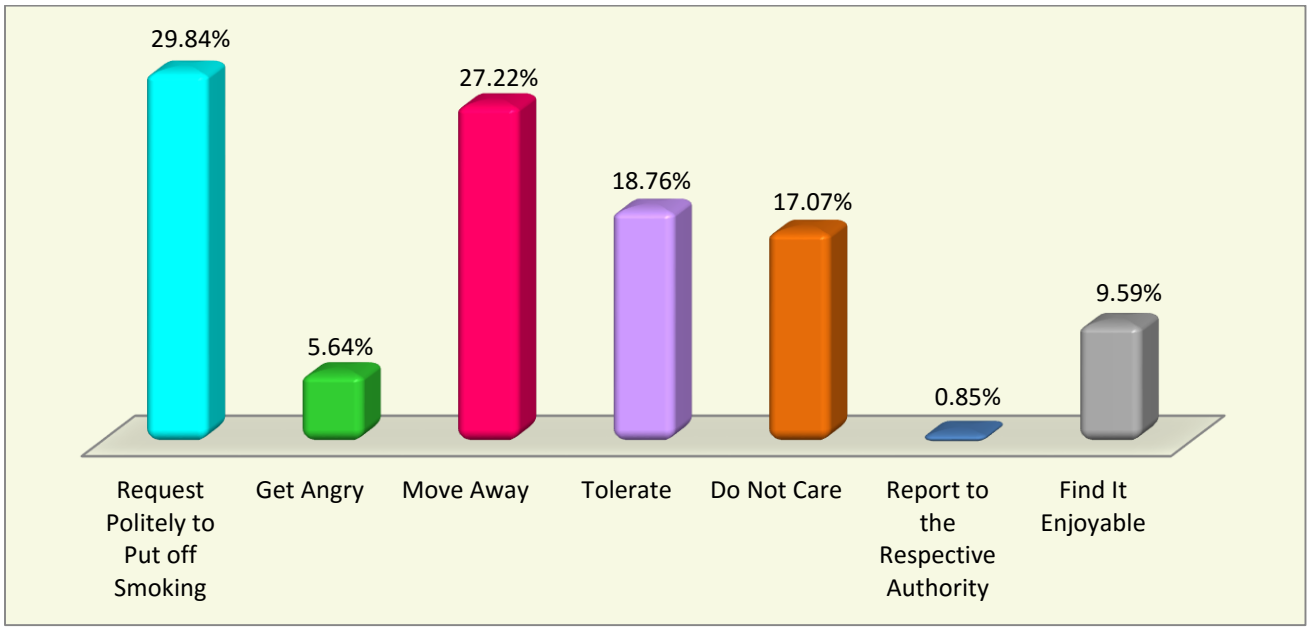

Fig 4.2.25: Way of Dealing with Passive Smoking

Most (29.84%) of the people said that they request the smokers politely to put the cigarette. Only 0.85% said that they report the respective authority.

4.2.26Restriction in Buying Cigarettes Aged Below 16

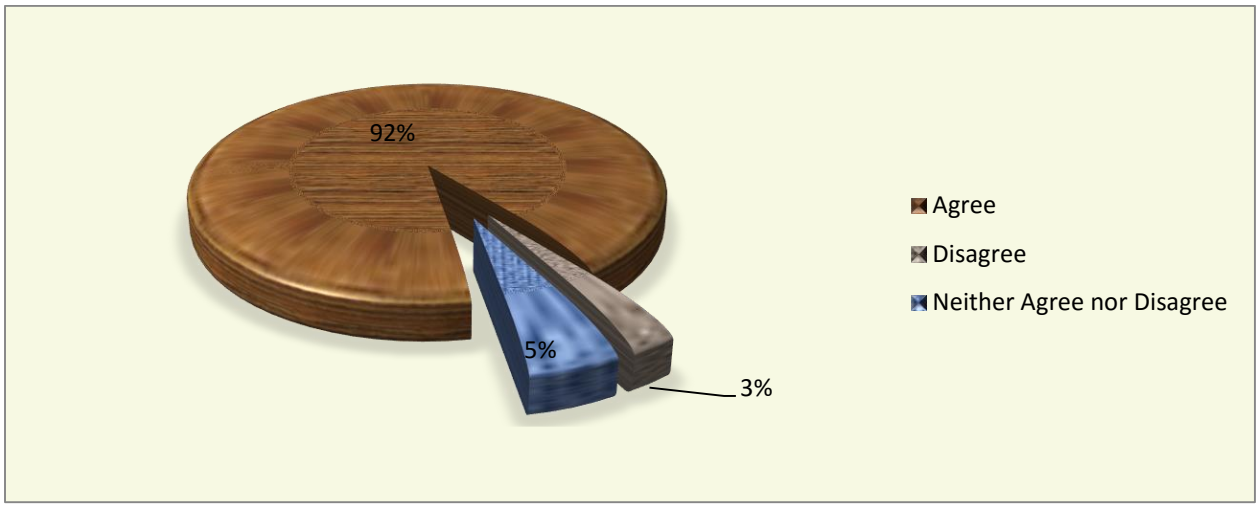

Fig 4.2.26: Restriction in Buying Cigarettes Aged Below 16

Most (92%) respondents agreed that no one under the age of 16 should be allowed to buy cigarette. Only 3% disagreed on this matter.

- **4.2.27Strict Law Enforcement to Stop Public Smoking**

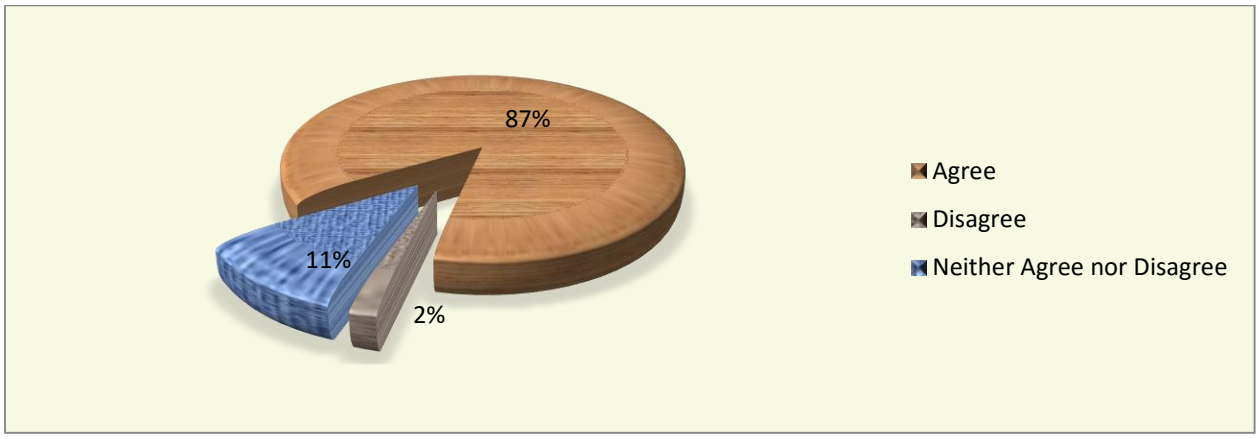

Fig 4.2.27: Strict Law Enforcement to Stop Public Smoking

Most (87%) respondents agreed that strict law should be enforced to stop public smoking.

Only 2% disagreed on this.

4.2.28Restriction in Smoking Advertisement

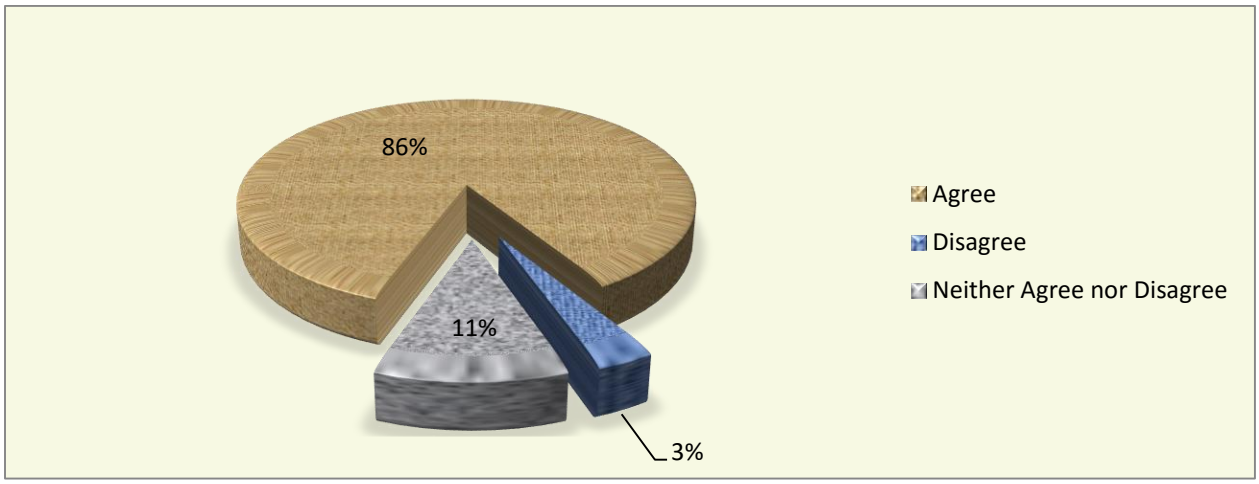

Fig 4.2.28: Restriction in Smoking Advertisement

Most (86%) respondents agreed that any advertisement about cigarette should not be presented in any form of media. Only 3% respondents disagreed on this matter.

4.2.29Organizing Awareness Programs

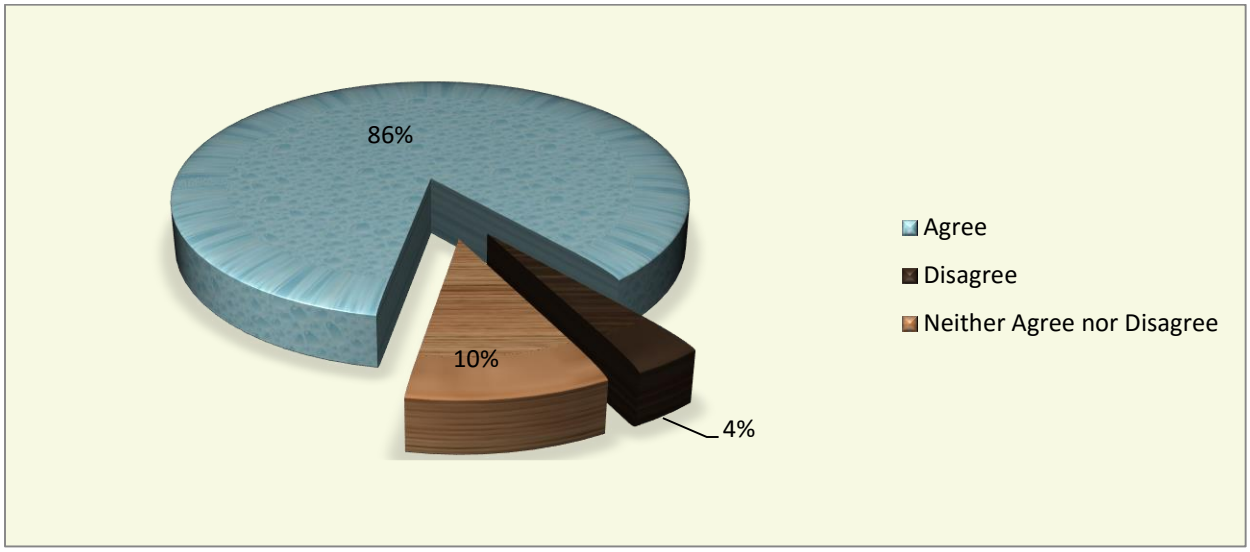

Fig 4.2.29: Organizing Awareness Programs

Most (86%) respondents said that awareness program should be organized to let people know about passive smoking. Only4% respondents disagreed on this matter.

4.2.30Input Information on Effects of Smoking in Academic Curriculum

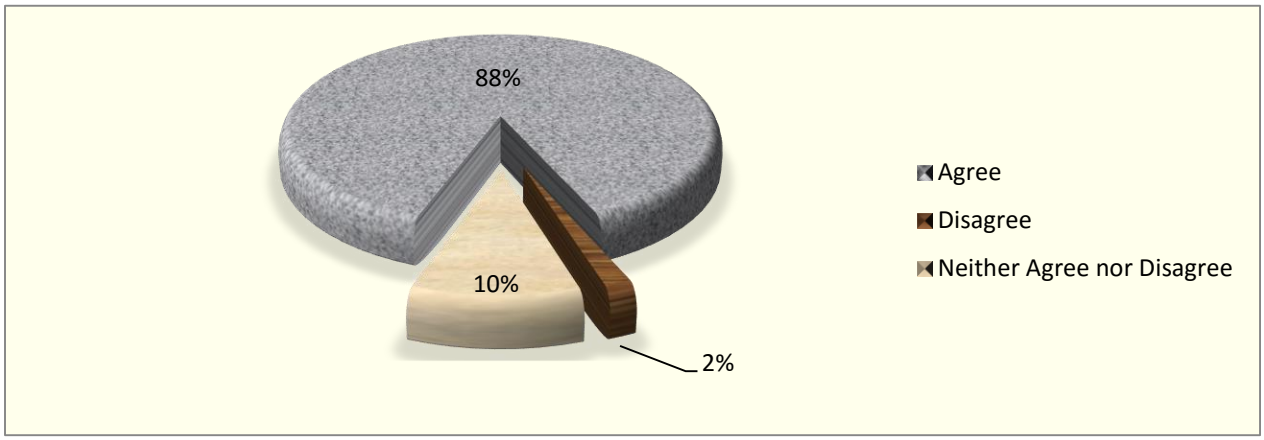

Fig 4.2.30: Input Information on Effects of Smoking in Academic Curriculum

Most (88%) respondents said that it would be good to add information about the passive smoking in the academic curriculum to raise awareness and to let the students know about the harmfulness of passive smoking. Only 2% respondents disagreed on this matter.

4.2.31Reasons of Smoking

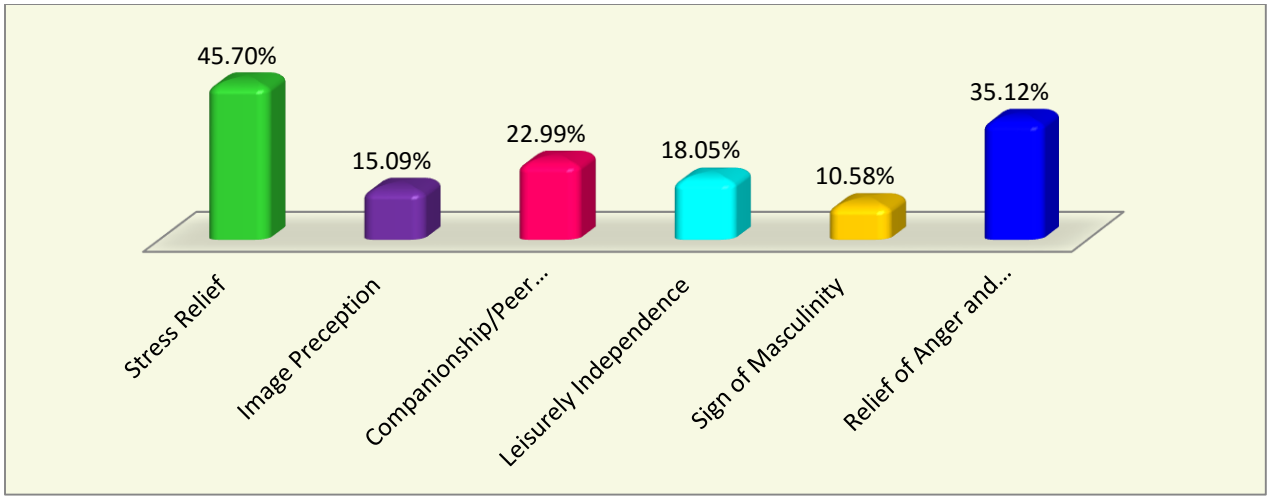

Fig 4.2.31: Reasons of Smoking

Most (45.70%) respondents thought that stress relief is the reason for smoking. Only10.58% respondents thought that sign of masculinity is the reason for smoking.

4.2.32Comparison between Smokers and Non-smokers on Public Smoking Ban

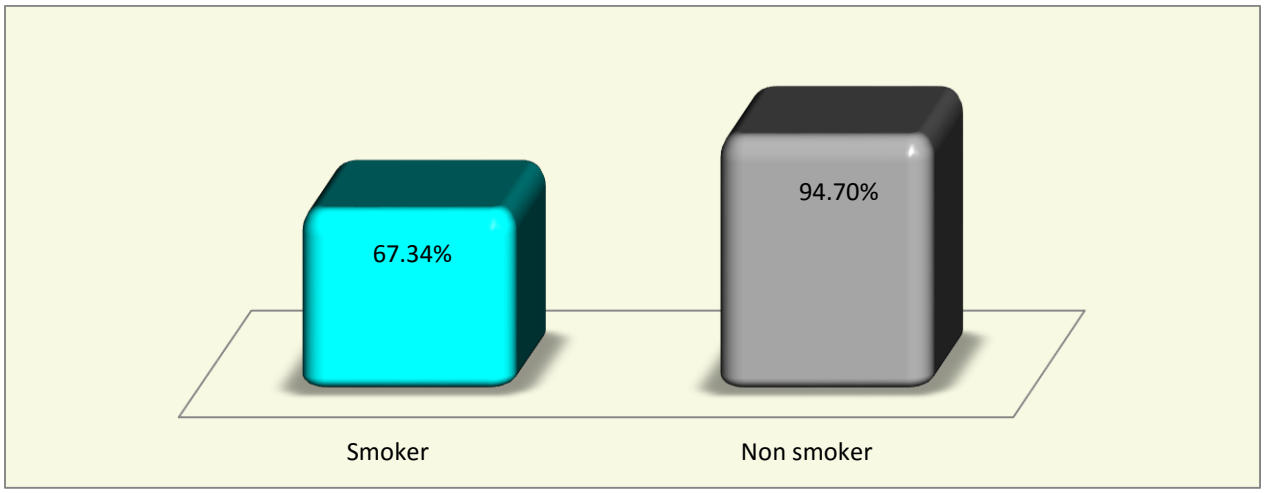

Fig 4.2.32: Comparison between Smokers and Non-smokers on Public Smoking Ban

Most (94.70%) of the non smoker person supported the ban of public place smoking. Among the smokers 67.34% supported the ban.

4.2.33Comparison between Smokers and Non-Smokers on Botheration in Smoke

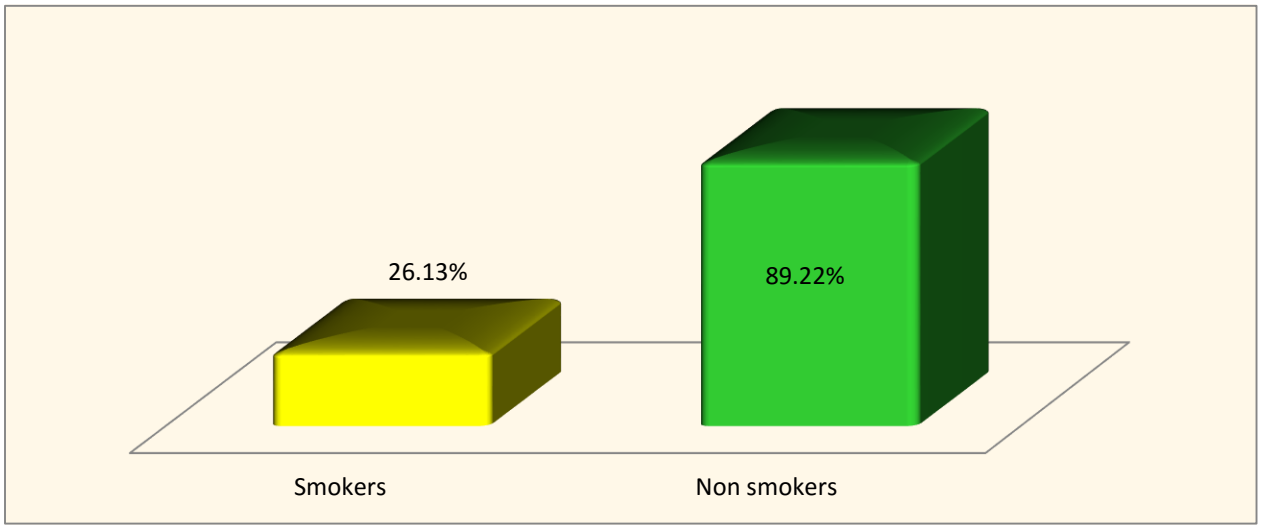

Fig 4.2.33: Comparison between Smokers and Non-Smokers on Botheration in Smoke

Most (89.22%) of the non-smoker felt bother in tobacco smoke. Only 26.13% smoker felt bother in passive smoke.

Chapter Five
Discussion & Conclusion

Discussion

Tobacco smoking has always been an issue of concern because it has destructive effect on health. Passive smoking can do the same kind of damage and even kill a person. Several studies have been conducted around the world, on the knowledge and attitude of people regarding passive smoking. In a study conducted inOsun state of Nigeria, 70.2% of the respondents had a good knowledge about passive smoking whereas in this study the percentage (71%) is higher. In Osun state studyamong the respondents 67.3% supported the ban of public place smoking and in this study 87% of the respondents supported it (Onigbogi *et al*., 2015).

In another study conducted in Australia, 87% of the respondents knew about passive smoking. On the other hand, in this study, 71% of the respondents knew about passive smoking (Makkai *et al.*, 1994).

In this study 88% of the respondents believed that passive smoking is harmful to health. On the other hand, in a study conducted in Bahrain, more than 80% responded knew that smoke from shisha or cigarette is harmful to other people around them (Al-Haddad, *et.al.*,2006).

In another study conducted on nurses, 100% of the respondent provided a positive response on the association of passive smoking and lung cancer (Zaborszczyket.al., 2009). In this study, 75% of the respondents agreed on the association of passive smoking with lung cancer.

According to the findings of this study 96.47% of the respondents were exposed to passive smoking and 70.10% respondents felt bother about that. All the respondents who were exposed to passive smoking, did not feel bothered. Only 28.07% of the respondents were current smoker, so it can be said that some smokers support the idea of banning the public place smoking. Most (87%) respondents supported the banning of public place smoking. Among the smokers 67.3% respondents agreed that smoking on the public places should be banned and 94.70% non-smokers agreed on this matter. Not only non-smokers but also smokers felt bother in passive smoke. Among the smokers 26.13% felt bother in passive smoke. Most people were exposed to tobacco smoke in the area where tobacco is sold (tea stall, 44.01%).

Conclusion

Passive smoking is a well-known fact to the general people of Bangladesh and most of them are well aware about the harms of passive smoking. Most of the people are exposed to passive smoking and most of them take steps to stop the smoke. Most of the people support anti-smoking initiatives. Thebiggest limitation of this study is that the respondent number is very low comparing with the total population. So the results of this study do not represent the whole situation.Further studies can be done on this topic by targeting larger population. Specific groups can be targeted based on their profession.

Chapter Seven References

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