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Smoking and Chronic Health Hazard: A Cross Sectional Study

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Abstract

Introduction: The World Health Organization predicts that by 2030, there will be more than 8 million tobacco-related deaths globally, with over 1 billion people already addicted to tobacco use and 5 million people dying annually from tobacco-related diseases. Smoking is already recognized as one of the main preventable causes of early death and disability worldwide and is a known risk factor for many chronic diseases. **Materials & Methods:** The purpose of this study, which was conducted via an online questionnaire, is to estimate the burden of disease caused by smoking in the Saudi population. In order to comprehend the prevalence of tobacco-related chronic diseases among smokers, a cross-sectional study was conducted. Without including foreign migrant workers, the research population was chosen from the Saudi population. **Results & Discussion:** In the smoker's group 27 out of 58 responders (46 %) reported that they have a family history of hypertension. Results analyzed show (OR) 3.425, 95% CI (1.7-6.7), Relative Risk 2.004 95%CI (1.358-3.078) P value 0.002. analyzed showers declared themselves with some gastric symptoms in the smoker's cohort and 11 (16.42%) people responded in the non-smoker's cohort (OR: 10.66, 95% CI (4.98- 22.19), RR: 4.122 95%CI (2.44- 7.288) P = 0.001 (Fisher Exact Test) Among smokers 45 people underwent angiography, out of which 6 people reported that there was some abnormality in the report. In the non-smoker group, 1 (4%) needed treatment for abnormal Angiography. RR 0.9 95% CI (0.81- 0.98). 58 % of the smoker demonstrated Hypertension (more than 130/85) with a mean age of 52.76(+/- 13.24), 6 % showed up with coronary artery disease and 67 % with chronic gastric symptoms. Besides this 13% had bronchitis, 16 % had breathing difficulties, 10% showed liver enzyme disorder and 18 % showed an altered lipid profile. **Conclusion:** Although our study has many limitations, it clearly demonstrates a relationship between chronic hypertension, gastrointestinal symptoms, and coronary artery disease. Liver function tests and diabetes do not correlate well. The general public needs to be made more aware

of the need to stop smoking to avoid an unexpected and preventable effects soon. More money spent on anti-smoking initiatives will encourage young folks to give up smoking and lead healthier lives.

Keywords: Smoking, Secondhand smoke, Hypertension, Gastric symptoms, Coronary artery disease.

Background

The culture of burning Tobacco and the resulted the smoke that is inhaled so that it can be tasted and taken into the bloodstream has been popular since last century (Wang et al., 2019). According to World Health Organization estimates, if this current trend continues, there will be over 8 million tobacco-related deaths worldwide by 2030, with over 1 billion individuals already addicted to tobacco use and the current death from tobacco-related disease is 5million per year (Almutairi, 2014; Rezk-Hanna et al., 2018).

Smoking is a recognized hazard issue for numerous long-lasting diseases and is already acknowledged as one of the foremost preventable reasons of premature deaths and morbidity in the world. (Liu et al., 2017). Despite cigarettes' terrible effects on our health, many people continue to use it long after it is too late. Almost a billion people smoke worldwide. The fact that these one billion people put a few more million people at risk on top of themselves is disturbing (Carreras et al., 2021).

Secondhand smoke (SHS) is typically present in both the workplace and the home for adults. SHS exposure is a significant burden in EU countries. South Eastern EU nations showed the largest burden (DALY 0.50%/0.70%), whereas Northern EU countries showed the lowest burden (with a proportion of DALY/deaths lower than 0.25%/0.34%). The burdens of chronic obstructive pulmonary disease (COPD), lung cancer, ischemic heart disease (IHD), breast cancer, asthma, stroke and diabetes were assessed according to gender in an original research study (Öberg et al., 2011).

Globally, secondhand smoking (SHS) exposure (2004) harmed 33% of male, 35% of female, and 40% of children. This exposure resulted in 603,000 fatalities in 2004 (1.0% of all deaths globally), 21400 deaths from lung cancer, 36900 deaths from asthma, 165,000 deaths from lower respiratory infections, and 379,000 deaths from ischemic heart disease. children (28%), males (26%), and women accounted for 47% of those who died from secondhand smoke. The three disorders with the highest illness burden were lower respiratory infections in children under the age of five (5,939,000), ischemic heart disease in adults (2,836,000), and asthma in adults and children (1,246,000) (Bassiony, 2009).

In Saudi Arabia, the prevalence of current smoking patterns ranges from 2.4 - 52.3% (median 17.5%); among school-age children, it is between 12.5 - 29% (median 16.5%); among females, it is between 2.4 - 37% (median 13.9%); among males, it is between 11.6 - 52.3% (median 22.6%); and among older people, it is 25%. In the male population as a whole, 13-38% of men smoke (median 26.5%), compared to 1-16% of women (median 9%) (Rai, Kafle, & Makaju, 2022).

A multistage nationally representative Saudi Health interview survey found that adolescents of 15 years and older, 12.1% of all Adults smoke cigarettes, with 21.5 % of men and 1.1% of women. Smokers consume an average of 15.0 cigarettes per day.

1.4% of females and 20.9% of males regularly consume shisha. and 0.5% of men and 0.2% of women regularly use goods that don't contain tobacco. Around age 18.7, Saudi Arabians start smoking (males at 19.0 and females at 21.6) In general, 60.9% of smokers started before they turned 18, and 29.7% started before they turned 15. In Saudi Arabia, people aged 55 to 64 have the highest percentage of current smoking (15.6%) (Grassi et al., 1994).

Materials and Methods

The study was steered from Jan 2021 to December 2021. It was an online questionnaire-based survey so no ethical approval was necessary from the institution (University of Shaqra). The aim of this study is to enumerate the liability of disease due to direct effect of smoking in the Saudi population. It was a cross sectional study to understand the occurrence of tobacco related chronic diseases among the smokers.

There are 10 tobacco-related diseases that have been recognized; the diagnosis of a tobacco-related chronic disease and the effect of quitting smoking have been determined and measured, respectively. Data was gathered for this reason using a questionnaire. The questionnaire includes demographic data along with details on 19 chronic diseases linked to tobacco use. The questionnaire's second section contains information about cigarette consumption (derived from CDC, NIHS).

Sampling population

The study population has been selected from Saudi population excluding migrant workers from other countries. the study will take place initially from the employees of the Shaqra university and their family members and gradually it will extend over other people who are Saudi national and living in other cities in Saudi Arabia. This will be a cross sectional study with quantitative and qualitative analysis on SPSS software with age and gender stratified and specific. It defines a smoker as a person, who smokes a cigarette per day for at least 6 months

Sample size

The sample size was determined using the formula $N= Z^2. P (1-P)/e^2$, where P=prevalence was taken as 0.3 (30%) for the maximum study indicating a prevalence rate of hypertension in smokers of 30%, and Z= 1.96 for 95% CI. As the margin of error, e = (0.05), is assumed to be 5%, $N = (1.96)^2.0.3.0.7 / (0.05)^2 = 322$

Data collection

A modified Arabic translation of the standard WHO questionnaire was used to obtain information on the prevalence of smoking among health workers. Based on the answers to the questions, current smokers were identified. Information will be treated in utmost confidence and the questionnaire was anonymous. These self-reported responses were reviewed using a computer to look for any consistency issues.

Statistical Analysis

Data from the Excel master spreadsheet was entered in Graphic Pad Prism version 9 for analysis. At a P value of 5%, the link between the categorical variables was statistically significant.

The odds ratio (OR) and relative risk (RR) were estimated simultaneously using the Koopmans Asymptomatic Score and Baptista-Pike technique.

Results

In our study, a total of 166 subjects took part in an online survey, out of which we got a total of 99 current smokers and 67 non-smokers. Among the cohort of 99 smokers 78 were regular smokers and 21 were occasional.

Demographic Analysis

Out of 67 non-smokers in our study, 7 responders said that they were past smokers, and 17 said they were neither smoking nor were they exposed to any passive smoking. Regarding second-hand smoke, participants said that they were exposed to passive smoking regularly and the remaining 31 said that they were exposed to passive smoking occasionally.

In the smoker's group, 93 were male and 6 females admitted to being smokers [n 30(<40), n 45(41-50), n31 (51-60)]. 48% had a doctorate degree, 25% had a master's, and 27% were graduates. 90 subjects were from the Saudi community and 9 were from other Arab communities. 57 people live with spouses and children and 6 responders declared that they live alone. 57 candidates were employed and 42 candidates were self-employed. 87 candidates were married and the rest 12 were unmarried**.** 81 are past active smokers and 18 are recent smokers. 87 smoke conventional cigarettes, 3 smoke Sisha (water pipe) .36 started their initial smoking from the age between 15-20 years and 45 started between 21 -25 years .18 smoked 11-20 cigarettes per day, 30 smoked 6-10 cigarettes per day, 3 smoked more than 20 cigarettes per day. 54 confessed that they tried to quit but could not, and resumed after some time. 30 people attempted to quit 5 times or more. 15 people knew smoking is injurious to health and that is why they quit and 18 people quit because of some other reason.

Table 1

Investigations for Smoker & Non smoker

Profile (**Smoker**)	CBC	TFT	KFT		Angiography		LFT		Lipid profile		Blood sugar	
Normal	54	51	60	60%	39	39%	53	54%	57	58%	63	63%
Slightly abnormal	3	0	0	0	3	3%	10	6	18	18	06	6%
Markedly abnormal	0	0	0	0	0	0	0	0	0	0	30	30%
Not done recently	42	48	39	40	55	55%	36	36	24	24	10	10%
Profile (**Nonsmoker**)	CBC	TFT	KFT		Angiography		LFT		Lipid profile		Blood sugar	
Normal	46	43	43		27	40%	44	64%	34	49%	33 (49%)	
Slightly abnormal	0	0	2		01	1%	03	6	14	21	19 (28%)	
Markedly abnormal	0	1	0		0	0	00	0	0	0	4 (5%)	
Not done recently	21	23	21		39	58	30	30	19	28	10 ((15%)	

S=Smoker: NS=Nonsmoker, PS=Passive smoker

Comparison between investigations between smoker and nonsmokers

	Angiography		LFT		Lipid profile	
	Sm	Nsm	Sm	Nsm	Sm	Nsm
Normal	39 (87%)	9 (96.5)	53 (84%)	44 (93.6%)	57 (76%)	34(71%)
Sl/ Abn	6 (13%)	01 (3.5%)	10 (15.87)	3 (6.4%)	18(24(%)	14(29.1%)
M/abn	0	0	0	0	0	0
ND recently	55	58	36	30	24	19

Sl/abn: Slightly Abnormal: M/Abn: Markedly abnormal: ND recently: Sm =Smoker /Nsm: Non smoker

Table 2

Disease spectrum for Smokers & Non smokers

	HT	CHD	Brain attack	Diabetes	bronchitis	Asthma	Gastritis	Hepatitis	cancer	Breathing difficulty	Cough	Wheezing
NS	19	2	0	15	0	3	11	1	0	2	5	1
F/H	13	3	0	11	0	3	1	0	3	0	1	0
Smoker	58	6	6	36	13	9	67	6	6	16	24	12
F/H	27	0	0	24	3	3	0	0	0	3	3	3
	NS = Non- smoker, F/H family history											

Table 3

Disease and investigations

Disease status	Disease (+)	Disease (-)	Not done recently	Result
1.Hypertension				OR: 3.425, 95% CI (1.7-6.7), RR: 2.004 95%CI (1.358-3.078) P = 0.002 (Fisher Exact test)
Smoker (n99)	**58**	**41**		
Non- smoker (n67)	**19**	**46**		
2.CAD				OR: 2.097, 95% CI (0.5-10.44), RR: 2.030 95%CI (0.487-8.654) P = 0.364 (Chi square test)
Smoker	**6**	**93**		
Non smoker	**2**	**65**		
3.Chronic Gastritis				OR: 10.66, 95% CI (4.98- 22.19), RR: 4.122 95%CI (2.44- 7.288) P = 0.001 (Fisher exact test)
Smoker	**67**	**32**		
Non smoker	**11**	**56**		
4.Diabetes				OR: 1.98, 95% CI (0.99- 4.034), RR: 1.624 95%CI (0.98- 2.747) P = 0.0612 (Fisher exact test)
Smoker	**36**	**63**		
Non smoker	**15**	**52**		
5. liver function test				OR: 0.33, 95% CI (0.12 – 0.90), RR: 0.89, 95%CI (0.79 – 0.98) P = 0.02 (Fisher exact test)
Smoker	**10**	**53**	**36**	
Nonsmoker	**3**	**44**	**20**	
6. Lipid profile				OR: 1.312, 95% CI (0.7067 – 1.280), RR: 1.075, 95%CI (0.9074 – 0.98) P = 0.4260 (Fisher exact test)
Smoker	**18**	**57**	24	
Nonsmoker	**14**	**34**	**19**	
7. Angiography				OR :0.27, 95% CI (0.096 – 0.87), RR: 0.9, 95%CI (0.81- 0.98) P = 0.02 (chi square test) (significant
Smoker	**6**	**39**	54	
Nonsmoker	**1**	**27**	**39**	

Pulmonary symptoms	Smoker		Non smoker		Analysis
	Dis +	Dis -	Dis +	Dis -	
Bronchitis	13	86	0	67	RR inf OR inf P = 0.002
Asthma	9	90	3	64	RR 2.0 (0.6- 6.7) OR 2.1(0.5-7.5) P= 0.2
Breathing Difficulty	16	83	2	65	RR 5.4 (1.4-20.8) OR 6.2 (1.6-28.1)P= 0.007
Cough + Mucous	24	75	5	62	RR 2.0 (0.6- 6.7) OR 2.1(0.5-7.5) P= 0.2
Wheezing	12	87	1	66	RR = 8.1 (1.4- 48.2) OR = 9.1 (1.5-98.9) P=0.01

Results of investigations

Out of 99 smokers responded, 58% people declared themselves as hypertensive, who were on regular medications and 41 people were normotensives. 27 out of 58 responders (46 %) reported that they have family history of hypertension. Among non-smokers (19) 30 % of the responders reported that they are hypertensive and 46 (68%) of them are normotensives. Results analysed in Graphic Pad prism 9 shows (OR) 3.425, 95% CI (1.7-6.7), Relative Risk 2.004 95%CI (1.358-3.078) P value 0.002 (significant).

Among smokers 67 (67.68%) responders declared themselves with some gastric symptoms, 11 (16.42%) people responded in the non-smokers cohort (OR: 10.66, 95% CI (4.98- 22.19), RR: 4.122 95%CI (2.44- 7.288) P = 0.001 (Fisher Exact Test) **36.36** % smoker declared themselves as diabetic, where as 22.39 % from the cohort of Nonsmoker has Diabetes. (OR 1.98 95%CI (0.99- 4.034) RR 1.6 95%CI (0.98 -2.74) P=0.06.) The trend is showing in favor of smokers who has more diabetic in their cohort. There is some derangement in the hepatic function in the group of smokers. Given the fact that liver function can vary with multiple reasons (infection, alcohol intake etc.)

Risk of developing dyslipidemia in smoker is almost same with nonsmokers. since it is a questionnaire based study and not all reports can be seen, a definite conclusion could not be established. In smokers' cohort 45 people underwent angiography, out of which 6 people reported that there was some abnormality in the report. In non-smoker group 1 (4%) needed a treatment for abnormal Angiography. [RR 0.9 95% CI (0.81- 0.98)].

Discussion

Smoking has been identified as the most important public health concern due to its well-documented negative effects on human health. Smoking is also often regarded as the leading preventable cause of death worldwide.

A. Hypertension and Coronary artery disease

Our results were corroborated with results of Rai et al who published their finding from Nepal (Feb 2022) showed that 54.28 % of the smoker demonstrated clinically recorded Hypertension (more than 130/85) with a mean age of 52.76(+/- 13.24) (Heitzer et al., 2000).

In order to study the immediate effects of smoking on heart rate, blood pressure, vascular resistance, calves blood flow and plethysmography, Grassi et al. recruited 9 healthy, normotensive participants. Plasma levels of post-ganglionic muscle sympathetic and norepinephrine and epinephrine. Smoking was found to significantly increase calf vascular resistance, heart rate, epinephrine levels and plasma norepinephrine although it was found to significantly decrease in sympathetic nerve activity (Thuy et al., 2010).

A malfunctioning endothelial NO synthase (NOS) caused by the loss of the cofactor tetrahydrobiopterin may contribute, at least in part, to endothelial dysfunction in chronic smokers, in addition to the burden of free radicals from cigarette smoke (Dochi et al., 2009).

In order to determine the risk factor for non-communicable disease in a population of Vietnamese men, Thuy et al. (2010) (n 910) used a population-based sample. After controlling for

age, BMI, and alcohol consumption, their analysis revealed a clear tendency of an increasing prevalence of hypertension with increasing years and pack years of smoking. The risk of hypertension among individuals who had smoked for 30 years or more and those who had smoked 20 pack years or more in comparison to never-smokers. RR 1.34 (95% CI 0.94-1.91), and 1.52 (95% CI 0.95-2.44), respectively (Primatesta et al., 2001).

Dochi et al. (2009) used three end points on Japanese male workers in a Japanese steel mill over the course of a 14-year longitudinal study: hypertension (BP >140/90), systolic hypertension (SBP >140), and diastolic hypertension (>90). According to their opinion, smoking is causally related to both the beginning of hypertension and systolic hypertension in Japanese male workers (Virdis et al., 2010).

A health survey was carried out in the UK by Primatesta et al. (2001) to examine any differences in blood pressure between the adult population of smokers and non-smokers. When age, body mass index, socioeconomic status, and alcohol consumption were taken into account, it was discovered that older male smokers had greater systolic blood pressure than non-smokers did (Endoh & Leung, 1994).

In another study, younger men and diastolic blood pressure in any age group did not differ in this way. Women who smoked between one and nine cigarettes per day had lower diastolic blood pressure than heavy smokers and never smokers (Nakamura et al., 2002).

B. Smoking and Chronic Gastric Symptoms

Nicotine from smoking has a variety of effects on the gastrointestinal mucosa. Nicotine weakens protective mechanisms while enhancing damaging activities in the stomach. It raises levels of free radicals and platelet-activating factors, endothelin production, and vasopressin secretion. It also increases acid and pepsin secretions, gastric motility, and duodena reflux of the bile salts. Prostaglandin production, stomach mucosal blood flow, mucous secretion, and epidermal growth factors are all decreased by nicotine (Tatsuta, Iishi, & Okuda, 1988).

Nakamura et al. (2002) looked at the connections between age, smoking status, Helicobacter Pylori (HP) infection, Atrophic Gastritis (AG) and Intestinal Metaplasia (IM). AG and IM, Gastric pH, TBA (Total Bile acid) were higher in smokers and in the middle and upper age range in HP positive participants. High TBA concentrations and cigarette usage may contribute to the development of AG and IM. Smoking (OR 9.31 95%CI (3.85-22.50), TBA (OR 2.92, 95%CI) were statistically linked to an elevated risk of severe AG/IM (OR 4.91 95%CI 1.19- 7.17) (Müller-Lissner, 1986).

Tatsuta et al. (1988) performed a multivariate analysis on smoking and other variables with the size of the acid-secreting region and intestinal metaplasia. Areas that were discolored and those that stained blue with Methylene blue, as well as areas of intestinal metaplasia, were identified as acid-secreting sites. They discovered that smoking was connected to a diminution in the size of the acid-secreting region, which subsequently resulted in the formation of intestinal metaplasia (Cho et al., 2009).

Müller-Lissner (1986) examined the effects of cigarette smoking on gastric emptying, gastric secretion, and bile salt reflux in 19 healthy smokers who consumed more than 20 cigarettes

per day and in 18 non-smokers. Even when they are not smoking, smokers were found to have higher levels of gastric bile salt concentration and bile salt reflux (P 0.01). While smoking during the study decreased gastric clearance and increased the rate and content of gastric bile salt reflux, smoking had both long-lasting and immediate impacts on gastric function and bile acid reflux (p 0.01) (Jee et al., 2010).

C. Smoking and Diabetes

In a 4-year survey conducted in Korea of 4041 men, Cho et al. found that the risk of type 2 diabetes was significantly increased in both former and current smokers and increased with cigarette consumption (Hu et al., 2001).

Those who smoke 20 or more cigarettes per day have a 1.55 (95% CI 1.51-1.60) higher risk of getting diabetes than nonsmokers, according to a 14-year prospective cohort study by Jee et al. (2010); Rimm et al. (1993).

According to a health study by American nurses that took into account other risk factors, smokers have a 1.42 percent increased risk of developing diabetes. The 16-year follow-up of the same cohort revealed a risk factor of 1.4 when compared to non-smokers (Global Initiative for Chronic Obstructive Lung Disease (GOLD), 2008; Lundbäck et al., 2003).

D. Smoking and Pulmonary Symptoms

Pulmonary symptoms in the cohort of smokers were exacerbated. Although symptoms have multifactorial origins smoking apparently causes enhancement of the symptoms among smokers.

Smoking is acknowledged as the primary contributing factor to chronic obstructive pulmonary disease (COPD), which is quickly emerging as a global public health emergency. According to guidelines from the Global Initiative for Chronic Obstructive Lung Disease (GOLD), 50% of smokers will eventually develop COPD (Alsalhen & Abdalsalam, 2014; Eisner et al., 2005).

Smokers can now be advised that, if they smoke for the rest of their lives, they have at least a one in two chance of having COPD (Rahmatallah et al., 2019).

E. Smoking and Liver function test

Alsalhen and Abdalsalam (2014) conducted a study on 30 male employees from Libya, ages 30 to 60, who had been smoking for at least 15 years and at least 10 cigarettes per day. Malon-di-Aldehyde (MDA), a marker of lipid peroxidation and oxidative stress in smokers, significantly increased in their blood sample as compared to the control group. The smoker group had higher levels of ALP, ALT, AST, total cholesterol, and triglycerides (Aminullah et al., 2021).

A study by Rahmatallah et al. (2019) on 30 male Sudanese workers to ascertain the impact of smoking on liver enzymes, ALT, AST, and Albumin level. (n 70 smokers Vs 30 nonsmokers). level of AST was 10% lower in smokers than in non-smokers. There is Insignificant differences in AST, ALT, Albumin level in cigarette smokers and non-smokers. Smokers who smoked (2 -10) cigarettes/ day, and those who smoked (> 10 cigarettes / day) had a substantial drop in albumin levels. This study came to the conclusion that smoking has a minor impact on liver function tests (Tan et al., 2008).

F. Smoking and lipid profile

A substantial correlation between hyperlipidemia and an increase in pack-years showed by Aminullah et al. (2021). Total cholesterol also significantly increased, whereas HDL-C levels were on the decline (Akbari, Bhatti, & Shakoor, 2000).

Low density lipoprotein cholesterol (LDL-C) levels were more commonly normal in very heavy smokers but Apoprotein B was more frequently aberrant in very heavy smokers compared to light smokers (Ramsdale et al., 1985).

Akbari et al. (2000) calculated the levels of total lipids, cholesterol, HDL, LDL, VLDL, triglycerides, and chylomicrons in a group of smokers. Except for HDL level, all of these indicators showed a considerable decline (Kojima et al., 2000).

G. Smoking and Angiography

180 males (mean age 54) and 207 women (mean age 54 +/-8 years, smokers N 229, non-smokers n 151) were studied by Ramsdale et al. (1985) in the UK. One patient had pulmonary stenosis, 149 had aortic, 122 had mitral, and 115 had combined aortic and mitral valve disease. DBP, cigarette use, age, the ratio of total cholesterol to HDL, and a history of angina were found through multiple regression analysis to be the most significant predictors of the severity of coronary artery disease (Libby & Theroux, 2005).

The risk of coronary plaque rupture increases with cigarette smoke exposure (Kondo et al., 2019).

It encourages the formation of thrombus at the lesion and causes acute coronary syndrome, including sudden cardiac death, to appear suddenly (Morita et al., 2014; Sugiishi & Takatsu, 1993).

Moreover, smoking causes coronary artery spasm, either with or without obvious substantial coronary constriction (Miettinen, Neff, & Jick, 1976).

According to a study by the Boston Collaborative Drug Surveillance Program, smokers who smoke one pack per day (age 40 to 49) had a risk ratio for nonfatal acute myocardial infarction of 3.0 compared to 1.4 for nonsmokers (age 50 to 59). Compared to rate ratios for non-smokers, both rate ratios were very significant.

Limitation of this Study

This study was undertaken with a questionnaire-based interview at the time of the Covid related restrictions. So the interviewer could not verify all investigations and related papers but rather has to rely on the patient's response. So unintentional misinformation from the patient response can be possible. Since this study is cross-sectional in nature we could not follow up the patients. some participants especially women did not agree to participate due to prevalent social culture.

Conclusion

The finding of this study showed positive correlation with smoking and chronic diseases like hypertension, coronary artery disease and chronic gastritis and poor correlation with diabetes

and lipid profile. Smoking was previously a fashion and now the awareness is grown among young adults and they could appreciate the deleterious effects of smoking and development of chronic disease in long term. However, more awareness is needed in public, particularly the young adults to quit smoking to avoid these unforeseen and avoidable consequence in short future. Investment in more antismoking campaign will help in young adults to quit smoking and lead a healthier life.

Abbreviations

DBP: Diastolic Blood pressure, DALY: Disability Adjusted Life Year, SHS: Second hand Smoke, AG: Atrophic Gastritis, IM: Intestinal Meta-analysis, COPD: Chronic obstructive Pulmonary Disease, MDA: Malon- di- Aldehyde, LDC –C: Low density Lipoprotein Cholesterol. CAD: Coronary artery disease.

Competitive interest

The authors declares that there are no conflicts of interest regarding the publication of this paper.

Authors contribution

1 has conducted cases studies and documented the data from the study population. 2 gave significant and important contribution in revising the text and 3and 4 significantly contributed to revise the article and preparing the tables .5 has performed the statistical analysis of this study

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