

Assessment of Hematological parameters of Young Male with Hookah Smoking in Rania City

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ABSTRACT

Background and aim: Hookah smoking is one of the major causes of cancer and cardiovascular diseases leading to millions of premature deaths each year all over the world. Scientists have identified about 4,000 different substances in tobacco all of which have certain degree of toxic effects, at least 43 of them known carcinogens. The aim of this study is to assess the extent of adverse effect of shisha on hematological parameters in male population of Rania City in Iraq.

Materials and Method: experimental study a purposive (non probability) sample of fifty-five male subjects participated in this study. The method of the study is the following; shisha smoker (n= 30) and non-smoker (n= 25). Fresh peripheral blood samples from healthy adult non-smokers and smokers (males) are collected and analyzed for Red Blood Cells (RBC) count, hemoglobin (Hb) content, packed cell volume PCV, MCV, MCH, MCHC and RDW, total and differential leucocytes (WBC) counts and total platelets count and its parameters by using fully automatic hematological analyzer.

Results: The smokers of shisha have non-significantly higher level of Hb, HCT, RBCs, WBC count, LYM and platelets counts and its parameters while NUET is insignificantly down in smoker. However, MXD and MCHC are significantly lower in cigarette smokers than that of non-smokers. The present study clarifies that age have no significant effect on hematological parameters except LYM NUET, MID and MCV in smokers. The study shows that the duration of smoking has no significant effect on hematological parameters except LYM and NUET. The current result reveals number of smoking weekly has no significant effect on hematological parameters except NUET.

Conclusion: The study concludes that smoking alters hematological parameter that is injurious to health.

Key words: Shisha, Smoking, Hematology

INTRODUCTION

The hookah pipe is a water pipe that originated in India and Persia over 500 years ago. Hookah is also known by other names such as; hubble-bubble, narghile, shisha, and goza. Although hookah pipes vary in size and shape, most have three pieces; a bowl, pipe and hose. Hookah pipes are often used in group settings, and the same mouthpiece may be shared among users (American Lung Association, 2007). Hookah was invented in India, in the court of Mughal Emperor Akbar (1542-1605 AD) when a physician Hakim Abdul Fateh Gilani raised concern on tobacco smoking and envisage a system to pass smoke through water in order to purify it (Chattopadhyay, 2000). Specially formulated flavored tobacco is typically used in hookah pipes. Hookah pipe smoking is not safer than cigarette smoking. Hookah pipe smokers may inhale as much smoke during one session as a cigarette smoker would inhale from 100 or

more cigarettes (American Lung Association, 2007). Smoking hookah is associated with three main detrimental health effects: cardiovascular damage, infection, and cancer formation. According to data reported from the World Health organization (WHO), there is about 2.4 billion people worldwide that have consumed tobacco in the forms of smoking, chewing, snuffing or dipping. WHO also estimates that tobacco-related deaths will amount to 6.4 million in 2015, 8.3 million in 2030 and one billion deaths during the 21st century (World Health Organization, 2009; Mathers and Loncar, 2006). The effect of smoking on hematological parameters has been studied previously but the literature is limited and controversial. However, there are paucities in studies on the effect of cigarette and WP smoking on hematological parameters in both human and animals. Therefore, this study aims to investigate the effect of WP smoking on hematological

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parameters in male population of Rania City. The hypothesis of the study are there is a significant relationship between hookah and accounting of WBC, RBC, and platelets, and there is a positive direction between hookah smoking and some sociodemographic characteristics.

MATERIALS AND METHOD

Experimental case, control study was conducted by measuring, white blood cells, red blood cell and platelets parameter in hookah smokers in Rania City in Iraq. The study was conducted from 15th December 2015 until 30th January 2016. A purposive (no probability) sample of 55 subjects for the two group (case and control) is set. The study includes 30 hookah smokers, as study group and 25 non-smokers as control group and they were taken in a homogenous way. This study is carried out on Iraqi volunteers from both smokers for (study group) and non-smoker for (control group) according to the following inclusion and exclusion criteria. Control group are males, their age range from 18 to 30 years old, and they appear to be healthy individuals. Inclusion criteria for the study group are regular hookah smoking, and the same is true for case group. Any individual have any disease on examination/investigation in any of control or study group was excluded from the study. And any individual who smoke both cigar and hookah were excluded form study group. Predesigned and pretested questioner are used to obtain biosocial information of participants like age, smoking dose, smoking duration and other diseases related to it. Reliability is determined using stability reliability (Test – Retest approach). A panel of four experts is involved in the determination of the questionnaire content validity. The data is put on computer file, and it is analyzed by using descriptive and inferential statistical measures by using the statistical package of social science (SPSS) version (21). The analyzed data is preformed through the following approaches: descriptive statistical data analysis approach, such as (frequency and percentage), and inferential data analysis approach, such as (Chi-Square, T-test). Three ml

K3EDTA anti-coagulated venous blood is withdrawn using 5 ml disposable syringe. All samples are checked for clots hemolytic and are mixed well before analysis. These samples are then subjected to apparatus analysis; 50 μ l from each sample is sucked by apparatus needle. Immediately the result of each sample is obtained, and results are kept until they were statistically analyzed. Two and half ml (2.5 ml) venous blood sample is collected in EDTA anticoagulant blood container in a proper way and is gently mixed in the hematology mixture immediately (not longer than one hour). The sample is then analyzed by Swelab-Alfa automated hematology analyzer. CBC (complete blood count), evaluations of the blood cell count are performed by Swelab-Alfa automated hematological analyzer, which could perform 20 hematological parameters with high accuracy and precision. Principally Swelab Alfa analyzer is based on the electronic resistance (impedance) detection method for counting and sizing recognition of the leukocytes parameter, red blood parameter and platelet parameter. Through using three preliminary hydraulic systems for leukocytes parameter, red blood parameter and platelet parameter, and display the mode of the cells blood count results on the liquid crystal displayer (LCD) with histogram and printed out the results in thermal paper (Dacie and Lewis, 2006). Quality control of Swelab-Alfa. all quality control of the machine done in instructed manner. The daily, weekly and monthly maintenance and calibration used to ensure quality assurance. Then before using the apparatus one of the last day samples was re-analyzed for delta check. All results are expressed as mean \pm standard deviation (Std). Comparison between study group and control group is performed by independent sample T-test. For all analyses, a value of ($P < 0.05$) is considered significant. Pearson's correlations were used to determine relationship between age, duration and time number of week smoking hookah with parameters studied taken $P \leq 0.05$ or $P \leq 0.01$ as the lowest limited of significant. All statistical analyses were performed statistical Package for Social Science (SPSS) V21.

RESULTS

Table (1): shows comparison of RBC and its related parameters in Nonsmokers and smokers.

Parameter	Type	N	Mean	Std. Deviation	F	Sig.
WBC($10^9/l$)	Control	25	5.0160	1.05975	8.606	.005
	Case	30	6.9600	1.94007		

LYM (%)	Control	25	38.4040	7.68415	.002	.966
	Case	30	38.9400	8.23820		
MID (%)	Control	25	5.0600	1.62327	65.471	.000
	Case	30	8.4767	4.76059		
NUE (%)	Control	25	55.7280	7.83318	2.509	.119
	Case	30	52.5400	10.95044		
RBC($10^{12}/l$)	Control	25	5.0968	.37663	.407	.526
	Case	30	5.7103	.53636		
Hb(g/dl)	Control	25	14.4000	1.39194	.000	.991
	Case	30	15.5333	1.54526		
HCT (%)	Control	25	45.1960	4.17168	1.348	.251
	Case	30	47.3067	8.30745		
MCV(fL)	Control	25	84.2960	14.74309	2.017	.161
	Case	30	84.8367	6.86588		
MCH(pg)	Control	25	28.6280	2.92098	.089	.767
	Case	30	27.3033	2.14934		
MCHC(g/dL)	Control	25	31.8680	.59702	31.493	.000
	Case	30	32.1967	1.48614		
RDW(%)	Control	25	13.3800	2.31247	3.490	.067
	Case	30	13.3733	.66016		
PLT ($10^9/l$)	Control	25	188.8000	57.83742	.619	.435
	Case	30	199.4000	50.20001		
MPV(fL)	Control	25	8.5240	1.80723	.844	.363
	Case	30	9.0767	.97333		
PDW (fl)	Control	25	11.6000	1.21518	1.448	.234
	Case	30	12.4667	3.73302		
PCT (%)	Control	25	.1736	.08669	1.731	.194
	Case	30	.2677	.51727		
LPCR (%)	Control	25	21.0040	6.80444	.234	.631
	Case	30	22.5433	6.97388		

Table (2): shows comparison of relationship between age and WBC parameter in Control and Case.

Control		WBC($10^9/l$)	LYM (%)	MID (%)	NUE (%)
Age	Pearson Correlation	.304	-.341	.023	.311
	Sig. (2-tailed)	.140	.095	.913	.130
	N	25	25	25	25
Case		WBC($10^9/l$)	LYM (%)	MID (%)	NUE (%)
Age	Pearson Correlation	.301	.488**	.462*	-.567**
	Sig. (2-tailed)	.106	.006	.010	.001
	N	30	30	30	30

Table (3): shows comparison of relationship between age and RBC parameter in Control and Case.

Control		RBC ($10^{12}/l$)	Hb (g/dl)	HCT (%)	MCV (fL)	MCH (pg)	MCHC (g/dL)	RDW (%)
Age	Pearson Correlation	.340	.275	.316	.214	.045	-.097	.002
	Sig. (2-tailed)	.097	.183	.123	.305	.832	.646	.993
	N	25	25	25	25	25	25	25
Case		RBC ($10^{12}/l$)	Hb (g/dl)	HCT (%)	MCV (fL)	MCH (pg)	MCHC (g/dL)	RDW (%)
Age	Pearson Correlation	.077	-.100	-.209	-.367*	-.179	.331	-.017
	Sig. (2-tailed)	.686	.598	.268	.046	.344	.074	.927
	N	30	30	30	30	30	30	30

Table (4): shows comparison of relationship between age and PLT parameter in Control and Case.

Control		PLT (10 ⁹ /l)	MPV (fL)	PDW (fl)	PCT (%)	LPCR (%)
Age	Pearson Correlation	.053	-.162	.196	-.197	.304
	Sig. (2-tailed)	.802	.439	.348	.346	.140
	N	25		25	25	25
Case		PLT (10 ⁹ /l)	MPV (fL)	PDW (fl)	PCT (%)	LPCR (%)
Age	Pearson Correlation	-.068	.124	.001	.014	.120
	Sig. (2-tailed)	.721	.514	.997	.942	.527
	N	30	30	30	30	30

Table (5): clarifies relationship duration of smoking and time number weekly with RBC parameter.

Case		RBC (10 ¹² /l)	Hb (g/dl)	HCT (%)	MCV (fL)	MCH (pg)	MCHC (g/dL)	RDW (%)
ysmok	Pearson Correlation	.114	.186	.046	-.056	.109	.290	-.089
	Sig. (2-tailed)	.550	.325	.810	.767	.566	.120	.642
	N	30	30	30	30	30	30	30
weekly	Pearson Correlation	.053	.165	.229	.196	.150	-.072	-.290
	Sig. (2-tailed)	.781	.382	.224	.298	.428	.707	.120
	N	30	30	30	30	30	30	30

Table (6): clarifies relationship duration of smoking and time number weekly with WBC parameter.

Case		WBC(10 ⁹ /l)	LYM (%)	MID (%)	NUC (%)
ysmok	Pearson Correlation	.140	.402*	.300	-.429*
	Sig. (2-tailed)	.459	.028	.108	.018
	N	30	30	30	30
weekly	Pearson Correlation	.005	.159	-.194	-.033
	Sig. (2-tailed)	.977	.401	.305	.861
	N	30	30	30	30

Table (7): clarifies relationship duration of smoking and time number weekly with PLT parameter.

Case		PLT (10 ⁹ /l)	MPV (fL)	PDW (fl)	PCT (%)	LPCR (%)
ysmok	Pearson Correlation	-.005	-.068	.085	-.206	-.094
	Sig. (2-tailed)	.980	.722	.655	.276	.622
	N	30	30	30	30	30
weekly	Pearson Correlation	-.146	-.034	-.031	-.129	-.088
	Sig. (2-tailed)	.442	.859	.870	.496	.644
	N	30	30	30	30	30

DISCUSSION

Total and differential WBC count: Analysis revealed a significant increase in total WBC counts in smokers. However, lymphocytes count, were insignificantly decreased in smokers than that of non-smokers, while neutrophils count and mixed white blood cells were significantly increasing in smokers than that of non-smokers (Table 1).

Red blood count and its related parameters: The current result clarified increasing non-significant of RBC, HB, HCT and MCV while increasing significant MCHC in smoker.

However, the MCH and RDW are decreasing non-significant in smoker (Table 1).

Platelets count and its related parameters: The PLT, MPV PDW, PCT and LPCR are elevation insignificant in smoker (Table 1).

Relationship : No statistically significant correlation is found between white blood cell parameters (table 2), Red blood cell parameters (table 3) and Platelets blood cell parameters (table 4) with age in non-smoker and smoker except positive correlation LYM and MID while negative relationship NUET and MCV in smoker. There is no statistically significant correlation is found between duration of

smoking and time number weekly with RBC parameter (table 5). There is also no statistically significant correlation between duration of smoking and time number weekly with WBC parameter except duration of smoking has positive correlation LYM while negative correlation NUET (table 6). There is no statistically significant correlation between duration of smoking and time number weekly with platelet parameter (table 7).

Since cigarette smoking leads to many health problems in people, the observations of this study also shows that cigarette smoking has severe effects on hematological parameters (e.g. Hb, Hct, WBC, RBC, Plt count) among the studied population. In the present study, leukocyte count is significantly ($p < 0.001$) increased in hookah smokers. Another published reports a significantly high WBC count in male smokers (Kawada, 2004; Freedman *et. al.*, 1996). Chronic tissue damage may be a possible mechanism for the increased total leukocyte count smokers (Silverman *et. al.*, 1975). Smoking has an irritant effect on the respiratory tree with resultant chronic inflammation. Prolonged smoking impairs ciliary movements, causes hypertrophy and hyperplasia of mucus secreting glands, hyper responsiveness of the airways and causes bronchiolar inflammation (Eric *et. al.*, 1997). Airway epithelium is regarded as a physical barrier which prevents the entry of inhaled noxious particles into the submucosa Exposure to smoke causes increased release of inflammatory cytokines from the epithelial cells. All of them can influence the growth, differentiation and activation of leucocytes. This possibly explains the Leukocytosis in smokers. Another mechanism put forward by some workers is that nicotine increases release of catecholamines which can increase the total leucocyte count (Armitage, 1965). Hemoconcentration attributed to cigarette smoking can also be considered as a possible explanation for the elevation of total leucocyte count. Nowadays, there is increasing evidence that apart from the known risk factors like cigarette smoking, diabetes, and hypertension, inflammation also plays an important role in the progression of coronary heart disease. Elevated WBC counts as observed in smokers along with high C reactive proteins are associated with increased incidence as well as mortality from coronary heart disease (Gillum *et. al.*, 1993; John, 2004).

Our study demonstrates change in differential leucocyte counts, slightly increasing

of Lymphocyte while decreased neutrophil that was non-significantly. However the mid was significantly increased in smokers compared to non-smokers. Our study also aims at Differential leucocyte counts because the association of cigarette smoking with total leucocyte count has been established by many but its effect on the differential leucocyte count is a matter of debate. According to some researchers, effect of smoking on differential count is not uniform and is influenced by the current smoking behavior.

Some studies have shown that neutrophil count rises and lymphocyte count shows a decrease (Schwartz and Weiss, 1994; Ogawa *et. al.*, 1998). While few studies have shown that both these counts are increased like Farhang and Fikry (2013). The lymphocytosis can be attributed to chronic tissue damage and inflammation produced by toxic smoke products. This corresponds with the findings of Silvermann *et. al.* (1975) that leukocytosis in smokers is mainly attributable to an increased lymphocyte count and that too of the 'T' lymphocytes. Similar findings have been reported by some other researchers also (Hughes and Haslam, 1985). Alteration in the T lymphocytes may explain the increased risk of infections and neoplasia in smokers. Other researchers revealed Significant decrease in Neutrophil count and increase in Lymphocyte count in smoker groups (Sunil *et. al.*, 2003; Taylor and Gross, 1988). The increase in lymphocyte count may be due to residual chronic inflammation of respiratory tract. As DLC is a relative count the decrease in Neutrophil count may be due to increase in lymphocyte count. There was significant difference in this study between cigarette smokers and non-smokers in MIDs%. These results agree with observations made by group Pankaj *et. al.* (2014) and Nadia *et. al.* (2015). The current study shows increasing RBCs, HCT and Hb but not reached to significant. However, other authors reported elevation of RBCs, HCT and Hb like the study of Saba (2015) reported that smoking cause elevated RBCs, HCT and Hb. Researchers suggested that the increased RBCs, HCT and Hb may be due to the combination of CO in tobacco with effects of nicotine disrupts oxygen delivery to tissue and stimulates the bone marrow to produce more RBCs and thereby increase HCT and Hb (Roethig *et. al.*, 2010).

Concomitantly the Hb concentration increase in smokers because the inhaled carbon monoxide result in increased carboxy

hemoglobin, which has no oxygen carrying capacity. Impaired tissue oxygen supply results from decrease oxygen carrying capacity and increase oxygen-hemoglobin affinity caused by carboxy hemoglobin (COHb), to compensate, Hb level increase (Sagone *et. al.*, 1973). Smoking is also considered as a major cause of polycythemia and elevated hematocrit levels (Attchison and Russell, 1988).

The study reveals significant higher MCHC while non-significant decreasing at RDW and MCH in smoker. Also non-significantly slight down MCV in smokers. The researchers suggested elevation of MCHC might be due to folic acid or vitamin B12 or thyroid problems (Ghosh *et. al.*, 2012). The study clarifies increasing PLT, MPV, PDW, PCT and LPCR in smokers compared to nonsmokers that were not significant. The results show that there is no statistically significant difference in PLTs count and indices when compared in study groups. According to these findings, we suggest that the effect of smoking on PLTs count and platelet morphological indices is insignificant. Similar results were also reported in other studies, Brummit and Barker (2000) found no statistically significant difference in PLTs count in healthy volunteer smokers. Also Dotevall *et. al.* (1992) noted no changes in PLTs count in female smokers and non-smokers. Similar finding also reported by Suwansaksri *et. al.* (2004) who observed no alterations in PLTs in male smokers and non-smokers. Our finding disagree with that of Chao *et. al.* (1982) who reported a significant increase in PLTs count, fibrinogen, and platelet factor-3 (PF-3) activity, and decrease in the lag period of collagen-induced platelet aggregation. It was reported that the hormonal pathways regulating platelet production may be potentially impaired following smoking inducing production of platelets and increased platelets count. Also our result disagree with another study by Ghahremanfar *et. al.* (2015) who reported that cigarette smoking in healthy individuals results in significant and considerable effects on platelet count and morphological indices compared with non-smokers. Variations in our study and these studies may be because of differences in the type of tobacco.

CONCLUSION

In the current study statistically significant positive correlation is found between Age and LYM and MID, while negative correlation NUET and MCV with age in the case

study. However, in the current study, statistically insignificant correlations is found between age and platelets, white blood cell, red blood cell count and their related parameters in control study. In the current study, statistically significant positive correlation is found between duration smoking and LYM while negative correlation NUET. In present study, non-significant positive correlation is found between time numbers smoking in week with hematology parameter study.

REFERENCES

- American Lung Association (ALA). (2007). *An Emerging Deadly Trend: Waterpipe Tobacco Use*. http://www.lungusa2.org/embargo/slati/Trendalert_Waterpipes.pdf
- Armitage, A.K. (1965). Effect of nicotine and tobacco smoke on blood pressure and release of catecholamines from the adrenal glands. *Bri J Pharmacol*. 25. P.p. 515-526.
- Attchison, R.; Russell, N.(1988).Smoking–A major cause of polycythemia. *J R Soc Med*. 81. P.p.89-91.
- Brummit, DR. and Barker HF. (2000).The determination of a reference range for new platelet parameters produced by the Bayer ADVIA TM 120 full blood count analyzer. *Clin Lab Hematol*. 22. P.p.103-107.
- Chao, FC.; Tullis, JL.; Alper, CA.; Glynn, RJ.; Silbert, JE. (1982). Alteration in plasma proteins and platelet functions with aging and cigarette smoking in healthy men. *Thromb Haemost*. 47. P.p. 259-264.
- Chattopadhyay, A. (2000). Emperor Akbar as a healer and his eminent physicians. *Bull Indian Inst History Med*. 30. P.p.151-8.
- Dacie, J.; and Lewis, SM. (2006) *Practical hematology*.(10th ed.). churchi 1 living stone.
- Dotevall, A.; Rongemarck, C.; Eriksson, E.; Kutti, J.; Wadenvik, H.; Wennmalm, A. (1992). Cigarette smoking increases thromboxane A2 formation without affecting platelet survival in young healthy females. *Thromb Haemost*. 68. P.p.583-588.
- Eric, G.; Honig, Roland H.; Ingram, Jr. (1997). *Chronic bronchitis, emphysema and airway obstruction, In- Harrison's Principles of Internal Medicine*. (14th ed.). USA:McGraw Hills publications. P.p.1452.
- Farhang, A. Aula; and Fikry A. Qadir.(2013). Effects of Cigarette Smoking on Some Immunological and Hematological Parameters in Male Smokers in Erbil City.

- Jordan Journal of Biological Sciences*. 6.(2). P.p.159-166.
- Freedman, DS.; Flanders, WD.; Barboriak, JJ.; Malarcher, AM.; Gates, L. (1996). Cigarette smoking and leukocyte sub population in men. *Ann Epidemiol*. 6(4). P.p.299-306.
- Ghahremanfar, Farahnaz; Vahid, Semnan; Raheb, Ghorbani; Farhad, Malek; Ali, Behzadfar; Mehrdad, Zahmatkesh. (2015). Effects of cigarette smoking on morphological features of platelets in healthy men. *Saudi Med J*. 36 (7). P.p.847-850.
- Ghosh, A.; Chowdhury, SD.; Ghosh, T. (2012). Under nutrition in Nepalese children: a biochemical and haematological study. *Acta Paediatr*. 101(6). P.p.671-676.
- Gillum, RF.; Ingram, DD.; Makuc, DM. (1993). White blood cell count, coronary heart disease and death: the NHANES I Epidemiologic follow-up study. *Am Heart J*. 125. P.p.855-63.
- Hughes, A.; Haslam P.L. (1985). Numerical and functional alterations in circulatory lymphocytes incigarette smokers. *Clin. Exp. Immunol*. 61 (2). P.p. 459- 456.
- John, Danesh MB. DPhil. (2004). CRP and other circulatory markers of inflammation in prediction of coronary heart disease. *NEJM*. 350. P.p.1387-1451.
- Kawada, T. (2004). Smoking-induced leukocytosis can persist after cessation of smoking. *Arch Med Res*. 35. P.p. 246–250.
- Mathers C., D.; Loncar, D. (2002). Projections of global mortality and burden of disease from to 2030. *PLoS Med*. 3(11). P.p.e442.
- Nadia, M.M.; Shamseldein, H.A.; and Sara, A.S. (2015). Effects of Cigarette and Shisha Smoking on Hematological Parameters: An analytic case-control study. *International Multispecialty Journal of Health (IMJH)*. 1(10). P.p.44-51
- Ogawa, Y.; Imaki, M.; Yoshida, Y.; Shibakawa, M.; Tanada, S. (1998). An epidemiological study on association between TLC and neutrophil counts and risk factors of I.H.D. by smoking status in Japanese factory workers. *Appl. Human Sci*. 17 (6). P.p.239-247.
- Pankaj, Jain.; Reena, jain.; Mal, K. L.; and Ketan Mangukiya. (2014) Department of biochemistry, Geetanjali medical college, Udaipur, Rajasthan. *I.J.S.N*. 5 (4). P.p.740-743.
- Roethig, HJ.; Koval, T.; Muhammad-Kah, R. *et. al.* (2010). Short term effects of reduced exposure to cigarette smoke on white blood cells, platelets and red blood cells in adult cigarette smokers. *Regul Toxicol Pharmacol*. 57. P.p.333-7.
- Saba Ibrahim Salih.(2015). Studying the Effect of Smoking on Some Blood Parameters in Young Adult Male Smokers. *Karbala J. Med*. 8 (2). P.p. 2287-2291.
- Sagone, Jr.; ALLawrence, T.; Balcerzak, SP. (1973). Effect of smoking on tissue oxygen supply . *Blood*. 41. P.p.845-51.
- Schwartz, J.; and Weiss, S.T. (1994). Cigarette smoking and peripheral blood leucocytedifferentials. *Ann. Epidemiol*. 4. P.p. 236-242.
- Silverman, N.A.; Potvin, C.; Alexander, J C Jr.; Chretien, PB. (1975). In vitro lymphocyte reactivity and T cell levels in chronic cigarette smokers. *Clin. Exp. Immunol* . 22. P.p. 285-292.
- Silverman, N.A.; Potvin, C.; Alexander, J C Jr.; Chretien, PB. (1975). In vitro lymphocyte reactivity and T cell levels in chronic cigarette smokers. *Clin. Exp. Immunol*. 22. P.p.285-292.
- Sunil Kumar Jena; Kanhu Charan Purohit; Akshaya Kumar Misra. (2013). Effect of Chronic Smoking on Hematological Parameters. *International Journal of Current Research*. 5(2). P.p.279-282.
- Suwansaksri, J.; Wiwanitkit, V.; Soogarun S. (2004). Effect of smoking on platelet count and platelet parameters: an observation. *Clin Appl Thromb Hemost*. 10.P.p. 287-288.
- Taylor, R.G.; and Gross, E. (1988) Smoking, allergy and the differential white blood cell. *Eur J Respir Dis*. 72(3).
- World Health Organization. (2004). *The Global Burden of Disease*. Update. WHO Press 2008.
- World Health Organization. (2009). *Report on the Global Tobacco Epidemic, 2009: Implementing smoke-free environments*. WHO Press. Geneva; Switzerland.