A Review Study of Cancer in Human Body by Tobacco or Smoking

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Abstract

The health risks of tobacco smoking have been documented in numerous studies and smoking rates have declined in developed countries. Today, we know that cigarette smoking is the major cause of preventable deaths due to tobacco smoke induced diseases. As a consequence of an increased awareness of smoking-related health risks, heated tobacco products (HTPs) are marketed as reduced toxicant alternatives to conventional tobacco products. including a short discussion on challenges with the adaption of standard analytical methods used for tobacco smoke. The reliability of analytical data is important for risk assessment approaches that are based on reduced toxicant exposure. In order to assess a putative reduction of health risks, an integrated study design is required that should include clinical studies and epidemiology data. However, data are not yet sufficient for a reliable assessment or recognition of putatively reduced health risks. Tobacco is a multisite carcinogen causing various cancers and high mortality in the world, tobacco is the only product that kills half of its consumers. This study aimed to review the various carcinogenic compounds present in tobacco, the harmful effects from tobacco consumption and the therapy for smoking (Nicotine) addiction. Tobacco causing 3 million deaths every year globally. About 1.3 billion smokers worldwide and half of them die due to smoking related diseases. Cigarette smoke contain nearly 4000 chemicals, most of them are carcinogens, causing various and other diseases.

Keywords-*Tobacco; Smoking; Nicotine; Cancer.*

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Introduction

Tobacco is one of the leading causes of cancer in the world, both in terms of incidence and fatality [1]. Tobacco is made from the plant Nicotiana tobacco, which belongs to the Solanaceae family and genus Nicotiana. Tobacco contains a lot of nicotine, which is the main drug. but Tobacco kills 3 million people per year

throughout the world especially smoking; if present smoking rates continue, yearly mortality would reach 10 million by 2030. Smokers die 14 years earlier than the general population. Almost half of children regularly inhale tobacco smoke-polluted air in public places, and 65000 children die each year as a result of passive smoking-related illnesses [2]. Tobacco smoking causes around 25% of

all cancers in males, 4% of all cancers in women, about 16% of all cancers in both sexes in most industrialized nations, and 10% in less developed countries [3]. Tobacco use is linked to nearly 40% of cancer diagnoses in the United States [4]. Tobacco is linked to a large number of incidences of lung cancer, according to a new study [5]. It is the major cause of malignancies of mouth and throat, vocal cords, oesophagus, stomach, pancreas, liver, bladder, cervix, colon, and rectum, as well as leukemia [6-9]. According to the Centre for Cancer Prevention (CDC), 66,0000 persons in the United States were diagnosed with tobaccorelated malignancies between 2008 and 2013, but 343,000 of them died [10]. There inverse association between endometrial cancer and tobacco smoking, according to available research [3]. Evidence also shows that the link between tobacco use and liver cancer has not to be conclusively This established. association can influenced by confounding factors such as hepatitis B and C [11]. Based on existing data and a review of the literature, this study attempted to analyze the association between tobacco smoking, particularly the use of cigarettes, and common malignancies of many organs of the human body, taking into consideration probable confounding variables for each organ.

Tobacco or Cigarette Toxic Chemical Composition

Nicotine is a tobacco-based chemical that induces cigarette addiction. It stimulates the neurological system, causing a rise in heart rate and blood pressure, as well as wrinkles due to the shrinking of blood vessels beneath the skin. Cigarette smoke includes almost 4000 distinct compounds, the majority of which are cancer-causing. Some of the harmful chemicals emitted during smoking, such as carbon monoxide, a poisonous gas that reduces the amount of oxygen taken up by RBCs, hydrogen cyanide, which causes the accumulation of various toxins in the lungs, and other dangerous substances, such as dimethylbenz(a)anthracene, mainly cause lung cancer by causing mutations in P53 genes, which are important for cell cycle deregulation and carcinogenesis.

Reviews Method

Through a search of PubMed, Scopus, and Web of Science databases, epidemiological evidence from all accessible randomised control trials, case-control, and cohort studies reporting tobacco-related cancer risks was included in this analysis. The key terms "cancer," "tobacco smoke," and "smoking" were used in the search approach. In addition, the reference lists of pertinent publications were manually checked to see whether there were any more works that would have been suitable. The current study also includes articles regarding smoking intensity.

Cancerin Human body by Tobacco and their products

Quitting tobacco and its products, regardless of how long you've used them, can help you avoid cancer and other chronic problems. More than 4,000 different types of chemicals have been found in tobacco and tobacco smoke by International Agency on Research in Cancer [12]At least 70 chemicals can cause cancer in smoke from

cigarettes, cigars, and pipes. When you inhale the smoke, the toxins enter your bloodstream, where they travel throughout your body. Many of these chemicals can harm your DNA, which determines how your body generates new cells and how each type of cell does what it was designed to do. Damaged DNA can cause cells to develop incorrectly. These out-of-the-ordinary cells have the potential to become cancerous.

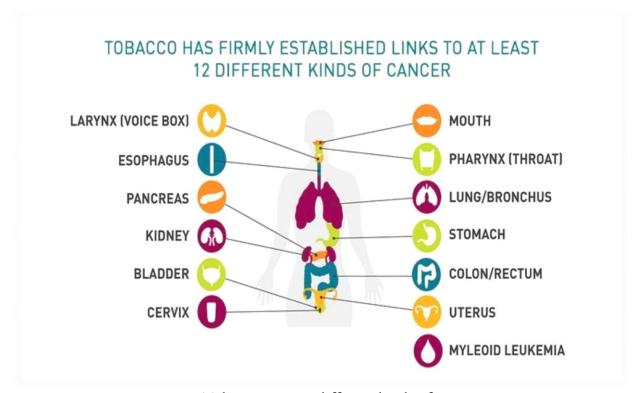


Figure No. 1: Tobacco causing different kinds of cancer.

Larynx cancer

Tobacco It's one of the most common causes of laryngeal cancer. You're more likely to have laryngeal cancer and any sort of head and neck cancer if you smoke cigarettes, cigars, or pipes, or chew smokeless tobacco. The more cigarettes you smoke, the higher your risk. Scientists believe that long-term exposure to secondhand smoking may increase your

chance of laryngeal cancer, although this is not proven.

Esophaguscancer

Esophageal cancer is the ninth most frequent malignancy worldwide and the sixth most common cause of cancer death [13]. Tobacco compounds, when taken in any form, damage the DNA in the oesophagus lining cells and make it difficult for damaged DNA to recover. Tobacco

smoking also impairs the esophageal sphincters, the muscle valves that keep stomach juices in and protect the oesophagus. Smoking causes damage to the oesophagus, allowing stomach acid to flow back into it, causing heartburn or gastroesophageal reflux disease (GERD), which is another risk factor for esophageal cancer [14].

Pancreas cancer

Smoking is one of the most significant risk factors for pancreatic cancer [15 - 16]. According to the conclusions of a metaanalysis research, smokers have a two-fold increased risk of pancreatic cancer compared to non-smokers. The number of cigarettes and the length of time spent smoking are both linked to an increased risk of cancer [17]. Smoking is responsible for 20-30% of pancreatic cancer cases. Most crucially, quitting smoking lowers the risk of pancreatic cancer to near-normal levels [18-19]. A meta-analysis of 82 cohort studies and case-control cases published between 1950 and 2007 found that the relative risk (RR) of pancreatic cancer septicemia is 1.7 (95 percent confidence interval [CI] 1.6-1.9) in current smokers and 1.2 (95 percent confidence interval [CI] 1.1-1.3) in former smokers [17]. The pancreatic cancer RR in former smokers was 1. 1 (95 percent CI 0.9-1.3) and in current smokers was 1.8, according to the multinational cohort research, which included 1481 cases and 1539 controls (95 percent CI 1.4-2.3). This risk was also linked to an increase in the number of cigarettes consumed and the length of time spent smoking. RR was 1.75

among those who smoked 30 or more doses per day and for at least 50 years, but RR was equivalent to those who had never smoked in people who had stopped smoking for more than 15 years [20].

Kidney cancer

In 1985, epidemiological data allowed for a causal link to be established between tobacco smoking and cancer of the renal pelvis, but not of the renal parenchyma [21]. Most research published since then agree that heavy smokers are at higher risk, however the link between smoking intensity and duration is weak. Most studies found a substantial negative trend as the number of years since quitting increased. Furthermore, the findings are similar across study designs and in both sexes. Hypertension and obesity are two more established risk factors for kidney cancer [22]. Because body mass index has been favorably related with an elevated risk of adenocarcinoma of the renal parenchyma and negatively associated with smoking, obesity cannot explain the link between tobacco smoking and renal cell cancer. Hypertension was also ruled out as a possible explanation for the observed link.

Gallbladder cancer

According to researchers, tobacco Smoking affects the hepatobiliary system and is linked to a higher risk of gallbladder cancer [23-24]. However, research on the link between gallbladder illness and the likelihood of gallstones are mixed. Several case-study and cross-sectional descriptive studies found no evidence of a link [25] [27] or even an inverse connection [28], but quantitative

analyses found an elevated risk [29-30]. Some of these research' conclusions appear to be impacted by selection or recollection bias. As a result, drawing firm conclusions from such research is challenging, and further studies with more robust designs, such as cohort studies, are required

Cervix cancer

One of the established risk factors for cervical cancer is smoking. This function of smoking is unrelated to sexual behaviour. Some researchers have discovered a link between the number of cigarettes smoked and the length of time spent smoking and the occurrence of cervical cancer. Those who have quit smoking appear to be in decreased danger [31-32]. Other research, on the other hand, have been unable to link their findings to smoking [33-34]. According to meta-analysis findings in women with human papillomavirus, smokers are twice as likely as non-smokers to develop cervical cancer [35]. The findings of a cohort study in women infected with the human papillomavirus also showed that women smokers and those who had previously smoked but had stopped were at least three times more likely than other women to develop cervical cancer [36]. The fact that nicotine metabolites may be discovered in the cervical mucus of women who smoke [37]. The effect of direct tobacco Smoking carcinogens on the cervix is supported. Suppression of local immune responses to human papillomavirus infection is another suitable approach [38]. More research is needed to prove the conclusive impact of tobacco Smokingon the incidence of oral cancer in general.

Mouth cancer

Mouth According the Cancer Foundation, 90% of people with mouth cancer use cigarettes. Though the exact origin of mouth cancer is unknown, there are several lifestyle factors that might increase one's risk of developing this deadly illness. Tobacco products of any sort, including cigarettes, pipes, cigars, and smokeless tobacco, can raise your risk of mouth cancer. Oral cancer is more likely to occur if you drink a lot of alcohol or chew cigarettes. Furthermore, when a person consumes cigarettes and alcohol at the same time, the problem becomes even worse.

Pharynx cancer

The cancer of the pharynx is more prevalent than the cancer of the larynx. Each year, around 31,000 persons in the United States receive these-diagnosis. Men are more prone than women to get throat cancer, owing to the fact that men consume more alcohol and/or smoke. While throat cancer is most frequent in adults over the age of 55, it is becoming increasingly common in younger people due to the rising incidence of HPV.Cancers of the oral cavity and pharynx combined increased by 0.6 percent per year on average between 2007 and 2016, as did cancers of the other oral cavity and pharynx (3.4 percent), base of tongue (1.8 percent), anterior tongue (1.8 percent), gum (1.9 tonsil (2.4)percent), percent), oropharynx (3.4 percent) (1.9 percent). The rates of malignancies of the soft palate and

uvula (3.7%), hard palate (0.9%), floor of mouth (3.1%), lip (2.7%), hypopharynx (2.4%), and nasopharynx (1.3%) decreased, but cancers of the cheek and other mouth and salivary gland remained steady. When oral cavity and pharyngeal cancers were categorised by HPV relationship, HPV-related cancers grew 2.1 percent per year on average, whereas cancers not associated with HPV decreased 0.4 percent per year.

Lung'scancer

Tobacco inflammation and are frequently associated to lung cancer. Tobacco smoking causes alterations in gene expression and cellular functioning not only in the pulmonary airway epithelium, but also in the nasal and buccal epithelium, alveolar macrophages, and peripheral blood [39, 40]. These findings support the previously proposed ideas about the systemic inflammatory process in COPD and lung patients. The link between cancer inflammation and EMT advancement in lung cancer progression and therapeutic resistance has lately been highlighted. As a result, inflammation may affect stem cell features in the development of lung cancer via EMT-dependent processes.

Stomach cancer

Studies examining the link between tobacco smoking and stomach cancer have found a twofold increase in risk for smokers compared to nonsmokers in the relevant cohort and case-control studies. Most studies found a favorable dose-response connection between smoking intensity and duration. In studies that differentiated

between cancer of the gastric cardia and cancer of the distal stomach, there was an effect of smoking on the risk for both locations, with a dose-response association with smoking intensity and duration, as well as time since stopping.

When relative risks for intestinal and diffuse histological types of stomach cancer were examined independently, the substantial connection remained. Other proven human carcinogens, such as Helicobacter pylori (H. pylori) infection of the stomach, might complicate the positive link between smoking and stomach cancer [41]. However, research on the relationship between H. pylori-positive and smoking has revealed that H. pylori have little or no impact on the possible confounding of the smokingstomach-cancer link. Indeed, classification based on H. pylori status revealed an independent link between stomach cancer and cigarette use.

Colon/Rectumcancer

Since then, a few studies have found increased hazards associated with cigarette use, but the majority of them either fail to achieve statistical significance [42-44] or are null [45-46]. However, a meta-analysis of case-control and cohort studies published in 2008 found that current smokers had a 7% higher risk of colorectal cancer [relative risk (RR) = 1.07, 95 percent confidence interval (CI): 0.99–1.16].A meta-analysis of 36 prospective trials found a 17 percent increased risk of colorectal cancer among current smokers (RR = 1.17, 95 percent CI: 0.97–1.40), and another meta-analysis of 28 cohorts found a 20 percent increased risk

(RR = 1.20, 95 percent CI: 1.10–1.30). In 2009, the International Agency for Research on Cancer classified colorectal cancer as a smoking-related malignancy based on available evidence [47].

Uterus cancer

It is worth noting that smoking is linked to a lower risk of uterine fibroids, endometriosis, and cancer, which might be due to smoke components' inhibitory effects on uterine cell proliferation and extracellular matrix interactions [48].

Myeloid leukaemia cancer

Benzene is a well-known carcinogen that has been found in cigarette smoke and has been proved to cause leukaemia. According to the extant research on tobacco smoking and leukaemia, there is a link between tobacco smoking and myeloid leukaemia, with the risk of myeloid leukaemia rising with the intensity and duration of smoking.

Conclusion

Tobacco or tobacco-related cancers are the leading cause of mortality worldwide. Tobacco includes carcinogenic chemicals such as methylnaphthalene and Dimethylbenz (a) anthracene, which cause malignancies of the lungs, mouth, uterus, kidneys, and pancreas. Tobacco kills 3 million people per year throughout the world, yet smokers live 14 years longer than non-smokers. Tobacco is also linked to coronary heart disease, cardiac arrest, COPD, and birth abnormalities. Diabetes, renal disease, and rheumatoid arthritis are just a few of the conditions that it

aggravates. Addiction treatment may involve the use of medicines such as buprioprin, verenicline, NRT therapy, clonidine, and nortryptyline, as well as medical counseling and attendance at smoking cessation programs. The major intervention should focus on the positive impacts of stopping smoking rather than the negative effects of tobacco use. Tobacco consumption and related mortality can be averted by making individuals aware of the health advantages of quitting tobacco and the adverse effects of consuming tobacco via the implementation of various plans and initiatives. Tobacco should be avoided if you want to live a happy and healthy life and, to be avoided tobacco-related cancer.

References

- 1. Chinnappan Ravinder Singh, Kandaswamykathiresan. (2015) Effect of cigarette smoking on human health and promising remedy by mangroves. Asian pacific journal of tropical biomedicine 5 (2): 162-167.
- 2. https://www.who.int/news-room/fact-sheets/detail/tobacco
- 3. Sasco, A. J., Secretan, M. B., &Straif, K. (2004). Tobacco smoking and cancer: a brief review of recent epidemiological evidence. *Lung cancer*, 45, S3-S9.
- 4.NStatistics. National vital statistics report. 2016.
- 5. Pope III, C. A., Burnett, R. T., Turner, M. C., Cohen, A., Krewski, D., Jerrett, M., ... & Thun, M. J. (2011). Lung cancer and cardiovascular disease mortality associated with ambient air pollution and cigarette smoke: shape of the exposure–response

- relationships. *Environmental health perspectives*, 119(11), 1616-1621.
- 6. Yadav, D., &Lowenfels, A. B. (2013). The epidemiology of pancreatitis and pancreatic cancer. *Gastroenterology*, 144(6), 1252-1261.
- 7. Chuang, S. C., Lee, Y. C. A., Hashibe, M., Dai, M., Zheng, T., &Boffetta, P. (2010). Interaction between cigarette smoking and hepatitis B and C virus infection on the risk of liver cancer: a meta-analysis. *Cancer Epidemiology and Prevention Biomarkers*, 19(5), 1261-1268.
- 8. Limsui, D., Vierkant, R. A., Tillmans, L. S., Wang, A. H., Weisenberger, D. J., Laird, P. W., ... & Limburg, P. J. (2010). Cigarette smoking and colorectal cancer risk by molecularly defined subtypes. *JNCI: Journal of the National Cancer Institute*, 102(14), 1012-1022.
- 9. Gandomani, H. S., Tarazoj, A. A., &Salehiniya, H. (2017). Cigarette: The Silent Killer in the World. *Biomedical Research and Therapy*, 4(9), 1624-1628.
- 10. Centers for Disease Control Prevention. Classification of Diseases, Functioning, and Disability(2016). https://www.cdcgov/media/releases/2016/p1110-vital-signs-cancer-tobacco.html.206.
- 11. K Tanaka, I Tsuji, K Wakai, C Nagata, T Mizoue, M Inoue. (2006). DRG for the, JE of Cancer Prevention Strategies in. Cigarette smoking and liver cancer risk: an evaluation based on a systematic review of epidemiologic evidence among Japanese. Japanese Journal of Clinical Oncology. 36: 445.
- 12. Global Tobacco Adults Survey. Available at

- www.mohfw.nic.in/WriteReadData/l892s/1 455618937GATS%20India.pdf;2009
- 13. Global Burden of Disease Cancer Center, Fitzmaurice C, Dicker D, et al. (2015). The global burden of cancer 2013. JAMA Oncol.1(4):505–527.
- 14. Can smoking cause esophageal cancer? |
 Roswell Park Comprehensive Cancer Center
 Buffalo, NY
- 15. E Giovannucci, EB Rimm, A Ascherio, GA Colditz, D Spiegelman, MJ Stampfer. (1999). Smoking and risk of total and fatal prostate cancer in United States health professionals. Cancer Epidemiol Biomarkers Prevention. 8:277-82.
- 16. DT Silverman, JA Dunn, RN Hoover, M Schiffman, KD Lillemoe, JB Schoenberg. (1994). Cigarette smoking and pancreas cancer: a case-control study based on direct interviews. Journal of the National Cancer Institute.86: 1510-6.
- 17. F CS, C GA, S MJ, G EL, H DJ, R EB. (1996). A prospective study of cigarette smoking and the risk of pancreatic cancer. Archives of internal medicine.156:2255-60. 18. S Iodice, S Gandini, P Maisonneuve, AB Lowenfels. (2008). Tobacco and the risk of pancreatic cancer: a review and meta-analysis. Langenbecks' Archives of Surgery. 393: 535-45.
- 19. JE Muscat, SD Stellman, D Hoffmann, EL Wynder. (1997). Smoking and pancreatic cancer in men and women. Cancer Epidemiol Biomarkers Prevention. 6:15-9.
- 20. SH Health UDo. (2004). The health consequences of smoking: a report of the Surgeon General.

- 21. IARC. (1986). IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans, Tobacco Smoking. *Lyon, France* IARC. 38.
- 22. IARC. (2002). IARC Handbook of cancer prevention, Weight Control and Physical Activity. *Lyon, France*: IARC Press.6.
- 23. P Ryan, MW Lee, B North, AJ McMichael. (1992). Risk factors for tumors of the brain and meninges: results from the Adelaide Adult Brain Tumor Study. International Journal of Cancer.51:20-7. . 24. D Wenbin, C Zhuo, M Zhibing, Z Chen, Y Ruifan, J Jie. (2013). The effect of smoking on the risk of gallbladder cancer: a meta-analysis of observational studies. European Journal of Gastroenterology& Hepatology.25:373-9.
- 25. D Aune, LJ Vatten, P Boffetta. (2016). Tobacco smoking and the risk of gallbladder disease.
- 26. S Kono, K Shinchi, N Ikeda, F Yanai, K Imanishi. (1992). Prevalence of gallstone disease in relation to smoking, alcohol use, obesity, and glucose tolerance: a study of self-defense officials in Japan. *American Journal of Epidemiology*. 136:787-94. 27. M Okamoto, Z Yamagata, Y Takeda, Y Yoda, K Kobayashi, MA Fujino. (2002). The relationship between gallbladder disease and smoking and drinking habits in middleaged Japanese. *J. ofGastroenterology*.37:455-62.
- 28. W Kratzer, V Kachele, RA Mason, R Muche, B Hay, M Wiesneth. (1997). Gallstone prevalence in relation to smoking, alcohol, coffee consumption, and nutrition. The Ulm Gallstone Study. Scandinavian

- Journal of *Gastroenterology*.32:953-8.
 29. H Pastides, A Tzonou, D Trichopoulos, K Katsouyanni, A Trichopoulou, N Kefalogiannis. (1990). A case-control study of the relationship between smoking, diet, and gallbladder disease. *Archives of Internal Medicine*. 150:1409-12.
- 30. AJ McMichael, PA Baghurst, RK Scragg. (1992). A case-control study of smoking and gallbladder disease: importance of examining time relations. Epidemiology (Cambridge, Mass.). 3: 519-22.
- 31. J Brisson, C Morin, M Fortier, M Roy, C Bouchard, J Leclerc. (1994). Risk factors for cervical intraepithelial neoplasia: differences between low- and high-grade lesions. *American Journal of Epidemiology*. 140: 700-10.
- 32. L Kjellberg, G Hallmans, AM Ahren, R Johansson, F Bergman, G Wadell. (2000). Smoking, diet, pregnancy and oral contraceptive use as risk factors for cervical intra-epithelial neoplasia in relation to human papillomavirus infection. *British Journal of Cancer.* 82: 1332-8.
- 33. AO Olsen, K Gj?en, T Sauer, I rstavik, O Naess, K Kierulf. (1995). Human papillomavirus and cervical intraepithelial neoplasia grade II-III: a population-based case-control study. *Int. J. of Cancer*.61:312-5.
- 34. M Schiff, TM Becker, M Masuk, L van Asselt-King, CM Wheeler. (2000).KK Altobelli. Risk factors for cervical intraepithelial neoplasia in southwestern American Indian women. *Amer. J. of Epidemiology*. 152:716-26

- 35. X Castellsague, FX Bosch, N Munoz. (2002). Environmental co-factors in HPV carcinogenesis. *VirusResearch*. 89:191-9.
- 36. PE Castle, S Wacholder, AT Lorincz, DR Scott, ME Sherman, AG Glass. (2002). A prospective study of high-grade cervical neoplasia risk among human papillomavirus-infected women. I. of the National Cancer Institute. 94:1406-14. 37. MH Schiffman, NJ Haley, JS Felton, AW Andrews, RA Kaslow, WD Lancaster (1987).Biochemical epidemiology of cervical neoplasia: measuring cigarette smoke constituents in the cervixCancerResearch.47:3886-8.
- 38. JM Palefsky, EA Holly. Molecular virology and epidemiology of human papillomavirus. 1995.
- 39. Hayes R.B., et al. (2005). Methods for etiologic and early marker investigations in the PLCO trial. *Mutat. Res.*, 592, 147–154. 40. Prorok P.C., et al. (2000). Design of the Prostate, Lung, Colorectal and Ovarian (PLCO) Cancer Screening Trial. *Control. Clin. Trials*, 21(suppl. 6), 273S–309S.
- 41. IARC. (1994).IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans, Schistosomes, Liver Flukes and Helicobacter pylori. Lyon, France: *IARC*. 16.

- 42. Giovannucci E., et al. (1994). A prospective study of cigarette smoking and risk of colorectal adenoma and colorectal cancer in U.S. men. *J. Natl Cancer Inst.*, 86, 183–191.
- 43. Tulinius H., et al. (1997). Risk factors for malignant diseases: a cohort study on a population of 22,946 Icelanders. *Cancer Epidemiol. Biomarkers Prev.*, 6, 863–873.
- 44. Wu A.H., et al. (1987). Alcohol, physical activity and other risk factors for colorectal cancer: a prospective study. *Br. J. Cancer*, 55, 687–694.
- 45. Nordlund L.A., et al. (1997). Cancer incidence in female smokers: a 26-year follow-up. *Int. J. Cancer*, 73, 625–628.
- 46. Wakai K., et al. (2003). Smoking and colorectal cancer in a non-Western population: a prospective cohort study in Japan. *J. Epidemiol.*, 13, 323–332.
- 47. Secretan B., et al. (2009). A review of human carcinogens—Part E: tobacco, areca nut, alcohol, coal smoke, and salted fish. *Lancet Oncol.*, 10, 1033–1034
- 48. Saraiya M, Unger ER, Thompson TD, et al.; HPV Typing of Cancers Workgroup. US assessment of HPV types in cancers: implications for current and 9-valent HPV vaccines. J Natl Cancer Inst 2015;107: djv086.