The Role of Smoking in the Development of Chronic Obstructive Pulmonary Disease

Sivtseva A.I., Neustroeva T.S., Ivanova M.A., Petrova E.R.

The main factor for chronic obstructive pulmonary disease (COPD) is smoking. Scientific researches conducted in recent years have established that smoking during index exceeding the number 120 (the number of cigarettes smoked per day multiplied by the number of months in the year, during which time a person smokes) typically develop symptoms of COPD. It is also proved that the COPD risk increases at the age of 40-60 years. Reputable studies have shown that smoking contributes to such social factors as loneliness, unemployment, etc. In Yakutia smoking has long been widespread and with severe climatic factor it is the factor for the development of COPD.

Keywords: COPD, smoking, epidemiology, risk factors.

Chronic Obstructive Pulmonary Disease (COPD) is a disease characterized not fully reversible bronchial obstruction. Airflow limitation is usually progressive and associated with abnormal inflammatory response of the respiratory tract to harmful particles or gases (mostly smoking) [2].

Currently smoking is considered as a socio-medical problem that involves two approaches:

1 –widely-spread perception of smoking as a habit being harmless enough or having possible but uncertain health risk [3, 21];

2 –perception of smoking asa habit having objective statistics of health damage. It is known that in male population aged 45 and elder over 20% of cardio-vascular disease and 22% of deaths from this disease are caused by smoking [6].It is proved that relative risk of lung cancer among smokers is 9.2 - 14.9 timeshigher depending on time period and intensity of smoking [15].

According to prognosis of the World Health Organization by 2020 tobacco epidemic will move from Western Europe and America whereantismoking propaganda was conducted during the last 20-30 years to developing countries where public health system is unable to fight the epidemic because of lack of antismoking programsfunding. This can be fully applied to Russia [31].

There are two types of smoking: passive and active. Active smoking is the most aggressive risk factor.Significant period of time (sometimes more than 10-15 years) usually pass before clinical manifestations of COPD make people to recognize pathogenic effect of smoking [13]. According to the authors [20] frequency of chronic bronchitis (CB) among smokers is 2.5-4 times higher than

among non-smokers.

Representative sampling of the population of Russia has shown that in our country more than half of men (63.2%) and one in ten women (9.7%) aged 15 and older smoke [10].

Of great importance is the study of patterns of spread of smoking in the population, that is, its dependence on various demographic and social characteristics. It is well established fact that proportion of smokers in the populationdecreases with age increase. The decrease usually begins at first slightly after age of 40 and essentially after 60. Correlation is revealed between smoking and other social characteristics. It is known that low standard of living, social disorders, unemployment, loneliness, low level of educationpredispose smoking [34].

Today pathological effects of smoking on the respiratory system are well studied. Tobacco smoke getting into the respiratory tract damagesmucociliary apparatus [22], causes functional insufficiency of antiprotease in the lower respiratory tract leading to hyperplasia of bronchial glands that produce mucus, damages the immune system protecting lungs, causes allergic response of organism and release of histamine because of degranulation of basophils, results in difficulty of lymph drainage from bronchi and disorders microcirculation in pulmonary capillarybloodstream and impairs lung function [18].

It is suggested that under toxic influence of tobacco smoke alveolar macrophages are damaged with release of oxidizing agents and lysosomal enzymes that causes not onlydamage of lung tissue but also their infection. On the base of the carried out studies [32] it is suggested that tobacco smoke also destroys surfactant or inhibits its production.

There are also studies in which the authors are less categorical in estimation of the negative effect of smoking on lung function compared to the influence of such factors as age, sex, constitutional type, individual parameters of bronchial reactivity [26, 27, 30, 38].

In adults clinical manifestation of chronic bronchitis usually occurs after 3.5 years from start of smoking. With the number of smoked cigarettes increases the risk of disease and mortality from it. Thus, according to the publications,among smokers aged 40-49 and 50-59 chronic bronchitis occurs in 5.2 and 7.4% of cases, among those who smoke from 1 to 9 cigarettes a day - in 8.2 and 13.7% , among those who smoke from 10 to 19 cigarettes a day - in 16.2 and 20.9%,among those who smoke more than 20 cigarettes a day - in 21.4 and 27.4% and in those who smoke more than 40 cigarettes a day risk of chronic bronchitis is 45 times higher compared to non-smokers [7, 9].

Smoking is reliably established and the main risk factor of chronic obstructive bronchitis (COB). Giving up of smoking slows down the rate of decline of lung ventilation function. Tobacco smoking is the cause of higher mortality rate from COPD and there are insignificant differences

observed between various types of tobacco and method of smoking. In Kazakhstan COB casesamong intensive smokers were revealed 2.1 times morefrequently than among non-smokers, 1.7 times more frequentlythan among those who gave up smoking, and 1.4 times more frequentlythan among moderate smokers ($P < 0.05$) [1, 12, 14].

Correlation of cigarette smoking and COPD is more complex than it is usually represented. Despite the close relationship between them smoking itself is not enough for development of the disease. COPD occurs only in minority (about 15%) of long-term tobacco smokers and this fact suggests the presence of some unknown factor that promotes the development of respiratory tract injury. According to the "Dutch hypothesis"there may be some genetic predisposition to respiratory tract injury for COPD development in smokers.The problem is complicatedby the fact that a small number of never-smokers also sufferCOPD which does not differ from that caused by smoking [19].

According to the publications dealing with the problem, smoking increases negative impact on pulmonary system not only environmental but also occupational factors (dust, cement, lime, etc.). For example, smoking increases the negative impact of grain on pulmonary system. Besides of damaging effect of tobacco smoke, tobacco dust also harmfully influences pulmonary systemof people who come in contact with tobacco dust while working in tobacco production. Thus, the prevalence of chronic bronchitis in women was 22% and in men - 15%. However, among smokers chronic bronchitis prevailed in men rather than in women. Thus, according to T.N.Bilichenko et al. (1991) chronic bronchitis occurs among men-smokers 5 times and among women - 3 times more frequently than among never-smokers [4].

Study in Norway showed that professional factors 2-3 times increase risk of chronic bronchitis among farmers but when combined with smoking the risk becomes 6 times higher [23, 24, 25, 28, 29, 33, 35, 37, 39].

Recently conducted studies have established the following,if smoking index is higher than120 (the number of cigarettes smoked per day multiplied by the number of months in the year during which a person smokes),as a rule, COPD symptomsdevelop. It is desirable ofa doctor toregister smoking index in patient's medical card to judge about smoking as a risk factor for the disease in each patient. Those having index 240 (a person smokes 20 cigarettes per day all year through)are defined as “hard smokers”. There is another calculation formula which takes into account time period of smoking and is expressed by the formula “pack / years” i.e. if smoking period is 20 years and a person smokes a pack of cigarettes per dayhis or her smoking index is 20 pack / years [8].

In recent years researchers pay great attention to study of the state of vascular endothelium

in various pathologies. Vascular endothelium is metabolically highly active monolayer of cells lining all blood vessels of a human body. Endothelial cells, specifically responding to different molecular signals, perform a variety of functions, including transport and barrierones, they are involved in metabolism of extracellular matrix, biosynthesis of various cytokines, angiogenesis regulating the process of blood coagulation and platelet aggregation, vascular tone, and immunological reactions. Endothelial activation or damage isfundamentallyimportantfor development of a wide range of disease processes. Obviously, evaluation of endothelial state may have very important clinical implication for understanding of pathogenesis of many human diseases [5, 11, 16, 17, 36].

There are many epidemiological papers concerning tobacco smoking (Table № 1.) [15].

Table 1.**Epidemiological Characteristics of Smoking Prevalence among Adults in the Russian Federation and Abroad.**

Author, year	Contingent surveyed	Place ofepidem.survey.	Men.%	Women.%
R.Liardet al.,1980	899 people	France, Paris	57.0	41.4
M.R. Pandley,1984	2826 people,20-80 years old	Nepal	78.3	58.9
K.Yan,1985	922 people, 18-80 years old	Australia	25.2	18.0
M.Krazyzanowskyet al.,1986	4335 people	Poland, Krakow	59.6	26.7
G.Vegy,1988	3289 people, 20-64 years old	Italy, Venice	49.2	26.9
S.L.Aleynikov, A.N.Kokosov, 1982	5935 men, 6770 Women,adult population	Southern coast of Crimea	85.0	6.29
S.L.Oleynikov et al.,1983	1913 men, 40-59 years old 2361 men,40-59 years old	Moscow, Kaunas	48.2 43.1	
R.F.Mukhametzhanova, 1987	6619 men,40-59 years old	Moscow	47.2	
M.A.Staponkene,1987	5208 men,40-59 years old	Lithuania	33.2	
A.E.Korolkov,1988	1110 people, 16-64 years old	Moscow, medical area	50.3	11.0
L.V.Shuteeva, S.L.Garnitsky. 1990	1500 men,20-69 years old	Kiev	51.1	
I.S. Petrukhin,1991	1785 men, 1621	Tver	63.1	8.6

	women,adult population			
L.V.Chazova et al., 1991	935 men, 1452 women,25-64 years old	Moscow	50.9	11.4
L.N.Korchagina,1996	475 men, 16-67 years old	Stavropol	62.3	
P.Aarva et al., 1999	1378 people,adult population	Vologda	57.0	10.0
E.Y. Mishineva,1999	1379 men, 335 women, builders	Moscow	41.0	49.0
AA Lenknovich et al., 1999	1006 men, 19-55 years old	Dzerzhinsk, Nizhny Novgorod	53.4	
E.R.Iskhakov, 2000	1138 men, 611 women, adult population	Bashkortostan	62.1	3.6
V.A.Glushchenko et al., 2000	316 men, 30-50 years old	Samara	46.5	
V.VI Shchekotov et al. 2001	420people, adult population	Perm	68.2	19.5
I.V.Drozdov, 2004	2004 people, adult population	Chelyabinsk	72.9	20.1
E.I. Bayanov. 2005	230 men,370 women	Leningrad Region.	68.7	17.6

Thus, data analysis of published papers concerning tobacco smoking as exogenous factor of COPD allows the following conclusions:

1. As it is seen from the above, all the researchers note that cigarette smoking contributes to the development of chronic bronchitis and point to a direct correlation of severity of chronic bronchitis to duration of smoking.

2. Currently, there are few studies of the state of vascular endothelium at COPD caused by smoking. Practically there are no data on insufficiency of endothelial function in COPD patients while there are enough factors determining development of endothelial dysfunction, namely hypoxia with increased content of various biologically active substances including cytokines, etc.

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Authors:

1. Sivceva Anna Innokent'evna – MD, Head of Respiratory Disease Department Research Institute of Health NEFU named after M.K. Ammosov, 677000, ul. Vinokurova 21 kv. 24 g. Yakutsk, the Russian Federation, cell-phone: 89141036523, e-mail: sannai@inbox.ru

2. Neustroeva Tat'jana Semenovna – PhD, Senior Researcher, Respiratory Disease Department Research Institute of Health NEFU named after M.K. Ammosov, 677009, ul. Dzerzhinskogo 36/1 kv. 22. g. Yakutsk, the Russian Federation, cell-phone: 89141064537, e-mail: sannai@inbox.ru

3. Ivanova Marianna Adol'fovna – Junior Researcher, Respiratory Disease Department Research Institute of Health NEFU named after M.K. Ammosov, 677005, ul. Lermontova 58, kv. 32, g. Yakutsk, the Russian Federation, cell-phone: 89241704624, e-mail: mariv.81@mail.ru

4. Petrova Evgenija Robertovna - Junior Researcher, Respiratory Disease Department Research Institute of Health NEFU named after M.K. Ammosov, 677000, Sergeljahskoe sh. 4 km. 2S, g. Yakutsk, the Russian Federation, cell-phone 89241712395, e-mail: mariv.81@mail.ru.