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Influence of smoking on exercise capacity
Influenţa fumatului asupra capacităţii de efort

Daniela Pintican, Dan Mihu
„Dominic Stanca" Obstetrics and Gynecology Clinic; University of Medicine and Pharmacy, Cluj-Napoca, Romania

Abstract

Smoke resulting from the burning of a cigarette contains up to 4000 chemical compounds, of which at least 50 are carcinogenic. The main chemical substances released after absorption in the respiratory system are nicotine, carbon monoxide and hydrocyanic acid.

Smoking is a risk factor for the development of respiratory, cardiovascular, muscle, bone, immune system diseases, as well as cancer. Exercise capacity represents the capacity of the circulatory, respiratory and muscular system to provide oxygen during sustained physical activity. Smoking is associated with a reduction of aerobic exercise capacity due to low oxygen supply and an impaired heart rate response (negative chronotropic effect), both of which represent important predictive factors of mortality. Nicotine induces the release of catecholamines, aggravates sleep disorders and difficulties falling asleep, and has a negative influence on overall performance capacity. Resistance to infectious bronchial and catarrhal diseases is crucial for smoking athletes, because optimal health is a basic requirement for sports training or the improvement of performance.

Keywords: smoking, aerobic capacity, anaerobic capacity, nicotine, smoking cessation

Rezumat

Fumul rezultat în urma arderii unei ţigări conţine până la 4000 de compuşi chimici, din care cel puţin 50 sunt carcinogeni. Principalele substanţe chimice eliberate, după absorbţia la nivelul aparatului respirator, sunt: nicotina, monoxidul de carbon şi acidul cianhidric.

Fumatul reprezintă un factor de risc în apariţia bolilor respiratorii, cardiovasculare, sistemului muscular, sistemului osos, sistemului imunitar şi în apariţia cancerului. Capacitatea de efort reprezintă capacitatea sistemului circulator, respirator, muscular de a furniza oxigen în timpul activităţii fizice susţinute.Fumatul este asociat cu reducerea capacităţii aerobe de efort datorită aprovizionării reduse a oxigenului şi un raspuns depreciat al ritmului cardiac (efect cronotrop negativ), ambele reprezentând factori predictori importanţi ai mortalităţii. Nicotina induce eliberare de catecolamine, accentuează tulburările de somn şi adormirea, acţionând defavorabil asupra capacităţii globale de performanţă. Rezistenţa la bolile infecţioase bronşice şi catarale este de o importanţă determinantă pentru sportivul fumător, deoarece o sănătate intactă reprezintă condiţia de bază în vederea antrenamentului sportiv sau a creşterii performanţelor.

Cuvinte cheie: fumat, capacitate aerobă, capacitate anaerobă, nicotină, renunţare la fumat.

General considerations

Smoke resulting from the burning of a cigarette contains up to 4000 chemical compounds, of which at least 50 are carcinogenic. The main chemical substances released after absorption in the respiratory system are nicotine, carbon monoxide and hydrocyanic acid (Genbacev-Krtolica, 2005; Weineck, 1995).

According to the World Health Organization (***, 2014), there are over 100 billion smokers worldwide. In the 20th century, smoking was responsible for about 100 million deaths; over the past years, there have been approximately 6 million deaths per year from smoking, a figure that is estimated to increase by 2030 to 8 million per year, of which more than 80% will be represented by developed countries (World Health Organization).

Carbon monoxide

The carbon monoxide content of a cigarette amounts to 1-3%, that of a pipe to approximately 2%, and that of a cigar to 6% (Staicu, 2009).

CO concentration in the exhaled air (COEA) is measured using a CO analyzer, into which the subject blows after an inspiration, followed by a 15 second apnea. In the absence of active or passive smoking, COEA is very low, below 5 ppm (particles per million). In active smokers, COEA exceeds 20 ppm and can reach higher values, over 70 ppm in heavy smokers. A percentage of 20 ppm can be found in passive smokers. The measurement of COEA is the most effective test for detecting non-smokers (below 5 ppm) and smokers (over 10-30 ppm).

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Address for correspondence: University of Medicine and Pharmacy, Obstetrics and Gynecology Clinic Dominic Stanca, 21 Decembrie 1989 Av. No 57 , 400124 Cluj-Napoca, Romania
E-mail: dana_bodnar@yahoo.com
Corresponding author: Daniela Pintican

Carbon monoxide (CO) rapidly diffuses through alveolar membranes and pulmonary capillaries, having an increased affinity for hemoglobin, with which it combines to form carboxyhemoglobin (COHb), an affinity 245 times higher than that of oxygen, thus reducing the oxygen carrying capacity and oxygen release, by shifting the oxygen dissociation curve to the left (Rudra et al., 2010). The formation of COHb is reversible by removal of the CO source in favor of inspired oxygen, so that after 4 hours of tobacco abstinence, its value is reduced to half. Carbon monoxide is also an inhibitor of carbonic anhydrase (Ganong, 2005).

The basal COHb level in humans is about 0.1-1% in the absence of environmental contamination or smoking. In the blood of a smoker, the COHb percentage usually ranges between 5-15% or more. COHb levels higher than 20% are usually associated with clinical toxicity symptoms, which can lead to neurotoxicity, cognitive disorders, unconsciousness, and death at chronic or increased concentrations (Gorman et al., 2003).

Recent studies have shown the fact that low CO concentrations can influence the intracellular signal transduction pathways. CO may exert vasoregulatory properties, as well as inflammation, modulation, apoptosis, cellular proliferation in vitro and in vivo.

Cellular exposure to CO has been demonstrated to directly or indirectly modulate the activity of a number of intracellular signaling molecules. Similarly to NO, CO can act as a heme ligand and an activator of soluble guanylyl cyclase (sGC), to increase the production of cyclic guanosine monophosphate (cGMP). Experimental evidence indicates that NO activates sGC in vitro and corresponding vasodilatory action in vivo with greater potency (Furchgott & Jothianandan,1991).

CO is implicated in the first place as a regulator of the sGC/cGMP axis in the context of olfactory transmission (Verma et al., 1993). CO may exert vasorelaxant effects in the liver and other vascular beds that are considered to be cGMP dependent (Durante et al., 2006). Alternatively, CO may also regulate vascular function through additional mechanisms, including the inhibition of cytochrome P450 and the activation of calcium dependent potassium channels in smooth muscle vascular cells (Wang, 2006).

CO can modulate the activation of mitogen-activated protein kinases (MAPK), which are important mediators of inflammatory and stress responses. A potent anti-inflammatory effect of CO has been demonstrated in bacterial lipopolysaccharides (LPS) – stimulating the macrophages, which depend on the modulation of mitogen-activated protein kinase kinase 3 (MKK3)/p38 MAPK (Zhang et al., 2003).

Nicotine

Nicotine, α-3-pyridyl-N-methylpyrrolidine, is a pyrrolidine alkaloid extracted from the leaves of Nicotiana species (tabacum, rustica americana), which contain 1-8+ % active substance. The nicotine content in various tobacco products is variable: 13-32 mg/cigarette, 5-7 mg/cigarette butt, 15-40 mg/cigar, 12-16 mg/1g snuff tobacco.

Nicotine can be introduced into the body by several routes: skin, lungs or mucosae (nasal, gingival). The most common method for introducing nicotine into the body is its inhalation, by smoking. After inhalation, nicotine enters the blood, where it attains an optimal concentration and rapidly reaches (7 seconds) certain brain cholinergic receptors. It stimulates the brain and is responsible both for pleasant sensations during smoking and for irritability specific to the moment when a smoker tries to quit smoking. Nicotine takes its effect in 10-15 seconds, and this effect lasts for about 60 minutes. After 6 hours, 0.031 mg of the nicotine content of a cigarette (0.8-0.7 mg nicotine/ cigarette) remain in the body (Buzoianu, 2002). 80% of nicotine is destroyed by the liver and 19% is eliminated by the kidneys in an unchanged form.

Nicotine has physiological and pathological effects on a variety of organs and systems of the body:

- lymph node system;
- nicotinic acetylcholine receptors (nAChRs) of chromaffin cells through catecholamines;
- stimulation of the central nervous system by nAChRs (Aseem et al., 2015).

Through the catecholamines released from the sympathetic adrenomedullary system, nicotine determines:

- cardiovascular changes: peripheral vasoconstriction, increase of the heart rate and output, increase of blood pressure;
- metabolic changes: increase of glycemia and free fatty acids;
- antidiuretic hormone secretion;
- increase of platelet aggregation (Schneider & Henrion, 1979).

Nicotine and CO concentration are important markers of smoking in the short term. The elimination half-life of nicotine is short (2-3 hours); 80% is metabolized into cotinine, which is found in all biological media (blood, urine, saliva, milk, amniotic fluid, placenta, hair, meconium). This is a good marker of daily smoking, and its half-life is about 15 hours in active smokers and 27 hours in passive smokers. Morning cotininemia has a high sensitivity, specificity and stability in active smokers. It also allows to evaluate passive smoking.

Hydrocyanic acid

Cigarette smoke contains small amounts of cyanide that combines in the body with hydroxocobalamin and thiosulfate, forming hydroxocobalamin and thiocyanate (which has a hypotensive effect). Cyanide inhibits the activity of essential enzymes involved in cellular respiration, such as cytochrome oxidase and carbonic anhydrase (Schneider & Henrion, 1979).

Passive smoking is the secondary exposure to cigarette smoke or other tobacco derivatives of non-smokers, who live in indoor environments with smokers. It is associated with a high risk of COPD, bronchial asthma, lung cancer, ischemic heart disease, acute myocardial infarction, cerebrovascular disease and respiratory infections (Costa et al., 2006).

The effects following the short-term exposure to tobacco smoke, such as irritation of the eyes and airways, among passive smokers (Junker et al., 2001). Even short-term exposure to tobacco smoke can generate significant negative effects on the human respiratory system, as shown in a recent comment (Flouris & Koutedakis, 2011). Finally, Pope et al. (2001) suggest that the effects of acute

exposure to tobacco smoke on autonomic cardiac function may contribute to pathophysiological mechanisms that link passive smoke exposure to an increased risk of cardiovascular mortality (Flouris et al., 2010).

Active smoking is defined as the smoking of at least one cigarette per day, involving nicotine dependence and the maintenance of nicotine levels above a certain threshold. Active smoking induces a number of negative effects on all the organs of the human body.

Smoking - a risk factor for health

a) *The bone system*

Smoking is associated with low bone mineral density, regardless of age and sex (Ortego et al., 1997).

The effect of smoking on bone mineral density increases with age, particularly in postmenopausal women. The association of smoking with low bone density and hip fracture is undoubtedly a cause-effect relationship. Risk is reduced in persons who quit smoking, with a dose-response relationship depending on the number of cigarettes smoked. Smokers have a high risk to develop osteoporosis and fractures, because of a low bone mineral mass (Ward & Klesges, 2000).

The study performed by Fusby et al. (2010) showed that exposure of mice to smoke induced a 50% diminution of B220+ CD43- bone marrow cells - premature, immature and mature B cells, but not B cell precursors, and increased the percentage of CD8+ mature splenic T cells to the detriment of CD4+ T cells. These effects are reversible after the cessation of smoking, the bone marrow having the capacity to restore B cells after smoke exposure. These data provide the first proof that cigarette smoke has a negative impact on the bone marrow. Because B cells that are negatively affected are able to produce osteoprotegerin that determines the formation of osteoclasts, smoking induces a reduction of B cells in the bone marrow, which suggests a plausible mechanism for the way in which smoking contributes to the development of osteoporosis, but also suggests that the restoration of bone marrow B cells lost by smoking exposure can be achieved by smoking cessation (Fusby et al., 2010).

b) *The muscular system*

In patients with chronic pulmonary disease, exercise intolerance has been found. In about 40% of the cases, exercise capacity is limited by skeletal muscle alterations rather than pulmonary problems. Chronic obstructive pulmonary disease (COPD) is frequently associated with a reduction in the number of muscle fibers and a change of their composition, which induces an earlier onset of muscle fatigue (Wüst & Degens 2007).

Healthy smokers frequently complain of an increased sensation of fatigue (Corwin et al., 2002). The etiology of this early onset of fatigue in smokers is unknown and it is not clear whether smoking or other factors such as a reduced level of physical exercise contribute to this phenomenon (Larsson et al., 1988). Studies carried out in young male subjects undergoing physical activity, compared to control subjects, have demonstrated that smoking itself induces a significant decrease of skeletal muscle mass and thus, a lower resistance to fatigue (Morse et al., 2007). The reduction of exercise capacity in healthy smokers suggests that long-term smoking may cause a progressive skeletal deterioration and muscle fatigue (Bernaards et al., 2003).

c) *The cardiovascular system*

Chronic exposure to cigarette smoke determines a prothrombotic as well as an atherogenic effect, increasing the risk of coronary disease, acute myocardial infarction, sudden cardiac death, cerebrovascular accident, aortic aneurysm and peripheral vascular diseases. Even very low exposure doses increase the risk of acute myocardial infarction.

Smoking cessation decreases cardiovascular mortality and morbidity by up to 5 years, regardless of the age and sex of the smoker (Bullen, 2008).

Nicotine stimulates the excitoconductor system of the heart, due to catecholamine release, which determines an increase in the heart rate by 10-20 pulsations/minute. This effect decreases in 15-45 minutes and is no longer detectable after 2-3 hours (Aseem et al., 2015).

In the vascular territory, nicotine induces vasoconstriction, followed by an increase of blood pressure. After smoking one cigarette, finger temperature decreases due to vasoconstriction by 0.6-3.8 degrees in 2 minutes, this effect lasting for 3-4 hours.

Tachycardia and vasoconstriction determine an increase in systolic and diastolic blood pressure, which leads to an intensification of cardiac strain and to a corresponding increase of oxygen requirements.

In a number of observational studies, smoking has been associated with high plasma concentrations of total cholesterol, low-density lipoprotein (LDL cholesterol) and triglycerides, low concentrations of high-density lipoprotein (HDL cholesterol), central obesity, an increase of blood pressure and heart rate (Asvold et al., 2014).

d) *The respiratory system*

Smoking induces a decrease of expiratory volume, a deterioration of alveolar gas exchange, a reduction of respiratory flow and an increase in the oxygen requirements of respiratory muscles.

The diminution of respiratory flow and the increased oxygen requirements of respiratory muscles are due, among others, to the fact that resistance to the passage of air through the airways increases during effort in smokers compared to non-smokers (by 40-50%), which is explained by mucosal edema and mucus overproduction (Flouris et al., 2012). Smoking as well as other factors can potentiate the effect of a respiratory disease on the human body. Studies have shown an association between smoking and respiratory diseases (Murin et al., 2000). Smoking may affect the immune system of the body, and the recovery time after disease can be longer than usual (Murin & Bilello, 2005). Smokers are more predisposed to influenza than non-smokers, and they have an increased risk of bacterial pneumonia, tuberculosis, COPD (Wüst & Degens, 2007; Ryter & Choi, 2013).

In conclusion, smoking has been found to double the number of respiratory disease cases. About 6 million people die every year because of smoking or its complications through respiratory diseases. However, the number of smokers increases every year. This is why smoking is a serious problem in many countries (World Health Organization).

Smoking increases the risk of developing respiratory

diseases by two times and reduces defense against infections, so that infections in smokers are more frequent than in non-smokers. Anti-smoking campaigns can significantly reduce the spread of respiratory diseases (Aldila & Apri, 2014).

Acute effects on respiratory function have been less significant and mainly limited to ventilation-perfusion abnormalities.

e) *The endocrine system*

Nicotine determines an intensification of the formation of thyroid hormones, with an enhancement of general catabolism. This leads, in the case of usual smokers, to a transient weight gain when smoking is stopped, due to a decrease in the lipolytic activity of protein lipase in the absence of nicotine.

Nicotine also induces the release of ACTH and ADH (Staicu, 2009).

f) *The nervous system*

The effects of nicotine are mediated centrally. Nicotine has a neuroregulatory impact on the nervous system, affecting biochemical and physiological functions. Circulating epinephrine and noradrenaline levels are increased, and the bioavailability of dopamine is also changed. Neuroendocrine effects include the release of arginine vasopressin, β-endorphin, adrenocorticotropic hormone and cortisol. Some of these neurochemical substances are psychoactive and/or involved in the modulation of behavior. Thus, emotional and cognitive states can be influenced by the dose of nicotine. When nicotine is inhaled, the neuroregulatory effects described above are immediately available.

In small doses, nicotine induces an increase of dopamine levels in the brain, through the inhibition of monoamine oxidase. Dopamine, an important neuromediator of the perception of pleasure, generates a state of good humor and focused attention. In high or repeated doses, it inhibits synaptic transmission. Under the influence of nicotine, changes in brain waves (desynchronizations) occur (Pomerleau, 1992).

g) *The excretory system*

The risk of chronic renal diseases in smokers is increased. Smoking increases albumin excretion in the urine, decreases the glomerular filtration rate, increases the incidence of renal artery stenosis, and is associated with an increase of mortality in patients with end-stage renal disease. The pathogenesis of renal involvement is due to the action of nicotine on the COX-2 isoform, which causes an increase of acute glomerular inflammation, glomerulonephritis and ureteral obstruction. Thus, nicotine inhibits diuresis (Jaimes et al., 2009).

h) *The immune system*

The qualitative and quantitative effects of cigarette smoke on the immune system depend on the duration of smoking, sex, ethnicity of the studied subjects.

Chronic smoking alters a wide range of immunological functions, both innate and acquired immunity.

Innate immunity

The lungs are an important route of exposure to pathogenic agents and antigens; specific and non-specific defense mechanisms are involved in lung defense. Protection against foreign substances reaching the pulmonary alveoli is ensured by the innate immune system and adaptive immune response. Alveolar macrophages and other monocytes are the most important part of the innate immune system in the lungs. Smoking is a risk factor for acute respiratory diseases and COPD (Wüst & Degens, 2007), alveolar macrophages from the alveolar lavage playing a major role in the pathogenesis of these diseases (Sopori, 2002).

Because smoking is associated with an increased risk for different types of cancer, the effects of smoking on the function of natural killer (NK) cells, lymphoid cells with a role in the monitoring of tumor growth, have been evaluated. The activity of NK cells in melanomas and other cancer cells is significantly reduced in smokers compared to non-smokers. The development of cancer might partially result from the effects of cigarette smoke on the immune system (Sopori, 2002).

Acquired immunity involves specific responses that are triggered by antigens of various origins and are mainly executed by T cells and B cells. A well documented effect of cigarette smoke is leukocytosis; however, the function of these cells is significantly reduced. Smoking is an important cause of morbidity during an influenza epidemic. This might lead, in part, to small titers and a decreased half-life of influenza specific antibodies. Studies have shown that long-term smoking significantly reduces serum immunoglobulin levels (Sopori, 2002) .

Based on the particle size, cigarette smoke is composed of two phases – a vapor phase and a particle phase – each containing thousands of chemical substances. Chronic exposure to the vapor phase does not suppress the immune system, which indicates the fact that one or more components of the particle phase is immunosuppressive. In the particle phase, the most important part of nicotine is found. Animals that are chronically treated with nicotine have a significant antibody loss and T cell proliferation (Sopori, 2002).

i) *The male reproductive system*

Nitric oxide (NO) released by the parasympathetic system plays an essential role in erectile function by vasodilation and relaxation of the cavernous body. Nicotine induces NO insufficiency. This may lead to erectile dysfunction. Various studies on animals suggest that nicotine determines the degeneration of seminal canals, disturbing spermatogenesis in germ cells. Nicotine diminishes testosterone levels secondary to a decreased production of StAR – a protein that plays an important role in testosterone biosynthesis (Aseem et al., 2015).

j) *The female reproductive system*

- *Effects on the menstrual cycle*

Nicotine, through the inhibition of 21-hydroxylase, determines a hypoestrogenic status, the formation of androgenic hormones, which induces chronic anovulation and irregular menstrual cycles. Nicotine acts on the endometrium by inadequate cytokine production and irregular bleeding (Jin et al., 1997).

- *Effects on oocytes*

In studies performed on animals, it has been found that nicotine affects the ovaries and changes oocyte production. Oocytes treated with nicotine lose their spherical shape, they have a rough, disrupted surface, and the zona pellucida is irregular. Nicotine also prevents the maturation of the

oocyte. The blood flow in the oviduct is reduced and thus, fertility is affected (Hammer et al., 1981).

Effects of smoking on exercise capacity

Exercise capacity is the ability of the active muscular system to release the energy required for maximum mechanical work and its maintenance for as long as possible.

Exercise capacity is the basic factor for obtaining performance by sports training (Duck-chul et al., 2010).

Aerobic capacity

Aerobic exercise capacity is the ability of the body to make efforts up to a submaximal level, using the energy produced by aerobic pathways, without lactate accumulation.

The term aerobic system designates all reactions and metabolic pathways in which ATP can by synthesized from food sources, but only in the presence of oxygen. The aerobic system includes aerobic glycolysis, the Krebs cycle and the electron transport system (ETS). ETS, also referred to as the respiratory chain, represents a series of chemical reactions that occur in mitochondria, in which electrons and hydrogen ions combine to form water, and ATP is resynthesized (Afzal et al., 1998).

Smoking is associated with a reduction of aerobic exercise capacity due to low oxygen supply and an impaired heart rate response (negative chronotropic effect), both representing important predictive factors of mortality (Thier de Borba et al., 2014).

Active smokers have an aerobic capacity that can be reduced up to 12% because of the increased carbon monoxide concentration in the blood. During exercise, the muscles under strain are supplied with carbon monoxide from the blood, and the heart rate increases to maintain adequate oxygen requirements for the muscles. Smoking determines during physical exercise an additional energy cost due to the increased activity of respiratory muscles. Smoking cessation and the practice of regular physical activities can cause the restoration of aerobic capacity (Costa et al., 2006).

Persons with a reduced cardiorespiratory capacity are more susceptible to develop systemic arterial hypertension (SAH) (Barlow et al., 2006), diabetes (Sawada et al., 2010), and metabolic syndrome (LaMonte et al., 2005), and have higher mortality rates because of cardiovascular diseases (Bullen, 2008) and cancer (Sopori, 2002) than persons with a good cardiorespiratory capacity.

Wüst et al. (2008) found that the reduction of exercise capacity in smokers was similar in men and women and was not related to a long history of smoking. The decrease of exercise capacity might be caused by neuromuscular transmission failure, changes in the contractile properties of skeletal muscles, reduced oxygen supply to muscles and/or muscle oxidative capacity (Degens et al., 2005). Previous studies found that smokers had a lower activity of mitochondrial enzymes such as cytochrome oxidase (Alonso et al., 2003).

The inhalation of tobacco smoke leads to an alteration of nicotine dependent aerobic capacity, regardless of the level of physical exercise performed (Tchissambou et al., 2004).

The absence of a significant correlation between a long history of smoking (Wüst et al., 2008), muscle fatigue and muscle fiber composition changes that might explain the decreased exercise capacity in smokers suggests that acute smoking prevents oxygen absorption. Low oxygen absorption may occur if the blood flow is diminished or if the blood oxygen content is lower than normal.

Physical exercise induces lower vasodilation in smokers compared to non-smokers (Gaenzer et al., 2001), which can be due to an insufficient production of nitric oxide (Montes de Oca et al., 2008) and/or increased oxidative stress (Gaenzer et al., 2001; Tsuchiya et al., 2002).

Low oxygen absorption might occur when carbon monoxide (CO) competes with oxygen in binding to hemoglobin (Hb), resulting in hypoxemia.

Consequently, it seems unlikely that hypoxemia is the factor that differentiates smokers from non-smokers. The effect of CO through its action on Hb cannot be completely excluded in contrast to hypoxemia, the oxygen dissociation curve shifting to the left, inhibiting oxygen release from Hb.

CO inhalation acutely affects maximum oxygen consumption both in healthy subjects and in patients with COPD (Wüst & Degens, 2007). Corroborated by data from the study of Wüst et al. (2008), this suggests that smoking can have an acute and reversible effect on exercise capacity, caused by CO from the cigarette smoke. This leads to a lower but reversible exercise capacity in smokers, which suggests that smoking itself acutely affects exercise capacity. This mechanism can take place through a reduced and potentially reversible acute oxygen absorption in the contractile muscle and/or a reduction of Hb and myoglobin function because of CO.

CO and other substances from the cigarette smoke inhibit respiratory chain enzymes (such as cytochrome oxidase). Given that smoking has an acute negative effect on exercise capacity, smoking cessation would lead to its recovery whether the subjects have or not clinical symptoms of chronic diseases, such as COPD or heart failure.

Reduced aerobic capacity and smoking were independently associated with a higher probability of injury in men and women during a standardized physical training program (Knapik et al., 2001).

In conclusion, skeletal muscles in smokers are much more fatigable than in non-smokers of the same age who perform the same physical activity, while the contractile properties of skeletal muscles are not different. The reduction of resistance to fatigue is similar in men and women and is not related to the history of smoking.

Anaerobic capacity

Anaerobic exercise capacity is the capacity of the body to perform maximum efforts using the energy produced by anaerobic pathways, with lactate accumulation.

Anaerobic means in the absence of oxygen. Anaerobic power is the development of maximum power during effort, measured as work (force in kg x distance in m) expressed per time unit (min). The anaerobic threshold is the intensity of the work load at which anaerobic metabolism is accelerated (Afzal et al., 1998).

The study carried out by Chia-Lun & Chang (2013) included 12 female smokers and 21 female non-smokers

who performed intermittent sprint tests (IST) and 20 m running tests. Intermittent sprints consisted of 6 sprints x 10 seconds with 60 seconds of active recovery between each sprint.

The mean power of smokers decreased during sprints 4-6 (in smokers: 95% confidence interval = 6.2-7.2 joules/kg, in non-smokers: 6.8-7.6 joules/kg; $P < 0.05$), and the fatigue index increased (smokers: 35.8% ± 2.3%, non-smokers: 24.5% ± 1.76%; $p < 0.05$) during the sprint. Maximal oxygen consumption in non-smokers was significantly higher compared to non-smokers ($P < 0.05$).

Smoking does not affect maximum power during sprint. The mean power of non-smokers during sprints 4-6 was significantly higher compared to smokers. Smoking does not affect sprint performance in initial stages, but reduces muscle strength in subsequent stages. The study performed by Morse et al. (2008) found that the fatigue index of smokers increased by 17%, which shows that smokers were less able to cope with fatigue compared to non-smokers. The increase of the fatigue index during IST indicates that smokers do not have the capacity to resist fatigue in the later phases of the test, which are part of the anaerobic exercise.

Another cause of a high fatigue index among smokers can be the limitation of cardiorespiratory function, because aerobic metabolism plays a critical role in providing energy during intermittent sprint. Because smoking reduces exercise capacity, the body can compensate by limiting the amount of energy provided by the aerobic system for exercise.

The results of lactate concentration in the blood before and after IST suggest that smoking does not influence the use of energy from the matter. High-intensity intermittent sprint requires an effective ATP system and glycolytic metabolism.

In the study of Chia-Lun & Chang (2013), a variation in blood lactate concentrations depending on the type of exercise performed was found.

Smoking mainly causes a decrease of sports performance in the case of endurance sports. Starting from the premise that nicotine induces the release of catecholamines, aggravates sleep disorders and difficulties falling asleep, having a negative action on overall performance capacity, resistance to infectious bronchial and catarrhal diseases is crucial for smoking athletes, because an optimal health is a basic requirement for sports training or the improvement of performance. It can be said that smoking limits health and performance capacity in various ways and is a behavioral error of athletes, in contradiction to their sports performance ambitions. A study carried out in USA on adolescent smokers evidenced that if these performed physical exercise 3 times/week or more and ate a vegetable diet once a week or more or dairy products daily, differences were significant compared to non-smokers. Smoking is usually associated with a reduction of physical exercise and healthy nutrition (Wilson et al., 2005).

Effects of physical exercise on smokers

Smoking cessation is frequently associated with insomnia, irritability, a reduction of the concentration capacity, depression, and an excessive wish to smoke.

Regular physical exercise seems to improve the intensity and frequency of the wish to smoke associated with smoking cessation (Vaughan et al., 2012).

The systematic review performed by Ussher et al. (2012), comprising 15 studies, seven of which including at least 25 persons in each group, with different durations and intensities of the smoking cessation and physical exercise program, studied the influence of physical exercise on smoking cessation. Three studies demonstrated significantly higher abstinence rates in the group performing physical exercise compared to the control group at the end of treatment. One of these studies found a significant benefit for the physically active group compared to the control group during the course of 3 months, and a limited benefit over a 12-month period. One study reported a significantly higher abstinence rate in the physically active group compared to the control group at 3 months, but not at 12 months. The other studies indicated no notable effect on smoking cessation in the physically active group. Only one study provided data indicating that physical exercise contributed to smoking cessation over the 12-month period monitored. The other studies were conducted over a too short time period or physical exercise was not sufficiently intense to attain the required exercise level.

The preventive role of physical exercise on smoking

In addition to the beneficial effect of physical exercise regarding smoking cessation, regular physical activity contributes to the reduction of the negative effects of smoking.

The study carried out by Menegali et al. (2009) on animals exposed to cigarette smoke analyzed the therapeutic effects of physical exercise on oxidative stress markers. The results showed that animals exposed to cigarette smoke had an extensive destruction of alveolar septa and a significant increase in macrophage and neutrophil counts, as well as in the amount of collagen. The results also showed a decrease in the volume density of elastic fibers and an increase in the volume of air spaces. However, physical exercise partially improves these markers. In addition, physical exercise reduces oxidant production and increases the activity of the enzymatic antioxidant defense system, but not of lipids and oxidized proteins induced by cigarette smoke. These results suggest that physical exercise partially improves the histological aspect and the parameters of oxidative stress in the lungs of animals chronically exposed to cigarette smoke.

Another study performed by Toledo et al. (2012) demonstrates that moderate-intensity aerobic training reduces the development of pulmonary embolism induced by exposure to cigarette smoke.

Prospective data from a study conducted in a large group of women indicate that both walking and vigorous physical exercise are associated with a considerable decrease of the risk of coronary events. A strong correlation between walking or vigorous physical exercise and the incidence of coronary disease was evidenced. Women who walked at a normal pace for at least 3 hours a week or those who had vigorous physical exercises for 1.5 hours a week had a 30-40% lower risk. These findings support the practice of moderate exercise, which can be achieved by the majority of the population. Although vigorous exercise should not be discouraged by those who choose it, the results of the study indicate the fact that huge public health benefits might be achieved through the performance of regular

moderate-intensity physical exercise by persons who are currently sedentary (Manson et al., 1999).

Conclusions

1. Smoking is a risk factor for respiratory and cardiovascular diseases, and has a negative effect on the quality of life.

1. The aerobic and anaerobic exercise capacity of smokers is reduced compared to that of non-smokers.

2. Smoking cessation results in restoration of exercise capacity.

3. Regular physical exercise is associated with smoking cessation and has a therapeutic role in pulmonary and cardiovascular diseases.

Conflicts of interest

Nothing to declare.

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