What is Tobacco Smoke? Sociocultural Dimensions of the Association with Cardiovascular Risk

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Abstract: The definition of smoking as the inhalation of the smoke of burned tobacco that may occur occasionally or habitually as a consequence of a physical addiction to some chemicals, primarily nicotine, cannot be fully accepted today since several clinical, biological, metabolic, epidemiologic, statistic and socio-economic factors which play a basic role in determining individual damage due to smoking are missing in this assessment.

The analysis of findings shows undoubtedly that several constituents of cigarette smoking play a strong role in the development and progression of cardiovascular damage, primarily atherosclerotic lesions.

Nicotine and its metabolites, carbon monoxide and thiocyanate seem to be the most specific markers of damage that, in the time, become irreversible.

Cigarette smoking is addictive because of nicotine and nicotine withdrawal causes many side effects of quitting smoking as well as nicotine itself usually increases cardiovascular risk.

Therefore, what is smoking? Smoking must be defined as a chemical toxicosis which is able to cause detrimental effects either of acute or chronic type on different structures of the body being some of these like cardiovascular system, respiratory system and epithelial glands target organs. Smoking also causes physical addiction, primarily due to nicotine, that adversely influences smoking cessation.

From these observations there is evidence that a large number of socio-economic and epidemiologic implications arise in smokers and that requires the necessity of specific structures which may help to face up the problem.

Keywords: Smoking, cardiovascular risk, socio-economic impact, heart damage.

INTRODUCTION

Commonly, smoking is defined as the inhalation of the smoke of burning tobacco of cigarettes, pipes or cigars. It may be an occasional habit or, more often, a smoking habit involving a physical addiction to tobacco products, primarily nicotine. Such behaviour is able to cause serious health consequences in the long run.

This definition is too generic and also far to clarify the negative impact of tobacco to individual health since there are a lot of harmful factors that must be emphasized when cigarette smoking is analysed. Clinical, biological, metabolic, epidemiologic, statistic and socio-economic factors [1-22] interact to lead to only one result which is an irreversible damage of individuals’ health primarily for some organs like lungs, cardiovascular system and epithelial structures which may be identified as a target of smoking toxics. Passive smoking also plays a strong role to cause health damage.

Large population findings have identified that there is a strong association between tobacco smoke and different diseases, particularly cardiovascular diseases, and the rate of their appearance increases statistically in smokers even in absence of other risk factors so that smoking may be considered as an independent risk factor for cardiovascular system. However, human risks due to cigarette smoke vary widely depending on factors related to smoke characteristics and factors due to health status.

Adverse effects of tobacco smoke are mediated through the action of many chemical compounds that are usually concentrated and condensed into tobacco mixtures [23]. Over 4,000 chemicals have been identified in tobacco smoke mixture, and a large majority of these have carcinogenic and adverse cardiovascular and respiratory effects in humans and animals. Moreover, smoking habit seems to be the gateway to illicit drug abuse [24]. People who start smoking as particularly children are almost four times more likely to be regular users of an illicit drug and three times more likely to use cocaine.

Worldwide [25-27], more than 3 Million people currently die each year from smoking, half of them before the age of 70 and more than one third of current smokers meet partial or permanent disability. Therefore, there is an enormous human and social cost to pay to smoking. Yet, smoking habit seems to be scarcely influenced by anti-smoking campaigns since there is evidence that 1 billion of smokers in the world exist and an increased/decreased/ and again increased rate characterises cigarette consumption [27].

Many smokers begin to smoke during adolescence or youth and the early stages of smoking provide a pleasant sensation associated with a positive reinforcement which is responsible of a progressive increase in the number of smoked cigarette. The pleasant sensation caused by tobacco smoke is primarily due to structural stoechiometry of nicotine [28,29]. There are, spatially, two types of isomeric aggregation of those elements that take part in nicotine composition. Evidence indicates that nicotineS(-) isomer is the main nicotine isomer responsible of the pleasant taste of cigarettes for smokers but not never smokers. Generally, the other nicotine isomer, chemically named nicotineS(+), provides unpleasant taste. Therefore, quitting smoke should be ordered as early as an individual begins to smoke.

This review will describe the main characteristics of cardiovascular damage from smoking, major chemicals responsible, social and economic impact on individuals and, finally, how one can reduce the potential related-risk.

CARDIOVASCULAR DAMAGE FROM SMOKING

Different types of cardiovascular damage from smoking have been described [27]. They are due primarily to two pathogenic mechanisms strongly related among them: a chemical injury of
tobacco components and a hypoxic injury which is associated with the reduction in oxyhaemoglobin concentration with a markedly strong increase of carboxyhaemoglobin into the erythrocytes of blood.

A different susceptibility of myocardial and blood vessel cells characterises the final result of smoking action. Moreover, cardiovascular pathology caused by smoking exposure is almost similar qualitatively for that concerning both active and passive smoking, although some difference due to type of lesions or incidence may be seen. Active smoking seems to be associated particularly with clinical and anatomical alterations related to ischaemic pathology, whereas passive smoking triggers particularly functional disorders acutely and chronically it is associated with late atherosclerotic changes [5, 30-33].

Experimental findings have identified ultra-structurally and microscopically typical myocardial cell alterations due to a direct action of smoking compounds, primarily carbon monoxide [34-37] (Table 1). These findings recognize, generically, smoking as factor responsible of damage without any detail about the type of tobacco smoke (active or passive smoking). Strictly speaking, the changes observed should be attributed to passive smoking since exposed animal inhales smoking involuntary. At first sight, clarifying this concept could seem a trite sentence but, on the contrary, it has a basic value. Indeed, there is evidence that it permits to define in detail those alterations met in passive smokers and differ them from those due to active smoke.

### Table 1. Experimental Myocardial Change Due to Chronic Smoking Exposure

<table>
<thead>
<tr>
<th>Myocardial focal necrosis</th>
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<tr>
<td>Perivascular infiltrates</td>
</tr>
<tr>
<td>Punctate haemorrhage</td>
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<tr>
<td>Mitochondrial alterations</td>
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</tbody>
</table>

#### Active Smoking

Usually, active smoking damages cardiovascular system chronically. It causes clinical, anatomical, and functional alterations which can be associated and lead to irreversible damage. Therefore, three types of cardiovascular damage may be seen as a consequence of an acute or chronic action of active smoking: clinical damage, anatomical damage and functional damage.

Clinical damage causes primarily ischaemic heart disease. Acute myocardial infarction, recurrent infarction, and chronic angina, which are characterised by a progression of coronary atherosclerosis, are closely related to active smoking that is found with statistically significant rate [5-6, 38]. Also coronary vasospasm and sudden death [5, 39] affect active smokers with a lower incidence, although, sometimes, with more evident catastrophic results.

Clinically, the gravity of the damage seems to be correlated statistically with smoking trend. If the trend in smoking increases, clinically alteration frequency increases; conversely, if the trend decreases, the number of affected people decreases. Also the relationship between active smoking and functional damage is worthy to be discussed.

Functional alterations which are before transient may trigger a mechanism that leads, as time goes, to irreversible changes. Usually they are a close consequence particularly of the action of nicotine and carbon monoxide on sympathetic system and catecholamine release [40-43].

Harm from active smoking depends strongly on the biochemical marker concentrations in the blood and cardiac tissue and, therefore, on the number of smoked cigarettes that influence actively their level.

#### PASSIVE SMOKING

Cardiovascular alterations from involuntary smoking exposure differ widely in relation to different factors which are listed in Table 2.

### Table 2. Factors Influencing Cardiovascular Damage from Passive Smoking

<table>
<thead>
<tr>
<th>Environment pollution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Environment volume</td>
</tr>
<tr>
<td>Ventilation rate</td>
</tr>
<tr>
<td>Duration of exposure</td>
</tr>
<tr>
<td>Number of smoked cigarettes</td>
</tr>
<tr>
<td>Number of living people</td>
</tr>
</tbody>
</table>

All these variables may be combined differently and the degree of cardiovascular damage usually depends on the type of this combination as well as acute or chronic exposure.

Functional damage usually following an acute exposure in the first phases is transient and recognises autonomic system involvement as a main pathogenic factor [44-46].

Primarily, some cardiovascular parameters are adversely involved in functional damage. Heart rate and systolic blood pressure usually increase, exercise tolerance is impaired and rhythm disorders may appear particularly during exercise stress testing for those people with a pre-existing myocardial infarction [47].

Clinical damage affects primarily those individuals who are customary exposed to passive smoke everywhere although home and workplaces play a basic role. Symptoms of angina pectoris, particularly effort angina, myocardial infarction, arrhythmias and heart failure may exist or may be worsened when this type of damage pre-exists [48,49].

Anatomical damage is the final step of chronically prolonged exposure to passive smoking. It develops gradually but progressively and reaches a character of irreversibility.

Fixed alterations of the coronary and systemic arteries cause myocardial alterations, hypertension, cerebrovascular and peripheral artery disease [1, 6, 16].

The main factors that support anatomical damage are the pro-atherosclerotic effects of nicotine and carbon monoxide which are able to trigger those functional, biochemical, humoral and thrombogenic mechanisms which are the door of atherosclerotic disease.

#### CHEMICAL COMPOUNDS OF TOBACCO SMOKE

Tobacco plant and its leaves produce those chemical substances which cause both increase in cardiovascular risk and cardiovascular damage.

Both active and passive smoke has similar chemical composition although some differences may be observed.
Adverse effects on heart and vessels are mediated through the action of many chemical compounds that are concentrated and condensed into tobacco mixture, although three main chemicals (Table 3) are usually identified as an indicator of both active and passive smoke exposure in epidemiological surveys [50,51].

Table 3. Chemical Compounds Primarily Involved in Cardiovascular Damage

<table>
<thead>
<tr>
<th>Chemicals</th>
<th>Production</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Nicotine and its metabolites (particularly Cotinine)</td>
<td>Fresh tobacco leaf</td>
</tr>
<tr>
<td></td>
<td>Lighted cigarette</td>
</tr>
<tr>
<td>2. Carbon monoxide</td>
<td>Lighted cigarette</td>
</tr>
<tr>
<td></td>
<td>Environment</td>
</tr>
<tr>
<td>3. Thiocyanate</td>
<td>Fresh tobacco leaf</td>
</tr>
<tr>
<td></td>
<td>Lighted cigarette</td>
</tr>
</tbody>
</table>

These components, when are absorbed into the blood, act like free or bound plasma constituents and have a different half-life that is about 5 hours for carbon monoxide when it is bound to haemoglobin, 16 hours for cotinine, and 6.5 days for thiocyanate.

Although inhaled after tobacco manufacturing and/or burning but not in fresh leaves, carbon monoxide plays a strong role in long-term damage from both active smoking and passive smoking. Nicotine intoxication may occur also acute and depends on both fresh tobacco exposure and exposure to any type of smoking. Thiocyanate toxicity is too hard to establish since different foods, which are rich in some vegetables, may adulterate and confuse obtained responses.

Many other chemicals, as already said, may influence adversely heart and blood vessels, but their action is limited when they are compared to that due to carbon monoxide and nicotine. On the contrary, other chemicals, such as ammonia, benzene, 2-naphthylamine, nitrosamines, benzoanthracene and benzopyrene [14] have primarily heavy carcinogetic effects.

DIFFERENCE IN CHEMICAL COMPOUNDS FOR ACTIVE AND PASSIVE SMOKE

As previously mentioned, essentially a similar composition characterizes active and passive smoke. However, some differences exist between the two types either for that concerning chemical phase or their concentration and mechanism of action.

Changes in concentration of tobacco chemicals, which depend on the number of cigarettes smoked, amount of passive smoke residue, type and structure of the environments, individual’s smoking pattern are the most common difference between active and passive smoking.

Another factor to be considered is the duration of exposure to smoking that is short in active smokers (about 2 minutes for each smoked cigarette) but longer in those individuals who are exposed to passive smoke since cigarette end continues to exhale toxic substances into the environment for a long time. Such a factor involves primarily the inhalation of carbon monoxide which is a compound of burned tobacco but not fresh tobacco. Therefore the higher chemical concentration absorbed by active smoking is partially balanced from the longer duration of exposure to passive smoking [52]. This longer exposure to smoke residue may be responsible of an effective major mixture and chemical combination among the different toxic products with changes which determine difference, in the long time, in the type of constituents of active and passive smoke. Mixing of environmental smoke is a consequence of gaseous and steam phase of tobacco chemicals which have moving particles. They may aggregate or break away and that depends strongly on their concentration and temperature into passive smoke residue. Therefore, environmental tobacco smoke consists of a dynamic mixture in ambient air, the concentration and composition of which vary widely because of their physical phase. This dynamic process will determine continuously difference in chemical compounds and their concentration in active and passive smoke.

Three main phases characterize passive smoking (Table 4): sidestream smoke which is defined as the smoke that drifts from the end of the lit cigarette, mainstream smoke, which is the smoke inhaled directly through the burning cigarette by the smokers, and the exhaled mainstream smoke which is the smoke breathed out by the smoker from their lungs. Also the composition of mainstream smoke and exhaled mainstream smoke can likely differ since some of the compounds in smoke are retained by the smokers or otherwise altered by the process of combustion and, then, exhaled smoking.

Table 4. Main Phases Which Characterize Passive Smoking

| 1. Sidestream smoke: | the smoke that drifts from the end of the lit cigarette |
| 2. Mainstream smoke: | the smoke inhaled by smoked cigarette |
| 3. Exhaled mainstream smoke: | the smoke breathed out by a smoker |

The particles of sidestream smoke are smaller than those of mainstream smoke, meaning that they can be inhaled more deeply into the lungs [32].

Then, once the smoke has left the cigarette, its physical nature and relative chemical composition will change dramatically since its particles evaporate rapidly or condense according to some characteristics of the environment, particularly its level of pollution due to other chemical pollutants which strengthen the power of smoking ambient pollutants.

Studies conducted in human non-smokers exposed to passive smoke dose measurable levels of nicotine or cotinine and carbon monoxide in the blood [27, 53-55] with an incidence up to 90 per cent of exposed subjects.

SOCIAL AND ECONOMIC IMPACT

The absolute method to avoid or strongly reduce the damage caused by tobacco smoke for exposed individuals is, of course, to ban smoking anywhere but there is evidence that such an obvious assessment is hard and far to be realized.

A large series of social and economic aspects interfere among themselves and they tend to impede positive results of antismoking campaigns.

Despite the tremendous human and financial impact of tobacco-related diseases, it is unfortunately still true that tobacco control activities require, at present, as similar economic costs as those related to hospitalization and treatment of people affected by the harmful effects of smoking.

Findings consistently show that concentrations of toxins exhaled by a lighted cigarette pollute individuals and environmental air heavily, whereas clean air may be dosed when tobacco smoke does not play its action [56-58]. Consequently, preventive antsmoking measures currently need to help active smokers to stop smoking and non-smokers exposed passively to tobacco chemicals to breathe clean air. It has been well documented [59] that people living in both outdoor and indoor environment usually breathe from
3 to 9 litre air per minute and environment is stably polluted also by industrial toxics that may potentiate tobacco smoke effects.

The surveillance of tobacco consumption and the estimation of its consequences on the health of population is, therefore, one of the major public trends together with social and psychological support to individuals who are resolute to stop smoking.

The first step to put into practice is translating the desire of individuals to stop smoking into a stable decision to obtain that by an approach that involves several experts in different fields related to smoking habit of smokers. Therefore, educational programmes conducted by physicians, psychologists, politicians, epidemiologists and others must address their efforts to persuade people not to smoke and make a healthy choice [60,61] by helping and influencing smokers in the course of their daily lives, in the places when they learn, work or recreate.

To maximise the number of people who quit smoking and, consequently, reduce the rate in morbidity and mortality from smoking it is necessary to follow closely heavy smokers as well as those people who live with groups of smokers.

Today, antismoking structures directed by different experts play their support to smokers who decide quitting smoking. A great number of these structures are specialised also in treating drug abuse since also this disease causes addiction as an underlying substrate similarly to what occurs for smokers.

Cometa Consortium in our county began in the seventy years engaging its work in the social field. It assists those people who are affected particularly by drug and alcohol abuse and are also, in a large majority of cases, heavy smokers. However, every type of addiction or discomfort is treated by a medical, psychological and socio-economic staff with the cooperation of volunteers who have the precise goal to re-integrate assisted people to an active job. The number of assisted individuals varies widely since all those people who turn to the Centre and are motivated to quit abuse are customary accepted.

The main goal of antismoking campaigns addressed to the control of individual health is to warrant clean air.

To have absolutely clean air environmental and policy interventions would be the most cost-effective approaches to reach positive results.

There is evidence that several legal acts against smoking habit are continuously promulgated in different countries. The United States are quickly progressing to this way [62-64]. Some local health departments have been authorised by legal policy to bring about reforms of clean air defence forbidding smoking in public as well as private buildings where people may go. Also tobacco advertising has been banned in several countries, although it financed a lot of cultural and sport events which had no financial support. All these measures are addressed to control social and economic habit of living people but, in our opinion, they will continue to be ineffective whether international guidelines on the prevention of smoking damage involving also specialised structures are lacking. Also schools and fitness places would be involved.

There is a strong association between cigarette smoking and socio-economic position [65,66]. Cigarette smoking is more prevalent among manual social groups than among non-manual groups, and is lowest among higher managerial and professional classes. In 2006, 29% of men and 27% of women in manual households smoked compared to 18% of men and 16% of women in non-manual households. This class difference has persisted since the 1990s, and recent data suggest no narrowing of the gap.

Also changes in cigarette prices seem to have had a strong impact on smoking; the greatest impact is on groups that are least responsive to health publicity measures but have the highest prevalence of smoking. This suggests that real increases in the price of cigarettes could reduce smoking habit and as well as the differences in the prevalence of smoking and smoking related diseases among socioeconomic groups. Special measures are necessary to ameliorate any effects on the cost of living of the most deprived families.

Since there is evidence of health risks derived from smoking other preventive measures have been carried out like banning television advertisement for cigarettes, smoking from international and domestic flights and insert information of the harm from smoking on the parcel labels of cigarettes.

Despite all these measures, smoking problem is far to be overcome. Therefore, socioeconomic aspects related to tobacco consumption should be more studied, specialised centres to assist smokers with or without drug abuse potentiated in an attempt to reduce, at a maximum, the harmful effects of smoking on people health.

Such behaviour needs since many smokers begin during adolescence or early adulthood and, during the early stages, smoking is pleasant and, socially, serves as a source of positive reinforcement. Moreover, after many years of smoking habit withdrawal symptoms occur making quitting smoking spontaneously very hard to be achieved.

**CARDIOVASCULAR-RELATED RISK**

Three main aspects must be considered to approach correctly the role of smoking as a cardiovascular-related risk factor (Table 5): 1 Action of smoking for those healthy individuals who have begun smoking recently; 2 action of smoking for those individuals where smoking caused or contributed to cause cardiac alterations; 3 reduced harm from smoking following smoking cessation.

Table 5. Main Approaches to Assess Cardiovascular –Related Risk of Smoking

<table>
<thead>
<tr>
<th>Smoking</th>
<th>Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Action on healthy individual</td>
<td>No cardiovascular risk factor</td>
</tr>
<tr>
<td></td>
<td>-Transient sympathetic effects</td>
</tr>
<tr>
<td></td>
<td>-Transient endothelial dysfunction</td>
</tr>
<tr>
<td></td>
<td>-Atherosclerosis door</td>
</tr>
<tr>
<td></td>
<td>Major cardiovascular risk factors</td>
</tr>
<tr>
<td></td>
<td>-Hypertension</td>
</tr>
<tr>
<td></td>
<td>-LDL Cholesterol concentrations</td>
</tr>
<tr>
<td></td>
<td>-Diabetes mellitus</td>
</tr>
<tr>
<td></td>
<td>-Metabolic syndrome</td>
</tr>
<tr>
<td>2. Action on individuals with cardiac disease</td>
<td>Active smokers</td>
</tr>
<tr>
<td></td>
<td>Passive smokers</td>
</tr>
<tr>
<td>3. Smoking cessation</td>
<td>Time of risk reduction or disappearance</td>
</tr>
</tbody>
</table>

Tobacco products in recent smokers with no disease usually cause two main types of response which is able to trigger that mechanism which leads, in the long run, to the development of an atherosclerotic lesion. It is a close consequence of a transient but frequent sympathetic activation as well as endothelial dysfunction which interact together.

Initially, these responses are functional disorders which begin as early as initial tobacco exposure.
Disorders from smoking acting as a risk factor in a healthy individual are usually the result of catecholamine and endothelium stimulation by the main compounds of environmental tobacco. Some of these, particularly nicotine, interfere actively with catecholamine stimulation [40, 67,68], some cause changes in cardiovascular parameters particularly due to sympathetic or parasympathetic stimulation which causes variability in heart rate and systolic blood pressure [27] often associated with haemodynamic changes, others determine endothelial cell activation by triggering an atherosclerotic mechanism [69].

Four main sources (Table 6) release catecholamine particularly after nicotine stimulation: sympathetic nerve endings, neurotransmitters within the myocardium, adrenal medulla and chromaffin tissue. The first two structures release primarily norepinephrine, while the latter two mainly epinephrine, but also norepinephrine. Norepinephrine release acts as one of the main factors to triggering atherosclerotic mechanism since it prolongs in time its effect. To be in the right, norepinephrine release acutely due to smoking products may be identified as a basic mechanism which opens the “atherosclerotic door”.

Table 6. Structures Involved in Catecholamine Release

<table>
<thead>
<tr>
<th>Structure</th>
<th>Main Metabolite</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sympathetic nervous endings</td>
<td>norepinephrine</td>
</tr>
<tr>
<td>Myocardial neurotransmitters</td>
<td>norepinephrine</td>
</tr>
<tr>
<td>Adrenal medulla epinephrine,</td>
<td>norepinephrine</td>
</tr>
<tr>
<td>Chromaffin tissue epinephrine,</td>
<td>norepinephrine</td>
</tr>
</tbody>
</table>

The effects of catecholamine have been well documented by a study of Benowitz et al. [70]. Authors found that nicotine infusion achieved plasma concentrations of catecholamine and caused increase in heart rate and haemodynamic response similar to those of cigarette smoking up to a limit, beyond which there was no further increase in stimulated parameters. Increased catecholamine concentration due to nicotine stimulation was shown almost similarly by Cryer et al. and Hill and Wynder [42, 43].

The final result of nicotine effect is a transiently increased demand in oxygen in both healthy and diseased individuals. Reduced oxygen availability from smoking has adverse effects on heart and muscle metabolism contributing to the occurrence of those alterations that already defined the “door” to atherosclerosis.

Carbon monoxide follows usually two ways to act as risk factor: it may activate catecholamine release and, then, induce functional disorders of cardiovascular system as well as can exert a directly toxic influence. Usually, however, the latter influence causes more often anatomical changes than functional disorders.

Since there is a close relationship between catecholamine release and sympathetic stimulation consequently acting of these two factors potentiates the adverse effects on cardiovascular system.

Endothelial cell activation is a very important factor of functional disorders that begin acutely but act chronically.

As it is known, physiologically endothelial function [71] is the result of a balanced response between vasodilator chemicals as nitric oxide and vasoconstrictors as endothelin. Decreased release of nitric oxide following different types of stimuli and consequently prevailing of vasoconstrictor response may cause endothelial dysfunction, that is, as it an identified marker of early vascular damage that leads to atherosclerosis.

Findings of Celermajer et al. [72] compared endothelial function in the arteries of three groups of healthy individuals. The first group included active smokers, the second group lifelong nonsmokers who were exposed to environmental tobacco smoke, and the third group consisted of subjects exposed irregularly to passive smoke. Effective exposure to passive smoking was considered of at least one hour per day anywhere: home, work or both environments.

Endothelial function was assessed by vascular ultrasonography measuring vascular reactivity of brachial artery that has been established to be a correct marker of the endothelial function. Authors showed that passive smokers had significantly impaired arterial endothelial function with reduced nitric oxide release as an effect of environmental smoking products.

Impaired availability of nitric oxide, the endothelium-derived relaxing factor, can stimulate platelet aggregation [15, 73] that initiates the vascular damage leading to atherosclerosis.

There is evidence that healthy individuals with major cardiovascular risk factors increase their risk as a consequence either for effect of smoking alone or for that of smoking combined with them.

Interesting data derive from MRFIT cohort large study [74] that analyses the possible interaction of cardiovascular risk factors.

From the analysis of this study, it emerges that everyone of the major coronary risk factors, namely LDL-cholesterol level, hypertension, smoking and diabetes mellitus, exerts a strong effect in increasing the rate of appearance of coronary events either acting alone or together. In the last case, their effects increase exponentially and the increase may be also measured [75,76]. Usually, strong effects in increasing cardiovascular risk show the association between level of LDL-cholesterol, smoking and hypertension in young people, whereas diabetes mellitus, isolated systolic hypertension and both systolic and diastolic hypertension associated play a heavy effect in elderly subjects. However, the degree of risk is absolutely increased when risk factor association occurs.

Adverse effects of smoking on cardiovascular system are primarily potentiated by high LDL-Cholesterol concentrations since both these factors lead, on the long run, to formation of an atherosclerotic plaque.

A study conducted to establish the interaction among the major coronary risk factors [77] analyzed the association of cholesterol, triglycerides, blood pressure, obesity, and cigarette smoking. Two methods were used: standard correlation analysis, and percentile analysis method applying of which was limited to associations at higher levels of these risk factors. Percentile analysis requires that known values of a variable are divided into 100 sub-sets with equal frequencies. Mathematical result of a percentile is easy to be calculated being the ratio of the number of observations below the value to be analyzed divided for the total number of observations multiplied 100. The study population consisted of 4,839 men and women aged from 30 to 39 years, a number statistically significant for the purpose of the finding. In the percentile analysis, subjects with moderate or greater (equal or over 70th percentile) or high (equal or over 90th percentile) levels on one of the examined risk factors showed clustering of elevations in other risk factors in that expected ratios were generally greater than unity, again excepting smoking comparisons. However, from these data there is a clear incidence of the strongest power due to interactions of the major coronary risk factors.

Data provided by the report of Gotto [76] demonstrated that the mortality rate from coronary heart disease was from two to three times greater in those people who were heavy smokers, representing smoking an independent major risk factor for coronary heart disease. Moreover, the risk was even higher when smokers were also hypertensive and with elevated levels of cholesterol, a pathological status of a frequent observation in ischaemic patients.

Interaction between smoking and other major coronary risk factors has been also analyzed by other papers [78-81] which demonstrated the exponential increase of risk when they exerted combined effects.
When smoking acts as a cardiovascular-related risk factor for those individuals who have cardiovascular pathology induced by smoking or, generally, ischaemic heart pathology, two groups of individuals can be identified: active smokers and passive smokers. Both these groups of individuals suffer from harmful effects of smoking products. However, there are some differences [82-87] with regards to coronary, carotid and cerebrovascular arteries, where passive smokers meet usually the same effects although of lesser degree.

Active smokers usually show a rapid progression of atherosclerotic lesions particularly some alterations of a limited extension with a more graded development. Such a progression well helps to understand what occurs in those people who quit smoking.

Coronary artery alterations in smokers involve both epicardial vessels as well as small intramural coronary arteries causing different degree of luminal narrowing where there is a strong possibility of thrombi formation or atherosclerotic plaque erosion or rupture. Moreover, coronary artery lesions are strongly related to the number of smoked cigarettes [88]. On the contrary, only a few smokers with myocardial infarction display normal or nearly normal coronary arteries [89,90].

Smoking cessation is determined by the balance between two opposing forces: motivation to stop smoking and level of nicotine dependence [91]. Positive motivation towards the first force associated with a control of nicotine addiction may induce changes in smoking habit with, consequently, easier result in quitting smoke. However, there is evidence that guidelines to reach smoking cessation require often repeated and repeated interventions because of chronic dependence determined by tobacco use [92]. Since smoking trend is characterised by a variable course with increase, decrease and, often again increase in smoking habit without any evident reason, there is evidence that positive results obtained by smoking cessation may vary widely in time and also depend on smoking habit itself.

Yet today, large-scale studies concerning cardiovascular risk reduction associated with smoking cessation are very limited. However, observational findings would seem to demonstrate encouraging benefits and results time-related begin to appear.

A reduction of excess risk in cardiovascular events of about 50 percent has been seen in quitting smokers within the first two years of cessation [93,94]. This decline meets a lesser reduction in time-rate during the following years reaching a level of risk for former smokers as similar as that of never smokers between 5 and 15 years. However, smoking cessation is highly cost-effective in both primary and secondary prevention similarly to that has been seen for other drug abuse diseases and such a consideration supports the action of those centres, like Cometa Consortium or similar structural teams to change smoking habit and lifestyle of smoker people.

CONCLUSION

In conclusion, a question arises spontaneously: how can we define correctly what is smoking?

Smoking must be defined as a chemical toxicosis which is able to cause detrimental effects either of acute or chronic type on different structures of the body being some of these like cardiovascular system, respiratory system and epithelial glands target organs.

Smoking also causes physical addiction, primarily due to nicotine, that adversely influences smoking cessation. Therefore, cigarette smoking must be included among those diseases to be treated by different approaches involving medical, psychological and educational teams to change smoking habit and lifestyle of smoker people.

CONFLICT OF INTEREST

None.

REFERENCES


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