CHAPTER-2

REVIEW OF RELATED LITERATURE
In this chapter, it is proposed to discuss various forms of tobacco use including smoking of cigarettes and the resulting health hazards in general and with reference to athletes in particular.

Traditional forms of tobacco use in developing countries

In assessing the tobacco habits in developing countries, indigenous forms of smoking, as well as chewing, which are characteristic of certain regions, have to be taken into account even though cigarette smoking is already the predominant type of smoking in some countries and regions.

**Bidi:** A bidi consists of about 0.5 g of sun-dried and cured tobacco flakes hand-rolled in a rectangular piece of dried leaf of Temburni (Diospyros melanoxylon). This traditional form of smoking is practised in several countries (e.g., Bangladesh, India, Nepal) and a similar smoking material is used in other countries (e.g., Indonesia, Thailand).

**Chutta:** This is a coarsely prepared medium-sized cigar made from cured tobacco leaves rolled and wrapped in a dried tobacco or a jackfruit leaf, it is smoked with the burning end inside the mouth. Its use is common in parts of India and Latin America e.g., Peru.

**Chilum and sulpa:** Though they are not identical in form, both are clay pipes in which tobacco is smoked. Chilum smoking is practised in parts of India and sulpa smoking in parts of rural Nepal.

**Hookah, goza and hubble-bubble:** Basically in this form of smoking, tobacco mixed with molasses is burnt and the smoke is passed through water before inhalation. The hubble-bubble
apparatus is usually passed round from person to person in a form of group smoking. Hookah smoking is prevalent in Bangladesh, India, Nepal, and Pakistan and goza smoking in Egypt and the countries of South-West Asia.

**Tobacco chewing:** Tobacco chewing is practised in several countries. In some it is chewed along with pan (which consists of sliced betel nut, catechu quid, and various amounts of other spicy ingredients rolled in the piper betel vine leaf) or with betel leaf and slaked lime only. Naswar for chewing, as used in Afghanistan, contains only slaked lime and tobacco, the green leaves being dried in the shade. In India, on the other hand, chewing tobacco is sun-dried. Burnt tobacco (mishri) is used for cleaning teeth, particularly by women in India, and is often retained in the mouth. In parts of Africa, too, many women retain tobacco in the mouth; in Gabon, for instance, adult women in rural areas put dried tobacco mixed with wood ash (adzeghane) under the tongue.

**Snuff:** Besides being smoked and chewed, tobacco in powdered form can also be inhaled, by taking up a pinch through the nostrils. Another method is to place the snuff between the lower lip and gum.

** Constituents of the smoke **

Cigarette smoke is a complex mixture of 4,000 constituents which are distributed in particulate or gaseous phase. The particulate phase contains nicotine, aromatic hydrocarbons and

The gaseous phase has a number of irritants like hydrocyanic acid, acetaldehyde and others which damage the lining of airways to the lungs.

There are about 10,000 chemicals in cigarette smoke. The smoke contains various vaporised chemicals. These chemicals are drawn into nose, throat and lungs. In this inhaled smoke the nicotine content varies from 0.5% to 8% and on an average it contains 1.5% of nicotine, or an average yield of 6 to 8 mg. A 10 gm cigar contains 15 to 40 mg of nicotine. The nicotine is present in the salt of organic acid and the free base liberated during burning.

A cigarette releases from 1% to 2.5% of its volume in cigarette smoke. It is mentioned above that the cigarette smoke consists of 10,000 chemicals. It may be noted that there are only 200 to 300 different kinds of chemical compounds in tobacco leaves but during burning thousands of products are released. At least 16 different chemicals are found in tobacco which can initiate cancer in test animals. The best known is benzo pyrene.

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About 1% of total cigarette smoke condensate consists of Alcohols/Phenols. Each cigarette consists of aldehyde and also volatile acids like formic acid, acetic acid and Benzoic acid. This smoke contains arsenic too.

Physicochemical Composition of Tobacco Smoke

More than 4,000 known compounds are estimated to be present in tobacco smoke. These occur as volatile constituents forming a vapor phase, and in particulate form suspended in the vapor phase. The majority of carcinogens and mutagens are found in the particulate phase. Currently, 43 carcinogens have been identified in tobacco smoke, including polynuclear aromatic hydrocarbons (PAH), nitrosamines, heterocyclic hydrocarbons, benzene, and radioactive polonium-210. Tobacco-specific nitrosamines (TSNA) are the carcinogens present in the highest concentration (they occur in even higher concentrations in smokeless tobacco). For purpose of risk assessment, tobacco smoke is classified as mainstream smoke (MS) and sidestream smoke (SS). The former represents smoke which is directly inhaled through the butt end of the cigarette, while the latter is continuously emitted from the burning end into the environment. Although most of the components that have been identified in tobacco smoke occur in both MS and SS, the concentrations of many toxic and tumorigenic agents are higher in SS (e.g., nitrosamines).


As the main and most potent alkaloid of tobacco, Nicotiana tabacum, the major interest of nicotine is concerned more with its widespread use as a recreational and addictive drug than with its therapeutic use in medical practice.

**Chemistry**

**Nicotine**

\[
\text{C H N}\]

\[10\ 14\ 2\]

\[(S)-3-(1\text{-Methyl-2-pyrrolidinyl})\text{pyridine}\]

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**Molecular weight**

162.2

**pKa**\(-\text{N} = , \ -\text{N} <\)

3.2, 8.0

**Solubility**

- in alcohol > 1 in 30
- in water > 1 in 30

**Octanol/water partition coefficient**

-  

A volatile strongly alkaline liquid, it is colourless when pure but undergoes auto-oxidation and turns yellow to brown on exposure to air or light and gives off the pungent, characteristic smell of tobacco. It has an acid, peppery taste. It is highly soluble in water and forms water-soluble salts and double salts with many metals and acids. Under atmospheric pressure, it boils at 246 °C. It is volatilized in the cone of burning tobacco, 800-900 °C, and the free base is present in the minute (0.3-1.0 um) semi-liquid droplets of the so-called particulate phase of the smoke. Nicotine is a natural product in tobacco plants (Nicotiana tabacum and N.rustica), from which it is obtained.
Between 1954 and 1975, the average tar content of a cigarette in the U.S. declined by 50%, from 38 to 19 mg; by 1985 the average tar content was between 12.7 and 13 mg per cigarette. During the period from 1954 to 1985, the average nicotine content was reduced from 2.3 to 0.9 mg.

The progression from high tar, to unfiltered high tar, to filtered middle-tar, and to filtered low tar cigarettes has also been observed in western European countries, including Germany, Switzerland, the United Kingdom and France.

'Safe' and 'less hazardous' low tar and low Nicotine cigarettes, since their introduction to the U.S. cigarette market in the late 1960's and early 1970s, have had a rapid increase in market share, from 2% in 1971 and 17% in 1976 to more than 60% in 1981 but declined to 54% in 1985 and 1986, indicating a return to a Fuller-flavoured cigarette". In addition, the ultra-low tar brands, since their introduction in the late 1970s, have captured 22.3% of the low tar market. (low-tar cigarettes are defined as having <15 mg of tar per cigarette and ultra low tar brands as having upto 10 mg of tar per cigarette). Similar patterns of


consumption of low-tar cigarettes also occurred in Canada during this period. The significant growth of the low-tar-cigarette market in the last decade is attributable to increased public awareness that cigarette smoking, particularly exposure to tar and nicotine, is detrimental to health.

**Cigarettes:** Cigarettes marketed in China and India have been shown to have high tar (21-33 mg and 19-27 mg per cigarette, respectively) and high nicotine (0.7-1.2 mg and 1.0-1.4 mg, respectively) yields. In Egypt, too, similar tar and nicotine values were observed in 1981, but these are now lower as a result of a government action. A study in India has shown that there is no difference between the tar and nicotine delivery of filter and non-filter cigarettes marketed there, so that a filter gives no protection to Indian smokers; also "king-size" filter cigarettes yield one-and-a-half times more tar and nicotine than ordinary cigarettes.

**Bidi:** Bidi, in India, bidis have been shown to deliver high tar (more than 23 mg) and nicotine levels (1.7-3 mg).

**Hookah:** When 24g of hookah tobacco was smoked through 150 ml of water, the yield of total (dry) particulate matter was 9.1 mg and of nicotine 0.55 mg, for a total smoking time of 35 minutes.

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For other types of smoking no estimates for tar and nicotine delivery are available, and there are no data available on gaseous components of the smoke of cigarettes marketed in any of the developing countries.

The smoker may think he is getting more value for his money by taking a large number of puffs per minute, which is obviously more hazardous to his health. That was indeed the behaviour observed in a Bombay smoker who took 1.9 puffs per minute at a cigarette and 4.7 puffs per minute at a bidi.

The tobacco habit is widely prevalent in the developing countries, where the risk is even greater than in developed countries owing to the high yield of emission products of the smoking materials used there and to the prevalence of other hazardous smoking and chewing habits.

Health risks related to cigarette smoking in developing countries

Since the smoke was found to have a number of chemicals which get into the biological systems through the respiratory tract and lungs, it would be worthwhile to briefly review various health hazards of smoking before any mention is made on the effect of smoking on athletes and sportsmen. The following are the physiological and other hazards on various systems.

CIGARETTE SMOKING

The major health risk related to cigarette smoking in developing countries is lung cancer. The "kreyberg group" tumours are common among cigarette smokers in developing as well as in developed countries. The rate of incidence of lung cancer among the Natal Bantu (South Africa) are among the highest in the world: 72.2% of male cases are diagnosed between the ages of 40 and 64 years, compared with 38% in the United Kingdom. Lung cancer among men increased six fold and about five-fold among women. Smoking was found to be positively associated with oesophageal cancer in African males in Johannesburg.

Several studies in India, Philippines and China have shown that cigarette smoking may be regarded as a significant risk factor for the development of ischaemic heart disease. A recent case control study in India has shown that cigarette smokers are more than twice as likely to develop coronary heart disease and three times more likely to develop myocardial infarction than non-smokers.

In a comparison of patients with chronic bronchitis in Egypt with a control group, 89% of the chronic bronchitis patients

and 62% of the controls were smokers. Smokers with bronchitis smoked an average of 27.5 cigarettes daily compared with 11 cigarettes per day for those in the control group. The chronic bronchitic patients had also been smokers for a significantly longer average time (25.5 years compared with 15.3 years for the control group).

Cigarette smoking itself and especially in combination with some other disease factors (Schistosomiasis in Egypt, for example) considerably increase the relative risk of bladder cancer. Research suggests that there exists a relatively high risk of perinatal mortality among the infants of anaemic women, older women and women with high parity who smoked during pregnancy. These aggravating factors are common among women in developing countries, and so the impact of smoking on perinatal mortality can be expected to be greater in these countries than in the developed world. In a recent study in Dhaka, perinatal mortality rates in 1987 and 1988 were more than twice as high in woman who smoke than in non-smokers, most of such women smokers consumed, daily 1-10 hand-rolled cigarettes of home-grown tobacco. In another study of women attending a maternity hospital in India the 'still birth' rate for tobacco chewers was found to be 50 per 1000 birth as compared with 17.1 per 1000 for

54. WHO op cit
non-chewers. Even for healthy women, smoking during pregnancy may increase the risk of low birth-weight.

Bidi Smoking

Bidi smoking may be regarded as a major contributor to the development of cardiovascular disorders—especially ischaemic heart disease—the risk of bidi smokers developing coronary heart disease and myocardial infarction being three times and almost four times greater, respectively, than that of non-smokers. Young men in Sri Lanka who are heavy smokers of bidis are particularly prone to occlusive disease of the small arteries. Bidi smoking also results in adverse effects on the cardiovascular system in general, such as increase in pulse rate and in blood pressure, as well as in the plasma level of free-fatty acids and in platelet aggregation. The countries where bidis are smoked they are among the main smoking-related causes of lung cancer, oropharyngeal cancer and cancer of the larynx.

A correlation has also been found, both for men and for women, between cancer of the oesophagus and bidi smoking. Bidi smoking carries a higher lung cancer risk than cigarette smoking, owing to the higher concentration of carcinogenic hydrocarbons in the smoke.

Bidi smoking (as well as cigarette smoking) has been found to hasten the decline of pulmonary function, especially if combined with occupational hazards. Furthermore, like cigarette smokers, bidi smokers also have a higher total mortality rate than non-smokers.

**Chutta, sulpa, hookah and other forms of smoking**

Of the main tobacco-related diseases, the highest risk to chutta smokers is of developing oral cancer. Reverse smoking of chutta is also significantly associated with greater risk of epidermoid carcinoma of the hard palate. Clay-pipe smoking has also been identified as a possible cause of precancerous changes in the mouth. Furthermore, between 70% and 80% of those who

indulged in this habit were found to be suffering from stomatitis.

Hookah smoking seems to be associated with a prevalence of chronic bronchitis that is higher than among the non-smoking population and may also be a cause of leukoplakia.

Goza smoking, being a shared type of smoking (several smokers use the same smoking apparatus at the same time) is regarded as playing an important role in the transmission of respiratory and other communicable diseases in Egypt.

Pipe and cigar smoking (Western style)

This type of smoking is also prevalent in some developing countries. In spite of the fact that most smokers apparently do not inhale the smoke, there is well established evidence that cancer mortality rates for cigar and for pipe smokers are higher than for non-smokers (although lower than for cigarette smokers) and demonstrate a dose-response relationship. A study in African patients with oesophageal cancer in Johannesburg showed a close association between pipe smoking and this type of cancer. In Sudan, of 135 patients with oesophageal cancer nearly half were women.


Tobacco chewing

Certain forms of tobacco chewing are clearly associated with cancers of the upper alimentary tract, particularly oral cancer, cancer of oropharynx, hypopharynx, larynx and oesophagus. Increased risk was connected with life-time chewing and the predominant histological type of the oral cancer was epidermoid carcinoma.

Oral and pharyngeal cancer is prevalent in central and South-East Asia. Colombo and Bombay have the highest mortality rates from pharyngeal cancer. Oral cancer is a major health problem in India and accounts for 50-70% of all cancers diagnosed. Chewing of tobacco, especially in the form of pan, has been found to play an important role in the etiology of oral cancer in most parts of central and South-East Asia. In a large epidemiological study in India, the proportion of tobacco-chewers was significantly higher among the oral cancer group than among the controls, regardless of sex or race. The same study also showed that the risk of developing oral cancer increases with the frequency of chewing, and is higher in prolonged chewers and chewers who started the habit at an early age. In most cases the side of the mouth affected is the side in which the quid of tobacco is kept, and the risk is 36 times higher than for non-chewers if the quid is kept during sleep. A large study in

Pakistan showed that tobacco smoking and/or chewing greatly increased the risk of oral cancer.

There are also data in support of the view that tobacco chewing in coronary heart disease patients may significantly decrease the blood coagulation time, the prothrombin time, and fibrinolytic activity, while increasing the platelet count and the platelet adhesive index, thus worsening the prognosis. Different forms of tobacco use i.e. chewing and smoking, are frequently combined and have a synergistic effect on disease development.

CARDIOVASCULAR DISEASE: (CHD)

Coronary Heart Disease

CHD is related to several risk factors, one of which is tobacco use. Of the various disease manifestations associated with tobacco use, CHD is the leading cause of excess death and disability in the United States. In 1987 more than 45% of the 2,123,323 deaths in the United States were due to disease of the circulatory system. Of Cardiovascular deaths, 53% or 412,138, were due to CHD, and 23% of the total CHD deaths were attributable to cigarette smoking.

These studies, accounting for more than 20 million persons over years of observation, revealed a higher incidence of myocardial infarction (MI) and death from CHD in cigarette smokers than in nonsmokers. This set of studies also demonstrated that

whether in the United States, Canada, the United Kingdom, Scandinavia, or Japan, smokers as a group have excess CHD mortality that is approximately 70% greater than that of nonsmokers (Table D).

Male and Female smokers have an increased relative risk of sudden cardiac death that is two to four times greater than that of non smokers. This risk is greater among young men and appears to be related to the number of cigarettes smoked per day.

Smoking also appears to increase the risk of angina pectoris. After 24 years of follow-up, smokers in the Framingham Study had a slightly higher incidence of angina in all age groups compared with nonsmokers. Among smokers in the youngest age group (30 to 40 years of age), however, the incidence of an angina pectoris was more than double than that among nonsmokers.

A study using all female subjects has reported that women smokers less than age 50 years who smoke a pack of cigarettes or more a day have twice the risk of having a nonfatal MI, compared with nonsmokers of the same age.


<table>
<thead>
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<th>Sex</th>
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<th>No. of cigarettes per day</th>
<th>Ratio</th>
</tr>
</thead>
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<tr>
<td></td>
<td></td>
<td>1-9</td>
<td>1.24</td>
</tr>
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<td></td>
<td>21-39</td>
<td>1.76</td>
</tr>
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<td>40 or more</td>
<td>1.94</td>
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<td>Non-smoker</td>
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<td>1.29</td>
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<td>21-40</td>
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<td>41 or more</td>
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<td></td>
<td>1-9</td>
<td>1.90</td>
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<td></td>
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<td>10-20</td>
<td>1.58</td>
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<td>physicians</td>
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<td>1-14</td>
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<td></td>
<td>1-7</td>
<td>1.60</td>
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<td>8-15</td>
<td>1.70</td>
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<tr>
<td></td>
<td></td>
<td>About 1/2 pack</td>
<td>1.39</td>
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<td>About 1 pack</td>
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<td>About 1 1/2 packs</td>
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<td>15-24</td>
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<td></td>
<td>16 or more</td>
<td>3.00</td>
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</table>

From US Department of Health & Human Services, 1983.
In a recent large study of female nurses, about half of the coronary events were attributable to smoking.

Diabetes confers an increased risk of CHD that is further increased if the women smoke.

Cigarette smoking also significantly increases CHD risk in those with familial hyperlipoproteinemia or hypercholesterolemia or both.

The pooled data (Table 2) showed that in those who smoked a pack of cigarettes or more per day at the time of their initial examination, the risk of having a first major coronary event was 2.5 times as great as in nonsmokers. The risk was greatest in heavy smokers (more than 1 pack a day), and the relative risk for smokers compared with nonsmokers tended to increase with age, up to 50 to 54 years of age, as shown in Table with a dose response relationship. After that age the relative risk decreased. This paradox is due to the rapid increase of CHD incidence with age (without cigarette smoking). Results from cohort studies.


Table 2  Average Annual Risk (per 1000 man-years) of first major coronary event by smoking pattern and age

<table>
<thead>
<tr>
<th>Smoking pattern</th>
<th>Age at risk (year)</th>
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<tbody>
<tr>
<td></td>
<td>40-44</td>
</tr>
<tr>
<td>All</td>
<td>3.1</td>
</tr>
<tr>
<td>Nonsmokers</td>
<td>[1.5]</td>
</tr>
<tr>
<td>Never smoked</td>
<td>[1.9]</td>
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<tr>
<td>Past smoker</td>
<td>[0.9]</td>
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<tr>
<td>&lt; 1/2 pack per day</td>
<td>[1.7]</td>
</tr>
<tr>
<td>Cigar and pipe only</td>
<td>[2.1]</td>
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<td>Cigarette smokers</td>
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<tr>
<td>About 1/2 pack per day</td>
<td>[3.1]</td>
</tr>
<tr>
<td>About 1 pack per day</td>
<td>3.9</td>
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<td>4.9</td>
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<td>Risk ratio</td>
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<tr>
<td>&gt; 1 pack per day vs.</td>
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<tr>
<td>nonsmokers</td>
<td>4.1</td>
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[ ] Based on fewer than 10 first events.
From US Department of Health & Human services, 1983.
clearly demonstrate that the risk of death from CHD among both male and female smokers is increased by early smoking initiation (Table:3), number of cigarettes smoked per day, and depth of smoke inhalation. Data from the cohort studies (Table:4) show that pipe and cigar smokers generally have a substantially lower risk of a major coronary event and subsequent CHD than do cigarette smokers. The risk of CHD-related death for pipe and cigar smokers, however, is still in the range of 1.02 to 1.40 compared with that for nonsmokers, with deeper smoke inhalation increasing the risk. Among pipe and cigar smokers, former cigarette smokers tend to inhale and to have much higher venous blood carboxyhemoglobin levels than do those who have never smoked cigarettes, and they are likely to be at higher risk for CHD.

Peripheral Arterial Occlusive Disease

The most powerful risk factor predisposing persons to atherosclerotic peripheral arterial occlusive disease is cigarette smoking. Cigarette smoking has been shown to be directly related to 'lower extremity atherosclerotic disease' of both large and small arteries.

### Table 3  Coronary Heart disease mortality ratios by age at start of smoking (Cohort studies)

<table>
<thead>
<tr>
<th>Study</th>
<th>Age (year)</th>
<th>Non-smoker ratio</th>
<th>Smoker Mortality Ratio by Age (year) of initiation</th>
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<td></td>
<td></td>
<td></td>
<td>&lt; 14</td>
</tr>
<tr>
<td>U.S. veterans</td>
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<td>1.96</td>
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<td></td>
<td>65-74</td>
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<td>American Cancer Society, 25-state study</td>
<td>45-64</td>
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<td>55-64</td>
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<td></td>
<td>65-74</td>
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</table>

From US Department of Health & Human services, 1983.
Table 4 Coronary Heart disease mortality ratios for male cigarette, pipe, cigar and mixed pipe and cigar smokers (cohort studies)

<table>
<thead>
<tr>
<th>Study</th>
<th>Non-smoker</th>
<th>Cigarette smoker</th>
<th>Pipe smoker</th>
<th>Cigar smoker</th>
<th>Mixed pipe and cigar smoker</th>
</tr>
</thead>
<tbody>
<tr>
<td>U.S. Veterans</td>
<td>1.00</td>
<td>1.68</td>
<td>1.02</td>
<td>1.12</td>
<td></td>
</tr>
<tr>
<td>American cancer society, 9-state</td>
<td>1.00</td>
<td>1.70</td>
<td>-</td>
<td>1.28</td>
<td></td>
</tr>
<tr>
<td>study</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Swedish</td>
<td>1.00</td>
<td>1.70</td>
<td>1.40</td>
<td></td>
<td></td>
</tr>
<tr>
<td>American cancer society, 25-state</td>
<td>1.00</td>
<td>1.90-2.56</td>
<td>1.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td>study</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>British physicians</td>
<td>1.00</td>
<td>1.62</td>
<td></td>
<td></td>
<td>1.03</td>
</tr>
</tbody>
</table>

a. Smokers groups are "pure" smokers only.
b. Ages 56 to 84 only
From US Department of Health & Human services, 1983.
Smoking prevalence is high among victims of aortoiliac (98%) and femoropopliteal (91%) disease.

One epidemiological study found that in smokers consuming less than one pack of cigarettes per day, the relative risk of acquiring peripheral arterial occlusive disease was 11.53, compared with that in nonsmokers, and the relative risk in those who smoked more than one pack per day was 15.56 compared with that in nonsmokers.

Cerebrovascular Disease

Both ischemic and hemorrhagic cerebrovascular diseases are major causes of death in the United States and together accounted for approximately 150,000 (7%) of all deaths in the United States in 1989. Each year there are more than 400,000 new events.

The Framingham study estimated that the chances of suffering a stroke before the age of 70 years are 1 in 20, with incidence doubling each successive decade after the age of 45 years.

Smoking has been well demonstrated as a causal factor in stroke. The majority of cohort and case control studies have shown smoker/nonsmoker stroke ratios ranging from 1.2 to 4.7.


Female smokers who use oral contraceptives have been reported to be at increased risk of stroke, especially subarachnoid hemorrhage and thromboembolic events. In one case-control study of female smokers not using oral contraceptives, the relative risk for these events was 5.7 times greater than that for a non smoker and for a female smoker and oral contraceptive user the relative risk increased to 21.9.

The risk increment associated with smoking, for both MI and stroke, is multiplicative in women older than 35 years of age who also use oral contraceptives.

Mechanisms of Cardiovascular disease Development related to Smoking

The systemic hemodynamic response to smoking includes increases in blood pressure, heart rate, cardiac output, myocardial contractile force, and velocity of contraction, with gradual return to baseline levels approximately 15 minutes after smoking.

These acute physiological effects are thought to result from nicotine stimulation of sympathetic ganglia, affecting the release of norepinephrine.


Another component of cigarette smoke, carbon monoxide, significantly reduces the oxygen-carrying capacity of normal hemoglobin by binding to it in an irreversible fashion. As a result, carboxyhemoglobin levels rise, oxygen binding affinity is diminished, and availability of oxygen for cellular aerobic metabolism is reduced.

The net result of the combined actions of nicotine and carbon monoxide on the cardiovascular system is an increased demand for myocardial oxygen in the presence of a limited supply, a set of conditions that could potentially precipitate myocardial ischemia.

Smoke products may also directly cause coronary artery spasm and increase platelet adhesiveness and aggregation.

Ischaemic heart disease

The evidence that cigarette smoking is probably the major modifiable risk factor in ischaemic heart disease is now very strong and is supported by numerous large-scale studies of various kinds-clinical, experimental, epidemiological, retrospective, and prospective-which have been carried out in several countries. There is evidence that the influence of smoking is independent of, but also synergistic with, other risk factors such as hypertension.


and high blood cholesterol. This means that the effects are more than additive. It has been shown that the relative risk is greater at younger ages and that the risk for the smoker increases with the amount smoked.

A study on British male doctors showed a decrease in death rates from coronary heart disease concurrent with a decrease in the number of doctors who smoked. In contrast, the death rate increased for all other men in the study who, as a group, did not markedly decrease their smoking habits. Atherosclerosis of the myocardial arterioles is more common in smokers than in non-smokers of a corresponding age. It is also more common in the aorta and other arteries. Peripheral vascular disease is highly correlated with cigarette smoking.

An important proportion of all deaths certified as due to ischaemic heart disease under the age of 65 is attributable to cigarette smoking. The proportion is much smaller than is the case in lung cancer, but ischaemic heart disease is so much more common than lung cancer that the total number of deaths attributable to cigarette smoking is greater. Thus ischaemic heart disease may well be the greatest single health risk of cigarette smoking—at least in certain developed countries.


Cigarettes seem to be particularly important in causing sudden death from coronary heart disease, especially in men under 50 years of age.

Women appear to be less liable to ischaemic heart disease than men but in some developed countries mortality from this cause among women is increasing. In Britain between 1958 and 1971, the death rate in women aged 35-44 went up by over a third. During this period cigarette consumption by women increased by 35% and the contraceptive pill was also coming into use. This has now been shown to have some independent effect in causing myocardial infarction. The risk, however, is increased by the presence of any of the other risk factors, of which a very important one is smoking.

Among women under 50 years of age who smoked 35 or more cigarettes a day and did not use oral contraceptives, the rate of myocardial infarction was estimated to be 20 times higher than among those who had never smoked.

A large prospective study of 46,000 women of child-bearing age showed a death rate from circulatory diseases among those who used oral contraceptives five times greater than that of controls.

The death rate increased with the duration of oral contraceptive use, the age of the woman, and the smoking of cigarettes. A second study gave similar results.

After reviewing these investigations the United Kingdom Committee on the Safety of Medicines concluded that the findings were in line with the trend noted in earlier investigations that the risk of arterial thrombosis with oral contraceptives increases with age—particularly in the later part of reproductive life—and that this risk is aggravated by cigarette smoking.

Women who use oral contraceptives and smoke have significantly lower mean serum levels of high density lipoprotein cholesterol. Low levels are known to be a major risk in the development of coronary heart disease and may explain the reported increase in mortality from circulatory diseases in women smokers using oral contraceptives.

CANCER

Lung Cancer

In the United States and other affluent industrial nations, carcinoma of the lung accounts for more deaths than any other cancer. Lung Cancer mortality in the United States has risen sharply, from 18,300 in 1950 to 61,800 in 1969, 98,400 in 1979, and an estimated 142,000 in 1989.

The preliminary results from a 1956 cohort study of 40,000 British physicians older than 35 years of age, which showed that the age adjusted death rate for lung cancer increased from 7114 per 100,000 for nonsmokers to 166 per 100,000 for heavy smokers.

Eighty three percent of lung cancer deaths are directly attributable to smoking, making smoking the leading cause of cancer deaths. In 1989, it is estimated that 93,000 men and 49,000 women died of lung cancer.

Ninety percent of malignant lung tumors belong to four major cell types: Squamous cell, small cell, large cell and adenocarcinoma. Together these are commonly designated bronchogenic carcinoma. Of these cell types that account for the largest number of lung cancer, adenocarcinoma (35%) and small cell carcinoma (25%) have the strongest relationship to smoking and are usually found in persons with a smoking history of more than one pack per day for 10 years.

Lung cancer is replacing breast cancer as the leading cause of cancer death among American women.


Bidi smoking carries a higher lung cancer risk than cigarette smoking, owing to the higher concentration of carcinogenic hydrocarbons in the smoke.

The rise in lung cancer rates in male smokers preceded that of female smokers. In 1964 the male/female ratio of death rates from lung cancer was 6:7. Whereas the incidence rate in males appears to have peaked in the 1980s, the rate for females has grown rapidly, by as much as 7% per year. Current lung cancer rates for women approximate male rates of three decades ago, and the male/female ratio has declined to about 2:1. Lung cancer is replacing breast cancer as the leading cause of cancer death among American women. In 1989, it is estimated that 93,000 men and 49,000 women died of lung cancer.

Table 5 provides an outline of the lung cancer mortality ratios for smokers and nonsmokers from eight cohort studies used to establish this association. Overall, the subjects of these studies represent 17.5 million person-years of experience in five different parts of the world. Although the mortality ratios vary among studies, smoker mortality rates for lung cancer ranged from 2 to 14 times those of nonsmokers. Strength of association was further enhanced by the dose-response relationship clearly shown.


Table 8 Lung Cancer Mortality Ratios for Men and Women, by current number of Cigarettes smoked per day (Cohort studies)

<table>
<thead>
<tr>
<th>Population</th>
<th>Men</th>
<th>Women</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cigarettes Smoked/day</td>
<td>Mortality Ratios</td>
</tr>
<tr>
<td>American Cancer Society,</td>
<td>Non-smoker 1.00</td>
<td>Non-smoker 1.00</td>
</tr>
<tr>
<td>25-state study</td>
<td>1-9 4.62</td>
<td>1-9 1.30</td>
</tr>
<tr>
<td></td>
<td>10-19 8.62</td>
<td>10-19 2.40</td>
</tr>
<tr>
<td></td>
<td>40+ 18.71</td>
<td>40+ 7.50</td>
</tr>
<tr>
<td>British physicians study</td>
<td>Non-smoker 1.00</td>
<td>Non-smoker 1.00</td>
</tr>
<tr>
<td></td>
<td>1-14 7.80</td>
<td>1-14 1.28</td>
</tr>
<tr>
<td></td>
<td>25+ 25.10</td>
<td>25+ 29.71</td>
</tr>
<tr>
<td>Swedish study</td>
<td>Non-smoker 1.00</td>
<td>Non-smoker 1.00</td>
</tr>
<tr>
<td></td>
<td>1-7 2.30</td>
<td>1-7 1.80</td>
</tr>
<tr>
<td></td>
<td>8-15 8.80</td>
<td>8-15 11.30</td>
</tr>
<tr>
<td></td>
<td>16+ 13.70</td>
<td>16+ -</td>
</tr>
<tr>
<td>Japanese study all ages</td>
<td>Non-smoker 1.00</td>
<td>Non-smoker 1.00</td>
</tr>
<tr>
<td></td>
<td>1-19 3.49</td>
<td>&lt;20 1.90</td>
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<td></td>
<td>40+ 6.45</td>
<td>40+ -</td>
</tr>
<tr>
<td>U.S. veterans study</td>
<td>Non-smoker 1.00</td>
<td>Non-smoker 1.00</td>
</tr>
<tr>
<td></td>
<td>1-9 3.89</td>
<td>1-9 3.89</td>
</tr>
<tr>
<td></td>
<td>&gt;40 23.70</td>
<td>&gt;40 23.70</td>
</tr>
<tr>
<td>American Cancer Society,</td>
<td>Non-smoker 1.00</td>
<td>Non-smoker 1.00</td>
</tr>
<tr>
<td>9-state study</td>
<td>1-9 8.00</td>
<td>1-9 8.00</td>
</tr>
<tr>
<td></td>
<td>10-20 10.60</td>
<td>10-20 10.60</td>
</tr>
<tr>
<td></td>
<td>20+ 23.40</td>
<td>20+ 23.40</td>
</tr>
<tr>
<td>Canadian veterans</td>
<td>Non-smoker 1.00</td>
<td>Non-smoker 1.00</td>
</tr>
<tr>
<td></td>
<td>1-9 9.50</td>
<td>1-9 9.50</td>
</tr>
<tr>
<td></td>
<td>10-20 15.80</td>
<td>10-20 15.80</td>
</tr>
<tr>
<td></td>
<td>20+ 17.30</td>
<td>20+ 17.30</td>
</tr>
<tr>
<td>California males in</td>
<td>Non-smoker 1.00</td>
<td>Non-smoker 1.00</td>
</tr>
<tr>
<td>9 occupations</td>
<td>About 1/2 pk 3.72</td>
<td>About 1/2 pk 3.72</td>
</tr>
<tr>
<td></td>
<td>About 1 1/2 pk 8.58</td>
<td>About 1 1/2 pk 8.58</td>
</tr>
</tbody>
</table>

From US Department of Health & Human services, 1982.
illustrated by Table: C. Data in Table: C demonstrate the gradient of increasing risk of death from lung cancer as the number of cigarettes smoked per day increases. Increased consumption of cigarettes per day, whether filter or nonfiltered, results in an increased relative risk for both male and female smokers.

Four large cohort studies confirm a direct relation between number of years of smoking and lung cancer mortality (Table: C ). Lung cancer incidence appears to increase with the square of the amount smoked daily, but with the duration of smoking raised to a power of 4 or 5. Smoking mechanics also affects lung cancer mortality; the degree of inhalation varies directly with smoking-associated mortality. However, even smokers who report slight inhalation or none have a relative risk of cancer as much as eightfold and ninefold that for non-smokers.

Both case-control and cohort studies have demonstrated some reduction in lung cancer risk in smokers who switched from nonfilter to filter cigarettes. For those who have always smoked


Table 6  Lung Cancer Mortality Ratios for Males, by Age at start of smoking (Cohort studies)

<table>
<thead>
<tr>
<th>Study</th>
<th>Age(year) at start of smoking</th>
<th>Mortality ratios</th>
</tr>
</thead>
<tbody>
<tr>
<td>American Cancer Society, 25-state study</td>
<td>Nonsmoker 1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>25+</td>
<td>4.08</td>
</tr>
<tr>
<td></td>
<td>20-24</td>
<td>10.08</td>
</tr>
<tr>
<td></td>
<td>15-19</td>
<td>19.69</td>
</tr>
<tr>
<td></td>
<td>&lt;15</td>
<td>16.77</td>
</tr>
<tr>
<td>Japanese study</td>
<td>Nonsmoker 1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>25+</td>
<td>2.87</td>
</tr>
<tr>
<td></td>
<td>20-24</td>
<td>3.85</td>
</tr>
<tr>
<td></td>
<td>&lt;20</td>
<td>4.44</td>
</tr>
<tr>
<td>U.S.Veterans</td>
<td>Nonsmoker 1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>25+</td>
<td>5.20</td>
</tr>
<tr>
<td></td>
<td>20-24</td>
<td>9.50</td>
</tr>
<tr>
<td></td>
<td>15-19</td>
<td>14.40</td>
</tr>
<tr>
<td></td>
<td>&lt;15</td>
<td>18.70</td>
</tr>
<tr>
<td>Swedish study</td>
<td>Nonsmoker 1.00</td>
<td></td>
</tr>
<tr>
<td></td>
<td>19+</td>
<td>6.50</td>
</tr>
<tr>
<td></td>
<td>17-18</td>
<td>9.80</td>
</tr>
<tr>
<td></td>
<td>&lt;16</td>
<td>6.40</td>
</tr>
</tbody>
</table>

From US Department of Health & Human services, 1982.
filter cigarettes, the risk of lung cancer may be reduced as much as one half compared with that for lifelong nonfilter smokers.

Several cohort studies have reported an increased risk of lung cancer among those who smoke pipes, cigars, or both. In general, the risk of lung cancer is much less for pipe and cigar smokers than for cigarette smokers, but greater than for non-smokers. Among pipe and cigar smokers, lung cancer death rates also exhibit dose-response relationships to smoking, as seen in Table 7.

In Denmark and the Netherlands, where the style of smoking pipes and cigars involves deeper inhalation than is generally practiced in the United States, rates of lung cancer for pipe and cigar smokers approach those for cigarette smokers. This suggests that condensates of cigar and pipe smoke have a carcinogenic activity similar to cigarette condensate, but the degree of exposure to susceptible organs is affected by the depth of inhalation.

LARYNGEAL CANCER

Cigarette smoking is the major cause of laryngeal cancer. More than 25 retrospective and 6 major prospective studies have examined the relationship between smoking and cancer of the larynx. Cigarette smokers in the prospective studies have up to 13


Table 7 Lung Cancer Mortality Ratios for Cigar and Pipe Smokers by amount smoked

<table>
<thead>
<tr>
<th>Smoking Type</th>
<th>Mortality Ratio</th>
<th>Number of deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-smoker</td>
<td>1.00</td>
<td>78</td>
</tr>
<tr>
<td>Cigar smokers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;6 cigars per day</td>
<td>1.14</td>
<td>12</td>
</tr>
<tr>
<td>6-8 cigars per day</td>
<td>2.64</td>
<td>12</td>
</tr>
<tr>
<td>&gt;8 cigars per day</td>
<td>2.07</td>
<td>2</td>
</tr>
<tr>
<td>Pipe smokers</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;5 pipefuls per day</td>
<td>0.77</td>
<td>2</td>
</tr>
<tr>
<td>5-19 pipefuls per day</td>
<td>2.20</td>
<td>12</td>
</tr>
<tr>
<td>&gt;19 pipefuls per day</td>
<td>2.47</td>
<td>3</td>
</tr>
<tr>
<td>Cigar and pipe</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;8 cigars, &lt; 19 pipefuls</td>
<td>1.62</td>
<td>18</td>
</tr>
<tr>
<td>&gt;8 cigars, &gt; 19 pipefuls</td>
<td>2.19</td>
<td>2</td>
</tr>
</tbody>
</table>

From US Department of Health & Human services, 1982.
times more deaths from laryngeal cancer than nonsmokers, and relative risk ratios for the retrospective studies were consistently above 2.0. Cigar and pipe smokers experience a risk for cancer of the larynx similar to that of cigarette smokers.

The risk of developing laryngeal cancer increases with increased exposure to cigarette smoke; heavy smokers have cancer mortality ratios 20 to 30 times greater than nonsmokers. Furthermore, alcohol can act synergistically with cigarette smoking to increase the risk for cancer of the larynx up to 50% more than the sum of the excess risks posed by either behavior alone.

**ORAL CANCER**

Cigarette smoking is a major cause of cancers of the oral cavity (including malignant tumors of the lip, tongue, salivary gland, floor of the mouth, mesopharynx, and hypopharynx). A similar risk for oral cancer is posed by pipe and cigar smoking. In studies of U.S. populations, the deaths from oral cancer are from 3 to 14 times greater for smokers than for nonsmokers. This risk is also dose-related. Comparing those who smoke 25 or more cigarettes a day (the standard definition of heavy smokers) to nonsmokers, there is evidence that oral cancer mortality ratios are 5.5 to 33 times greater than in the nonsmoker.

As is true for laryngeal cancer, alcohol synergistically increases the risk of cancer of the oral cavity on a dose-related basis. Smokers who consumed 7 or more ounces of alcohol per day
had a fivefold increase in risk for oral cancer even if they smoked less than half a pack of cigarettes a day. The risk rose to 20-fold for 11 to 20 cigarettes and to 24-fold for more than a pack a day.

**ESOPHAGEAL CANCER**

Smoking of cigarettes, cigars, and pipes cause carcinoma of the esophagus. Death rates for esophageal cancer are up to six times greater for smokers than for nonsmokers; the risk is dose-related, and as is true for larynx and oral cancers, a number of studies have found that alcohol consumption is synergistically with smoking to increase the risk for developing esophageal cancer.

**OTHER CANCERS**

Cigarette smoking is a contributory factor in the development of bladder, kidney, and pancreatic cancers. An association has also been noted between cigarette smoking and cervical and stomach cancers.

The estimated deaths attributable to smoking for each of these cancers are shown in Tables 8, 9, displays attributable risk. Most cases in both sexes for each of these three cancers are attributable smoking, with strong dose-response relationships at 127 each of these sites 127.

---

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD, age &lt; 66 yrs.</td>
<td>34 a</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>[30-38]</td>
<td>[9-12]</td>
</tr>
<tr>
<td>CHD, age &gt; 66 yrs.</td>
<td>44</td>
<td>26</td>
</tr>
<tr>
<td></td>
<td>[36-54]</td>
<td>[20-34]</td>
</tr>
<tr>
<td>COPD</td>
<td>37</td>
<td>20</td>
</tr>
<tr>
<td></td>
<td>[35-39]</td>
<td>[18-21]</td>
</tr>
<tr>
<td>Cancer of lip, oral cavity, and pharynx</td>
<td>5.1</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td>[4.4-5.4]</td>
<td>[1.2-2.0]</td>
</tr>
<tr>
<td>Cancer of larynx</td>
<td>2.3</td>
<td>0.6</td>
</tr>
<tr>
<td></td>
<td>[1.6-2.7]</td>
<td>[0.4-0.7]</td>
</tr>
<tr>
<td>Cancer of esophagus</td>
<td>5.0</td>
<td>1.6</td>
</tr>
<tr>
<td></td>
<td>[4.0-5.7]</td>
<td>[1.3-1.9]</td>
</tr>
<tr>
<td>Cancer lung</td>
<td>76</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>[74-77]</td>
<td>[20-32]</td>
</tr>
<tr>
<td>Cancer of pancreas</td>
<td>3.3</td>
<td>3.4</td>
</tr>
<tr>
<td></td>
<td>[2.1-5.0]</td>
<td>[2.8-5.1]</td>
</tr>
<tr>
<td>Cancer of bladder</td>
<td>3.1</td>
<td>1.1</td>
</tr>
<tr>
<td></td>
<td>[2.1-4.2]</td>
<td>[0.6-1.9]</td>
</tr>
<tr>
<td>Cancer of kidney</td>
<td>2.6</td>
<td>0.4</td>
</tr>
<tr>
<td></td>
<td>[1.8-3.5]</td>
<td>[0.1-1.5]</td>
</tr>
<tr>
<td>Cerebrovascular disease, age &lt; 65 yrs</td>
<td>5.5</td>
<td>5.2</td>
</tr>
<tr>
<td></td>
<td>[3.9-7.0]</td>
<td>[4.3-6.2]</td>
</tr>
<tr>
<td>Cerebrovascular disease, age &lt; 65 yrs</td>
<td>12</td>
<td>4.8</td>
</tr>
<tr>
<td></td>
<td>[8-17]</td>
<td>[1.9-11.4]</td>
</tr>
<tr>
<td>Ten causes</td>
<td>231</td>
<td>106</td>
</tr>
<tr>
<td></td>
<td>[220-242]</td>
<td>[98-116]</td>
</tr>
</tbody>
</table>

Numbers in parentheses are 95% confidence intervals. From US Department of Health & Human Services, 1989.
Table 9  Estimated Attributable Risks for 10 selected causes of death in Male and Female cigarette Smokers
(United States, 1986)

<table>
<thead>
<tr>
<th>Cause of death</th>
<th>Males (%)</th>
<th>Females (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD, age &lt; 65 yrs.</td>
<td>45 a [40-50]</td>
<td>41 [34-48]</td>
</tr>
<tr>
<td>CHD, age &gt; 65 yrs.</td>
<td>21 [17-26]</td>
<td>12 [9-16]</td>
</tr>
<tr>
<td>COPD</td>
<td>84 [78-88]</td>
<td>79 [73-83]</td>
</tr>
<tr>
<td>Cancer of lip, oral cavity, and pharynx</td>
<td>92 [79-87]</td>
<td>61 [45-76]</td>
</tr>
<tr>
<td>Cancer of larynx</td>
<td>81 [57-93]</td>
<td>87 [56-97]</td>
</tr>
<tr>
<td>Cancer of esophagus</td>
<td>78 [62-89]</td>
<td>75 [57-87]</td>
</tr>
<tr>
<td>Cancer of lung</td>
<td>90 [88-92]</td>
<td>79 [75-82]</td>
</tr>
<tr>
<td>Cancer of pancreas</td>
<td>29 [18-43]</td>
<td>34 [25-44]</td>
</tr>
<tr>
<td>Cancer of bladder</td>
<td>47 [31-63]</td>
<td>37 [18-61]</td>
</tr>
<tr>
<td>Cancer of kidney</td>
<td>48 [32-64]</td>
<td>12 [3-43]</td>
</tr>
<tr>
<td>Cerebrovascular disease, age &lt; 65 yrs</td>
<td>51 [36-85]</td>
<td>55 [45-66]</td>
</tr>
<tr>
<td>Cerebrovascular disease, age &lt; 65 yrs</td>
<td>24 [16-35]</td>
<td>6 [2-14]</td>
</tr>
</tbody>
</table>

a Numbers in parentheses are 95% confidence intervals.
From the U.S. Department of Health and Human Services, 1989.
About 50% of bladder cancer cases in men and 40% in women are attributable to smoking, accounting for more than 4000 deaths per year.

In both men and women, smoking increases the risk of pancreatic cancer. Relative risks range from 2 to 3 in most studies.

The studies have established that smoking consistently increases risk of cancer of the stomach, but to a limited degree, with an average relative risk of about 1.5 and a dose response on number of cigarettes smoked.

At least 15 epidemiological studies have consistently shown an increased risk of cervical cancer in cigarette smokers.

Recent findings of nicotine and cotinine in the cervical mucus of cigarette smokers and mutagenic mucus in the cervix of smokers provide physical evidence supporting a causal relationship.


Chronic obstructive Pulmonary Disease (COPD)

Recent studies have identified the likely mechanism by which cigarette smoking induces COPD as an imbalance of levels of lower respiratory tract proteases and their inhibitors.

It is hypothesized that smoking results in an increased number of neutrophils in association with inflammation. They, in turn, secrete elastase, which can degrade elastin, a structural element of lung tissue. Oxidizing agents in smoke reduce the activity of α1-antiproteinase (α1-antitrypsin), which normally blocks elastase activity.

Increased elastase activity eventually breaks down the alveolar walls, causing emphysema.

One of the first common symptoms of smoking-related respiratory problems is "smokers cough", which is associated with excess secretion of mucus. On average, the prevalence of cough and phlegm increases threefold among male smokers and two fold among female smokers. Smokers with cough and phlegm have reduced pulmonary function and are at increased risk for symptomatic COPD.

An estimated 71,000 Americans died of COPD in 1986; 82% of these deaths were attributable to smoking. Rates increased with age and were 1.8 times higher in males than females and 2.8 times higher in white than in black people.

Data from both case-control and cohort studies consistently demonstrate a uniform increase in mortality for COPD among cigarette smokers compared with nonsmokers, with mortality ratios of 2.2 to 24.7 for one-pack-a-day cigarette smokers (Table 7). The reported relative risk of COPD for heavier smokers compared with nonsmokers is in the range of 30. Dose-response relationships have been consistently observed, with the risk of death from COPD influenced not only by the number of cigarettes smoked per day but also by the depth of smoke inhalation and by the age at smoking initiation.

Chronic bronchitis and emphysema

Surveys in many countries show that smokers cough more than non-smokers and produce more sputum. This can occur very


Table 10  COPD Mortality Rates for Men and Women by Number of Cigarettes smoked per day (Cohort studies)

<table>
<thead>
<tr>
<th>Study</th>
<th>Men</th>
<th>Women</th>
<th>COPD disease classification</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Cigarettes Mortality Ratios per day</td>
<td>Cigarettes Mortality Ratios per day</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>British physicians</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonsmoker</td>
<td>1.00</td>
<td>Nonsmoker</td>
<td>1.00</td>
</tr>
<tr>
<td>1-14</td>
<td>17.00</td>
<td>1-14</td>
<td>10.50</td>
</tr>
<tr>
<td>15-24</td>
<td>28.00</td>
<td>15-24</td>
<td>28.50</td>
</tr>
<tr>
<td>25+</td>
<td>38.00</td>
<td>25+</td>
<td>32.00</td>
</tr>
<tr>
<td>U.S. Veterans</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonsmoker</td>
<td>1.00</td>
<td>Chronic</td>
<td>1.00</td>
</tr>
<tr>
<td>1-9</td>
<td>3.63</td>
<td>bronchitis</td>
<td></td>
</tr>
<tr>
<td>10-20</td>
<td>4.51</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21-39</td>
<td>8.31</td>
<td></td>
<td></td>
</tr>
<tr>
<td>40+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Non smoker</td>
<td>1.00</td>
<td>Emphysema</td>
<td></td>
</tr>
<tr>
<td>1-9</td>
<td>5.33</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10-19</td>
<td>14.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>21-39</td>
<td>17.04</td>
<td></td>
<td></td>
</tr>
<tr>
<td>40+</td>
<td>25.34</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Canadian veterans</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonsmoker</td>
<td>1.00</td>
<td>Chronic</td>
<td>1.00</td>
</tr>
<tr>
<td>1-9</td>
<td>4.84</td>
<td>bronchitis</td>
<td></td>
</tr>
<tr>
<td>10-19</td>
<td>11.23</td>
<td>and emphysema</td>
<td></td>
</tr>
<tr>
<td>21-39</td>
<td>17.45</td>
<td></td>
<td></td>
</tr>
<tr>
<td>40+</td>
<td>21.98</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Japanese</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nonsmoker</td>
<td>1.00</td>
<td>Chronic</td>
<td>1.00</td>
</tr>
<tr>
<td>&lt;100,000</td>
<td>0.51</td>
<td>bronchitis</td>
<td></td>
</tr>
<tr>
<td>&lt;200,000</td>
<td>2.57</td>
<td>and emphysema</td>
<td></td>
</tr>
<tr>
<td>&gt;300,000</td>
<td>1.93</td>
<td></td>
<td></td>
</tr>
<tr>
<td>California men in various occupations</td>
<td>1.00</td>
<td>Emphysema</td>
<td></td>
</tr>
<tr>
<td>About 1/2 pk</td>
<td>8.18</td>
<td></td>
<td></td>
</tr>
<tr>
<td>About 1 pk</td>
<td>11.80</td>
<td></td>
<td></td>
</tr>
<tr>
<td>American Cancer Society, 9-state study</td>
<td>1.00</td>
<td>All pulmonary disease other cancer</td>
<td></td>
</tr>
<tr>
<td>Nonsmoker</td>
<td>1.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1-9</td>
<td>1.67</td>
<td></td>
<td></td>
</tr>
<tr>
<td>10-20</td>
<td>3.00</td>
<td></td>
<td></td>
</tr>
<tr>
<td>20+</td>
<td>3.64</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Data for the Japanese study are for lifetime exposure by total number of cigarettes consumed.
Non smokers in the California occupations study also include smokers of pipes and cigars.
Pneumonia, influenza, tuberculosis, asthma, bronchitis, lung abscess, and so on. From the U.S. Department of Health and Human Services, 1984.
soon after the onset of smoking and even when the number of cigarettes smoked is small. It increases in prevalence as the number of cigarettes increases. Respiratory infections, including postoperative ones, are more common in smokers, who take longer to recover. Lung function is also impaired, and the normal decrease in efficiency of the lung with age is accelerated. Even young smokers with no respiratory symptoms may have impaired lung function.

Cough, sputum, and impairment of lung function are all increased by greater inhalation of smoke, high puff frequency, keeping the cigarette in the mouth, and the use of plain rather than filtered cigarettes and of high-rather than low yield cigarettes. In a study of 800 men it was shown that non-smokers hardly ever develop a significant chronic obstructive lung disease, but a highly significant correlation was found between smoking habits and rate of loss of forced expiratory volume, thus confirming that smoking is a direct cause of this loss. In another study on over 34000 British doctors, only three of the many lifelong non-smokers were certified as possibly dying of chronic bronchitis and emphysema, while among the smokers there were hundreds of deaths due to chronic bronchitis and emphysema and to pulmonary heart disease. The certified death rate from chronic obstructive lung diseases among non-smokers was found to be only 10% of that among cigarette smokers.


There is greater likelihood of the most severe and disabling bronchities developing when heavy cigarette smoking is combined with exposure to atmospheric pollution. A nation wide survey in Great Britain showed that when cigarette smoking and exposure to high pollution are both present, the prevalence of chronic bronchitis is considerably higher than the prevalence that each factor would cause separately.

There is an increase in the number and size of the mucous glands in the bronchial epithelium of smokers, and the mucous ciliary defence of their bronchi is reduced. Emphysematous changes are more common in smokers and are rare in non-smokers. These effects are related to the number of cigarettes smoked.

Cigarette smoking acts independently of and synergistically with the other risk factors contributing to bronchitis. In developed countries it is now the most important cause of chronic bronchitis. The importance of cigarette smoking as a cause of chronic bronchopulmonary disease is relatively much greater than that of atmospheric pollution or occupational exposure. In many countries, a large percentage of bronchitis mortality in men under 65 is attributable to cigarette smoking.

In purely economic terms bronchitis is probably the most expensive of all the smoking related diseases.

Gastrointestinal Disease

Smoking contributes to the development of symptomtic gastroesophageal reflux by lowering oesophageal sphincter pressure and decreasing the competence of the oesophagogastric barrier to reflux.

Nicotine decrease pyloric sphincter pressure, permitting increased reflux of duodenal contents into the stomach, including bile salts, exocrine pancreatic enzymes, and phospholipids, all of which can injure gastric mucosa.

Smokers of both genders have a high prevalence of peptic ulcer disease, with a clear dose-response relationship based on amount smoked per day. Relative risks for prevalence of peptic ulcers and for duodenal ulcer mortality are in the range of 1.5 to 2.5 for smokers vs nonsmokers.


Diseases of the mouth

Epidemiological studies from several countries have shown that cigarette smokers have more periodontal disease than nonsmokers, suggesting a possible causal role for cigarette smoking in the development of periodontal disease.

Among adult users of smokeless tobacco or snuff, the risk of oral disease has been well documented, and changes in the hard and soft tissues of the mouth (including gum recession and leukoplakia), discoloration of teeth, decreased ability to taste and smell, and advanced periodontal destruction have been reported.

Effects of smoking on children and young people

Children are mainly harmed by the example of adults, but several studies have shown that they may also be harmed by the passive inhalation of cigarette smoke. The risk of a young infant developing bronchitis or pneumonia in the first year of life is doubled if its parents smoke. The risk is further increased if the parents also cough and produce phlegm. Wheezing in young children up to the age of five is also more common if the parents smoke.


Children under the age of two who experience a serious chest illness for any reason are more likely to have poor ventilatory function in childhood (as measured by peak expiratory flow rate). This effect persists at least into early adult life, and if such children start to smoke they are likely to develop a persistent cough. Without smoking, cough tends to decrease at 20-25 years of age. A study of nearly 4000 Paris school children and students aged 10-20 showed that adenoidectomy and/or tonsillectomy, considered as an index of repeated upper respiratory tract disease in early childhood, was very significantly related to the amount of smoking by each parent.

Children of smoking parents are more likely to smoke themselves, and if they do they experience more respiratory symptoms than those who do not. One large study involving nearly 10000 ten year old children showed that cough was significantly more common in those who smoked regularly at least one cigarette a week.

Another study showed that 12-13 year olds who smoke regularly at least one cigarette a day experience more cough, produce more sputum, and have more chest illness than non-smokers and those who have recently given up smoking. These observations suggest that at this age the damage is reversible.

Studies in 17 years olds and in young adults showed ventilatory impairment in smokers in addition to an increased incidence of coughs and of colds 'going to the chest'. This effect was dose-related, both to the amounts smoked and the years in which smoking had occurred. On giving up smoking, ventilatory function in the 17 years olds improved in a matter of weeks. Children 11-15 years old who smoked reported more shortness of breath on exertion. This was also reported by 15-19 years olds in another study.

The shortness of breath on exertion already referred to may be due partly to an effect on small airways and the ventilation of the lung. There may also be a reduction of the oxygen carrying capacity of the blood from the inhalation of carbon monoxide, which has an affinity for haemoglobin 200 times greater than that of oxygen. This could have a significant effect on athletic performance.

There is thus increasing evidence that smoking in childhood results at a very early stage in an increase of cough and sputum, more complications from infections of the upper respiratory tract, and a greater degree of ventilatory impairment. At least in the early stages the lung damage is reversible. If smoking persists, the damage is likely to progress and chronic

bronchitis in the familiar adult form may manifest itself. The earlier the age at which a child starts to smoke, the greater is his or her chance of having a less healthy life and of dying prematurely.

Pregnancy and smoking

Maternal smoking during pregnancy gives rise to retarded foetal growth, resulting in an average weight of 200 g less than that of children born to non-smoking mothers, independently of the other factors that tend to reduce birth weight such as low socio-economic status, high parity etc. The baby is small-for-dates rather than small because of a shorter gestation. Almost twice as many smokers as non-smokers produce babies weighing less than 2500g. The weight deficiency is related to the number of cigarettes smoked during pregnancy rather than to the number smoked before the pregnancy began.

Recent studies have shown an immediate effect of smoking on the foetus - an acceleration of the foetal heart rate and a decrease in breathing movements. Carbon monoxide in cord blood is higher than that in maternal blood taken at the same time, suggesting that the consequent decreases in oxygen carrying capacity of the blood in the foetus is a significant factor.


A study of 12,000 patients, Murphy et al. found that smoking mothers are more likely to have spontaneous abortion, premature delivery, and perinatal loss than are non-smoking mothers.

The association of smoking with abortion later in pregnancy is stronger and is probably due to a combination of placental damage and foetal hypoxia—the factors believed to be responsible for the increased perinatal mortality in babies born to smoking mothers.

There is some suggestion that there may be an increased risk of congenital malformation in children whose mothers smoke during pregnancy. Perinatal deaths include stillbirths, deaths during labour, and deaths in the first week after birth. One very large survey showed a 28% increase in perinatal deaths when the mother smoked. Pre-eclampsia has been reported as less common among smokers than non-smokers; however, the risk to the baby if a smoker does develop pre-eclampsia is greater than for the non-smoker's baby.

Smoking and occupation

The difference in health between smokers and non-smokers may be substantially aggravated by occupation. The combined effect of smoking and other environmental hazards, mainly occupational in nature, have been extensively studied during the past few years. In a large number of studies on occupational morbidity, a very significant difference between smokers and non-smokers has been discovered.

Respiratory diseases (other than cancer)

Many studies of workers in the mechanical, chemical, ceramics, foundry, marble, corn-mill, mining, tyre cutting, asbestos, construction, cement, rubber, cork and other industries have shown higher incidence of respiratory diseases among smokers than among non-smokers exposed to the same occupational hazards. Smokers present a higher prevalence of morning cough and production of phlegm, and dyspnoea, chronic bronchitis, and other chronic non-specific bronchopulmonary diseases are more frequent among them. In certain occupations, higher prevalences of different kinds of pneumoconiosis are often found among smokers.

Cancer

Since the 1950s, numerous investigations have indicated cigarette smoking as one of the most important factors in the etiology of lung cancer in non-occupationally exposed subjects.

Studies on industrial workers suggest that smoking habits potentiate the effect of some carcinogens such as arsenic, nickel, chromium and chromates, cadmium, beryllium, silicates, ionizing radiation and asbestos.

Specific occupational groups such as uranium miners, workers in the chromium, nickel and asbestos industries, painters and carpenters have an increased risk of lung cancer, and the risk is considerably augmented if the individuals also smoke cigarettes. The multiplicative effect of smoking on lung cancer in asbestos workers was confirmed in a study on the mortality and morbidity of asbestos workers in Finland. An excess of mortality for both lung cancer and asbestosis was found. Cancer of the larynx was also found to be associated with cigarette smoking in asbestos workers.

Sickness absence and industrial accidents

Many studies have shown that workers who are smokers are more susceptible to increased morbidity and absenteeism from work.


<table>
<thead>
<tr>
<th>Occupation</th>
<th>Exposure</th>
<th>Disease</th>
<th>Smoking-occupation interaction</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asbestos workers</td>
<td>Asbestos</td>
<td>Lung cancer</td>
<td>Multiplicative</td>
</tr>
<tr>
<td>construction workers and others</td>
<td></td>
<td>Chronic lung disease</td>
<td>Additive</td>
</tr>
<tr>
<td>in contact with asbestos</td>
<td></td>
<td>(restrictive, obstructive)</td>
<td></td>
</tr>
<tr>
<td>Aluminium smelter workers</td>
<td>Polynuclear hydrocarbons</td>
<td>Bladder cancer</td>
<td>Additive or multiplicative</td>
</tr>
<tr>
<td>Cement workers</td>
<td>Cement dust</td>
<td>Chronic bronchitis, obstructive</td>
<td>Additive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lung disease</td>
<td></td>
</tr>
<tr>
<td>Chlorine manufacturing workers</td>
<td>Chlorine</td>
<td>Chronic bronchitis, obstructive</td>
<td>Additive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lung disease</td>
<td></td>
</tr>
<tr>
<td>Coal miners</td>
<td>Coal dust</td>
<td>Chronic bronchitis, obstructive</td>
<td>Additive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lung disease</td>
<td></td>
</tr>
<tr>
<td>Copper smelter workers</td>
<td>Sulphur dioxide</td>
<td>Chronic bronchitis, obstructive</td>
<td>Additive</td>
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<td></td>
<td></td>
<td>lung disease</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Arsenic</td>
<td>Lung cancer</td>
<td>Additive or multiplicative</td>
</tr>
<tr>
<td>Grain workers</td>
<td>Grain dust</td>
<td>Chronic bronchitis, obstructive</td>
<td>Additive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lung disease</td>
<td></td>
</tr>
<tr>
<td>Organic chemicals</td>
<td>Carcinogens</td>
<td>Cancer of various organs and</td>
<td>Additive or multiplicative</td>
</tr>
<tr>
<td></td>
<td></td>
<td>tissues</td>
<td></td>
</tr>
<tr>
<td>Rock cutters, foundry workers</td>
<td>Silica dust</td>
<td>Chronic bronchitis, obstructive</td>
<td>Additive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lung disease</td>
<td></td>
</tr>
<tr>
<td>Textile workers</td>
<td>Cotton, hemp, flax, dust</td>
<td>Acute airway obstruction (byssinosis) chronic bronchitis</td>
<td>Possibly multiplicative</td>
</tr>
<tr>
<td></td>
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<td></td>
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</tr>
<tr>
<td>Uranium miners and many other</td>
<td>Alpha radiation (Radon)</td>
<td>Lung cancer</td>
<td>Possibly multiplicative</td>
</tr>
<tr>
<td>miners in radioactive environment</td>
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<td></td>
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</tr>
<tr>
<td>Welders</td>
<td>Irritant gases, metal fume, dust</td>
<td>Chronic bronchitis, obstructive</td>
<td>Additive</td>
</tr>
<tr>
<td></td>
<td></td>
<td>lung disease</td>
<td></td>
</tr>
</tbody>
</table>

Element taken from Occupational Medicine, Appleton and Lange publishers
when compared with non-smokers doing the same work. High morbidity and absenteeism were reported in a number of occupations. Some investigators have found a higher frequency of occupational accidents among smokers. The significantly greater number of days lost at work among smokers and the accidents (including fire and explosions) caused by the act of smoking raise obvious managerial problems and result in serious economic losses. Excess sickness and accidents also increase the demand for medical services.

Other aspects of smoking and occupation

Associations between tobacco smoking and pathology, other than pulmonary, have often been reported. Smoking is considered to be an important risk factor in the development of coronary heart disease, notably in office workers, bus drivers and coal miners.

Investigations to identify ischaemic heart disease risks in airline pilots showed a clear-cut dose-response relationship between smoking and various pathological findings. A 45 year old male pilot who smokes 20 cigarettes a day has a risk of sudden death 2.8 times greater than that of a non-smoking pilot, irrespective of other risk factors. Flying personnel were also


studied to evaluate the risk of pathologies of vision connected with tobacco use, which seem to be produced by cyanide and not by nicotine. Increased levels of thiocyanates in urine and plasma were found among the smokers. Tobacco smoke significantly interferes with the physical and mental abilities that are so important for airline pilots. The limitations include visual impairment, timing or temporal impairment, and impairment in decision-making and coordination. Many of these impairments are due to increased carboxyhaemoglobin level in the blood. Non-smoking pilots may also be subject to an increase in carboxyhaemoglobin levels due to passive smoking.

Many people maintain that air pollution rather than their own smoking habit is the main factor to be indicated in the development of lung cancer, respiratory diseases, and other pathologies. That this is not so has been shown by several investigations. In one of them measurements were made of the influence of smoking on the fraction of carboxyhaemoglobin in the blood of a group of Parisian taxi-drivers. This fraction was nearly four times as high for those smoking more than 15 cigarettes a day as it was for those smoking less than 5


cigarettes a day. In another study, the carboxyhaemoglobin levels in blast-furnace workers were determined at the end of the work shift. Non-smoking workers had levels of 4.0–4.9% of total haemoglobin while workers who smoked more than 20 cigarettes a day had levels of 8.5%. It is evident that if air pollution is a pathogenic factor it is small in comparison with smoking.

**HEALTH RISKS OF PASSIVE SMOKING**

**Constituents of Passive Smoke**

Evidence of an adverse health effect of passive smoking is increasing. Involuntary smoking occurs when nonsmokers are exposed to the tobacco smoke exhaled by smokers in an enclosed environment. Tobacco smoke in the environment is derived from two sources: 1. Main stream smoke emerging into the environment after being drawn through the cigarette, filtered by the smoker's lungs, and then exhaled, and (2) sidestream smoke arising from the burning end of the cigarette and entering directly into the environment. The two types of smoke share similar components, including oxides of nitrogen, nicotine, carbon monoxide, and various carcinogens and cocarcinogens. However, undiluted sidestream smoke has a higher pH, smaller particles, and higher concentration of carbon.


Sidestream smoke contains a higher concentration of potentially dangerous gas-phase constituents and accounts for about 85% of the smoke in a room with smokers. However, because of dilution, non-smokers are exposed to smaller doses of the products of tobacco combustion than active smokers.

Analysis of the smoke issuing from the end of the cigarette (side-stream smoke) shows that it contains more of the combustion products of tobacco than the mainstream smoke inhaled through the cigarette. In fact it contains five times the carbon monoxide, three times the tar and nicotine, four times the benzo(a) pyrene, and 46 times the ammonia, as well as higher concentrations of many other noxious substances.

Nitrosamines, which are potent carcinogens in animals, are present at higher concentrations in side-stream smoke than in mainstream smoke.

The smoke in a smoke-filled atmosphere consists chiefly of side-stream smoke, which has given rise to concern that exposure to this is harmful to the non-smokers.

Considerable work to develop sensitive and specific markers of exposure to passive smoking has identified


continine and, to a lesser extent, nicotine as the best short-term markers for epidemiological studies. There is a strong dose-response correlation between urinary cotinine levels and self-reported exposure to tobacco smoke.

Passive Smoking and Children's Health:

Urinary cotinine concentrations in infants and young children correlate with the number of smokers reported in the home and the number of cigarettes smoked by the mother during the prior 24 hours.

Between 1974 and 1987, four prospective and nine case control studies examined the possible effects of exposure to parental tobacco smoke on the frequency and severity of acute respiratory illness in children. Although a number of different research designs were used, the results have demonstrated a


consistent increase in the frequency of both upper and lower respiratory tract problems among young children of smoking parents compared with the children of parents who do not smoke. Pneumonia, bronchitis, bronchiolitis, and tracheitis, both as mild illnesses and for illnesses requiring hospitalization, have all been shown to increase significantly in children whose parents smoke. A dose-response curve has been observed for number of smoking parents and for level of maternal smoking, with one study estimating that an increase in the mother's smoking of five cigarettes per day results in an annual increase of 2.5 to 3.5 incidents of lower respiratory tract illness per 100 children at risk. A French study correlated higher incidence of tonsillectomy and adenoidectomy, as indexes of repeated respiratory infections, with parental smoking. Other studies have demonstrated significant associations between parental smoking and an excess incidence of chronic middle-ear effusions and related ear infections.


Most of the observed effects are limited to the first year or two of life, although residual effects later in life cannot be ruled out.

A substantial number of cross-sectional studies also support an increased prevalence of chronic respiratory symptoms in children of smokers. Significant associations have been found between parental smoking and chronic wheeze, cough, and phlegm, although not all correlations in each study achieve statistical significance. Overall, existing studies suggest that an excess prevalence of 30% to 80% of chronic respiratory disease symptoms in children of smokers, with effects observed throughout childhood.

Whether parental smoking leads to a decline in lung function in exposed children is a question that has so far defied clearcut answers. During the decade ending in 1987, a total of 17 cross-sectional studies in the United States, China and Italy assessed the physiological effects of exposure to environmental tobacco smoke on children's lung function.

Passive Smoking and Adults

Among healthy adults, the most common complaints after exposure to passive smoking are eye irritation (69%), headache (33%), nasal symptoms (33%), and cough (33%). Exposure to tobacco smoke both precipitates and aggravates allergic attacks.

in some individuals with respiratory allergies and exacerbates other symptoms associated with allergies such as eye irritation, nasal symptoms, headaches, cough, wheezing, sore throat and hoarseness.

Passive Smoking and Lung Cancer

The National Research Council's review estimated the true relative risks (relative to subjects not exposed to smoking in the environment) for nonexposed nonsmokers married to smokers and for nonexposed nonsmokers married to nonsmokers as 1.41 to 1.87 and 1.09 to 1.45, respectively. These estimates mean that between 2500 and 8400 of the approximately 12,200 annual lung cancer deaths in the United States not caused by smoking are attributable to environmental tobacco smoke.

At least three prospective studies and 15 case-control studies have been published on this subject, with most investigators assessing exposure on the basis of nonsmokers' reporting that they were living with a smoker. Each of the three prospective studies reported a slightly higher risk of lung cancer in nonsmokers married to smokers, with reported relative risks of 1.18 to 2.25 and a weighted risk/ratio value


of 1.44 (95% confidence intervals of 1.20 to 1.72). An increased risk of lung cancer was found in nonsmokers married to smokers in 10 of the 15 case control studies, with increases in 6 of the 10 studies reaching statistical significance. The odds ratios weighted average for these studies was 1.24 (95% confidence interval 1.04 to 1.50).

In Utero Effects of Maternal Smoking

The effects on the fetus of exposure to maternal smoke in utero have been extensively studied. It is well documented that infants born to women who smoke during pregnancy weigh an average of 200 g less than those born to nonsmokers. The incidence of low birth weight (<2500 g) in infants born to mothers who smoke is twice that in infants born to nonsmokers.

Although fetal growth is diminished among maternal smokers, placenta/birth-weight ratios are larger in comparison.


with maternal nonsmokers, probably because of the larger placental surface necessary to provide adequate fetal oxygenation in smokers. Smoking increases the level of carboxyhemoglobin in both maternal and fetal blood, with subsequent reduction in oxygen binding capacity and the pressure at which oxygen is delivered to fetal tissues, resulting in fetal hypoxia. Smoking also increases the risk of maternal bleeding, including bleeding caused by abruptio placentae, during pregnancy.

Maternal smoking is associated with higher fetal, neonatal, and infant mortality. One large study showed adjusted infant mortality rates of 15.1 per 1000 for white nonsmokers and 23.3 for white women who smoked more than one pack per day. Comparable infant mortality rates for black women were 26.0 and 39.9, respectively. Maternal smoking increases the risk of spontaneous abortion. The few studies that have examined the long-term consequences of maternal smoking in offspring are:


suggest that there may be a slight increase in the incidence of mental retardation, cerebral palsy, epilepsy, hyperactivity, a shortened attention span, lower test scores, and electroencephalographic abnormalities.

Review of some Research studies on Tobacco smoking

Tobacco smoking and Athletic performance

As a rule coaches are against smoking during training while there is no general agreement regarding the harmfulness of smoking, no one as yet has seriously recommended smoking as an adjunct to training.

The most frequently quoted reference is a report, made of students in a military school, stating that non-smokers were faster in cross-country running than smokers. On the other hand, after a marathon race at Pittsburgh it was reported that the first five winners were smokers. To this we may add, mention of many athletes who continue smoking during training without any apparent detrimental effect on performance. Numerous studies have been conducted to determine the effect of cigarette smoking on physical performance. Many of the studies have reported detrimental effects of smoking on various individual physiological adjustments to exercise. In general,


they have found (a) a decrease in airway conductance; (b) a decrease in lung diffusing capacity; (c) an increase in airway constriction in lung bronchioles; (d) a decrease in the efficiency of the heart as determined by a higher heart rate and lower stroke volume; (e) an increase in the oxygen cost of breathing, (f) a larger oxygen debt for a standardized work load; and (g) an increase in blood pressure.

Three investigators have shown that smoking had no apparent effect on strength of the hand grip, speed of tapping a telegraph key, the Sargent jump test, the Harvard Step Test score, oxygen intake, oxygen debt and the net oxygen cost of an exercise performed on a bicycle ergometer.

The effect of smoking evidently may not be too well pronounced and not always the same. Moreover, man's physical performance has a range of fluctuation which may mask the effect of tobacco.

One such experiment was made by Karpovich and Hale. It took two years, but only thirteen subjects were tested: eight men who were habitual smokers and five men who were nonsmokers. All of the men practiced riding a bicycle ergometer for one to


two months, two or three times a week, until they became proficient, as judged by their riding time with a prescribed load and number of pedal revolutions. After the training period was over, the experiment itself started. The subjects were asked to complete a prescribed number of pedal revolutions in the shortest possible time, while the load remained the same during each test. (For most subjects it was 425 pedal revolutions with an 8 pound load, an activity similar to a 1-mile run on the track.) Each man served as his own control, smoking during certain periods of the test and abstaining from smoking during other periods. The number of tests made on individual men varied from 24 to 31.

On the strength of this experiment it is possible to draw the following conclusions:

1. Although the average performance of the group was worse after smoking, than without smoking, the difference was not statistically significant.

2. Three habitual smokers and two nonsmokers did better when they did not smoke. This difference was statistically significant.

Thus it appears that not all people are noticeably affected by smoking. Some are tobacco sensitive and some are not. This study indicated that as high as 37.5 percent of young men may be tobacco sensitive, and their speed will be slowed by smoking. True enough, this slowing may not be observed each time, as is shown by Fig.1. One may see that, occasionally,
performance with smoking (dark disks) was better than without smoking, but that these occasions were exceptions. As a rule, speed was lower after smoking.

This means that by smoking, a tobacco-sensitive athlete may jeopardize his own and his team's chances for victory. Since the percentage of tobacco-sensitive men is relatively high, a nonsmoking rule for athletic teams is a wise precaution which should be firmly supported.

In another study to determine if non-invasive pulmonary stress testing would be useful in screening for obstructive lung disease in teenage smokers, twenty four apparently healthy, untrained boys, aged 16 to 18, participated as subjects. Twelve had smoked more than 20 cigarettes per day for over five years. The other 12 boys were similar to the smokers in age, height, weight, and percentage body fat, and served as a control group of non-smokers. Resting pulmonary function tests showed no significant difference between groups in vital capacity, residual volume, total lung capacity, and timed expiratory volumes. The groups did not differ in resting heart rate (HR), resting blood pressure (BP), maximum HR, maximum BP, or electrocardiographic responses to graded treadmill exercise. Maximal oxygen consumption averaged 3.38 L/min (48.0 ml/kg/min) for the smokers and 3.50 L/min (50.2 ml/kg/min) for the non-smokers, the difference between groups not being statistically significant. There was also no

significant difference in exercise tidal volume or respiratory exchange ratio. However, the smokers had 17% lower breathing frequency, 19% lower expired minute volume, and 16% lower ventilatory equivalent for oxygen uptake at the point of volitional fatigue, all of which were highly significant (P < 0.01). Exertional dyspnea was reported more often by the smokers. Thus, the smokers breathed less during maximum exercise because of a lower respiratory rate, despite similar carbon dioxide production. This study reveals an association between chronic teenage smoking and reduced maximum exercise ventilation. Impaired ventilatory capacity on pulmonary stress testing may be an early sign of obstructive lung disease.

In another study to determine the effect of drinking and smoking patterns of university students when participating in "endurance" (jogging, conditioning exercises) and "participatory" (riflery, billiards, archery) classes three times a week for 40 minutes for a period of 15 weeks.

For this study, all students during the academic year 1978-1979 enrolled in the beginning elective endurance and participatory courses at a large midwestern university were asked to participate.

The students were administered the Student Alcohol Questionnaire and items to determine their smoking patterns at

the beginning and at the end of the semester. The sample of students that completed both the pre and post test conditions contained 156 students in the participatory and 135 in the endurance activities.

A Friedmans Analysis of Variance for Ordinal Data, comparing the difference in rankings between two groups, was used. The results of the study showed that 15 weeks of participation in either endurance or participatory classes had no effect on drinking or smoking patterns of university students in the sample.

Investigative Studies on Tobacco Smoking

First Study:

A multivariate model of adolescent smoking decisions was examined in order to assess the role of health beliefs versus social perceptions and demographic factors. Utilizing a technique which increases the validity of self-reports, an indepth survey of 431 ninth graders was undertaken. Females reported more overall exposure to cigarette smoking \( F(1,416) = 5.29; p = 0.2 \) and a greater likelihood of having a steady who smokes \( X(4) = 16.89; p = 0.02 \) than males. Smokers reported being exposed to others who smoke significantly more than nonsmokers \( \text{F}(1,416) = 174.56; p = .001 \). Evaluation of the model predicting present smoking decisions indicated that exposure to smoking by others \( \text{F}(10,339) = 36.05; p < 0.001 \)

accounted for 45% of the variance while the normative expectation of others ($F(2,339) = 13.99; p = .001$) and health beliefs ($F(1,339) = 17.20; p = .001$) accounted for 3.6% and 2.1% of the variance respectfully. In contrast, the model predicting future smoking intentions, controlling for present smoking behavior, resulted in health beliefs ($F(1,338) = 30.45; p = .001$) accounting for 4.5% of the variance followed by 3.2% by traditional demographic measures ($F(1, 338) = 1.97; p = .05$), 2.2% by perceived social pressure to smoke ($F(1,338) = 14.86; p = .001$) and 1.6% by expectations of others ($F(2,338) = 4.82; p=0.01$). Social psychological perceptions were found to be material to both present and future smoking decision, although exposure to smoking models was most predictive of present behavior. Important differences were found between smokers and non-smokers as well as males and females.

Another study in the Utah Smoking Education Risk Reduction program was to develop, implement, and evaluate a multiple stage smoking education risk reduction program designed to prevent/delay smoking onset. The Smoking Risk Reduction Curriculum was comprised of three components; (1) knowledge of the health consequences of smoking, (2) health decision making skills, and (3) resistance to peer, media, and adult pressures to smoke.

A stratified sample of urban and rural school districts participated in the study targeted for sixth grade students (N=700). A quasi-experimental pre-test, repeated post-test design was used to evaluate the risk reduction program. Student outcome measure included knowledge, locus of control, behavioral intent, decision making skills, resistance to persuasion, and selected aspects of smoking behavior. No significant differences (p .06) between experimental and control groups were hypothesized. Results of a repeated analysis of variance and covariance indicate significant differences on selected student outcome measures.

Third study:

Determinants of the smoking behavior of college women were studied in a survey of 953 women selected at random from four Boston-area colleges. Whether or not the woman ever smoked was significantly related to the smoking behavior of the mother, but not of the father. Multiple regression was used to ascertain the relative contributions to the woman's current amount and frequency of smoking of the smoking behavior of each parent and the woman's age cohort, as well as the contributions of the smoking behavior of her five closest male and female friends and boyfriend. The equations relating the primary socialization agents showed the smoking of the mother (Beta=.137) and the age cohort (Beta=.140) to be significantly

related to the woman's frequency of smoking, while the age cohort alone was related to the amount. With regard to the peer network, only the smoking of female friends (Beta = .171 versus .017 for boyfriend and .008 for male friends) was related to the woman's frequency of smoking. The same pattern held for amount (Beta = .168 for female friends; .084 for boyfriend; and -.039 for male friends). The failure to find any relationship between the smoking behavior of the father, male friends and boyfriend and that of the woman in contrast to that found for the mother and female friends is intriguing. It suggests that smoking may have a special meaning to women linked to sex role and that it is transmitted through identification with the mother in the socialization process. Other research has shown higher levels of smoking for college and professional women than for their male peers. For these women, who perform in "liberated" sex roles, smoking may have acquired a secondary meaning related to aggression, power, and other traditionally masculine traits.

Fourth study:

The specific objective of the study was to identify and compare the age and grade variables as related to beginning marijuana, tobacco, and beer/wine use of students located in a metropolitan area. Data for this study were gathered through the use of a STUDENT SURVEY instrument which was developed by R. Nell H. Gottlieb. "Sex Role and the Determination of the Smoking Behavior of College Women". Research Quarterly, p. 137, April, 1981.
PRIDE and used in many similar studies prior to this one. Responses (38,986) from a sample size (N=12,962) representing grades six (6) through twelve (12) were catalogued as follows; Marijuana use (N=13,867); Tobacco use (N=14,677), and Beer/wine use (N=10,441). Based on the responses given by this sample the following results were found; Yes responses to marijuana use; 53% to 12th grade; 51% for 11th grade; 41% for 9th grade and 10 grade; 21% for 7th & 8th grade; and 13% for 6th grade. Yes responses to tobacco use; 61% for 12 grade; 60% for 11 grade; 67% for 9th & 10th grade; 48% for 7th and 8th grade; and 34% for 6th grade. YES responses for beer/wine use; 78% for 7th and 8th grade; and 23% for 6th grade. It can be concluded from the results of this study that the use of all these substances increased with age and the rate of use increases over grades six through twelve as follows; Tobacco (twice the percentage); Beer/wine (three times (+) the percentage); and marijuana (four times the percentage). Students begin using tobacco and alcohol at a younger age than marijuana but the percentage of increase is much greater.

Fifth Study

The purpose of the study was to identify correlates of expected future use of tobacco and alcohol among elementary school students. A total of 622 first, third and fifth grade children from four elementary schools located in four different cities in Arkansas served as subjects for the study. Each child

212. Thomas J. Gleaton, "Relationship between Grade level, Age, Marijuana, Tobacco and Beer/Wine use of students in Grades Six through Twelve." Res.Qtrly., p.22, April,1982.
was interviewed individually, with their responses recorded on audio tape. In addition to general background questions, each child was asked the questions concerning tobacco and alcohol products. In order to identify correlates of expected future use of tobacco and alcohol, the SPSS subprogram crosstabs were utilized to analyse the data in a series of 2 x 3 contingency tables. It was found that significant relationships (\( p = 0.05 \)) existed between expected future use of tobacco and (1) personal use and (2) having seen the product used at home. Data were further analysed controlling first for grade level and then for whether or not the child lived with one or both parents. It was found that the relationship between expected future use of tobacco and personal use was significant at the first grade level and when the child lived with both parents. The relationship between expected future use of tobacco and whether or not the student has seen the product used at home existed at both the first and fifth grade level and when the student lived with both parents. Significant relationships were also found between expected future use of alcohol and (1) knowing someone who used alcohol, (2) personal use and (3) having seen the product used at home. Reanalysis of these findings revealed that the relationship between expected future use of alcohol and all three preceding variables was significant at all grade levels and regardless of whether the child lived with one or both parents.

Sixth Study

Experimental simulation of human smoking was conducted on beagle dogs by Auerbach and colleagues. Cigarettes were held in a Teflon tube that was inserted in a tracheostoma, and smoke was delivered by deep voluntary inhalation of the dog. After a maximum of 14 months of exposure, no tumors developed in 10 exposed dogs, but sections of bronchial epithelium exhibited atypical nuclei, loss of ciliated columnar cells with squamous replacement, and hyperplasia. These histologic changes, which were similar to those seen in tissue from lung cancer patients, were almost entirely absent in sections from unexposed dogs.

Only limited success has been achieved in inducing respiratory cancers in animals in response to tobacco smoke inhalation, due both to acute toxic effects and animals' resistance to deep inhalation of tobacco smoke. In these studies, animals are placed in exposure chambers and exposed to alternating short periods of tobacco smoke diluted with air, followed by air alone. Excess incidence of respiratory tumors has been demonstrated in mice, rats, dogs, and hamsters (the latter develop laryngeal tumors only) compared to unexposed controls. However, overall incidence has been low, and dominated by adenomas and alveogenic adenocarcinomas.


The majority of carcinogenesis bioassays in animals have used direct application of tobacco smoke condensates ('tars'), or subfractions of the particulate phase. Tumor initiation and cocarcinogenesis have primarily been associated with the neutral subfractions that are rich in polynuclear aromatic hydrocarbons (PAH), while promoters are found in the weakly acidic subfractions. Intrapulmonary administration of tobacco smoke condensates or fractions have produced lung carcinomas and adenomas in rats and hamsters, respectively. Tumors of the nasal cavity, lung, liver, and esophagus have been produced in rats by intraperitoneal administration of tobacco-specific nitrosamines (TNSA). The most extensive assays have examined tumorigenesis of tars on mouse skin. Skin-painting of cigarette smoke condensates has consistently produced papillomas and carcinomas in over 40% of mice, compared to near-zero incidence in control animals. Condensates from pipes and cigars have also produced high tumor incidence in animal bioassays.

Seventh study

A large number of in vitro studies have been conducted with tobacco smoke (both MS and SS) as well as condensates. Mutagenicity has been demonstrated for smoke and condensates in Salmonella typhimurium (the Ames test). Urine from smokers


has also been shown to be mutagenic in this system, with some
evidence of a dose-response. In mammalian tissue culture
systems, condensates, fresh tobacco smoke, and the gas phase of
smoke have induced sister chromatid exchange and other
mutations, cell transformation, and inhibition of both DNA
repair and intercellular communication.

A number of markers of genetic damage occur with
significantly greater frequency in smokers than non-smokers.
Excess prevalence of sister chromatid exchanges and micronuclei
has been observed in peripheral blood lymphocytes and bone
marrow of smokers compared to non-smokers. Many of these
studies have demonstrated dose-response trends with number of
cigarettes or duration of smoking. DNA adducts (addition
products) represent covalent bonding of carcinogen with DNA, and
serve as a marker of initiation. These adducts have been found
in the lung, bronchus, larynx, esophagus, bladder, kidney,
blood and heart of smokers; adduct levels correlate with dose
and duration of smoking. The adducts probably arise from
exposure to PAH and other aromatic compounds, since adducts are
not seen in association with smokeless tobacco and polycyclic
aromatics are generated by burning tobacco. Detectable DNA

219. IARC. Op. cit

220. IARC. Ibid.

221. Perera, F.P. Santella, R.M. Brenner, D. Poirier, M.C.
Munshi, A.A. Fischman, H.K. and Van Ryzin, J: "DNA adducts,
protein adducts and sister chromatid exchange in cigarette
Adduct levels are observed in less than 20% of smokers presumably representing heterogeneity in ability to metabolize PAH or repair adducts. This is in good agreement with the 10-20% of heavy smokers who develop lung cancer.

In an effort to find a short cut that will reduce the time and effort required to become competitive or to meet the challenge of the moment, it has been all too common an occurrence in the history of man to try chemicals of various types to improve both mental and physical performance. Examples of this range from students trying to obtain a better grade, to athletes trying to shave seconds off the time required to perform an athletic feat, to the military's use of chemicals to help soldiers perform heroic feats of endurance in an effort to defeat the enemy. The first reported use of drugs to increase or improve athletic performance predates the birth of Christ. It is reported in the writings of Homer that Greek athletes consumed mushrooms prior to athletic events to improve their performance. More recently, during the nineteenth century, there were widespread reports of athletes using caffeine, alcohol, nitroglycerine, ethyl ether, and opium. However, it was not until the unfortunate death of a Danish cyclist during the 1960 Olympic Games in Rome that the attention of the world was drawn to this serious problem. Today, drug use by athletes involves several classes of drugs.


Cocaine use is clearly on the rise. Intense media coverage of the deaths caused by cocaine, particularly of sports figures have brought the deadly potential of cocaine into the public eye. However, in 1986, there were 100,000 deaths related to alcohol in the USA and only 613 deaths due to cocaine. Alcohol-related deaths account for more than 30 times the number of deaths attributed to all illegal drugs. Alcohol continues to be the primary recreational drug abused by athletes and society in general. Alcohol is readily available, legal in most areas, and socially acceptable. Cocaine, heroin, hallucinogens, stimulants, and depressants are sold primarily on the black market.

However, the following discussion will be a brief synopsis of the pharmacology of other recreational drugs like alcohol, cocaine, marijuana and its effects on endurance and physical performance.

OTHER RECREATIONAL DRUGS

Alcohol

Alcohol is the recreational drug most frequently used by both amateur and professional athletes. In a survey of one of the major athletic NCAA conferences, more than 60% of college athletes were regular users of alcohol. Alcohol is classified as a depressant and acts primarily on the central nervous system.

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nervous system. The action of alcohol takes place in four stages as blood alcohol levels rise. In stage one, there is increased freedom of speech and action, a sense of well-being and greater self-confidence. Stage two allows the subject to become more talkative, with blurred speech. This is followed in stage three by indistinct and incoherent speech. In stage four, there is uncontrollable behavior with emotional disturbances and reduced sensibility, followed by loss of consciousness.

Recently, alcoholic beverages have been used by marathon runners as a carbohydrate and electrolyte replacement fluid. There are no data to support beneficial effects. There are numerous studies which demonstrate fine motor skill retardation and discoordination under the influence of alcohol. The American Medical Association and the National Safety Council consider an individual intoxicated with 150 mg alcohol/100 ml blood (0.15%) or its equivalent in urine, saliva, or breath.

The toxic effects of alcohol abuse are also well documented. Chronic ingestion leads to addiction and serious neurological and mental disorders such as brain damage, memory loss, sleep disturbances, and psychoses. Liver damage and acute
documentation.


228. Casal DC, Loc.cit
Although alcohol, in some circles, is believed to be a central nervous system stimulant, it is really a depressant and if consumed in large quantities, it may bring about partial or complete loss of sensibility.

Numerous studies have been conducted to observe the effects of alcohol on strength, local muscular endurance, psychomotor skills, aerobic and anaerobic work capacities. While there appears to be agreement that large amounts of alcohol (such as 6 ounces or more) may be detrimental to physical performance, there is no general agreement as to the effects of small and moderate amounts on work performance. Some studies, in using small and moderate amounts of alcohol, found performance to be either enhanced, retarded or unchanged. This should be apparent, however, since the tolerance varies from person to person and probably from time to time in the same person.

Old experiments showed that small amounts of alcohol increased muscular endurance. Hellsten, using Johansson's ergograph, which involves the pulling of weights with both hands, found that small doses of alcohol taken five to ten


minutes before the exercise did increase work for the first twelve to forty minutes, after which there was a definite drop in performance lasting for two hours. Amounts of alcohol up to 80 cc. of 38 percent brandy taken half an hour before Hellsten's test caused a decrease in work output from the beginning of the test. On the other hand, Atzler and Meyer found that even 240 cc. of alcohol in the form of beer or brandy given immediately, or as much as four hours before their test would increase the work output, provided the men were habitual drinkers. The same amount of alcohol taken the night before caused a marked drop in work capacity.

Herxheimer observed a deleterious effect of alcohol upon speed in swimming or running short distances. Herring observed the same effect in 100, 400 and 1500 meter races. The amount given was rather large; 100 cc of 52 per cent alcohol, or the equivalent of about half a tumbler of whiskey. Simonson reported a decrease in oxygen debt after work done following the intake of a small amount of alcohol, whereas Meyer found no change.


The common use of alcohol for warming up seems to draw supporting evidence from observations that small doses of alcohol increase the endurance of chilled muscles. This coincides with a feeling of superficial warmth, caused by a larger amount of blood coming to the skin blood vessels, which have been dilated by alcohol. However, the loss of heat is also increased, and the danger of greater chilling is enhanced.

Marijuana

Marijuana is probably the recreational drug second most frequently abused by athletes. It is used to obtain a general sense of well-being and release from the surrounding environment. Its active ingredient is 1-8-9 tetrahydrocannabinol (THC). Approximately 50% to 75% of the THC in a marijuana cigarette is absorbed through the lungs when it is smoked. Drug action occurs in 5 to 15 minutes after smoking and has a duration of 3 hours. The primary observed effects are produced by alterations in central nervous system function, but the drug is also found in the liver, lung, kidney, and spleen.

There are no studies to date that indicate any benefits to the athlete from marijuana use. Motor coordination, short-term memory, and perception are impaired. Work motivation, critical to the athlete for adequate training, may be impaired.

decreased. In chronic users, the toxic effects of marijuana abuse have been decreased plasma testosterone levels, oligospermia and gynecomastia.

Cocaine

Cocaine is the third most frequently used recreational drug. However, its abuse is increasing in high school, college, and professional athletics. Cocaine is a naturally occurring chemical isolated from the leaves of the coca plant (Erythroxylon coca), which grows in the Andes Mountains of Peru. For several centuries, the local inhabitants of these mountains have chewed on the leaves of the plant. In fact, it is estimated that almost nine million kilograms of leaves are consumed annually by the two million inhabitants of the region. The popularity of cocaine has waxed and waned through the centuries. It has been, at times, hailed as a 'magical drug' capable of curing a variety of mood disorders as well as alcohol and morphine addiction. At other times, it has been placed under such tight legal restrictions as to make its use by humans legally impossible. Clearly, the reason for the high degree of consumption of the coca leaves by the inhabitants of the Andes, its wide acceptance as a 'magical drug', and the legal restrictions on its use are probably more closely related to its mood-altering and reinforcing properties than to any therapeutic benefit of the drug.


Abusers describe an "incredible feeling of well-being, a feeling of omnipotence. Cocaine is provided through black market channels and is usually of unknown purity. Cutting agents include lactose, procaine, benzocaine, and lidocaine. Cocaine is usually snorted but the current trend is towards free-basing/smoking cocaine. The cocaine used for this route of administration is called crack. Preliminary evidence indicates that this form of cocaine is much more addictive and causes a very intense, quick drug response. It is a much more deadly than the traditional forms of cocaine because of its very intense and quick action. The death of Len Bias, an amateur basketball player at the University of Maryland, and Don Rogers, a professional football player for the Cleveland Browns, within the past year emphasizes the danger of cocaine abuse. The supply of cocaine has become a major political issue in several South American countries which depend upon cocaine as a major source of revenue.

Cocaine acts on catecholaminergic neurons in preventing reuptake of both norepinephrine and dopamine. The physiological effects of cocaine are similar to those of amphetamines. However, the mood effects are more profound. Moderate effects include both euphoria and dysphoria while


strong stimulant effects include irritability, withdrawal, hostility, anguish, fear, and extreme paranoia. These effects would appear to discourage use during competition. However, the increased energy level, hyperactivity, and anorexia produced appeal to some athletes.

The toxic effects of cocaine use are dizziness, tremor, extreme irritability, confusion, and hallucinations. There are also effects on the cardiovascular and respiratory systems which produce chest pain, palpitations, hypertension, sweating, and cardiac arrhythmias. Extreme cases of cocaine abuse are fatal with hyperpyrexia (increased body temperature) and convulsions.

The most important therapeutic activity of cocaine is its activity as a local anesthetic. Aside from this local anesthetic activity, its most prominent effects are on the CNS. In man, the effect of cocaine on the CNS is a generalized stimulation. The initial effect upon administration is euphoria and a feeling of well-being, although dysphoria is sometimes reported. No effect on motor function is observed at low doses. However, as the dose is increased, tremors and potentially lethal convulsions may be seen. The stimulation is followed by a depression, and eventually the respiratory centers become

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depressed. At high doses, the cause of death is respiratory depression.

At low doses, a bradycardia is sometimes seen due to a central vagal stimulation. However, as the dose is increased, the heart rate and mean blood pressure increase. At high doses, acute myocardial infarctions, often associated with potentially lethal arrhythmias have been reported.

The mechanism of action for the CNS effects and probably some of the cardiovascular effects of cocaine appears to be related to its ability to block the neuronal reuptake of catecholamines at nerve endings. Thus, cocaine causes a potentiation or at least an increase in the duration of action of catecholamines in the synaptic cleft. Cocaine is well absorbed from all sites following application, including nasal and gastrointestinal mucosa and the lungs. However, due to its vasoconstrictor properties, its absorption is somewhat self-limiting. Once absorbed into the bloodstream, cocaine is rapidly metabolized by plasma esterases and in some species by hepatic enzymes. The plasma half-life in man is approximately.


Mosso showed that 0.1 gm. of cocaine postpones the onset of fatigue. Thiel and Essig found that the endurance of men and women riding bicycle ergometers was increased when they were given 0.1 gm of cocaine hydrochloride by mouth. The maximum effect of the drug was noticed thirty minutes after intake. Herbst and Schellenberg using the same amount of cocaine, noted that the speed of recovery after riding stationary bicycles was increased.

Physical fitness

Modern research in Physical Education and Education proved that, the student product as an entity, a unity of the mind, body, spirit and social being. These unified and integrated, individuals are composed of many elements and properties and are the end product of many forces and conditions that play upon them.

A nation's true wealth lies not in its lands and waters, not in its forests and mines, not in its flocks and herds, not in its dollar's but in its healthy and happy men, women and children. In other words, it lies the energy and


initiative and moral fitness of its people. For example, 1438 AD, there was a "Black Death" in England. Then England was called the country's graveyard.

Physical Fitness is the basic fitness of all other fitness. Physical fitness is not only one of the most important keys to a healthy body, it is also the basis of dynamic and creative activity.

It can be said that there are three types of fitness. Fitness for living, fitness for holding a job and fitness for recreational hobbies. All of them are affected by age and sex.

President "Johnson" emphasized that "Physical fitness is a matter of fundamental importance to individual well being and to the progress and security of our nation". President Kennedy referred to Physical fitness as "the basis of all other forms of excellence". It has been said that fitness adds not only "years to one's life, but life to one's years"

The old saying, "If you don't use it you lose it", certainly applies to physical fitness.

Strictly speaking, physical fitness means that a person possessing it meets certain physical requirements. These


requirements may be anatomical (structural), physiological (functional), or both. Anatomical fitness may require a person to be of a certain height or weight, or have specified dimensions of various parts of the body. Physiological fitness may require a person to be able to withstand certain temperatures or altitudes or able to perform specific physical tasks involving muscular efforts. A person may be perfectly fit to meet some of these requirements and yet be unfit for others. A person physically fit in all respects does not exist.

From an occupational point of view, physical fitness may be defined on "the degree of the ability to execute a specific physical task under specific ambient conditions".

DEFINITION OF PHYSICAL FITNESS

1. "Physical fitness may be defined as the organic capacity of the individual to perform normal tasks of the individual to perform normal tasks of daily living without undue tiredness or fatigue, having a reserve of strength and energy to meet satisfactorily and emergency demand".

   -Clarke.

2. "Fitness is that state which characterises the degree to which a person is able to function. Fitness is an individual matter. It implies the ability of each person to live most effectively with his potential. Ability to


function depends upon Physical, mental, emotional, social, and spiritual components of fitness, all of which are related to each other and are mutually interdependent".

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3. "The ability to carry out daily tasks with vigor and alertness, without undue fatigue, and with ample energy to enjoy leisure time, pursuits and to meet unforeseen emergencies .... Physical fitness is the ability to last, to bear up and to preserve under difficult circumstances where an unfit person would give up. It is the opposite to being fatigued from ordinary efforts, to lacking the energy to enter zestfully into life's activities, and to becoming exhausted from unexpected, demanding physical exertion".

-Clarke.

Motor Fitness

"The quality of life which enables one to live most and serve best". - William.

Generally speaking, Motor fitness is an inherent, innate and inborn quality of the individual. The ascribed quality of motor fitness is a part and parcel of human motor performance. Motor fitness is generally judged by performance and this performance is based on a composite of many factors.

The Physical Educator understands the term motor fitness because it is more limited in scope and loses some of the elusiveness of Physical or total fitness. It includes the

capacity of the individual to move efficiently and with strength and force over a reasonable length of time. It is highly related to total fitness in the same manner. This view is consistent with the idea that each individual is an integer and cannot be separated into divisible parts for development.

Definition of Motor fitness

1. "Motor fitness may be defined as a readiness or preparedness for performance with special regard for big muscle activity without undue fatigue. It concerns the capacity to move the body efficiently with force over a reasonable length of time."
   - Unknown

2. "Motor fitness may be defined as a limited phase of motor ability, giving importance for the capacity to do vigorous work, various aspects that are included in motor fitness are, endurance, power, strength, agility, flexibility, balances and stamina."
   - BARROW.

The term motor fitness came into being during World War-II as tests that could be given quickly to many subjects with a minimum of equipment were constructed for use by various branches of the armed forces and by schools and colleges. Actually, motor fitness is a limited aspect of general motor ability, with emphasis placed on the underlying element of vigorous physical activity, but does not include the neuromuscular coordination involved in motor skills.

The basic physical fitness components are muscular strength, muscular endurance, and circulatory-respiratory endurance. Muscular power, agility, speed, and flexibility are added to compose motor fitness.

**Muscular Strength**

Muscular strength refers to the maximum contraction power of the muscles. How strong muscles are is commonly measured with dynamometers or tensiometers, which record the amount of force particular muscle groups can apply in a single effort.

Muscular strength is also referred as the amount of external force which a muscle can exert. In other words, muscular strength may be defined as "the capacity of individual to exert muscular force". "Strength can be also defined as the maximal one-effort force that can be exerted against a resistance". Ex: Six Pound Medicine Ball Put.

**Muscular Endurance**

Muscular endurance is the ability of muscles to perform work. Two manifestations of muscular endurance are recognized: isometric, whereby a maximum static muscular contraction is held; and isotonic, whereby the muscles continue to raise and lower a submaximal load, as in weight training or performing pushups. In the isometric form, the muscles maintain a fixed

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length; in the isotonic form, they alternately shorten and lengthen.

Muscular endurance is also referred as the ability of the muscle or a group of muscles to continue to function under a heavy work load without undue fatigue, over a period of time. In other words, 'ability of a muscle or muscle group to sustain effort for a prolonged period of time'.

"Muscular endurance can be defined as the ability of muscle or muscle group to apply force repeatedly or to sustain a contraction for a certain period of time". For example, if we want to measure the endurance of the abdominal muscles, we could determine how many times these muscles can contract in a given period. Ex.: Bent Knee Sit-ups (one minute)

Muscular endurance is of two types. (i). Static endurance (ii) Dynamic endurance.

Static endurance
The ability to sustain effort in a fixed position.

Dynamic endurance
The ability to sustain effort in movement.

Circulatory-Respiratory Endurance:
Circulatory-respiratory endurance, also called cardiovascular endurance, is characterized by moderate contractions

of large muscle groups for relatively long periods of time, during which maximal adjustments of the circulatory-respiratory system to the activity are necessary, as in distance running and swimming.

Cardio-vascular system is the body's vital transport system as it serves as a carrier through which materials are moved by means of a network of arteries, veins and capillaries. It transports the necessary food materials and oxygen to the cells and serves as the garbage collector, removing waste products from the cells and transferring them to the elimination centres.

Cardio-vascular may be defined "the ability to sustain a series of repetitions of an activity without unduly taxing the physiological systems that furnish the fuel and oxygen to the muscles". In other words, "Endurance is the result of a physiologic capacity of the individual to sustain movement over a period of time".

Cardio vascular fitness has been defined as "the ability of the organism to maintain the various internal equilibria within the body as closely as possible to the resting state during submaximal task and to restore promptly after exercise and equilibria which have been disturbed". According to this definition, a fit person will adapt more efficiently to the stress improved by a specific task and will recover much faster following the task.

There are various types of tests are there to measure cardiovascular endurance. Some important among them are as follows:

1. One and one-half mile run test
2. 12 - Minute Run Test
3. Havard Step Test

Muscular Power

Muscular power is the ability to release maximum muscular force in an explosive manner, that is, in the shortest possible time. Muscular power is also referred as "the ability to transfer energy into force at a fast rate of speed" in other words, the capacity of the individual to bring into play maximum muscle contraction at the fastest rate of speed. Example: Standing Broad Jump.

Agility:

Speed in changing body positions or in changing directions or "The ability to change the entire position of the body in space". In other words, "the ability of the body or parts of the body to change directions rapidly and accurately". Examples: squat thrusts; dodging run.

263. Yobu, Loc.cit
264. Harrison, H. Loc.cit
265. Yobu, Loc.cit
Speed:
Rapidity with which a movement or successive movements of the same kind may be performed or "the ability to perform a movement in a short period of time". In other words, "speed may be defined as the capacity of the individual to perform successive movements of the same pattern at a fast rate".
Example: 50 yard-dash.

Flexibility:
Flexibility is the range of movement in a joint or sequence of joints or "The ability of a joint to move with 100% of its possible movement range".

Flexibility is concerned with the range of movements in a joint. In limits the degrees to which some parts of the body can bend, twist or move by a mean of flexion and extension of muscles. It also depends upon the ligaments that surround the joints. Flexibility varies from one joint to another.

Adequate flexibility of joint can contribute to increased work performance, increased resistance to muscle injury and soreness and general state of good health.

Flexibility is classified as (a) Extend flexibility (b) Dynamic flexibility. Extend flexibility concerns to extend joint motion or movement as far as possible. Dynamic

266. Harrison, H. Op.cit
268. Harrison, H. Loc.cit
flexibility is concerned with the speed in flexing in extend movement.
Examples; Knee flexion-extension movement; touching fingers to floor without bending knees.

World No-Tobacco Days

As a tool for public information and education WHO coordinates the worldwide celebration of the World No-Tobacco Days held on 31st May each year. Originally, this day was for appealing to all those who smoke and to those who chew tobacco to quit for at least 24 hours, as a first step to breaking their addiction to a harmful and wasteful habit.

Currently, World No-Tobacco Days are also seen as opportunities to call for action in the tobacco or health area and also to initiate research on specific themes and subsequently disseminate information. WHO has designated some of the years as follows;

1992 : "Tobacco-free workplaces"
1993 : "Health services, including health personnel, against tobacco"
1994 : "The media against tobacco"
1995 : "The economics of tobacco"
1996 : "Sports and the arts without tobacco"
1997 : "The United Nations and specialized agencies against tobacco"
