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The physiological effects of smoking on the respiratory system

Active and passive smoking damages the lungs. There are thousands of toxic substances found in tar and as gases in the vapour phase of cigarette smoke. Dawn Milner explains how smoking damages the respiratory system with a focus on the lungs

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Knowledge about the harmful effects of smoking on the lungs is high among the general population. This damage can occur to both active and passive smokers. Although scientific understanding in this area is incomplete we do have some knowledge about how individual toxic substances in cigarette smoke act on the lung tissues. There is great variation in smokers' susceptibility to the harmful effects of tobacco dependence. The reasons for this are complex and the role of genetic factors requires more research.

Smoking causes fatal diseases to develop in many parts of the body including cancers of the upper and lower respiratory tracts (mouth, nasopharynx, larynx, and lung), the oesophagus, and the kidney.

Smoking also increases the risk of cardiovascular disease, aortic aneurysm, Crohn's disease, gastric and duodenal ulcers, cataracts, and age-related macular degeneration (causing a loss of central vision).

The two most common respiratory diseases caused by smoking are lung cancer and chronic obstructive pulmonary disease (COPD). In western countries smoking is the major risk factor for these two diseases. According to a study of male British doctors between 1951 and 1991, smoking caused 81 per cent of lung cancer deaths and 78 per cent of deaths from COPD (Doll et al, 1994).

The exposure of infants and children to passive smoking increases the risk of wheezing, the severity and frequency of asthma attacks, cough, and lower respiratory tract infections including bronchitis and pneumonia. It also increases the risk of acute and chronic middle-ear disease in children (World Health Organization, 1999).

A considerable body of evidence links passive smoke exposure in adults with increased risk of lung cancer and ischaemic heart disease (Poswillo, 1998). Smoking precipitates asthma attacks and increases their severity. New evidence indicates that the onset of asthma in adults may be induced by passive smoke exposure (Jaakkola et al, 2003).

Toxic components of tobacco smoke To assist our understanding of the lung diseases associated with smoking we should first look at the toxic components of cigarettes. When the tobacco leaf is burnt, the smoker is exposed to over 4,000 chemicals (see the Action on Smoking and Health website at www.ash.org.uk).

A general search on the internet using the phrase 'smoke constituents' will lead to websites that provide a detailed list of these groups of substances and their concentrations in a single cigarette.

Tobacco smoke contains chemicals in the form of particulate substances and gases. Smoke is arbitrarily divided into gas and particulate phases. A number of the substances found in tobacco smoke are known human carcinogens (substances that can cause cancer) (Box 1).

Most of the carcinogenic components are in the particulate phase that forms the tar. The introduction of low-tar cigarettes has been a response to this finding but may not necessarily reduce the inhalation of carcinogens (Box 2). Many of the constituents of the gas phase of smoking are also very damaging to the respiratory tract when inhaled (Box 3).

Tobacco smoke is made up of side-stream smoke from the burning tip of the cigarette and main-stream smoke from the filter or mouth end. Many of the toxins are present in higher concentrations in side-stream rather than main-stream smoke.

Cigarettes also contain additives that are used for a range of purposes. For example, humectants produce a smoother smoke and impart the distinctive flavours of

BOX 1. EXAMPLES OF KNOWN HUMAN CARCINOGENS FOUND IN CIGARETTE SMOKE

- Aromatic amines (2-naphthylamine and 4-aminobiphenyl)
- Benzene
- Vinyl chloride
- Ethylene oxide
- Arsenic
- Nickel
- Chromium
- Cadmium
- Radioactive element (polonium-210)

There are a number of substances that are considered to be significant carcinogens in animals and possibly or probably carcinogenic in humans:

- Benzo(a)pyrene
- Nitrosamines
- Aldehydes
- Acrylonitrile
- DDT
- Cobalt
- Lead

BOX 2. LOW-TAR CIGARETTES

Exposure to tar is not reduced by smoking low-tar cigarettes. Smokers are known to 'compensate' when they change from high-tar to low-tar cigarettes. This compensation is driven by the need to achieve a required nicotine intake. Smokers do this subconsciously by taking deeper and more frequent puffs, by smoking more of the cigarette, and by covering the ventilation holes on the filter with the mouth, fingers, or saliva. Some smokers have even been known to tape up the filter to achieve a 'stronger' smoke.

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BOX 3. SOME OF THE CONSTITUENTS OF THE GAS PHASE OF CIGARETTE SMOKING

- **Acetone**
- **Acrolein** – this is ciliatoxic (the minute hair-like structures [cilia] of the lining of the upper airways of the respiratory tract are paralysed and can no longer sweep particulate matter up the airways and away from the lungs)
- **Ammonia** – this irritates the respiratory tract
- **Carbon monoxide** – binds with haemoglobin to produce the stable compound carboxyhaemoglobin. This reduces the blood's oxygen-carrying capacity, inhibits respiration, and induces atherosclerosis (hardening of arteries)
- **Carbon dioxide**
- **Nitrogen**
- **Hydrogen**
- **Methane**
- **Nitrogen oxides** – these cause lung inflammation
- **The polycyclic aromatic hydrocarbons**
- **Phenol**
- **Hydrogen cyanide** – this is ciliatoxic
- **Hydrogen sulphide** – irritant to the respiratory tract and is ciliatoxic
- **Volatile nitriles**
- **Toluene**
- **Formaldehyde**
- **Benzene**

Nicotine is found in the particulate phase of tobacco smoke but increasingly enters the gas phase as the pH of the smoke becomes more

particular brands. A link to a complete list of about 600 additives permitted for use in UK cigarettes is available on the ASH website (*www.ash.org.uk).*

There are concerns that certain additives may increase the addictiveness of the product and others may make smoke easier to inhale.

For example, nicotine present in the gas phase of cigarette smoke is more readily absorbed than from the particulate phase. There are concerns that tobacco additives increase the proportion of nicotine in the gas phase and this results in its greater absorption during smoking. The effect would be to increase the addictiveness of the product.

ASH notes that 'additives are regulated with minimal concern for public health, and with no attempt made to assess whether the additive is making the bad problem of tobacco consumption worse' (*www.ash.org.uk*).

Mechanisms of clinical damage Constituents of tobacco smoke cause damage throughout the respiratory tree from the main airways (bronchi) to the peripheral airways (bronchioles), right down to the terminal alveoli (air pockets), as well as to the immune system. Loss of cilia and mucous gland hypertrophy occur in the upper airways; inflammation, epithelial changes, fibrosis and secretory congestion occur in the peripheral airways, and alveoli are destroyed with loss of gas exchange surface area and airways flexibility.

There are vascular changes to the small arteries and capillaries of the bronchioles and the alveoli. Smoke also causes inflammation of the cells of the bronchial tree leading to squamous metaplasia (a precancerous condition), smooth muscle hypertrophy, and peribronchial fibrosis.

Damage is evident in the results of bronchoalveolar lavage (a fibreoptic scope is placed into the lung of a patient, and sterile water is injected into the lung). The overall cell count in the lavage is increased with people who smoke, with many more neutrophils and eosinophils but fewer lymphocytes. Concentrations of the antibodies immunoglobulin M and immunoglobulin E (markers of sensitisation) are increased, showing that allergic processes are involved.

Lung disease associated with smoking

Chronic obstructive pulmonary disease COPD is characterised by airflow obstruction. This obstruction is usually progressive, not fully reversible, and does not change markedly over several months (National Institute for Clinical Excellence, 2004).

The changes induced by the irritant tobacco smoke produce the recognisable symptoms including:

- Productive and persistent cough;
- Regular chest infections requiring antibiotics;
- Shortness of breath at first on exercise, later after simple non-strenuous activities, and finally at rest.

This disease has a very gradual onset and should be suspected in people aged over 35 who smoke, have a chronic productive cough with winter chest infections, and are breathless on exertion.

Patients do not generally present with symptoms until their 50s or 60s after many years of smoking. Spirometry is the recommended way of assessing airflow limitation.

The patient will have a reduced FEV_1 and FEV_1/FVC ratio. FEV_1 is the forced expiratory volume in one second and FVC is forced vital capacity.

A landmark study on COPD was published in 1977 (Fletcher and Peto, 1977). The authors demonstrated the key features of the disease including a wide range of susceptibility among smokers. They identified that stopping smoking was the only way to slow down the progressive decline of lung function.

The disease is most prevalent in socioeconomically

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deprived people, a group that also has the highest prevalence of smoking.

Genetic factors are thought to play a part in the different susceptibilities to COPD between smokers. Deficiency of alpha-1 antitrypsin has been shown to be a risk factor but more studies are needed to find other genetic links.

It is important to note that there are some other atmospheric pollutants associated with COPD, such as dust from cotton, grain, coal, and cement, as well as oil and cadmium fumes.

The National Institute for Clinical Excellence has recently produced guidelines on the management of COPD (NICE, 2004).

It is hoped that these guidelines will lead to earlier diagnosis and less variable standards of care. Earlier diagnosis of COPD and referral to NHS smoking cessation services are key to reducing the burden of this disease (see p48).

Asthma For some time it has been known that passive exposure to tobacco smoke increases the frequency and severity of asthma attacks in children and adults. A recent study has demonstrated for the first time that passive tobacco smoke inhalation increases the risk of developing asthma in adults (Jaakkola, 2003).

Not surprisingly cigarette smoking has a harmful effect on the lung capacity of people with asthma, which is demonstrated by impaired lung function tests (Althius et al, 1999). People with asthma should be advised not to start smoking or be given strong encouragement and support to quit.

Pneumonia The risk of pneumonia is increased by the irritant effect of smoke inhalation accompanied by mucous gland hypertrophy and damage to the immune system. Pneumonia is not only more common among smokers but is also much more likely to be fatal. In 1995, a total of 9,900 deaths from pneumonia were attributed to smoking (Health Education Authority, 1998).

Lung cancer The risk of dying from lung cancer rises with the number of cigarettes smoked per day, although duration of smoking is the strongest determinant.

In 1999, lung cancer was the most common form of cancer, accounting for 22 per cent (34,240 people) of all cancer deaths (Cancer Research Campaign, 2001). Over 80 per cent of all lung-cancer deaths are caused by smoking.

Mortality from lung cancer in men fell from about 880 deaths per million of the population in 1990 to 628 per million in 1999, continuing the downward trend that began in the 1970s. This reflects the fall in tobacco consumption in the male population.

The female mortality rate from lung cancer is still about half that of the male: 301 deaths per million in 1999. This rate has remained stable throughout the 1990s (CRC, 2001). In total, 95 per cent of patients die within five years of diagnosis, with most dying within the first year (CRC, 2001).

Factors contributing to lung cancer Inhaled carcinogens from cigarette smoking play a major part in the development of all lung cancer (squamous cell, adenocarcinoma, small cell, and undifferentiated carcinomas) but lifestyle and genetic factors are also important. This is a highly complex area and understanding could be said to be in its infancy.

Since the 1950s there has been a shift towards a relative increase of adenocarcinoma over squamous carcinoma. It is thought to be due to reduced polynuclear aromatic hydrocarbons (PAHs) from filtered cigarettes or the deeper inhalations needed by smokers who compensate for lower nicotine yields from filtered cigarettes (Thun et al, 1997).

Unfortunately adenocarcinoma is less easy to treat than squamous carcinoma.

When smokers give up, their risk of getting lung cancer starts decreasing so that after 10 years an ex-smoker's risk is about one-third to one-half that of those who continue to smoke.

Smoking cessation and respiratory symptoms A recent study from the Netherlands reviewed the evidence for the impact of smoking cessation on the lungs. It confirmed that giving up smoking prevents further deterioration of lung function and results in an improvement in inflammation of the airways in smokers without respiratory symptoms.

This improvement in inflammation is not seen in those with bronchitis or COPD although giving up smoking prevents an excessive decline in lung function in all groups of smokers (Willemse et al, 2004).

The researchers concluded that more research is needed on how smoking cessation affects the lungs of people with COPD.

Conclusion Nurses have an important role to play in helping patients to understand the health implications of starting smoking or failing to stop. Understanding the pathophysiological processes that link smoking to ill health can assist practitioners in developing this important part of their health education role. ■