Tobacco in Australia | Facts & Issues
A comprehensive online resource
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The health effects of active smoking

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3.0 **Introduction**

Smoking of tobacco as we know it today, in the form of manufactured or ‘factory-made’ cigarettes, became common in Australia in the late 1800s. Pipe and cigar smoking was already widespread among men, but the convenience and ready availability of the cigarette soon made it a popular alternative.1 Manufactured cigarettes were supplied to Australians and their allies in the trenches of World War I,1 and by the end of World War II, nearly three quarters of Australian men and one quarter of women were smokers, the majority using cigarettes (see also Chapter 1, Section 1.1).

Similar changes in smoking behaviour had occurred in Western Europe and North America, and with them, a marked escalation in lung cancer death rates and the growing suspicion that cigarette use was implicated in this trend. By 1950 several studies had been published in the medical literature2 and the finding that cigarette smoking and lung cancer appeared to be causally linked was reported.3,4 Several series of authoritative, landmark reports have since been published by national and international agencies,1 documenting the damaging effects of smoking and calling for action to help halt the smoking epidemic. Of these, the most regular series has been that issued by the Office of the US Surgeon General. Since 1964, comprehensive and rigorous reports on various aspects of tobacco and health have been issued by the US Surgeon General, repeating the conclusion that smoking is “the single greatest cause of avoidable morbidity and mortality in the United States,” and never finding reason to reverse any earlier conclusions of causality.5

The 2004 report of the US Surgeon General, *The Health Consequences of Smoking*, published the following four major conclusions:6

1. Smoking harms nearly every organ of the body, causing many diseases and reducing the health of smokers in general.
2. Quitting smoking has immediate as well as long-term benefits, reducing risks for diseases caused by smoking and improving health in general.
3. Smoking cigarettes with lower machine-measured yields of tar and nicotine provides no clear benefit to health.
4. The list of diseases caused by smoking has been expanded to include abdominal aortic aneurysm, acute myeloid leukaemia, cataract, cervical cancer, kidney cancer, pancreatic cancer, pneumonia, periodontitis and stomach cancer.

In addition, the 2014 report of the US Surgeon General, *The Health Consequences of Smoking – 50 Years of Progress*, published these major conclusions in regard to the health effects of smoking:7

1. The century-long epidemic of cigarette smoking has caused an enormous avoidable public health tragedy. Since the first Surgeon General’s report in 1964 more than 20 million premature deaths [in the U.S] can be attributed to cigarette smoking.
2. The tobacco epidemic was initiated and has been sustained by the aggressive strategies of the
tobacco industry, which has deliberately misled the public on the risks of smoking cigarettes.

3. Even 50 years after the first Surgeon General’s report, research continues to newly identify diseases
caused by smoking, including such common diseases as diabetes mellitus, rheumatoid arthritis, and
colorectal cancer.

4. In addition to causing multiple diseases, cigarette smoking has many other adverse effects on the
body, such as causing inflammation and impairing immune function.

5. The burden of death and disease from tobacco use in the United States is overwhelmingly caused by
cigarettes and other combusted tobacco products; rapid elimination of their use will dramatically
reduce this burden.

3.0.1 Defining causality

The US Surgeon General’s reports have provided a detailed review of definitions of causality of disease, and
how measures of causality may be applied. Causality is determined by evaluating the range of available
evidence and considering it against well-established criteria. The more that an observed association fulfils
the criteria, the more likely it is that a causal relationship can be inferred. These criteria are outlined in the
US Surgeon General’s Report for 2004.8

Consistency: This refers to the persistence of the finding of an association between exposure and outcome
in a number of methodologically valid studies undertaken in a range of settings. This helps ensure that
possible confounding effects are eliminated, and also increases the statistical validity of the finding through
the accumulation of additional evidence.

Strength of association: Strength refers both to magnitude of the association, and to its statistical strength.
The greater the measured association and the more sound its statistical basis, the less likely it is that the
findings are influenced by chance, bias, or unmeasured or poorly controlled confounding factors. However
the observed association must also have a plausible basis in understood biological processes.

Specificity: Specificity refers to the degree to which exposure to the suspected disease causing agent can
predict outcome. Other biological and epidemiological factors may need to be taken into account. For
example, not all smokers develop lung cancer, and not all cases of lung cancer are caused by smoking.
However, the extremely high relative risk for lung cancer in smokers, and the high percentage of lung
cancers attributable to smoking, gives the association between smoking and lung cancer “a high degree of
specificity”.

Temporality: Exposure to the causative factor must precede the onset of the disease. Considered alone,
temporality is a poor predictor of causality, but no association can be considered to fulfil the criteria for
causality if temporality is not satisfied.

Coherence, Plausibility and Analogy: Taken together, these three criteria require that the proposed causal
relationship must not defy known scientific principles, and that it must be biologically plausible and consistent
with experimentally demonstrated biological mechanisms and other relevant patterns.

Biologic Gradient (Dose-Response): This criterion refers to the observation of increased effect (for example
incidence of disease) in response to increased dose (heavier and/or longer duration of smoking). Meeting
this criterion forms a strong support for causality, except in the unlikely event that there is an unidentified
confounder, which happens to be varying in the same manner as the observed dose and which could
account for the measured association. Virtually all health outcomes causally linked to smoking have
demonstrated a dose-response relationship of some description.

Experiment: This criterion refers to naturally occurring “experiments” that might be considered to imitate the
conditions of a properly conducted experiment in a scientific environment, and whose outcomes might have
the force of a true experiment. An example of a ‘natural experiment’ in the smoking arena is assessing the
health consequences of quitting smoking. To attribute observed improvements in health outcomes to factors
other than smoking cessation would necessitate identifying alternative influences and demonstrating that
those who continued smoking had also attained a health benefit where that alternative influence was
present.
The more closely an association fulfils the above criteria, the stronger its claim to causality. Not all inferences of causality will necessarily satisfy all criteria. For example where biological mechanisms may not be completely understood, causality may still be justified by satisfaction of other criteria, such as consistency and strength of association. Those applying the criteria must weigh the all of the scientific evidence and make a multidisciplinary judgement.  

### 3.0.2 Tobacco—a leading preventable cause of death and disease

Smoking is one of the leading preventable causes of death and disease in Australia, responsible for about 15,000 deaths annually. In 2003, tobacco caused more than 1 in every 10 deaths in Australia, and taking into consideration sickness and disability as well as deaths, tobacco caused more disease and injury in Australia than any other single risk factor (Table 3.1). Tobacco is also responsible for most (90%) of all drug-caused deaths. In 2004-05, smoking caused 14 times as many deaths as alcohol, and 17 times the number of deaths due to illicit drug use. (see Table 3.0.1).

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Number of deaths</th>
<th>Percentage of total deaths from all causes</th>
<th>Percentage of total burden of disease and injury</th>
</tr>
</thead>
<tbody>
<tr>
<td>High blood pressure</td>
<td>22,504</td>
<td>17.0</td>
<td>7.6</td>
</tr>
<tr>
<td>Tobacco</td>
<td>15,511</td>
<td>11.7</td>
<td>7.8</td>
</tr>
<tr>
<td>High blood cholesterol</td>
<td>15,351</td>
<td>11.6</td>
<td>6.2</td>
</tr>
<tr>
<td>Physical inactivity</td>
<td>13,491</td>
<td>10.2</td>
<td>6.6</td>
</tr>
<tr>
<td>High body mass</td>
<td>9,525</td>
<td>7.2</td>
<td>7.5</td>
</tr>
<tr>
<td>Alcohol*</td>
<td>1,084</td>
<td>0.8</td>
<td>2.3</td>
</tr>
</tbody>
</table>

*Net effects, i.e. offsetting beneficial effects against harmful effects.

Source: compiled from Begg et al.

It has been conservatively estimated that smoking kills about one half of all persistent users. A more recent study puts this figure at closer to two in three. Over the decades, the death toll from tobacco use has been vast. In the 50 years from 1960 to 2010, smoking is estimated to have killed 821,000 Australians. Tobacco use is also responsible for a global pandemic of death and disease, causing nearly six million deaths a year. More than five million of those deaths are caused by direct tobacco use, while more than 600,000 are caused by exposure to second-hand smoke (see Section 3.36).

References


Chapter 3: Health Effects » 3.1 Smoking and heart disease

3.1 Smoking and heart disease

Cardiovascular disease (CVD) covers all disease processes of the heart and blood vessels. In 2007, 46,623 Australians died from CVD, accounting for just over a third of all deaths in that year.\(^1\) CVD is the second largest contributor to the burden of disease in Australia after cancer—estimated at 18% and 19% respectively in 2003.\(^2\)

Premature CVD is highly preventable. Tobacco smoking, raised blood pressure, elevated blood cholesterol, insufficient physical activity, overweight and obesity, poor nutrition, drinking at harmful levels and diabetes are major preventable risk factors for CVD. There is also recognition that socio-economic and psychosocial factors, such as low income, unemployment, depression and social isolation influence the development of CVD. Disease trends in Australia show that CVD impacts most heavily on population groups that suffer socio-economic disadvantage, including Australia's Aboriginal people and Torres Strait Islanders.\(^1,2\)

Cigarette smoking contributes to CVD in a number of ways. Toxic products from cigarette smoke, in particular nicotine and carbon monoxide (CO), circulate in the bloodstream, interfering with the efficient working of the endothelium (the inner cellular layer of the arterial wall), eliciting blood fat abnormalities and impairing glucose regulation. Each effect is implicated in the development of atherosclerotic lesions (collections of cholesterol, fat and other matter) in the arterial walls. These collections narrow the arteries, gradually impairing blood flow, and making the arteries harder, less elastic, and more liable to rupture. The process leading to atherosclerosis—plaque (fatty streaks) deposited within the inner layers of the arteries—is slow and complex, often starting in childhood and progressing with age. Smoking also has a direct effect on platelets (blood cells involved in the clotting process), leading to increased activation and stickiness. This in turn causes an increased risk of thrombosis, or development of blood clots.\(^3\)

Smoking a cigarette also temporarily increases heart rate and blood pressure and also affects the ability of the heart to contract. These circulatory changes result in increased work for the heart muscles, which in turn raises the body's demand for oxygen. At the same time, the body is deprived of oxygen through the effects of CO on reducing transport oxygen. The resulting imbalance in oxygen supply and demand promotes the complications of atherosclerosis. These include ischaemia (lack of oxygen due to poor blood supply), with resultant angina pectoris (chest pain or tightness) or myocardial dysfunction (poor heart muscle function).\(^3,4\)

While nicotine and CO in tobacco smoke are strongly implicated in the processes leading to development of CVD, other chemicals may also be involved.\(^3\) There is now strong evidence that exposure to secondhand cigarette smoke is also a cause of coronary heart disease in non-smokers (see Chapter 4).

3.1.1 Coronary heart disease

Coronary heart disease (CHD), also known as ischaemic heart disease, is the most common form of CVD,
and the most common cause of sudden death in Australia.\textsuperscript{5} It occurs when the arteries supplying the heart become progressively narrowed by a fatty fibrous plaque or atheroma. This reduces the blood flow, forcing the heart to work harder to compensate, and can lead to symptoms of angina. If the plaque breaks up, a blood clot may form, blocking the artery completely. If not promptly treated, this can lead to death of vital heart muscle due to oxygen starvation (termed acute myocardial infarction or heart attack), or, in the worst case, sudden death.\textsuperscript{3}

Smoking is a cause of CHD,\textsuperscript{3,6} increasing the risk of disease incidence by between two- and four-fold, the risk increasing with heaviness of smoking.\textsuperscript{7} Smoking in adolescence and young adulthood is also associated with an increased risk of coronary artery atherosclerosis in adulthood.\textsuperscript{8} Even light smoking significantly increases the risk of dying from CHD, the steepest increase in risk occurring in smokers of up to four cigarettes a day.\textsuperscript{9}

Smokers who have CHD are more likely to die of the disease than non-smokers with the disease. A 2013 study looking at 50-year trends in smoking-related mortality found that although overall death rates from CHD have declined, these declines were proportionately larger for never smokers than for current smokers. The mortality rate was more than three times higher for current smokers compared with never smokers aged between 55 and 74, with two thirds of the deaths among smokers being attributable to their smoking. There was also a significantly increasing risk of CHD mortality for both men and women with increasing consumption through 40 or more cigarettes per day.\textsuperscript{10} The heaviest burden of excess death due to tobacco-caused CHD is felt in early middle age. In Australia in 2004–05, 40% of all deaths due to CHD occurring in males between the ages of 35 and 39 were due to smoking. Among women aged 40–44, smoking caused about 34% of all deaths due to CHD. The effects of active smoking compared with no exposure are likely to be underestimated because most CVD studies have not excluded persons who had secondhand smoke exposure from the comparison group.

Lower tar and nicotine cigarettes have not been shown to reduce the incidence of CHD due to smokers increasing the number of cigarettes smoked per day or by taking deeper, faster, more or longer puffs. Thus, such cigarettes do not provide a lower risk alternative for smokers who cannot or do not wish to quit.\textsuperscript{3}

The risks of myocardial infarction and death from CHD are lower among former smokers than among continuing smokers. The 2010 report of the US Surgeon General states that the risk halves within the first year and takes between 10–14 years before it approaches that of a lifetime non-smoker.\textsuperscript{3} The International Agency for Research on Cancer\textsuperscript{11} describes the benefits of quitting more conservatively:

‘... there is a substantial reduction in risk of CHD compared with that of continuing smokers with the first two to four years of smoking abstinence, followed by a slower decline of risk, with risk approaching that of never smokers in fifteen to twenty years. For methodological reasons, the assessment of risk reduction is problematic within the first two years of cessation.’(p 342)

The International Agency for Research on Cancer also cites a reduced risk of 35% within two to four years for persons already suffering from CHD, and says studies on subjects without diagnosed CHD are ‘compatible with this conclusion and point toward similar relative risk reduction.’\textsuperscript{11}(p 336).

3.1.2 Cerebrovascular disease (stroke)

A stroke occurs when blood flow to the brain is interrupted, leading to injury or death of brain tissue. This occurs most commonly because of arterial blockage caused by atherosclerosis or a blood clot, an event known as an ischaemic stroke. Happening less often, but more likely to be fatal when it does arise, is a haemorrhagic stroke, in which bleeding occurs from a leaking or ruptured arterial wall at a point weakened by atherosclerosis. Sometimes the artery stretches at the site of weakness, causing it to balloon out, forming an aneurysm. The bigger the aneurysm, the more likely it is to rupture, causing haemorrhage and a resultant stroke.

One in five people experiencing their first stroke episode will die within four weeks, and one in three will die within 12 months. Among the people who survive the first month after their first-ever stroke, about half will
survive five years. Stroke is a major cause of disability in Australia. By the end of the first year following a stroke, about half of stroke survivors still require assistance with daily activities.

Smoking is an important cause of stroke, with the risk of having a stroke rising with the amount of tobacco smoked. Smokers are one and a half times more likely to have a stroke than non-smokers.

As with CVD, the impact of stroke caused by tobacco is greatest among the middle aged. In Australia in 2004–05, 40% of all deaths due to stroke in men aged between 35 and 39 were caused by smoking. The greatest impact occurred in women aged 40–44, among whom 35% of all stroke deaths were due to tobacco. Research has shown that the risk of having a stroke decreases steadily after quitting smoking, ex-smokers having the same risk as never-smokers after 5–15 years, depending on the study.

3.1.3 Atherosclerotic peripheral vascular disease

Atherosclerotic peripheral arterial disease (PAD) occurs when blockages within the blood vessels prevent proper blood circulation. PAD most commonly occurs in the legs and feet, but it can also develop in the arms and hands. This may result in severe pain (claudication), especially when physically active. PAD can lead to death of part of the limb. Amputation may be necessary for relief of pain, and to prevent the development of gangrene. Given that it’s the same atherosclerotic disease process, it is not uncommon for individuals with PAD to die from heart attack or stroke.

Smoking is a cause of PAD. There is a strong dose–response relationship between the number of cigarettes smoked and the likelihood of developing PAD even after adjustment for other CVD risk factors. In Australia in 2004–05, about 37% of all deaths due to PAD in males aged over 35 were attributable to smoking, as were 30% of all PAD deaths in women aged over 35.

Patients who suffer vascular-related leg pain are less likely to suffer serious obstruction of the arteries in their legs if they quit. Quitting smoking also reduces the risk of re-occlusion after peripheral vascular surgery.

3.1.4 Abdominal aortic aneurysm

Abdominal aortic aneurysm is a weakening of the wall of the aorta (the major artery carrying oxygenated blood from the heart to the body) in the region below the diaphragm. The weakening occurs as a result of atherosclerotic lesions developing in the aortic wall. The wall may eventually stretch and then leak or burst. Abdominal aortic aneurysm is frequently fatal.

Smoking is a cause of abdominal aortic aneurysm, the risk rising with increased exposure to tobacco smoke. Further, active smoking in adolescence and young adulthood can cause early abdominal aortic atherosclerosis in young adults. Smoking is one of the few currently modifiable risk factors for this disease. With increasing time after stopping smoking, the risk of developing an abdominal aneurysm appears to slowly decline.

3.1.5 Sudden cardiac death

Sudden cardiac death describes death occurring due to sudden, unexpected loss of heart function. Most sudden death is due to CVD, in particular CHD accompanied by smoking. Cardiac dysrhythmias (irregular muscular contractions of the heart, also referred to as cardiac arrhythmias) also cause sudden cardiac death. Smokers have a three-fold greater risk of suffering sudden cardiac death than non-smokers. Importantly, cigarette smoking may be the only modifiable risk factor for sudden cardiac death in the presence or absence of CHD.

In Australia it is estimated that smoking causes 30–40% of all deaths due to cardiac dysrhythmias in men aged 35–59, and about one-third of all deaths due to cardiac dysrhythmias in women aged 35–44.
3.1.6 Congestive heart failure

Congestive heart failure (CHF) occurs when the heart becomes less able to pump blood through the body effectively. The heart may become enlarged or thicken, and fluid may collect in lungs (causing shortness of breath) or in other parts of the body (causing swelling or weight gain). CHF usually occurs in individuals with a history of heart problems such as high blood pressure or coronary heart disease. As well as contributing to the disease processes that primarily lead to CHF, smoking is an independent risk factor for CHF.

CHF sufferers experience high levels of disability and have a reduced life expectancy. In Australia, it is estimated that smoking causes 30–40% of all deaths due to CHF in men aged 35–59, and about one-third of all deaths due to CHF in women aged 35–44.

In conclusion, cigarette smoking and exposure to secondhand cigarette smoke are major causes of CVD. The risk arises from atheroma narrowing the affected blood vessels and with a higher risk of acute thrombosis. The cardiovascular risks attributable to cigarette smoking escalate with smoking just a few cigarettes per day, low level exposure to secondhand cigarette smoke and the duration of smoking. Stopping cigarette smoking and eliminating exposure to secondhand cigarette smoke rapidly and substantially reduces the risks of various CVDs.

References


8. US Department of Health and Human Services. Preventing tobacco use among young people: A report of


Chapter 3: Health Effects

3.2 Respiratory diseases (excluding lung cancer)

The airways and lungs, being the route of tobacco smoke exposure, are exposed to higher concentrations of the toxic constituents of smoke than any other system in the body. The adverse effects of smoking range from impairment of the protective mechanisms in the lungs that reduce the risk of infection to actual lung destruction.\(^1,2\)

### 3.2.1 Impairment of pulmonary immune and protective responses

The lungs are continually exposed to gases, particles and micro-organisms in the air. To avoid the ill effects from these potential insults, the respiratory system employs a set of protective mechanisms. Cigarette smoke can impair or overwhelm the lungs’ defences, leading to chronic disease.\(^1\)

Every time we breathe, we inhale particles. Some are harmless dust and others are potentially injurious particles, viruses, or bacteria. Large and very small particles are mostly trapped in the nose and upper airway and cleared without reaching the lung at all. Intermediate sized particles, between 0.001 and 10 microns, penetrate deep into the lungs.\(^1\) From here they are cleared by the mucociliary system: particles become trapped in the mucus blanket on the surface of the cells lining the airways, and are swept out of the lung by the synchronised movement of cilia, which are tiny hair-like structures on the surface of airway cells.\(^3\)

About 60\% of the particles from cigarette smoke are deposited in the lung. Exposure to cigarette smoke reduces the clearance rate of particles from the lung.\(^1\) This is in part due to shortening, loss or discoordination of cilia, but may also be due to changes in the thickness of mucus that reduces the effective propulsion of the mucus by the cilia.\(^3-6\) This impairment of the mucociliary system increases the risk of infection.\(^1\) Smokers also become increasingly reliant on coughing to clear mucus, rather than the normal clearance process which is more effective and less irritating.\(^7\) Long-term smokers retain a substantial amount of particles in their lungs.\(^5\) Smoking cessation improves mucociliary clearance in the nose after two weeks, and in the lungs after three months.\(^4,8\)

Lying under the mucociliary system, the outer layer of lung cells lining the airway form a physical barrier between lung tissue and airspace. Chronic exposure to cigarette smoke damages this protective barrier, increasing its permeability, which leads to inflammation.\(^1,9,10\)

Smoking compromises the immune system, provoking an inflammatory response and increasing the potential for infection.\(^1,9,11\) The ability of the lung’s immune system to sense and eliminate viruses and bacteria is impaired.\(^10,12\) All smokers have inflammation in their lungs, which may persist for many years after smoking cessation.\(^1,13\)
3.2.2 The effect of smoking on acute respiratory illnesses

Adults who smoke are more likely to develop acute respiratory illnesses, including bronchitis, bronchiolitis, influenza, legionnaires disease, and pneumonia. The risk of pneumococcal infection, the most common cause of severe pneumonia, is two- to four-fold in smokers compared to non-smokers, with the risk increasing as daily cigarette consumption increases. Smokers are more likely to be infected with influenza in an epidemic. Seasonal influenza is more common and severe in non-vaccinated smokers.

Smoking also causes active tuberculosis disease and death from tuberculosis. Tuberculosis is not common in Australia, but there are groups vulnerable to TB infection including Aboriginal Australians, migrants from countries where TB is common and people with HIV. Smoking cessation and avoiding secondhand smoke reduces the risk of tuberculosis disease.

In Australia in 2004–5, it is estimated that about 15% of all deaths due to lower respiratory tract infection in men aged over 35, and 12% in women of the same age, were caused by smoking.

3.2.3 Smoking and respiratory symptoms

Active smoking causes respiratory symptoms in adults, teenagers and children, including coughing, phlegm, wheezing and dyspnea (difficulty breathing and shortness of breath). These symptoms are associated with a number of acute and chronic respiratory illnesses. They may also indicate underlying lung injury and disease. The population prevalence of these symptoms decreases with smoking cessation.

3.2.4 Smoking and lung function

Active smokers in childhood and adolescence have both reduced lung function and impaired lung growth. Smoking causes the early onset of decline in lung function during late adolescence and early adulthood. All adults experience a loss of lung function as they age, but this process occurs earlier and at a greater rate among smokers than non-smokers. Among smokers, there appears to be a sliding scale of susceptibility to loss of lung function. A few smokers may lose lung function almost as slowly as non-smokers, but for a significant minority of smokers, their rapid loss of lung function becomes disabling or fatal. Most smokers will fall between these groups. A diagnosis of chronic obstructive lung disease (COPD) can be made after a significant and non-reversible loss of lung function. In the population of smokers without COPD, the age-related rate of lung function decline slows down to that seen in people who have never smoked within five years of smoking cessation. However, they do not regain the lung function they have already lost.

3.2.5 Major diseases caused by smoking

3.2.5.1 Chronic bronchitis

Bronchitis is defined by symptoms of cough together with frequent and increased production of sputum or phlegm. Chronic bronchitis is diagnosed when these symptoms are present for three months in each of two successive years. It occurs in about half of all heavy smokers. Chronic bronchitis is associated with inflammation in the large and small bronchial airways, which results in the enlargement of mucus-producing glands and remodelling (thickening) of the airway walls. People with chronic bronchitis have a greater frequency of respiratory infections. In persons who also have chronic obstructive pulmonary disease (COPD), symptoms of chronic bronchitis increase the risk of death from respiratory infections.

Chronic bronchitis often co-occurs with COPD, but it does not influence airflow limitation unless the inflammation extends into the small airways. Having symptoms of chronic bronchitis is associated with an
accelerated decline in lung function as seen in COPD.\textsuperscript{1,12} It was previously thought that chronic bronchitis was a necessary first step in the development of COPD. However, since then research has shown that airflow limitation can develop without symptoms of chronic bronchitis.\textsuperscript{1} Also, in people with normal lung function, the presence of chronic bronchitis does not increase their likelihood of developing COPD.\textsuperscript{1,13}

Symptoms of chronic bronchitis decrease by one to two months after smoking cessation, and the population prevalence of cough and phlegm returns to the level of never smokers within five years.\textsuperscript{13} In people with severe COPD, chronic cough associated with chronic bronchitis is more likely to persist after smoking cessation.\textsuperscript{23}

3.2.5.2 Chronic Obstructive Pulmonary Disease (COPD)

Chronic obstructive pulmonary disease (COPD) is characterised by airflow limitation that is usually progressive and not fully reversible.\textsuperscript{1} In 2011, COPD was the fifth leading cause of death in Australia, representing 4.4% of all deaths in people aged 55 and over.\textsuperscript{25} These statistics may underestimate the contribution of highly prevalent, slowly progressive, chronic diseases such as COPD to mortality, as the reported number of deaths is based on the underlying cause of death only. In Australia, only 40% of all deaths with COPD as any cause had COPD recorded as the underlying cause of death for the period 2007–2011.\textsuperscript{25}

Death rates from COPD have declined over time.\textsuperscript{25} COPD is more prevalent among the elderly, when it has important interactions with many other acute and chronic illnesses.\textsuperscript{17,26} The evidence also suggests that women may be more susceptible to developing severe COPD at younger ages.\textsuperscript{11} Beyond the effect on mortality, the chronic nature of COPD means those who develop COPD may live for many years, with various degrees of discomfort and disability.\textsuperscript{17} Even individuals with mild COPD have reduced quality of life, which worsens as the disease becomes more severe.\textsuperscript{27}

The greatest cause of COPD by far is smoking.\textsuperscript{25} In 2004–5, it is estimated that of all deaths in Australians aged over 35 caused by COPD, 77% of cases in males and 71% of cases in females were attributable to smoking.\textsuperscript{19} In 2007-08, among Australians aged 55 years and over who had self-reported COPD, 20% were current smokers, 52% were former smokers, and 28% had never smoked.\textsuperscript{28} Limited data suggests that, of the current smokers who survive to their mid-70s, around half may develop mild to severe COPD.\textsuperscript{29}

COPD arises from progressive, permanent damage to the airways and airway sacs (alveoli) of the lungs. The main processes thought to be important in the development of COPD are inflammation, oxidative stress, and an imbalance of proteases (enzymes that affect proteins) and antiproteases in the lung. Oxidative stress is the result of highly reactive chemicals in tobacco smoke creating an imbalance between oxidants and antioxidants in the lung. Oxidative stress can directly damage lung cells, promote inflammation, and contribute to the protease-antiprotease imbalance. While all smokers have inflammation of the lungs, not all develop COPD. People who develop COPD are thought to have an enhanced or abnormal inflammatory response to noxious particles or gases.\textsuperscript{1,13}

Different disease processes result in the airflow limitation that characterises COPD. The main diseases are obstructive bronchiolitis and emphysema.\textsuperscript{1,13} Chronic bronchitis often co-occurs with COPD (as described above). Smokers have different susceptibilities to each disease process and this will influence their symptoms.

**Obstructive bronchiolitis**

Inflammation in the small airways is seen to some extent in all smokers.\textsuperscript{12,30} Obstruction of the small airways occurs when abnormally heightened inflammation and remodelling occur in the small bronchi and bronchioles in the lungs. The term 'remodelling' describes a cycle of injury and repair in the presence of inflammation, that results in the thickening of the airway wall and narrowing of the lumen (airway space).\textsuperscript{1} In addition, excess mucus accumulates in the small airway lumen.\textsuperscript{9} This process obstructs air flow through the small airways to the lung's air sacs (alveoli) where gas exchange occurs. For as long as smoking continues,
the condition progresses. The main symptom is breathlessness, because the gradually altered lung structure cannot allow increases in the flow of air that is needed to exercise comfortably. Smoking cessation slows lung function decline. In some smokers, airway inflammation persists, possibly for life, after stopping smoking.

**Emphysema**

Emphysema is irreversible loss of the walls of the alveoli—the small air sacs where gas exchange occurs. As this framework is lost, the alveoli walls cannot regenerate and air spaces enlarge. The resulting loss of the surface area where gas exchange occurs reduces the capacity of the lungs to transfer oxygen to red blood cells and remove carbon dioxide from the bloodstream—its essential functions.

Smoking causes oxidative stress, which tips the protease–anti-protease balance towards proteases. Proteases are enzymes that degrade structural proteins, such as elastin and collagen, in the lungs airways and alveoli. The lung becomes less elastic, restricting its capacity to contract and expand. The loss of elastic recoil reduces the force driving the air out the lungs, so it takes longer to breathe out. In advanced emphysema, the inelastic lungs enlarge leading to a large barrel-shaped chest.

**Course of COPD after smoking cessation**

Smoking cessation is the only action known to protect from rapid declines in lung function. In populations with COPD, there is a small improvement in lung function in the year after smoking cessation. Thereafter, age-related decline in lung function that is less than half of that seen in continuing smokers. Reduction in the number of cigarettes smoked does not change the loss of lung function. Former smokers have a reduced risk of hospitalisation related to COPD and death from COPD compared with those who continue to smoke.

**3.2.6 Other respiratory illnesses related to smoking**

**3.2.6.1 Asthma**

In Australia, one in ten people suffer from asthma. Smoking rates in people with asthma are at least as high as those without asthma. Active smoking is associated with an increased risk for asthma in adolescents and adults. Smoking exacerbates asthma in adults and research suggests that it may also do so in children and adolescents. Smoking increases asthma symptoms and impairs the response to asthma treatment. People with asthma who smoke are more likely to have accelerated loss of lung function. The risk of severe asthma events, such as hospitalisation, use of emergency services and death, are increased in smokers. Smoking cessation improves asthma control.

**3.2.6.2 Interstitial Lung Diseases (ILD)**

Idiopathic pulmonary fibrosis (IPF) is a progressive, fatal fibrotic interstitial lung disease. It has the worst prognosis of all idiopathic interstitial diseases of the lung, with a median survival time of three to four years. Although its cause is unknown, evidence suggests that both genetic and environmental factors are involved in its development. The latest Surgeon General's report concluded that cigarette smoking is associated with an increased risk of IPF.

Respiratory bronchiolitis-interstitial lung disease (RB-ILD) is seen in very heavy smokers, typically those smoking more than 30 cigarettes per day. Unlike typical COPD, it can be seen in young smokers. RB-ILD is a greatly exaggerated form of bronchiolitis that spreads to create inflammation in the nearby alveoli. RB-ILD impairs lung function and has an abnormal appearance on chest X-rays. Smoking cessation is recommended.
3.2.6.3 Histiocytosis X (Langerhan's Cell Histiocytosis)

Histiocytosis X is rarer than RB-ILD. It involves the development of inflammatory nodules in the lung along with cystic degeneration of the lungs themselves. It has a distinct appearance on X-rays. Patients are commonly young adults and the vast majority have a history of smoking.\(^\text{42}\) Smoking cessation is strongly encouraged, because case reports show improvement after quitting, and even restoration of normal or near-normal lung structure.\(^\text{42,40}\)

3.2.7 Other respiratory illnesses related to smoking

3.2.7.1 Sense of smell

Being a smoker is associated with having an impaired sense of smell (hyposmia).\(^\text{45,46}\) Smoke directly damages the olfactory sensory neurons, located in the nasal airways, which detect different odours.\(^\text{45}\) Smokers are about twice as likely to have olfactory impairment compared to non-smokers. Following quitting, sense of smell is restored to levels of a never smoker.\(^\text{46,47}\) (Also see section 3.22.5)

3.2.7.2 Snoring

Snoring is more common in smokers and former smokers than in never smokers. Frequency of snoring increases with the amount of tobacco smoked, and is independent of obesity, another well-established risk factor for snoring. Snoring is likely to occur in response to the effects of tobacco smoke on the airways, including upper airway inflammation, cough and sputum production.\(^\text{48}\) (Also see section 3.22.4)

References


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Two expert bodies, the International Agency for Research on Cancer (IARC) and the US Surgeon General’s office, periodically examine the evidence on smoking and cancer and issue comprehensive reports. Reports from both the IARC in 2004 and Surgeon General in 2014 stated that smoking causes cancers of the lung, upper aerodigestive tract (oral cavity, larynx, pharynx and oesophagus), pancreas, bladder, kidney, liver, cervix and stomach, and acute myeloid leukaemia.\(^1\)\(^2\) The Surgeon General’s report also concluded that smoking causes colorectal cancer.\(^\text{1}^\text{1}\)\(^\text{1}\) The Surgeon General’s report also highlighted an association between smoking and breast cancer, but concluded that there is insufficient evidence to infer a causal relationship.\(^2\)\(^2\) Details of the links between smoking and these cancers are discussed in Chapter 3, Sections 3.4 (lung cancer) and 3.5 (all other cancers).

In this section the mechanisms by which smoking causes cancer are summarised. Lung cancer is used as an example because it is one of the most thoroughly investigated cancers. Although more than 85% of lung cancers are attributable to smoking, not all smokers develop lung cancer and lung cancer does occur in non-smokers. In fact, lung cancer in non-smokers is believed to be a different disease from lung cancer in smokers.\(^4\) A compelling explanation of cancer causation needs to reflect these facts and the summary presented here discusses the issue of susceptibility to cancer.

This section draws heavily on the US Surgeon General’s 2010 report *How Tobacco Smoke Causes Disease: The Biology and Behavioural Basis for Smoking-Attributable Diseases*,\(^5\) and unless otherwise referenced the information has been sourced from this report.

The mechanisms by which cigarette smoking causes cancer are extremely complex, but nevertheless can be classified into two categories: genotoxic and non-genotoxic, or epigenetic.\(^6\) Genotoxicity means that the constituents of cigarette smoke damage the DNA of genes. This is believed to occur in a multi-step pathway: the carcinogens in cigarette smoke are activated by enzymes (which in turn have been activated, or induced, by cigarette smoke); the activated carcinogens bind to DNA to form compounds known as DNA adducts; DNA adducts disrupt normal DNA repair mechanisms causing gene mutations—inactivating tumour-suppressor genes and activating oncogenes (cancer promoters); and these gene mutations then disrupt normal cell growth control mechanisms, eventually resulting in cancer. The epigenetic pathway affects the last step of this pathway. The constituents of cigarette smoke alter the expression of genes (without affecting the underlying DNA), activating cell receptors, which then disrupt multiple processes involved in cell cycle regulation. These pathways are discussed in more detail below. Cigarette smoke also alters a range of immunologic functions and it is thought that these effects may promote tumours or act as co-carcinogenic stimuli.

Although much is known about smoking-associated carcinogenesis, particularly in relation to lung cancer,
and to a lesser extent bladder cancer, there are still many unanswered questions. Further, the available data are unhelpful in terms of cancer prevention. Smoking cessation is the only proven strategy to reduce the pathogenic processes that lead to cancer.

3.3.1 Carcinogens in cigarette smoke

There are thousands of compounds in cigarette smoke, including more than 60 known carcinogens from multiple chemical classes, including polycyclic aromatic hydrocarbons (PAHs), N-nitrosamines, aldehydes, volatile organic hydrocarbons and metals.

The fact that carcinogens in cigarette smoke are absorbed into the bloodstream of smokers has been confirmed by the measurement of these substances or their metabolites (biomarkers) in breath, blood and urine. Measurement of urinary biomarkers is the most convenient method to quantify carcinogen exposure. However, many carcinogens found in cigarette smoke are also found in food and the general environment, so their metabolites are also detected in the urine of non-smokers. For example, PAHs occur in grilled foods and engine exhausts. The phenolic compounds, catechol and caffeic acid, are common dietary constituents. However, the N-nitrosamine, NNK (4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone) is specific to tobacco. Its metabolite, NNAL (4-(methylnitrosamino)-1-(3-pyridyl)-1-butanol) is therefore the most discriminatory biomarker because the only source of its parent carcinogen (NNK) is tobacco products. NNAL is only detected in non-smokers if they have been exposed to secondhand tobacco smoke.

PAHs and NNK are the major carcinogens involved in the development of lung cancer. In rodents, NNK primarily produces the adenocarcinoma sub-type of lung cancer. The concentration of NNK in tobacco smoke increased from 1959 to 1997 as the nitrate concentration in tobacco increased, and the risk of adenocarcinoma also appears to have increased since the 1960s. The latest Surgeon General’s report suggested that ventilated filters and increased levels of tobacco specific nitrosamines in cigarettes since the 1950s might have played a role in this increased risk. Other compounds that could also be involved in lung cancer include: 1,3-butadiene, ethylene oxide, ethyl carbamate, aldehydes, benzene and metals.

3.3.2 Activation of carcinogens to form DNA adducts

The next major step in the carcinogenic process is binding of carcinogens to DNA to form the compounds known as DNA adducts. Most cigarette smoke carcinogens have to be converted to a form that can bind to DNA. This process is referred to as ‘activation’ and generally requires an enzyme, often one of the cytochrome P-450s (P-450s), to catalyse (speed) the process. These enzymes are ‘induced’ (essentially produced) by cigarette smoke.

Detoxification processes, which excrete carcinogen metabolites in harmless forms, compete with the activation process. The balance between activation and detoxification varies between people. This may be due to the existence of multiple forms (polymorphisms) of the genes that code for the enzymes that metabolise carcinogens. For example, the CYP2A13 gene, which is expressed primarily in the respiratory tract and participates in the activation of NNK, has a variant with one-half to one-third the capacity to activate NNK. In a study of about 700 lung cancer patients and almost 800 control subjects this variant was associated with a reduced risk of lung cancer. Genetic polymorphisms may be part of the explanation for differential susceptibility to cancer, but further research is required.

DNA adducts are central to the carcinogenic process. Measurement of DNA adducts is difficult because the amount of DNA available from routine procedures (such as bronchoscopy of the lung) is usually small, and adduct concentrations are typically low. Nevertheless, adducts of NNK and other carcinogens with deoxyguanosine and other DNA bases have been identified in human lung tissue, and DNA adduct levels are higher in most tissues of smokers than in corresponding tissues of non-smokers.

3.3.3 Conversion of DNA adducts to mutations
DNA adducts are not mutations *per se*, and can be removed by various DNA repair mechanisms that protect human cells. There is variability between people in this DNA repair capacity, and researchers hypothesise that this is also due to gene polymorphism, which leads to further differential susceptibility to tobacco-induced cancer.

When DNA repair is not completed before a normal DNA replication takes place, DNA synthesis can slow or halt. In some instances it continues, a process known as 'translesion DNA synthesis', which can result in the insertion of an incorrect nucleotide (a component of RNA and DNA). In other words, a mutation occurs.

Inactivation of a number of tumour-suppressor genes, and activation of a number of oncogenes that promote cancer, are thought to be part of the development of lung cancer. These inactivations and activations occur as a consequence of mutations. For example, 90% of patients with small-cell lung cancer, and 15% of patients with non-small cell lung cancer, have loss of function of the *RB* tumour-suppressor gene, and the *TP53* tumour-suppressor gene is mutated and inactivated in 70% of patients with small-cell lung cancer and 50% of non-small cell lung cancer patients. Activating mutations of the *KRAS* oncogene are seen in non-small cell lung cancers, but rarely in lung cancers of non-smokers.

Mutation is a complex process and subject. More than 22 000 mutations have been identified in a small-cell lung cancer cell line, highlighting the impact of the carcinogen cocktail in cigarette smoke.\(^8\)

### 3.3.4 Loss of normal cell growth control mechanisms

Gene mutations can lead to a disruption of the normal regulation process for cell proliferation (growth) and apoptosis (death). The latter is a natural process for eliminating injured or unstable cells and it prevents the malignant growth of cancer cells. Deregulation of apoptosis mechanisms is a characteristic of cancer cells.

As mentioned above, in addition to adversely affecting normal cell growth regulation though gene mutations, components of cigarette smoke can disrupt cell control through epigenetic pathways, and this disruption can eventually result in cancer. For example, nicotinic acetylcholine receptors (nAChRs), which are the first line of contact between cells and cigarette smoke, are believed to be activated by NNK and by nicotine. This activation of nAChRs then promotes the processes required for the development of lung cancer, for example by stimulation of kinases that mediate cancer cell survival, by proliferation and resistance to chemotherapy and by promotion of angiogenesis (the growth of new blood vessels and a fundamental step in carcinogenesis).\(^9\)

Genes can also be inactivated (silenced) and activated by hypermethylation, rather than chromosomal mutation. In lung cancer, more than 50 genes involved in regulating cell growth have been found to be affected by hypermethylation.

### References


The two lungs are located on either side of the heart, near the backbone. The function of the lungs is to transport oxygen from the air to the blood, and carbon dioxide from the blood to the atmosphere. These two gases are 'exchanged' in the alveoli (the thin-walled cells containing air) of the lungs.

There are four main types of lung cancer: squamous cell carcinoma, small-cell undifferentiated carcinoma, adenocarcinoma and large-cell carcinoma.

Lung cancer is now the most common type of cancer in the world. In 2008, the number of new cases that occurred was estimated to be 1.1 million with just under 949 000 deaths occurring in the same year. The number of lung cancer deaths worldwide attributable to smoking has been estimated to be 0.85 million, making lung cancer the third-ranking cause of smoking-attributable deaths, after cardiovascular disease and chronic obstructive pulmonary disease.

In Australia, lung cancer is the fifth most common cancer diagnosed; there were an estimated 11,280 new cases and 8,410 deaths in 2012. Five-year survival in the period 2006 – 2010 was only 13% for males and 17% for females. In Australian males, lung cancer is the most common cause of cancer death and is the third leading contributor to burden of disease, accounting for 4.5% of total disability-adjusted life-years (DALYs) lost due to illness or accident. In Australian women, lung cancer is the second most common cause of cancer death, after breast cancer.

3.4.1 Risk associated with smoking

The evidence that tobacco smoking causes lung cancer is unequivocal. Cigarette smoking causes most cases of lung cancer. In fact, in populations with prolonged cigarette use, up to 90% of cases of lung cancer are attributable to smoking.

Lung cancer was one of the first diseases to be causally linked with smoking. The story of the research that established this link, and the controversies the emerging data created, is told in articles by Colin White and Michael Thun; in the biography of the late Sir Richard Doll, one of the key researchers of the topic; and in the commentary published in conjunction with the re-publication of a pivotal 1959 review of the smoking and lung cancer relationship by Cornfield and colleagues.

The following is a brief summary. In the early 1900s, lung cancer was a rare disease. By the 1930s and 1940s an increase in lung cancer incidence was becoming obvious, and at least seven small studies found an association between smoking and lung cancer. However, it was thought that better diagnosis of the disease, or increased life expectancy, might be the explanation for the increase in lung cancer and these early studies were not widely accepted internationally, or perhaps even read.
In the late 1940s and early 1950s, it became obvious that the increase in lung cancer and lung cancer deaths was real and important. In 1950, two large case–control studies were published, one by Wynder and Graham in the US, and the other by Doll and Hill in the UK. Both found a large increase in the risk of lung cancer associated with smoking. The British study found that the risk for smokers compared with never smokers was 14-fold higher and the American study found that the risk was seven-fold higher. It is noteworthy that while conducting their research, Doll, Hill and Graham, who were all smokers, were doubtful that smoking was a cause of lung cancer. Their results provoked much controversy and disbelief, but nevertheless large prospective studies were initiated in Britain (by Doll and Hill) and in the US to investigate the possible link in a manner free of the potential bias in retrospective case–control studies. These large studies soon confirmed the relationship between smoking and lung cancer.

Doll and Hill's study warrants special mention. They sent questionnaires about smoking habits to almost 35 000 male British doctors in 1951, and periodically thereafter. Deaths and causes of death were monitored for almost 99% of these doctors over 50 years and results were published in 1954 and 1956 and after 10, 13, 14 20, 15 40 16 and 50 years follow-up. As early as 1956, Doll and Hill had concluded, in relation to lung cancer deaths, that smokers had a higher mortality than non-smokers, heavy smokers had a higher mortality than light smokers, cigarette smokers had a higher mortality than pipe smokers and those who continued to smoke had a higher mortality than those who gave up.22

The cohort studies initiated in the US reported similar findings, and expert bodies eventually found the evidence that smoking causes lung cancer compelling. The US Surgeon General, for example, concluded in 1964 that smoking causes lung cancer in men, and in 1968 concluded that smoking causes lung cancer in women.18

The 50-year follow-up of the British doctors study found that lung cancer mortality rates were 16-fold higher (averaged across all ages) for cigarette smokers compared with never smokers.17

3.4.2 How tobacco smoke causes lung cancer

This issue is discussed in Chapter 3, Section 3.3.

3.4.3 Factors affecting risk

3.4.3.1 Intensity and duration

The risk of lung cancer increases with the number of cigarettes smoked and the duration of smoking. The latter is the strongest determinant. A modelling study of the excess odds ratio for lung cancer per pack-year of smoking found a greater risk for a total exposure delivered for a longer duration than for an equivalent exposure delivered at higher intensity (shorter duration). So the earlier the age a person starts smoking and the longer they continue, the greater the risk. For example, the annual excess incidence of lung cancer increases approximately 100-fold for men who have smoked for 45 years compared with men who have smoked for 15 years. In contrast, American Cancer Prevention Study II found that the lung cancer mortality rate for women who had smoked for 21–30 years was about 35% higher in those who reported smoking more than 20 cigarettes per day compared with those who reported smoking 20 cigarettes per day.1

3.4.3.2 Smoking cessation

Stopping smoking decreases the risk of lung cancer. In fact, Julian Peto has pointed out that when a smoker quits, the lung cancer mortality rate stops increasing steeply and remains almost constant.20 The International Agency for Research on Cancer summarised this effect as follows: "Stopping smoking at any age avoids the further increase in lung cancer incurred by continued smoking. The younger the age at cessation, the greater the benefit."1
3.4.3.3 Histological type

Smoking increases the risk of all histological types of lung cancer, but the association between adenocarcinoma and smoking has become stronger over time and it is now the most common type of lung cancer in smokers.\textsuperscript{21,22} For example, in the American Cancer Prevention Study I (initiated in 1959) the relative risk of death from lung adenocarcinoma for male smokers compared with non-smokers was 4.6, whereas in the American Cancer Prevention Study II (initiated in 1982) the comparable risk was 19.\textsuperscript{23} The 2014 Surgeon General's report highlights the substantial changes that have taken place since the 1950s in the design and composition of cigarettes. Although these changes are not fully understood, they have resulted in different patterns of smoking (e.g., more intense puffing) and alterations in the chemical composition of cigarette smoke, and have caused the increased risk of lung adenocarcinoma from smoking. There is currently not enough research to specify which changes have caused this increase, but the evidence suggests that ventilated filters and increased levels of tobacco-specific nitrosamines are partially responsible.\textsuperscript{24}

3.4.3.4 Gender differences

Some studies have suggested that women who smoke are at greater risk for developing lung cancer than men who smoke. The weight of evidence now suggests that there is little, if any, difference between women and men in their vulnerability to the carcinogenic effects of cigarette smoke.\textsuperscript{1,25} For example, a US study that followed almost 300,000 men and almost 200,000 women aged 50–71 years from 1995 to 2003 found no statistically significant difference between sexes in the incidence of lung cancer for those who smoked more than two packs of cigarettes per day.\textsuperscript{26}

However, there is mounting evidence that the biology of lung cancer differs between the sexes.\textsuperscript{25} The US study mentioned above found a 30% higher incidence of lung cancer in women who had never smoked compared with men who had never smoked, and this difference was almost statistically significant.\textsuperscript{26} Although adenocarcinoma is currently the most common histological type of lung cancer in both men and women, women have proportionally more adenocarcinoma and less squamous cell carcinoma.\textsuperscript{22,25} Female hormones and reproductive factors may influence lung carcinogenesis.\textsuperscript{25,27} Data from the Nurses' Health Study in the US suggest that early onset of menopause and past oral contraceptive use increase lung cancer risk.\textsuperscript{28} In this study, postmenopausal hormone use did not affect lung cancer incidence, but a prospective cohort study in the US state of Washington found that oestrogen plus progesterone replacement therapy was associated with increased lung cancer risk.\textsuperscript{29} Further research will be needed to elucidate the impact of such factors on lung cancer incidence and their interaction, if any, with the influence of smoking.

3.4.3.5 Other factors

The role of alcohol in lung cancer is controversial. Some studies have suggested that alcohol consumption, particularly binge drinking, increases the risk of lung cancer in smokers but not in non-smokers.\textsuperscript{30,31} Another study found that moderate red wine consumption decreases lung cancer risk.\textsuperscript{32} Further studies are needed to clarify this association.

Emphysema (a form of chronic obstructive pulmonary disease), which is itself a smoking-associated condition, may increase the risk of lung cancer.\textsuperscript{33}

There are ethnic differences in susceptibility to lung cancer caused by smoking.\textsuperscript{1} In the US, the risk of lung cancer has been found to differ between racial/ethnic groups after taking into account the duration and intensity of smoking. Compared to Caucasians, African Americans and native Hawaiian smokers have an increased risk, whereas Latino and Japanese American smokers have a lower risk.\textsuperscript{31,32} Researchers have hypothesised that ethnic/racial differences in the activity of an enzyme that metabolises nicotine may lead to different smoking behaviour, resulting in different levels of tobacco smoke carcinogens for the same number of cigarettes smoked per day, in turn resulting in elevated lung cancer risk in groups with greater nicotine metabolism, and reduced risk in those with lower metabolism. A study of nicotine metabolism in a multiethnic
cohort supports this hypothesis.\textsuperscript{33}

3.4.4 Impact of smoking on prognosis

Smoking not only increases the risk of lung cancer, but appears to adversely impact prognosis once lung cancer is diagnosed. In patients with non-small cell lung cancer, the survival rate has been found to be lower in patients who had smoked for more pack-years.\textsuperscript{37, 38}

In the US, more than 80% of patients continue to smoke after being diagnosed with lung cancer.\textsuperscript{36} A meta-analysis published in 2010 has provided preliminary evidence that such patients have a worse prognosis than those who quit.\textsuperscript{39} Most patients in the studies had early stage tumours. For both small cell and non-small cell lung cancer, the risk of death (from any cause) was greater in patients who continued to smoke compared with those who quit.\textsuperscript{40} Most patients in the studies had early stage tumours. For both small cell and non-small cell lung cancer, the risk of death (from any cause) was greater in patients who continued to smoke compared with those who quit. Models developed by the authors suggested that the quitters’ improved prognosis was due to a reduction in cancer progression, rather than the cardiovascular benefits of quitting. The meta-analysis results were consistent with this suggestion—the risk of recurrence for both types of lung cancer was higher in continuing smokers.\textsuperscript{40}

Women appear to have a better response to therapy for lung cancer, irrespective of stage of disease, histological type and whether they are being treated with surgery, chemotherapy, radiotherapy or a combination of these modalities.\textsuperscript{25}

3.4.5 Temporal trends in lung cancer rates

The impact of tobacco-control efforts and falling smoking rates on smoking-associated illnesses is clearly of interest to researchers, public health program funders and the wider community. In this context, interest has focused on lung cancer, because most lung cancer is caused by smoking, and because reliable national mortality statistics are widely available. Two analyses of data in the US have found links between tobacco-control efforts and lung cancer rates. The first was based on data from California, where an enhanced tobacco-control program, involving increased taxes and comprehensive strategies to change social norms, was initiated in 1988. The analysis found that over the period 1988–1997 per capita cigarette consumption declined more rapidly in California compared with the rest of the US and that the decline in lung cancer incidence rates in men was 1.5 times greater than in other states where tobacco-control measures were less intensive. In women, lung cancer incidence rates fell by 4.8% in California, but increased by 13.2% in other states.\textsuperscript{41} The second analysis compared data from the 51 US states found that an index reflecting the intensity of tobacco-control efforts in a state was correlated with both lung cancer incidence and mortality in young adults.\textsuperscript{42}

Analyses of data in the UK,\textsuperscript{43} US\textsuperscript{44} and Australia\textsuperscript{45} have found lung cancer incidence and mortality trends in line with smoking patterns and the lag between smoking initiation and disease occurrence. The benefits of smoking cessation in men are now being reflected in national statistics. For example, in Australia, lung cancer incidence in men decreased by 32% between 1982 and 2007, but increased by 72% in women over the same period. However, by 2007 incidence in women was still only about half that in men.\textsuperscript{46} Similar trends have been seen in lung cancer death rates. In men, mortality fell by 1.9% per year between 1993 and 1998, but increased by 0.3% per annum, on average, in women.\textsuperscript{5}

In the US, the steadily increasing lung cancer death rate began to level off for men in the mid-1970s, peaked in 1991 and had decreased by 20% by 2003. In women, the death rate steadily increased until 1991, after which it began to level off, although still increasing by 9.6% between 1991 and 2003. Thun and Jemal estimated from these data that the reductions in tobacco smoking in the US over the previous half-century had prevented at least 146 000 deaths from lung cancer.\textsuperscript{44}

So, the payoff from tobacco control has only just begun, with lung cancer mortality in men peaking 20–25
years after the peak in smoking rates. Researchers anticipate that smoking cessation by women will soon be reflected in disease statistics.

i See the International Agency Against Cancer's Facts sheet at http://globocan.iarc.fr/factsheets/populations/factsheet.asp?uno=900

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3.5 Other cancers

3.5.1 Upper aerodigestive tract cancers

The upper aerodigestive tract comprises the oral cavity (the mouth—including the lip, cheek, gum, tongue and mouth lining), pharynx (throat), larynx (voice box) and oesophagus (the gullet—the muscular tube through which food passes from the throat to the stomach). In epidemiologic studies, cancers of these areas are often grouped together and referred to as upper aerodigestive tract cancer (UADTC). Another grouping is 'head and neck cancer', which comprises cancer of the oral cavity, pharynx and larynx (i.e. the fourth UADTC, oesophageal cancer, is excluded).

In Australia in 2007, there were 1996 new cases of cancer of the lip, tongue or mouth and 328 deaths; 719 new cases of cancer of the pharynx and 280 deaths; 581 new cases and 214 deaths due to cancer of the larynx; and 1264 new cases of oesophageal cancer and 1098 deaths.1 The five-year relative survival rate for cancers of the head and neck was 56.3%.2

3.5.1.1 Risk associated with smoking

Smoking causes all of the UADTCs.3-5 A meta-analysis of 85 studies that included data on more than 50 000 people with UADTC reported an almost four-fold higher risk of these cancers for smokers compared with never smokers and a two-and-a-half-fold higher risk compared with non-smokers.6 Concern about the increasing incidence of UADTC in young people prompted a case–control study in 10 European countries that included people aged under 50 years. Smoking was associated with a five-and-a-half-fold increase in the risk of UADTC.5

The strength of the association between smoking and cancer differs between the sub-types of UADTC. The larynx is directly exposed to tobacco smoke when it is inhaled through the space between the vocal chords, and smoking is particularly strongly associated with cancer of the larynx. Risks for smokers 20-fold higher than for non-smokers have been reported in some studies.3 The meta-analysis of 85 UADTC studies, referred to above, found that the risk of laryngeal cancer was nine times greater for current smokers than never smokers, whereas the risks of oropharyngeal and oesophageal cancer were about three-fold higher in smokers.6

Oesophageal cancer has two histological types: adenocarcinoma (which has further sub-types) and squamous cell carcinoma. An Australian case–control study that included more than 1000 cases found the risk of both squamous cell carcinoma and gastro-oesophageal junction adenocarcinoma was about four-fold higher in smokers compared with never smokers. The risk of oesophageal adenocarcinoma was about
two-and-a-half-fold higher in smokers. A pooled analysis of data from 10 case–control studies of adenocarcinoma also found that smoking doubled the risk. Another pooled analysis of 13 case-control and two cohort studies found a similar (85%) increase in the risk of oesophageal adenocarcinoma associated with smoking.

3.5.1.2 How tobacco smoke causes UADTC

The 2010 US Surgeon General's report (2010) suggests that the polycyclic aromatic hydrocarbons (PAHs) in the particulate phase of cigarette smoke have a role in the development of cancer of the larynx, and that PAHs and two other constituents of cigarette smoke—NNN (N’-nitrosonornicotine) and NNK (4-(methyl nitrosamino)-1(3-pyridyl)-1-butanoine)—are the cause of oral cancer. Such carcinogens can affect DNA repair, producing chromosomal aberrations, which have been found in increased numbers in the oral mucosa of smokers. NNN may also be the key cause of oesophageal cancer; N-nitrosamines are the most potent oesophageal carcinogen known and NNN is the most prevalent N-nitrosamine in cigarette smoke.

Although the links between smoking and UADTCs are well established, only a small proportion of people exposed to tobacco develop these cancers. Aided by emerging information about the human genome, scientists are therefore studying the impact of gene polymorphisms on differential susceptibility to UADTC (and other cancers) among tobacco users. A 2011 meta-analysis, for example, found that the glutathione S-transferase M1 (GSTM1) null polymorphism (i.e. absence of the gene), was associated with an increased risk of oral cancer in Asians but not Caucasians. However, in people with this null polymorphism, the study found that smokers were at lower risk of oral cancer than non-smokers or light smokers. The authors cautioned that the studies they considered had not been designed to investigate this interaction and called for further research.

3.5.1.3 Factors affecting risk

Intensity and duration of smoking

There is a dose–response relationship for smoking and each of the UADTCs. The risk of developing these types of cancer increases with the duration of smoking and the amount smoked. A pooled analysis of 15 case–control studies found that the risk of cancer of the larynx was more strongly associated with greater amounts smoked (cigarettes per day) than the duration of smoking (pack-years).

Alcohol consumption

Alcohol is also a cause of all UADTC sub-types, and exacerbates the effects of smoking on the risk of UADTC. In other words, the effects of smoking and alcohol are synergistic in relation to UADTC (meaning that the combined effect of tobacco and alcohol exceeds the sum of their individual effects). In the meta-analysis of 85 studies referred to above, 24 studies investigated the combined effects of alcohol and smoking. The risk of UADTC in people who had the highest consumption of alcohol and tobacco was almost five times higher than the risk among heavy smokers who did not drink alcohol, or drank moderately. The synergistic effect of tobacco and alcohol on cancer risk is strongest for cancer of the larynx and weakest for cancer of the oesophagus. A pooled analysis of 17 European and American case–control studies found that 4% of head and neck cancers (oral cavity, pharynx and larynx cancers) were attributable to alcohol alone, 33% were due to tobacco alone and almost 35% were due to tobacco and alcohol combined. For cancer of the larynx, these figures were: 3% due to alcohol alone, 52% due to tobacco alone and 33% due to tobacco and alcohol combined.

Smoking cessation

In 2007, the International Agency for Research on Cancer reported that, even after a long period of abstinence, the risk for laryngeal cancer and for carcinoma of the oesophagus does not return to that of
never smokers. Alternatively, a meta-analysis in 2009 found that risks of all UADTCs decline when people quit smoking relative to continued smoking, and can reach the level of non-smokers about 10 years after smoking cessation.

3.5.1.4 Impact of smoking on prognosis

Continued smoking after diagnosis of a UADTC increases the risk of a second primary tumour in the region. This is consistent with the concept of ‘field cancerisation’, which is the concomitant occurrence of carcinogenic alterations in multiple areas of the upper aerodigestive tract.

Recent studies of patients with head and neck cancer have shown poorer local-regional control, disease-free survival and overall survival for patients who were smoking at diagnosis or had smoked in the past, compared with never smokers. Patients who continue to smoke while receiving treatment have poorer local-regional control, disease-free survival and overall survival compared with those who quit.

3.5.2 Pancreatic cancer

The pancreas is an abdominal organ that secretes hormones that assist with digestion.

There were 2525 new cases of pancreatic cancer in Australia in 2007 and 2248 deaths. Pancreatic cancer has the lowest survival rate of any cancer, and is the seventh most common cause of cancer death in Australia. Between 1984 and 2004, the 5-year relative survival of patients with pancreatic cancer was only 4.6%, in contrast with 88% for breast cancer.

3.5.2.1 Risk associated with smoking

Smoking is a well-recognised cause of pancreatic cancer. A pooled analysis of 12 international prospective cohort studies involving almost 1500 cases found that current smokers had a 77% higher risk of pancreatic cancer than never smokers.

One of the European cohort studies in the pooled analysis found that exposure to environmental tobacco smoke (ETS) at home or work increased the risk of pancreatic cancer. Exposure to ETS during childhood was also associated with pancreatic cancer, but the increase in risk was not statistically significant.

3.5.2.2 How tobacco smoke causes pancreatic cancer

NNN and its metabolite NNK, mentioned in Chapter 3, Section 3.5.1.2, are the two known pancreatic carcinogens in tobacco products.

3.5.2.3 Factors affecting risk

The pooled analysis referred to above found that the risk of pancreatic cancer increases with smoking intensity (cigarettes per day), smoking duration (years smoked) and cumulative smoking dose (pack-years). The risk of pancreatic cancer decreases after quitting, reaching the risk level of non-smokers after about 15 years. In Australia, for example, male pancreatic cancer mortality has declined in line with reductions in tobacco consumption approximately 15 years previously. However, pancreatic cancer mortality has continued to rise in Australian women. This may be because of the later peak in female tobacco consumption (compared with male tobacco consumption) or because other factors, such as obesity, are also affecting female pancreatic cancer mortality trends.

3.5.2.4 Impact of smoking on prognosis
This issue does not appear to have been studied, presumably because of the short survival time after diagnosis.

3.5.3 Stomach cancer

The stomach is located between the oesophagus and the small intestine and has a role in digestion of food, secreting enzymes and acids.

Gastric (stomach) cancer is categorised according to the site in the stomach where it occurs. Cancers occurring at the gastric cardia, which is near the junction of the oesophagus and stomach, are referred to as gastric cardia cancers. Cancers of the gastric antrum, corpus or fundus are termed non-cardia cancers. The pathogenesis of gastric cancer is thought to differ between cardia and non-cardia gastric cancer.

There were 1897 new cases of stomach cancer in Australia in 2007 and 1129 deaths. The 5-year relative survival rate in Australia is 25%, the fifth lowest of cancers. Cancer of the stomach was the 10th most common cause of cancer death in Australia in 2007.

3.5.3.1 Risk associated with smoking

Smoking causes gastric cancer. A meta-analysis of 46 case–control studies found approximately a 50% increased risk for people who had ever smoked. For current smokers compared with never smokers the increase in risk of gastric cancer was about 70%. A meta-analysis of 21 case-control and three cohort studies found that smoking increased the risk of gastric cardia adenocarcinoma by 76%.

3.5.3.2 How tobacco smoke causes stomach cancer

As yet, there is no well-defined model of stomach cancer genesis. However, it appears that smoking is associated with intestinal metaplasia and dysplasia, which precede cancer, and DNA adducts (DNA bonded to a carcinogenic chemical) have been identified in tissue samples from the stomach of smokers. Nicotine itself also affects gastric physiology, although it is not clear whether this impacts on carcinogenesis.

3.5.3.3 Factors affecting risk

The 2004 International Agency for Research on Cancer (IARC) report stated that eight of 16 cohort studies found significant dose–response relationships between intensity of smoking and risk of stomach cancer and five studies found a relationship between duration of smoking and risk. Most of the case–control studies that have examined the dose–response issue have found relationships between both intensity and duration of smoking and gastric cancer risk. The IARC concluded that stomach cancer risk increases with duration of smoking and number of cigarettes smoked.

The risk of gastric cancer decreases with increasing duration of quitting.

The 2004 US Surgeon General's report concluded that: 'The evidence is suggestive but not sufficient to infer a causal relationship between smoking and noncardia gastric cancers'. This issue has been difficult to resolve because many studies of the risk of gastric cancer associated with smoking have not distinguished between cancer sub-site types. However, the meta-analysis referred to above identified five studies of non-cardia gastric cancer and four studies of cardia gastric cancer. Smoking significantly increased the risk of both types of stomach cancer; the risk of cardia cancer was increased by 47% and the risk of non-cardia cancer by 32%.

A prospective study of approximately 1000 Japanese men who were followed up for 14 years found that smoking and Helicobacter pylori infection had a synergistic effect on the risk of gastric cancer. The risk of gastric cancer for men who smoked but did not have H. pylori infection was almost six-fold higher than that of...
non-smokers without *H. pylori* infection, whereas the risk for men who both smoked and had *H. pylori* infection was 11-fold higher. In this population, 7.3% of gastric cancer was estimated to be due to smoking alone, 30.1% was due to *H. pylori* infection alone and 49.6% was due to cigarette smoking with *H. pylori* infection.

### 3.5.4 Kidney and bladder cancers

The urinary system includes the bladder, ureters, urethra and kidneys, which filter the blood and excrete waste (urine), which is transferred to the bladder via the ureters, and exits the body via the urethra. The kidneys also have homeostatic roles in relation to blood pressure, acid-base balance and electrolytes.

In Australia in 2007, there were 2580 new cases of kidney (renal cell) cancer, 2217 new cases of bladder cancer and 399 new cases of cancer of other urinary organs. Kidney cancer was the ninth most commonly reported cancer. Five-year relative survival rates for cancers diagnosed between 1998 and 2004 were 65.8% for kidney cancer and 60.4% for bladder cancer. Deaths from these cancers numbered 855, 925 and 70, for kidney, bladder and other urinary organ cancers respectively.

#### 3.5.4.1 Risk associated with smoking

Smoking causes cancer of the kidney, bladder and renal pelvis (a portion of the ureter). A meta-analysis that included more than 8000 cases of kidney cancer found a 50% higher risk of for men who had smoked cigarettes compared with never smokers. The corresponding risk for women was 22%. ETS may increase the risk of kidney cancer. Non-smokers with high combined exposure to home and work ETS have been found in one study to have a two- to four-fold higher risk of renal cell carcinoma compared with non-smokers who were not exposed to tobacco smoke.

A case–control study and accompanying editorial published in the Journal of the National Cancer Institute in 2009 suggest that the association between bladder cancer and smoking has increased substantially between 1994 and 2004. The risk of bladder cancer for current smokers relative to never smokers was 2.9 in 1994–1998, 4.2 in 1998–2001 and 5.5 in 2001–2004. The editorial points out that the concentrations of specific carcinogens in tobacco smoke have increased, and hypothesises that this may be the cause of the observed increase in smoking-attributable bladder cancer risk.

The IARC has estimated that 66% of bladder cancer in men and 30% in women is due to smoking.

#### 3.5.4.2 How tobacco smoke causes kidney and bladder cancers

Aromatic amines, including 2-naphthylamine and 4-aminobiphenyl (4-ABP), are combustion products of cigarette smoke and are known bladder carcinogens. They are thought to contribute to bladder cancer.

#### 3.5.4.3 Factors affecting risk

The risk of kidney cancer increases with the amount smoked, and declines after quitting. The risks of bladder cancer and cancer of the renal pelvis and the ureter increase with the amount of tobacco consumed and the duration of smoking, although for bladder cancer a levelling off of risk at high daily consumption levels, possibly due to under-reporting of consumption by heavy smokers, has been reported. The risk of bladder cancer declines after smoking cessation, rapidly in the first one to four years. However, even after 25 years the risk is not as low as for non-smokers.

#### 3.5.4.4 Impact of smoking on prognosis
A 2002 systematic review found that smoking cessation might favourably alter the course of bladder cancer, but the evidence was insufficient to conclusively recommend to patients that quitting will improve their prognosis. A recent study found that smoking status and a higher cumulative smoking exposure are associated with worse prognosis in patients with primary non–muscle-invasive bladder cancer, who had higher rates of disease recurrence and progression, and lower overall survival.

3.5.5 Cervical cancer

The cervix is the lower portion of the uterus, where it joins the vagina.

There were 739 new cases and 208 deaths from cancer of the cervix in Australia in 2007. The 5-year relative survival rate was 71.8%.

3.5.5.1 Risk associated with smoking

Infection with human papilloma virus (HPV) is the main risk factor for cancer of the cervix, and until recently it had been unclear whether the observed association between smoking and cervical cancer was due to confounding, as women who are HPV-positive have been found to be more likely to be smokers. The IARC and US Surgeon General concluded in 2004 that smoking causes cervical cancer, and two recent case–control studies and a review confirm that smoking is an independent risk factor for cervical cancer. The review cited articles that suggest smoking increases the risk of squamous cell cervical carcinoma, but not adenocarcinoma, and that risk increases significantly with intensity and duration of smoking.

Small studies have recently reported associations between smoking and neoplasia of other genital tract tissue. Neoplasia is the abnormal proliferation of cells and can progress to cancer.

3.5.5.2 How tobacco smoke causes cervical cancer

Smokers’ cervical mucus is mutagenic and contains the carcinogen NNK. Levels of DNA adducts are higher in the cervical tissue of smokers than non-smokers, indicating DNA damage, and it is thought that in combination with HPV these compounds may contribute to the development of cervical cancer in smokers.

3.5.5.3 Factors affecting risk

There is a dose–response relationship between smoking and cervical cancer; the risk of cervical cancer increases with the duration of smoking.

3.5.5.4 Impact of smoking on prognosis

A study in the US involving approximately 2500 women with cervical cancer, who were followed for up to 5 years, found that smokers were about 20% more likely to die from cervical cancer.

3.5.6 Acute myeloid leukaemia

Acute myeloid leukaemia (AML) is a type of cancer that affects the blood and bone marrow. Myeloid leukaemias involve overproduction of immature white blood cells called myeloblasts (in contrast to lymphoid leukaemias which arise in immature blood cells called lymphoblasts). This overproduction of myeloblasts prevents the bone marrow from making normal blood cells. AML develops quickly, with anaemia, bleeding and bruising occurring because of inadequate numbers of red cells or platelets. If untreated, AML is rapidly
fatal. In contrast, chronic myeloid leukaemia develops more slowly and urgent treatment is usually not necessary.

There were 849 new cases and 721 deaths from AML in Australia in 2007. AML is the second most common myeloid cancer, and myeloid cancers were the sixth most common cancers reported in 2007.

3.5.6.1 Risk associated with smoking

Smoking causes AML. Studies have generally reported an approximate doubling of the risk of AML for current smokers, but the Whitehall study (of almost 20 000 male government employees who were followed up from the 1960s to 2005) found a five-fold increase in risk of myeloid cancers for current smokers.

3.5.6.2 How tobacco smoke causes acute myeloid leukaemia

Benzene, which is contained in cigarette smoke, is thought to be the likely carcinogen in AML. Polonium-210 and lead-210, both of which emit ionising radiation, are also found in cigarette smoke. Ionising radiation is a recognised cause of leukaemia.

3.5.6.3 Factors affecting risk

The risk of AML increases with the amount smoked and the duration of smoking.

3.5.6.4 Impact of smoking on prognosis

A small study of 148 patients undergoing stem cell transplantation for treatment of acute leukaemia found that smokers had longer hospitalisation and poorer survival.

3.5.7 Liver cancer

The liver is an abdominal organ essential for metabolism and detoxification.

In 2007, there were 1169 new cases of liver cancer and 1109 deaths in Australia.

3.5.7.1 Risk associated with smoking

The IARC concluded in 2004 and the Surgeon General concurred in 2014 that tobacco smoking causes liver cancer. Researchers from the IARC and the US conducted a meta-analysis of 38 cohort studies and 58 case-control studies to clarify the potential association, and found a ‘moderate’ statistically significant 50% increase in risk for current smokers compared with never-smokers. More recently, the US Surgeon General reported a 60-70% increased risk of liver cancer in current smokers compared with never-smokers.

3.5.7.2 How tobacco smoke causes kidney and bladder cancers

The liver metabolises many circulating carcinogens from tobacco smoke, with NNK, other nitrosamines and furan identified as likely liver carcinogens. A number of potential mechanisms have been identified in liver carcinogenesis, including long-term exposure to carcinogens in cigarette smoke increasing the risk of cellular damage in the liver and in turn the development of cancer. Smoking also increases the risk of liver fibrosis, primary biliary cirrhosis, and chronic liver disease, which can progress to liver cancer.

3.5.7.3 Factors affecting risk
A meta-analysis found some evidence of an increase in risk with the number of cigarettes smoked per day, but this effect differed between studies. There is also limited evidence suggesting that the effect of smoking on risk for liver cancer may be modified by viral hepatitis, but further studies are needed to clarify this relationship.

3.5.8 Colorectal (bowel) cancer

The colon is the major part of the large intestine and its primary function is absorption of water from digested material. The rectum is the final (straight) portion of the large intestine, where faeces are stored before defecation through the anus.

Bowel cancer was the second most commonly reported cancer (after prostate cancer) and the second most common cause of cancer death (after lung cancer) in Australia in 2007. There were 14,234 new cases of bowel cancer and 4,047 deaths. The 5-year relative survival rate was 61.8%.

3.5.8.1 Risk associated with smoking

Four meta-analyses and an analysis of a large US prospective cohort study have concluded that cigarette smoking is associated with colorectal cancer. The increase in risk is about 20%. Similarly, the US Surgeon General concluded in 2014 that smoking causes colorectal adenomatous polyps and colorectal cancer.

In fact, colorectal cancer is a complex collection of diseases and causation appears likely to differ between molecularly defined subsets. A study in women from Iowa in the US found only a moderately increased risk of colorectal cancer associated with smoking, but much higher risks for sub-types defined by anatomical location, phenotype and BRAF mutation status.

A case–control study found the risk estimates for the association between smoking and colorectal cancer increased when smokers were compared with non-smokers who had not been exposed to ETS, suggesting that ETS may be associated with colorectal cancer.

The evidence that smoking is associated with colorectal cancer has led to a suggestion that screening guidelines for bowel cancer be amended to recommend that screening for smokers start at age 45 years rather than 50 years.

3.5.8.2 How tobacco smoke causes bowel cancer

The evidence strongly suggests an effect of smoking in increasing the formation of polyps, the precursor of colorectal cancer, and possibly also the development of malignancy. The many carcinogens in cigarette smoke can reach the bowel via the bloodstream. Higher concentrations of DNA adducts to metabolites of PAHs have been found in the bowel tissue of smokers than non-smokers. The identification of sub-types of colorectal cancer for which the causative link with smoking is stronger has led to suggestions that smoking’s impact on colorectal cancer is mediated through interference with normal DNA methylation pathways.

3.5.8.3 Factors affecting risk

The recent analyses have generally reported increases in risk with amount smoked per day, pack-years of smoking and longer duration of smoking.

Two of the meta-analyses analysed risk by sub-site of cancer and found that the risk associated with smoking is higher for rectal cancer than colon cancer.

Former smokers generally have lower risk than current smokers and the large US cohort analysis found a trend for decreased risk of colorectal cancer with longer time since cessation and no association for former
smokers who had quit before age 40 years or had been non-smokers for 31 years or more.\textsuperscript{51}

3.5.8.4 Impact of smoking on prognosis

Two of the meta-analyses investigated the link between smoking and death from colorectal cancer and found that mortality rates are higher in smokers.\textsuperscript{47,49} This could reflect an impact of smoking on prognosis as well as bowel cancer incidence. A small study of people with cancer of the anus found that recurrence rates and cancer-related deaths were higher in patients who continued to smoke.\textsuperscript{57}

3.5.9 Breast cancer

In Australia in 2007, breast cancer was the third most commonly reported cancer\textsuperscript{22} (and the most commonly diagnosed cancer in women)\textsuperscript{1} and the fourth most common cause of cancer death.\textsuperscript{22} There were 12 670 new cases and 2706 deaths.\textsuperscript{1} The five-year relative survival rate was 87.7%.\textsuperscript{2}

3.5.9.1 Risk associated with smoking

In 2011, a Canadian expert panel reviewed the evidence and concluded that active smoking causes breast cancer and that the association between exposure to ETS and breast cancer in young women who have never smoked is also consistent with causality.\textsuperscript{58} The results of a US cohort study involving almost 80,000 women were consistent with this conclusion.\textsuperscript{59} Most recently, the US Surgeon General concluded that there is sufficient evidence to identify mechanisms by which cigarette smoking may cause breast cancer, and both active and passive smoking are associated with an increased risk of breast cancer.\textsuperscript{44} Although there are at least 20 known or suspected mammary carcinogens in tobacco smoke,\textsuperscript{58} further data and analyses will be needed before the association between smoking and breast cancer can definitely be regarded as causative.

3.5.9.2 How tobacco smoke causes breast cancer

There are biologically plausible mechanisms, particularly for DNA adduct formation and unrepair DNA mutations, by which exposure to tobacco smoke could cause breast cancer. These mechanisms are supported by research to date; however, further studies are needed to identify a specific model of these effects.\textsuperscript{44}

3.5.9.3 Factors affecting risk

Having ever smoked relates to an increased relative risk for breast cancer by an average of 10%; smoking for 20 or more years, 20 or more cigarettes per day, or 20 or more pack-years of smoking has been shown to significantly increase this risk by 13-16%, depending on the study.

There is emerging evidence that premenopausal women may be at greater risk for breast cancer from smoking than postmenopausal women, with risks of 17% and 7% respectively.\textsuperscript{44}

3.5.9.4 Impact of smoking on prognosis

The US Surgeon General has highlighted the difficulty in inferring a causal association between smoking and breast cancer mortality; there are many confounding variables relating to treatment and other noncancer, smoking-related comorbidities that can contribute to mortality. There is currently insufficient evidence to conclude that either active or passive smoking influences breast cancer mortality.\textsuperscript{44}

3.5.10 Other cancers
3.5.10.1 Hodgkin lymphoma

Studies of Hodgkin lymphoma and smoking reviewed by the IARC reported weak or no association. A study published in 2009 found that people who smoked for 25 years or more were at increased risk of Hodgkin lymphoma, but this association is unconfirmed.

3.5.10.2 Prostate cancer

Both the IARC in 2004 and the US Surgeon General in 2014 concluded in 2004 that the evidence did not support a causal relationship between prostate cancer and smoking. A subsequent meta-analysis of cohort studies found no increase in risk of prostate cancer overall in smokers, but did find a slightly increased risk associated with higher daily consumption of cigarettes or greater pack-years of smoking. A large prospective US cohort study, involving over a quarter of a million men, found that current and former smokers may be at decreased risk of being diagnosed with prostate cancer, and a review of the epidemiologic evidence found that smokers are not at appreciably higher risk of developing prostate cancer.

The US Surgeon General concluded that the evidence suggests that smokers have a higher mortality rate from prostate cancer than non-smokers, as well as a higher risk of advanced-stage disease, less well-differentiated cancer, and a higher risk of disease progression. The elevated risk of death from prostate cancer noted by the US Surgeon General has also been reported in the meta-analysis and other studies, and the review of epidemiologic evidence concluded that smokers appear to have more advanced disease at diagnosis, a worse prognosis and a greater risk of fatal prostate cancer.

Smoking is known to alter sex steroid hormone levels, and this effect may be a confounder in studies to date. Further research is needed to clarify the role of smoking in prostate cancer.

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3.6 Reproductive health

3.6.1 Menstrual function, menarche and menopause

The 2001 US Surgeon General's report reached suggestive conclusions about the impact of smoking on menstrual cycles and reproductive lifespan: the period from the commencement of menstruation (menarche) to its cessation (menopause). The 2010 US Surgeon General's report expanded on these conclusions.

Women who smoke are, generally, at higher risk of dysmenorrhoea (painful menstruation) and a range of other symptoms such as premenstrual tension and heavy periods. In the US Nurses' Health Study, for example, smokers were twice as likely as non-smokers to develop premenstrual syndrome over a two- to four-year period. Smokers also tend to have a shorter and more variable menstrual cycle. The former has been attributed to a shortening of the follicular phase. A non-statistically significant higher risk of anovulation in smokers has been found in some studies. These effects are consistent with an increased risk of infertility (see Section 3.6.2) as the timing of ovulation is less predictable in women with variable cycle length and a shortened follicular phase may indicate abnormal formation of follicles and maturation of ova.

Smoking may also result in an earlier menopause. A meta-analysis found that smokers were between 0.8 to 1.7 years younger than non-smokers at menopause. More menopausal symptoms have also been reported, although hot flushes associated with smoking may result from a non-hormonal mechanism. It has been suggested that shorter cycles may deplete oocytes earlier leading to an earlier menopause and thus a shorter reproductive life span. An earlier age at menarche has been reported for the daughters of women who smoked heavily during pregnancy.

3.6.2 Fertility

Measures of fertility include fecundability (the monthly probability of conception), infertility (defined as lack of conception after one year of unprotected intercourse), and sub-fertility (reduced fertility, measured by time to conception or inability to conceive within six months). Smoking reduces fertility in women. Studies have found reduced pregnancy rates, longer time to pregnancy and decreased fecundability in women who smoke. A trend of decreased fertility with increasing number of cigarettes smoked has been reported. The American Society for Reproductive Medicine estimated that 13% of infertility may be attributable to smoking. Impaired fertility has been attributed to the polycyclic aromatic hydrocarbons in cigarette smoke and diminished oviductal functioning.

In relation to the impact of male smoking on sperm quality and fertility, the 2004 US Surgeon General's report concluded that although the evidence suggests that smoking may decrease semen volume and sperm
number, and increase the number of abnormal forms present, it was insufficient to establish causality.\(^6\) The 2010 report found strengthened evidence for decreased semen quality and fertility associated with exposure to tobacco smoke either \textit{in utero} or in adulthood. The report found consistent evidence linking smoking to chromosome changes or DNA damage in sperm, adversely affecting male fertility and pregnancy viability as well as anomalies in offspring. \(^2\)

### 3.6.3 Assisted reproduction

(See 3.15.5 Treatment of infertility)

### 3.6.4 Sexual function

The link between smoking and erectile dysfunction (ED; defined as the persistent inability to attain and maintain penile erection adequate for satisfactory sexual performance) has been studied extensively.\(^8\) The 2014 US Surgeon General’s report concluded that smoking causes ED.\(^8\) Vasospasm induced by the nicotine in cigarette smoke has been suggested as a mechanism for the acute deleterious effects of smoking on erectile function, while the chronic effects are caused by impaired vascular physiology of the erectile tissue. The Surgeon General has recommended promoting non-smoking to prevent ED, and cessation to limit the risk of ED.\(^8\) A study of about 130 Italian women found that smokers have decreased blood flow to genital blood vessels, which may impair sexual function.\(^9\)

### 3.6.5 Sexually transmitted diseases

(See 3.9.7 Infections of reproductive organs)

### References


3.7 Pregnancy and smoking

Smoking during pregnancy is harmful to the health of both the mother and the unborn child. The 2002 United States Linked Birth/Infant Death Data Set reveals that it remains one of the most prevalent preventable causes of infant death and illness.¹

In 2010, 11.7% of Australian women smoked during some or all of their pregnancy. In the period before they knew they were pregnant, 11.7% of pregnant women smoked and 7.7% reported that they smoked after they knew they were pregnant. The likelihood of smoking during pregnancy was higher among teenagers, women in disadvantaged circumstances and Indigenous women.² See Chapter 1, Section 1.10.

Many of the constituents of cigarette smoke are potentially toxic to the developing foetus, including lead, nicotine, cotinine, cyanide, cadmium, mercury, carbon monoxide and polycyclic aromatic hydrocarbons (PAHs).³⁴ Carbon monoxide (CO) reduces the oxygen supply to the baby, leading to hypoxia (insufficient oxygen). CO binds to haemoglobin with an affinity 200 times that of oxygen, and also has an inhibiting effect on the release of oxygen to the cells. Chronic mild hypoxia of foetal tissue can persist for five or six hours after the mother has stopped smoking.⁴ Cadmium, a carcinogen, which accumulates in the placenta and has been detected in umbilical cord blood, is associated with a reduction in foetal capillary volume.⁴ Nicotine is found in foetal blood, amniotic fluid and breast milk,³ and has short- and long-term effects likely to be related to several adverse pregnancy outcomes.⁴ Studies suggest that exposure to PAHs may disrupt hormones, alter enzyme levels or activity, and damage DNA, which, if not repaired, can lead to cell death, cancer or foetal abnormalities.⁴

There are various mechanisms through which smoking may affect the pregnancy and the development of the foetus. Both smoking and nicotine by itself change hormone patterns, affecting the pregnancy outcome and the endocrine profile of the infant. Smoking and nicotine affect the functioning and structure of the oviduct (fallopian tube) in ways that could impair fertility and complicate the pregnancy. Smoking disturbs the development of the placenta, disrupting the implantation process and interfering with the transformation of the uterine spiral arteries. Studies consistently show thickening of the villous membrane of the placenta in smokers, which decreases the ability of nutrients to diffuse through the placenta. Smoking and nicotine impair amino acid transport across the placenta, which the baby needs to make foetal proteins. Nicotine may decrease the pumping of fluid across the placenta, leading to lower oxygen levels in the foetus and acidosis (excessive acid in the blood and tissues). Nicotine can alter embryonic movements that are important in the early development of the organs. Consistent evidence shows that smoking can affect the development of the foetal lung and brain. Smoking a cigarette temporarily increases maternal heart rate and blood pressure and decreases foetal heart rate variability (a measure of the infant's wellbeing).⁴ Studies show a decrease in foetal movement for at least an hour after smoking one cigarette, consistent with a reduction of oxygen to the foetus.⁵
Smoking may present further risks to the pregnancy by increasing the mother's risk of infectious disease and altering the inflammatory response of her immune system. Smokers have lower levels of micronutrients that play a vital role in the health of the pregnancy, such as zinc (in cord blood) and vitamin C. Vitamin C is important for immune function and the formation of collagen. Genetic variation in enzymes that metabolise chemicals from tobacco smoke mediate the risk of adverse pregnancy outcomes. Several proposed mechanisms for the effects of smoking on pregnancy are presented in more detail in the US Surgeon General's report of 2010.4

### 3.7.1 Spontaneous abortion

Smoking during pregnancy is associated with spontaneous abortion or miscarriage (the involuntary termination of a pregnancy prior to 20 weeks of gestation).4,6 Because many miscarriages occur too early to be recognised and confirmed, and miscarriage can be caused by a number of preconditions, exposures or events, spontaneous abortions are difficult to study. However there are multiple ways in which smoking could potentially increase the risk for miscarriage. Proposed mechanisms include placental insufficiency, chronic reduced oxygen to the foetus, and direct toxic effects of constituents of cigarette smoke.4 Tobacco or nicotine may also affect the quality of the egg and embryonic development.6

### 3.7.2 Ectopic pregnancy

Maternal active smoking causes ectopic pregnancy (the implantation of a fertilised egg occurring outside the uterus, usually in the fallopian tubes).6 Nicotine slows down the movement of the fertilised ovum in the fallopian tubes, and impairment of oviduct function can lead to ectopic pregnancy.4 Smokers also have a higher risk of developing pelvic inflammatory disease, which is associated with ectopic pregnancy.3

### 3.7.3 Complications of pregnancy

Smoking affects the healthy development and function of the umbilical cord and placenta, and causes abnormalities or insufficiencies that can lead to serious complications for the success of the pregnancy and the safe delivery of the baby. Smoking causes premature rupture of the membranes (breaking of the amniotic sac before the onset of labour), placenta previa (when the placenta is attached to the uterine wall close to or over the cervix), and placental abruption (premature separation of the placenta from the wall of the uterus).3,4 These complications increase the risk of preterm delivery, haemorrhaging that requires a blood transfusion, and death of the mother or baby. Research indicates that stopping smoking between pregnancies reduces the risk of placental abruption, suggesting that, for this complication, the effects of smoking do not persist.4

### 3.7.4 Preterm delivery

Smoking is a cause of preterm delivery (birth at less than 37 completed weeks of gestation) and shortened gestation.3 Preterm delivery is a leading cause of neonatal death and illness.4 Australian data show that in 2003, babies born to mothers who smoked in pregnancy had a 60% higher risk of preterm delivery than babies of non-smoking mothers.7 It is not known how smoking contributes to preterm delivery, but researchers have proposed various mechanisms. Smokers are more susceptible to vaginal infection; for example they have two to three times the risk of bacterial vaginosis, which is a risk factor for preterm delivery. Some research suggests that smokers may be more sensitive to stimuli that lead to contractions. Smoking may affect collagen formation, leading to weakening and rupture of the membranes. Smokers are more likely to develop complications that are risk factors for preterm delivery, such as placental abruption and placenta previa.4

Limited research suggests that women who quit smoking within the first three months of pregnancy reduce their risk of placental complications at birth, premature birth, infant illness and perinatal death.8
Refer to Section 3.6 for discussion of smoking and reproductive health.

References


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3.8 Child health and maternal smoking before and after birth

There are several possible routes by which the effects of tobacco smoke may compromise infant health. Before birth, paternal active smoking may alter chromosomes or damage DNA in sperm, and the foetus may be exposed to maternal active smoking or maternal exposure to secondhand smoke during pregnancy.\textsuperscript{1,2} Following birth, infants may be exposed to parental secondhand smoke in the home,\textsuperscript{2} to thirdhand smoke in household dust and indoor surfaces,\textsuperscript{3} and to an increased bacterial load carried by a parent or carer who smokes.\textsuperscript{4,5,6} Both prenatal and postnatal exposure have been found to contribute to several health conditions in children.\textsuperscript{1,2} Maternal smoking also has negative effects on the quality and quantity of breast milk.\textsuperscript{7,8} Delineating the impact of each route of exposure in the causation of disease can sometimes be difficult, particularly for rarer conditions such as birth defects and childhood cancers.

Exposure to secondhand smoke during pregnancy is also a cause of reduced infant birthweight, and is associated with other health problems for the developing foetus.\textsuperscript{9,10} See Chapter 4, Section 4.11 for further information.

3.8.1 Foetal size and growth

3.8.1.1 Birthweight

Smoking in pregnancy causes restricted growth and low birthweight in the infant. Intrauterine growth retardation is reduced foetal growth during gestation. Babies born with low birthweight have a higher risk of subsequent illness, death and longer-term poor health outcomes through childhood and adult life.\textsuperscript{9} Low birthweight is associated with heart disease, type 2 diabetes, high blood pressure and being overweight in adulthood.\textsuperscript{11,12,13}

Babies born to smokers weigh, on average, about 150 g to 200 g less than babies born to non-smokers.\textsuperscript{9} Australian data show that babies of women who smoke during pregnancy are twice as likely to be of low birthweight (defined as weighing less than 2500 g) compared to babies whose mothers are non-smokers. They are also more likely to be admitted to special care nurseries or neonatal intensive care units.\textsuperscript{14,15} In Australia in 2004–5, it is estimated that about 14% of all deaths due to low birthweight were attributable to tobacco use in pregnancy.\textsuperscript{16}
The effect of maternal smoking on low birthweight is largely due to intrauterine growth retardation and to a lesser extent shortened gestation. Growth retardation is likely to be caused by chronic mild oxygen deprivation in the foetus from exposure to carbon monoxide, with a more minor role played by the effects of smoking on the placenta leading to nutritional deficiency of the foetus. The effects of smoking on birthweight appear to be stronger among older mothers and in mothers who have particular genotypes for drug-metabolising enzymes. The risk for low birthweight increases with the number of cigarettes the mother smokes per day. However, some research indicates that birthweight declines far more sharply at low levels of exposure, such as that experienced by women exposed to secondhand smoke and possibly by women who smoke a low number of cigarettes per day. This may help account for the observation that the benefits of cutting down the number of cigarettes smoked per day on birthweight are considerably smaller than for complete smoking cessation.

Mothers who stop smoking early in their pregnancy have babies with similar birthweights to babies of non-smokers. Even mothers who stop before their third trimester can avoid much of the effect of smoking on birthweight.

3.8.1.2 Respiratory health

Maternal smoking during pregnancy causes reduced lung function in infants, and may also cause an increase in the number of lower respiratory tract illnesses, including wheezing, during infancy. The effects of maternal smoking in utero may also be related to an increased risk of impaired lung function in childhood and adulthood. Australian research suggests that infants born to women who have smoked during pregnancy have weakened innate immune defences, and develop their acquired immune system more slowly than infants of non-smoking mothers. This may explain why infants of smokers are more prone to be asthmatic and to develop respiratory infections. In Australia in 2004–05, it is estimated that about 13% of all deaths due to lower respiratory tract infections in babies less than one year of age were attributable to exposure to maternal tobacco smoking before and/or after pregnancy. Infants living with smokers are also more likely to experience a range of respiratory symptoms and chest illnesses. These findings are discussed in greater detail in Chapter 4, Section 4.9.

3.8.2 Perinatal and infant death

3.8.2.1 Stillbirth

Maternal smoking is associated with an increased risk of stillbirth (foetal death after 28 weeks' gestation) and neonatal mortality (death of an infant within the first 28 days of life). Data from the Australian Institute of Health and Welfare's National Perinatal Statistics Unit show that in 2003, babies born to mothers who smoked during pregnancy had a 50% greater risk of perinatal death than babies of non-smoking women. Proposed mechanisms by which smoking increases perinatal mortality include complications of pregnancy (abruption, placenta previa), preterm delivery, premature and prolonged rupture of the membranes (“water breaking”), and through physiologic responses of the foetus and newborn to stress.

3.8.2.2 Sudden infant death syndrome

Sudden infant death syndrome (SIDS) is the sudden, unexplained, unexpected death of a child before one year of age. Smoking has been established as a cause of SIDS, whether the baby has been exposed to smoking before birth or in the home following birth. The biological pathway remains uncertain, but may be due to the effects of chronic oxygen deficiency on the development of the central nervous system and other neurotoxic effects of tobacco smoke on the foetal brain. Neurochemical changes to the cardiorespiratory control centres of the brainstem can result in changes in the development of respiratory control. Several studies have linked smoking during pregnancy to alterations in breathing patterns, ventilatory responses, and
arousal responses in infants.\textsuperscript{9,25} Almost one in five deaths from SIDS in Australia (19\%) is thought to be caused by maternal tobacco use.\textsuperscript{16}

For information on secondhand smoke and SIDS see Chapter 4, Sub-section 4.9.2.

3.8.3 Birth defects

A large meta-analysis of studies published between 1959 and 2010 found that maternal smoking is associated with an increased risk for limb reduction defects; oral clefts; clubfoot; defects of the eyes; and defects of the gastrointestinal system, especially gastroschisis and abdominal hernia. More modest associations were found for digit anomalies (abnormal number or formation of fingers); cryptorchidism (undescended testes); and defects of the heart and musculoskeletal system, including craniosynostosis (premature fusing of the skull bones).\textsuperscript{27}

The US Surgeon General’s report (2014) concluded that maternal smoking in early pregnancy causes orofacial clefts, and that maternal smoking is associated with other defects such as clubfoot, gastroschisis (the guts protruding through an opening in the abdominal wall), and atrial septal heart defects.\textsuperscript{25} Proposed mechanisms for oral clefts include the alteration of embryonic movements in early pregnancy that are important to the development of the organ systems, reduced supply of essential nutrients (such as vitamins and folate) for embryonic tissues, oxygen deficiency, and DNA damage. Mothers and babies with certain genotypes may be more susceptible to damage from tobacco smoke. Further work is needed to establish the mechanism. Studies also support an increased risk of oral clefts with paternal smoking, although it is not clear whether this is due to exposure of the mother to secondhand smoke or if it is due to the effects of tobacco smoke on sperm.\textsuperscript{1}

3.8.4 Health complaints in infancy

A few studies have reported an association between maternal smoking, both during and after pregnancy, and infantile colic or excessive crying.\textsuperscript{28,29} Infantile colic is characterised by the frequent sudden fits of irritability, inconsolable crying and screaming accompanied by clenched fists, drawn-up legs and a red face. It occurs in the first weeks after birth and usually resolves by four months of age.\textsuperscript{28} One study suggests that maternal smoking may be linked to colic through gastrointestinal tract dysregulation. More research is needed to confirm this theory.\textsuperscript{28}

The evidence is uncertain regarding the effect maternal smoking during pregnancy on risk of allergic sensitisation and atopic disease, including allergic symptoms, eczema, rhinitis and dermatitis. More studies addressing potential genetic determinants of susceptibility are needed.\textsuperscript{2} While smoking during pregnancy may increase the risk of wheezing illnesses in infants,\textsuperscript{9} the potential role of prenatal tobacco exposure as an independent cause of asthma is still unclear.\textsuperscript{2}

3.8.5 Long-term development

3.8.5.1 Neurodevelopment

Investigating the impact of maternal smoking on the cognitive and behavioural development in infants and children is difficult as many factors affect the outcomes, including genetic and environmental effects.\textsuperscript{1} This has produced mixed results in the research. So while various studies have found an association between smoking during pregnancy and poorer outcomes in children, including for impaired learning and memory, lowered IQ, cognitive dysfunction, later childhood conduct problems, substance use, and early adult criminality, their findings are called into question by other studies reporting no association and the problems of inadequate study design.\textsuperscript{30}
Recently, the US Surgeon General concluded that maternal prenatal smoking increases the risk of disruptive behavioural disorders, particularly attention deficit hyperactivity disorder, among children. However, there is insufficient evidence to infer a relationship between maternal prenatal smoking and anxiety, depression, Tourette syndrome, schizophrenia, and intellectual disability among children. General assessments of children's cognition and intelligence have been mixed. However, studies of children's general verbal skills and specific language and auditory tests have found a more consistent association between smoking during pregnancy and children's poorer performance on these tests. More comprehensively designed studies that take into account the many confounding factors on child development are needed.

3.8.5.2 Nicotine dependence

Evidence is emerging that suggests that exposure to nicotine in utero predisposes an individual to a greater likelihood of nicotine dependence later in life, independent of socio-economic and other factors that influence uptake of smoking. It is possible that this may occur by nicotine having a direct effect on the developing foetal brain, causing permanent abnormalities in neurotransmitter regulation. Other research, while confirming that offspring of women who smoked during pregnancy are more likely to become smokers in early adolescence, suggests that environmental influences on smoking uptake such as the mother's current smoking status and peer group behaviour are stronger predictors. This is an area requiring further study.

3.8.5.3 Physical development

Studies into the possible effects of smoking during pregnancy on subsequent physical growth of children have been mixed. Where differences have been found between children of smokers and non-smokers, they have generally been small. More research is needed.

3.8.5.4 Cardiovascular disease risk

Several studies have examined the link between smoking during pregnancy and the development of cardiovascular risk factors in the child. Among other risk factors, maternal smoking during pregnancy is associated being overweight or obese in childhood. This effect appears to be independent of the effects of smoking on foetal growth, and is likely to be an effect of smoking in early pregnancy. Two possible mechanisms are the effects of smoking on hypothalamic function affecting food intake and energy expenditure, or abnormalities in fat cells. Breastfeeding for more than six months may reduce the risk of child obesity associated with smoking in pregnancy. The evidence is unclear as to whether there is an increased risk of higher blood pressure in children born to women who smoked during pregnancy. However, limited research indicates that maternal smoking in pregnancy leads to impaired blood pressure regulation in infants and adverse lipid (cholesterol) profiles in adult offspring. Smoking throughout pregnancy is a risk factor for cardiovascular developmental changes, including aortic narrowing, in early childhood and adolescence. One study found that the foetuses of smoking mothers are more likely to have lesions in the walls of the foetal artery and adjoining vessels, which are the initial stages of atherosclerosis (narrowing of the arteries by fatty deposits).

3.8.5.5 Other long-term consequences

A few studies suggest that smoking during pregnancy may affect the reproductive development of male offspring, increasing the risk for lower sperm counts and quality, lower fertility, smaller testicles, undescended testes and hypospadias (a penis abnormality). Limited research suggests that smoking during pregnancy may also affect reproduction in female offspring. It is associated with a smaller uterus, a reduced number of somatic cells (which are necessary for egg survival), and slightly lower fertility in female
offspring. More research is required to confirm these findings.

The link between parental smoking and development of childhood cancers is discussed in Chapter 4, Section 4.9.9.

### 3.8.6 Breastfeeding and smoking

Breastfeeding has wide-ranging health benefits for both baby and mother. These include well-recognised benefits such as reduced risk of diarrhoeal illness, reduced risk of developing allergies to cow’s milk and possible reduced risk of obesity later in childhood. They also include less well-recognised benefits such as improved sight and psycho-motor development, reduced incidence of orthodontic problems resulting from under-development of the jaw and other facial bones, and possibly reduced risk of autoimmune diseases such as diabetes and inflammatory bowel disease.

Babies who are breastfed gain better levels of immunity to infectious disease, particularly against respiratory and ear infections associated with secondhand smoke exposure. Women who have breastfed have a reduced risk of developing breast cancer and possibly ovarian cancer, as well as possible improvements in bone mineralisation.

There is consistent evidence that women who smoke are less likely to breastfeed their infant and are more likely to wean their child earlier than mothers who do not smoke. This effect persists even after adjusting for other influences on the decision to breastfeed, such as socio-economic factors.

Tobacco smoke appears to have a direct negative effect on milk quality, as well as the quantity produced. It is thought that nicotine may affect the activity of prolactin, a hormone essential for milk production. Nicotine is found in the breast milk of mothers who smoke. Cotinine, one of the main metabolites of nicotine, is found in the urine of breastfed infants of smokers, as well as in the urine of infants who are exposed to secondhand smoke. Nicotine absorbed by infants through breast milk can produce short-term symptoms such as restlessness, insomnia, nausea, vomiting, diarrhea and rapid pulse, and may affect infants’ autonomic cardiovascular control and sleeping patterns. However, as nicotine has a short half-life in milk of about two hours, breastfeeding mothers who cannot quit can minimise the exposure of their baby to nicotine by prolonging the time between their last cigarette and breastfeeding.

Although smoking and breastfeeding is not ideal, the benefits of breastfeeding outweigh the risks associated with smoking and not breastfeeding.

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1. Perinatal death is defined as ‘a fetal or neonatal death of at least 20 weeks gestation or at least 400 grams birthweight’, Laws et al (2006) (p42).

### References


58. Dorea JG. Maternal smoking and infant feeding: breastfeeding is better and safer. Maternal and Child


Inhaling the complex chemical mixture of combustion compounds in tobacco smoke causes adverse health outcomes through mechanisms that include DNA damage, inflammation and oxidative stress. Smoking has substantial adverse effects on the immune system, both locally (such as in the respiratory tract and soft tissues in the lungs) and throughout the body. As a result, smokers are at increased risk of a wide range of infections. This chapter examines the evidence with respect to acute infections, pneumococcal and meningococcal disease, tuberculosis, complications among persons with existing HIV infection, as well as some other viral and bacterial infections. Evidence regarding periodontitis is covered in Section 3.11.1 and surgical infection is discussed in Section 3.15.1. Chronic respiratory conditions such as chronic obstructive pulmonary disease and asthma are covered in Section 3.2.

3.9.1 Acute respiratory infections

Evidence suggests that there is a causal relationship between smoking and acute respiratory illnesses, as well as all major respiratory symptoms among adults. Upper respiratory tract infections (URI or URTI) are the illnesses caused by an acute infection. This involves the upper respiratory tract: nose, sinuses, pharynx or larynx and commonly includes tonsillitis, pharyngitis, laryngitis, sinusitis, otitis media and the common cold.

Cigarette smoking is a major risk factor for acute respiratory tract infections, with both active and passive smoke exposure increasing the risk of infection. Smoking increases the incidence, duration and/or severity of respiratory viral infection. The mechanism of this enhanced susceptibility is multifactorial and includes alteration in structural and immune defences—a substantial report of immunologic effects of cigarette smoking was published in 2004; it described the harmful effects on cell counts and distribution in peripheral blood and lung fluids as well as impairment of the functioning of white blood cells, lymphocytes (natural killer cells) and humoral immune system function (production of antibodies). Recent studies provide additional detail of the adverse effects on the immune system, such as those on the retinoic acid-inducible gene I (RIG-I), inflammatory factors in nasal lavage fluids (NLF IL-6), NF-kappaB regulation (regulation of critical defence genes), pulmonary T-cell responses, type II interferon responses (antiviral mechanisms) in airway epithelial cells, and the functioning of intelectin 1 (an immune defence protein). There is also evidence that cigarette smokers have distortions to the normal microbial communities of the upper
respiratory tract, which are thought to contribute to the prevalence of respiratory tract complications in this population.\textsuperscript{12}

The patient problems most commonly managed overall by Australian general practitioners are respiratory related (accounting for 22 problems per 100 encounters in 2009–10) and this category also comprises the majority of new problems presented by patients (39\% of all problems, managed at a rate of 59 per 100 encounters).\textsuperscript{13} Cigarette smokers get more colds and worse colds, have much higher rates (several-fold higher) and more severe cases of influenza infection and are at increased risk of bacterial pneumonia compared with non-smokers.\textsuperscript{3, 5}

3.9.2 Chronic respiratory infections

For discussion of chronic respiratory diseases associated with infection such as chronic bronchitis and chronic obstructive pulmonary disease (COPD), see Section 3.2.5.

3.9.3 Pneumococcal and meningococcal disease

Pneumonia is an infection of the lungs, usually caused by bacteria or viruses. Bacteria and viruses living in the nose, sinuses or mouth may spread to the lungs; a person may also breathe some of these germs directly into the lungs. The most common cause of pneumonia in adults is infection with \textit{Streptococcus} \textit{pneumoniae} (pneumoccus). Viruses are a common cause of pneumonia, especially in infants and young children. Atypical pneumonia (sometimes called 'walking pneumonia') is caused by bacteria such as \textit{Legionella pneumophila}, \textit{Mycoplasma pneumoniae}, or \textit{Chlamydia pneumoniae}. \textit{Pneumocystis jiroveci} pneumonia is sometimes seen in people whose immune system is impaired (due to AIDS or to certain medications that suppress the immune system). \textit{Staphylococcus aureus}, \textit{Moraxella catarrhalis}, \textit{Streptococcus pyogenes}, \textit{Neisseria meningitidis}, \textit{Klebsiella pneumoniae}, or \textit{Haemophilus influenzae} are other bacteria that can cause pneumonia.\textsuperscript{14}

Exposure to tobacco smoke suppresses the activation of the innate immune system response to bacterial infection; this mechanism is considered important in people's susceptibility to pneumonia.\textsuperscript{15, 16} Evidence from several studies confirms that smoking is significantly associated with the development of bacterial pneumonia.\textsuperscript{5, 17} There is a dose–response relationship between the current number of cigarettes smoked per day, pack-years of smoking and time since quitting and invasive pneumococcal disease, with approximately 50\% of those with invasive pneumococcal disease being cigarette smokers.\textsuperscript{18}

Cigarette smoking is an especially prominent risk factor for pneumococcal pneumonia in patients with chronic obstructive pulmonary disease, but even without COPD, smoking remains a major risk factor. There are reported estimates of increased risk for pneumococcal pneumonia among smokers ranging from an almost two-fold (OR 1.88; 95\% CI, 1.11–3.19) to a four-fold increase in risk (OR 4.1; (95\% CI, 2.4–7.3) for active smoking. Exposure to secondhand smoke has been found to more than double the risk of this infection (OR 2.5; 95\% CI, 1.2–5.1) compared with non-exposed non-smokers.\textsuperscript{3} A recent review indicates active smoking\textsuperscript{19} and a recent case–control study indicates secondhand smoke exposure\textsuperscript{20} as factors that predispose the elderly population to pneumonia. Evidence from several longitudinal studies conducted in large populations confirms significantly increased pneumonia associated mortality in smokers compared with non-smokers (other evidence to date from cross sectional and meta-analytic studies is inconsistent).\textsuperscript{2} There is also strong evidence that smoking is an independent risk factor for Legionnaires disease, an atypical pneumonia that usually develops two to 14 days after exposure to \textit{Legionella pneumophila}.\textsuperscript{17, 21}

Meningococcal disease describes infections caused by the bacterium \textit{Neisseria meningitidis} (also called meningococcus). The meningococcal bacteria (\textit{Neisseria meningitidis}) are a significant cause of disease in Australia, especially in the very young, teenagers, young adults and those with medical risk factors. The meningococcal bacteria can cause meningitis (inflammation of the meninges, the membrane lining of the brain and spinal cord) and/or septicaemia (blood poisoning). Meningococcal disease is an uncommon but life-threatening infection. There are 13 different types of meningococcal bacteria with the most common in
Australia being meningococcal group B and C. Notifications of meningococcal disease in Australia for 2006–2007 were at average annual rate of 7.0/100 000 population. Neisseria meningitidis remains a major cause of bacterial meningitis and other invasive bacterial infections worldwide with major fluctuations in the incidence of endemic disease and the occurrence of outbreaks and epidemics. There is evidence from case–control studies that tobacco smoke exposure independently increases the risk of developing meningococcal disease. Children under 18 years of age are at almost four times the risk of infection from maternal smoking (OR 3.8; 95% CI, 1.6–8.9). All age groups have more than a doubling of risk from active smoking (OR 2.4; 95% CI, 0.9–6.6) or from exposure to secondhand smoke (OR 2.5; 95% CI, 0.9–6.9). There is a dose–response relationship between exposure to secondhand smoke and the risk of meningococcal disease in all age groups.

### 3.9.4 Tuberculosis

Tuberculosis is an infectious disease caused by various strains of mycobacteria but usually by Mycobacterium tuberculosis. It typically attacks the lungs but can also affect other parts of the body. Approximately one-third of the human population is skin test positive for the infection and is thus thought to harbour the bacterium. The infectious bacilli are inhaled as droplet nuclei that have been exhaled into the atmosphere. These droplets are small enough to remain airborne for several hours. Estimations of the minimum infectious dose range from a single bacterium upwards. In developed countries, tuberculosis is held in check by effective public health systems; in countries where the disease is truly endemic, control remains a huge challenge and one that is exacerbated as highly drug-resistant strains continue to evolve. Tuberculosis results in an estimated 1.7 million deaths each year and the worldwide number of new cases (more than nine million) is higher than at any other time in history. Overall, Australia has one of the lowest incidence rates of tuberculosis in the world; however it has a relatively high incidence rate among Indigenous people. In 2007 the crude incidence rate was 5.4 cases per 100 000 population but the rate among Indigenous people (6.6/100 000) was more than seven times that in the non-Indigenous Australian-born population (0.9/100 000). The incidence rate for Indigenous people in the Northern Territory was 32.2 per 100 000 population, 13 times that in the non-Indigenous Australian-born population. Looking at tuberculosis rates in countries with a comparable Indigenous population, Australia has a similar rate ratio between the Indigenous and non-Indigenous populations compared with New Zealand and the US and a much lower rate ratio compared with Canada. There are no grounds for complacency given the probability of ongoing transmission of tuberculosis as well as the obvious need to address the ongoing inequalities.

The US Surgeon General’s report in 2014 was the first in its series to address the evidence regarding smoking and tuberculosis. It concluded that smoking causes both an increased risk of Mycobacterium tuberculosis disease and mortality from tuberculosis, and is associated with higher rates of recurrent tuberculosis. More evidence is needed to determine whether active smoking causes tuberculosis infection, although the report notes that both active and passive smoking are risk factors. Other reviews confirm that active smoking is a risk factor for infection and that it increases the risk of progression to tuberculosis disease and death. Up to one in five deaths from tuberculosis could be avoided if patients were not smokers. As with active smoking, exposure to secondhand tobacco smoke is also a risk factor for the development of tuberculosis, especially for children. Quitting smoking and prevention of exposure to secondhand smoke are both important measures in the control of tuberculosis. These measures are underscored by the earlier comments about incidence rates among Indigenous people, given that smoking prevalence is markedly higher in this community than in the non-Indigenous population.

### 3.9.5 Risks for and complications of HIV

The authors of a recent systematic review suggested that smoking may be an independent risk factor for acquiring HIV infection but this finding remains controversial. Research into the association between smoking and HIV disease progression has produced inconsistent findings. One recent North American study of more than 2000 people living with HIV/AIDS found that current smokers (HR 1.8; 95% CI, 1.3–2.3) and individuals with an increased dose and/or duration of smoking were at greater risk of all-cause mortality.
compared to never smokers. Among people with HIV, smoking may compound the risk of developing COPD, and increase the risk of cervical cancer (in those who also have human papillomavirus; HPV) and liver cancer. A review concluded that social class, intravenous drug use and compliance with highly active antiretroviral treatment (HAART) are factors that may interact with smoking behaviour, and the independent role of these factors may be difficult to assess in relation to the outcome of HIV infection.

3.9.6 Other viral infections

Smoking may also have indirect adverse outcomes such as the increased risk of hepatocellular carcinoma (cancer of the liver) due to the potential smoking-related progression of chronic viral hepatitis.

3.9.7 Infections of reproductive organs

A small but growing number of research studies deal with the association between cigarette smoking and infections of reproductive organs. Bacterial vaginosis, although often asymptomatic, can cause considerable discomfort and is associated with the development of more serious infections, such as septicaemia and increased risk of poor pregnancy outcome. A recent review found that tobacco smoking is significantly correlated with bacterial vaginosis, typically being in the region of twice as common in smokers as non-smokers, with a greater prevalence noted in young women. Tobacco use was also independently associated with a higher prevalence of specific sexually transmitted bacterial infections—chlamydia and gonorrhoea.

Persistent infection with oncogenic human papilloma virus (HPV), leading to precancerous lesions and potentially cervical cancer, is a serious health burden. Cancer-causing HPV infections can result from interactions of the virus, the host, and many other cofactors; a recent prospective study found that smoking may induce impaired antibody response among HPV-infected young women. Non-smokers were five times more likely to develop HPV antibodies than young (<30 years old) female smokers (OR 0.2; 95% CI, 0.0–0.9). In addition these younger female smokers had a significantly decreased tendency of maintaining constant HPV antibody positive status by the end of the follow-up compared with non-smokers, who were 10 times more likely to do so (OR 0.1; 95% CI, 0.0–0.8).

3.9.8 Periodontitis

(See Dental 3.11.1.)

3.9.9 Surgical infections

(See 3.15.1.)

3.9.10 Other bacterial infections

There is evidence that smoking may cause an increased risk of peptic ulcer disease owing to an increased rate of Helicobacter pylori infection. There is mixed evidence on the relationship between active smoking, bacteraemia (bloodstream infections) and sepsis (a severe illness in which the bloodstream is overwhelmed by bacteria) with about half of studies recently reviewed finding an adverse effect (a positive association).
References


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Cataract and age-related macular degeneration (AMD) are the two leading causes in Australia of vision impairment not correctable by refraction (eye glasses). Cataract is responsible for 37% of such vision loss and AMD is responsible for 26%. AMD is responsible for 48% of blindness in Australia and cataract for 12%. Smoking increases the risk of both cataract and AMD. Smoking may also be associated with the rare eye condition Graves' ophthalmopathy, and recent studies suggest a link between smoking and ocular inflammation.

### 3.10.1 Cataract

The ocular lens, which is behind the pupil, focuses light onto the retina. It is normally a transparent organ but with age tends to develop opaque areas, which impair vision. These opaque areas are called cataracts.

There are three main types of cataract, classified by their location within the lens structure: nuclear, cortical and posterior subcapsular. Nuclear cataract, which occurs in the centre of the lens, is the most common. Each type of cataract has its own distinct risk factors. Smoking is a cause of nuclear cataract. A study of almost 4000 Australians aged 49 years and older who were followed up for 10 years found that people who had smoked at some time (ever smokers) had a 40% higher risk of developing nuclear cataract than people who had never smoked. Although the exact mechanism of causation is not known, many trace metals and other chemicals in cigarette smoke are capable of damaging the proteins in the eyes' lens. Quitting smoking may reduce the risk of developing nuclear cataract and of progression of cataract.

Smoking may also be associated with an increased risk for developing posterior subcapsular cataract (situated under the external membrane, usually behind the lens) but more research is required.

### 3.10.2 Age-related macular degeneration

The macula is the central area of the retina. It contains the fovea, which is responsible for high-resolution vision. There are two main types of AMD: neovascular (or exudative) and atrophic. The 2014 US Surgeon General's report concluded that smoking causes both types of AMD. A meta-analysis of large studies from the US, Netherlands, Australia, France and Japan also found that the evidence strongly suggests that smoking causes AMD. The pooled analysis found a four-fold increase in risk for neovascular AMD and a two- to three-fold increase in the risk of atrophic AMD associated with smoking. A further meta-analysis of studies published up until 2007 confirmed that smoking increases the risk of AMD (neovascular and atrophic...
There are a number of proposed mechanisms for smoking-related damage to retinal structures, primarily oxidative stress; cigarette smoke is a strong oxidant that causes systemic oxidative stress. Smoking may also increase oxidative stress on the macula by removing its defences and reducing macular pigment and plasma levels of antioxidants. Inadequate peripheral blood flow might also contribute to the development of AMD.

The first meta-analysis also found evidence of reversibility, because ex-smokers had a lower risk of AMD. Furthermore, in patients with neovascular AMD who were treated successfully, there was a higher recurrence rate in those who continued smoking compared with those who quit. Quitting smoking appears to reduce the risk of AMD, but several decades after quitting smoking, the risk remains higher for former smokers than for never smokers.

Australian researchers developed a model predicting the decline in risk of AMD after quitting smoking and used it to assess the cost-effectiveness of smoking cessation in relation to AMD. They found that because of the high cost of treating AMD, smoking cessation interventions are cost-effective in terms of their impact on AMD alone (see Section 17.4.1.3).

3.10.3 Graves' ophthalmopathy

Graves' ophthalmopathy is a complication of Graves' disease, a fairly rare autoimmune thyroid disease. The eye complications include protrusion of the eyeballs, double vision, inflammation of eye tissue and damage to the optic nerve. A number of studies have observed an increased risk among smokers for developing the ocular complications of Graves' disease. At this time the evidence is not conclusive, and further research is required.

3.10.4 Ocular inflammatory disease

Ocular inflammatory disease is inflammation of one or more part of the eye and encompasses uveitis (inflammation of the middle layer of the eye), scleritis (inflammation of the white outer coating) and inflammation of the ocular surface.

Two recent studies have suggested that smoking may be associated with ocular inflammation. One study found that smokers were twice as likely to have uveitis as never smokers. The second study found that ocular inflammation was more severe in patients who were smokers and recurred more quickly. Cigarette smoke has an inflammatory effect and this may be the mechanism of action.

References


3.11 Dental diseases

The oral cavity is the first part of the anatomy to be exposed to mainstream smoke in smokers and, as described in Chapter 3, Section 3.5.1, smoking causes oral cancers. Smoking also damages the soft and hard tissue structures that support the teeth, known as the periodontium. The periodontium includes the gingiva and the ligaments that attach the tooth root to the jaw. The gingiva is the soft tissue covering the gums and overlapping the teeth; it protects the root surfaces of the teeth. Gingivitis is an inflammation of the gingiva, triggered by the build-up of plaque, leading to reddening of the gums, bleeding and swelling. Untreated gingivitis can lead to chronic periodontitis, an inflammation of the gingiva and the adjacent tooth attachment apparatus. Plaque on the teeth spreads below the gum line behind the gingiva, triggering an inflammatory response. A range of symptoms, including bleeding, swelling, gum recession and separation of the gingiva from the surface of the tooth, lead to further infection. This in turn can lead to bone loss, loosening of teeth, development of abscesses in soft tissue and bone, a greater risk of decay of the exposed root surfaces of the tooth (root surface caries) and tooth loss.

3.11.1 Periodontitis

Smoking causes periodontitis. Increased tobacco use and longer duration of smoking are associated with more severe disease and a higher risk of dental damage. Cessation appears to reduce the risk.

Analysis of the Australian National Survey of Adult Health (2004–2006) suggested that about 32% of moderate to severe periodontitis is due to smoking. This extrapolates to an estimated 700 000 adults affected by periodontitis due to their smoking.

The precise means by which smoking causes periodontitis have not been determined, but three mechanisms have been suggested. First, smoking may increase the quantity of plaque and the likelihood that bacterial pathogens colonise the plaque. Second, smoking impairs the body's immune response, making the smoker more susceptible to bacterial infection and also impairing the regeneration and repair of periodontal tissues. Third, the vasoconstrictive effect of tobacco smoke and nicotine may reduce gingival blood flow and impair oxygen and nutrient delivery to gingival tissue.

Smoking also results in poorer bone regeneration after surgical treatment aimed at replacing all missing tissues of the periodontium. A meta-analysis found statistically significant less bone gain in smokers than non-smokers after such treatment.

3.11.2 Dental caries
Dental caries (cavities) is a disease that occurs when acids produced by bacteria dissolve the hard enamel of the tooth surface. It is then possible for bacteria to penetrate the tooth and reach the pulp tissue. Pain, infection and the need for tooth extraction can result.\(^1\)

The 2014 US Surgeon General’s report found that smokers are more likely to have dental caries, missing teeth due to decay, or fillings, although more research is needed to establish smoking as a cause.\(^8\) Data from a US national healthy survey of more than 5,000 women found that smoking is a risk factor for untreated caries and DMFS: decayed, missing (due to disease) and filled permanent tooth surfaces.\(^3\)

### 3.11.3 Tooth loss

The main biological causes of tooth loss (edentulism) are periodontal disease and caries. As outlined above, smoking is linked to the risk of both.

A systematic review found significant associations between smoking and tooth loss in each of the six cross-sectional and two cohort studies considered.\(^9\) Most studies found a dose-response relationship and a decrease in the risk of tooth loss for former smokers.

The Australian '45 and Up Study' investigated the association between smoking and the chance of being edentulous (having no teeth remaining) in approximately 100,000 residents of New South Wales. Smokers had a 2.5-fold higher risk of being edentulous compared with never smokers. The more and the longer people smoked, the higher their risk. Former smokers had lower risk than current smokers but the chance of being edentulous was still higher than that of never smokers 30 years after quitting.\(^10\) A Japanese study also found that smoking was associated with the number of missing teeth.\(^11\) The Australian study suggested environmental tobacco smoke might also increase the risk of edentulism.\(^10\)

### 3.11.4 Complications and failure of dental procedures

Because of the established adverse effects of smoking on the oral cavity, researchers have investigated the impact of smoking on the outcome of surgical procedures for periodontal disease\(^12\) and the success of prosthetic implants for missing teeth.

A meta-analysis published in 2007\(^13\) and an evaluation of implants in almost 500 patients published in 2010\(^14\) found that smoking approximately doubles the risk of implant failure. A review by the Massachusetts Dental Society noted that in a cross-sectional study of 109 patients, the prevalence of implant loss in smokers was 15.3%, compared with 2% in non-smokers.\(^15\) The review also cited studies reporting that smoking increases the risk of peri-implantitis and that implants failed earlier in patients who smoked more, and concluded that smoking is a relative contraindication to implant placement.

Root-coverage procedures for people with periodontal disease were less successful in smokers than non-smokers.\(^12\) The US Surgeon General concluded that the evidence suggests that smoking compromises the survival of dental implants, but more research is needed to confirm it as a cause of dental implant failure.\(^8\)

### References


3.12 Gastro-intestinal diseases

3.12.1 Peptic ulcer disease

Mucus and other secretions line the gastrointestinal tract, protecting it from gastric acid. If this protective mechanism is impaired or if there is an increase in gastric acid or other damaging agents, then ulceration may occur. Peptic ulcer disease involves the formation of ulcers in either the lining of the stomach (gastric ulcers) or the duodenum, the section of the small intestine closest to the stomach (duodenal ulcers).

The presence of the gastric bacterium *Helicobacter pylori* causes infection and damage to the gastrointestinal wall, greatly increasing the risk of developing peptic ulcers. The *Helicobacter pylori* organism is present in all people with duodenal ulcers and 70–90% of people with gastric ulcers. The risk of developing peptic ulcers is also increased among people who take non-steroidal anti-inflammatory drugs (NSAIDS).

Peptic ulcers were the eleventh most common cause of hospital admissions in Australia in 2007–2008, and almost 3% of Australians report having some sort of peptic ulcer. Smoking increases the risk of peptic ulcer disease in people who are infected with *Helicobacter pylori*. In Australia, about 9% of peptic ulcer disease in men and 6% in women has been attributed to smoking. Smoking affects the gastrointestinal tract in a number of ways: it reduces the production of gastric mucus and other protective secretions, promotes duodenal reflux and reduces blood flow to the lining of the tract. In this compromised environment, *Helicobacter pylori* is better able to spread and cause damage. Smoking may also be related to an increased risk of developing complications of peptic ulcer disease, such as ulcer perforation or bleeding, but this effect may be confined to people who are not taking NSAIDS.

The increased risk of peptic ulcer disease consequential to smoking appears to reverse with smoking cessation.

The higher prevalence of peptic ulcer disease in people with mental disorders has been largely explained by smoking and alcohol dependence in this population.

3.12.2 Inflammatory bowel disease

Inflammatory bowel disease (IBD) is a group of conditions in which the intestines are inflamed. The two major types of IBD are Crohn's disease and ulcerative colitis. Crohn's disease can involve any part of the gastrointestinal tract, but most commonly affects the small intestine or the colon. Ulcerative colitis is
restricted to the colon and the rectum. The presenting symptoms of these two IBDs are often similar (abdominal pain, vomiting, diarrhoea), but the pathophysiology differs. Crohn's disease is thought to be an autoimmune disease (see Section 3.17), but ulcerative colitis is not. Tissue inflammation tends to be deeper with Crohn's disease than with ulcerative colitis. Both conditions are treated with drugs and/or surgery.\(^6,7\)

The 2014 US Surgeon General's report concluded that smoking increases the risk of Crohn’s disease, but more evidence is needed to confirm whether it is a cause.\(^8\) A 2006 meta-analysis of the research found that smoking increases the risk of Crohn’s disease by about 76%.\(^9\) In Australia, about 36% of Crohn’s disease in men, and about 40% in women, has been attributed to smoking.\(^10\)

Patients with Crohn’s disease who continue to smoke have a worse prognosis. Their symptoms are exacerbated and, after surgery, they are more than twice as likely as non-smokers to have a disease recurrence. Smokers have a 2.5-fold increased risk of repeat surgery within 10 years. Quitting smoking reduces the risk of relapse.\(^11\)

Smoking affects the immune system in a variety of ways and it is not clear which of its immunologic effects are relevant to Crohn's disease.\(^7\)

In contrast to its impact on Crohn’s disease, smoking seems to decrease the risk of ulcerative colitis by about 40% and quitting increases the risk,\(^9\) although more research is needed to confirm that smoking causes this effect.\(^8\) Ex-smokers have an almost 80% higher risk of ulcerative colitis compared with never smokers.\(^9\) The reason or reasons for the contradictory effect of smoking on the two main IBDs is unknown.\(^7\)

### 3.12.3 Disorders of the liver and gallbladder

Smoking adversely affects the hepatobiliary system.

The 'Million Women Study' in the UK found that smoking increases the risk of liver cirrhosis two- to three-fold and the risk of gallbladder disease (symptomatic gallstones or cholecystitis) by about 10–30%.\(^12\) Ex-smokers were found to have elevated risks for these conditions compared with never smokers, but lower risks than current smokers.

Smoking appears to increase the severity of non-alcoholic fatty liver disease,\(^13\) and large case–control studies in the UK and US have found that smoking increases the risk of primary biliary cirrhosis (an autoimmune disease that results in the destruction of hepatic bile ducts) by about 50–60%.\(^14,15\) A 2011 meta-analysis of five published studies, that together included almost 2 000 cases of primary biliary cirrhosis, confirmed the association and found that smoking increases risk by almost 70%.\(^16\) See Section 3.17 for a discussion of autoimmune disease.

### 3.12.4 Disorders of the pancreas

Pancreatitis is inflammation of the pancreas. It can be acute or chronic. The most common symptom is severe abdominal pain. Gallstones cause acute pancreatitis and high alcohol intake is a risk factor for chronic pancreatitis. Smoking increases the risk of gallstones and smoking is also strongly associated with drinking alcohol. It has therefore been difficult to determine whether smoking \textit{per se} increases the risk of pancreatitis.

A cohort study of over 18 000 residents of Copenhagen found that smoking does increase the risk of pancreatitis, independently of its effect on gallstones and its association with alcohol consumption. In fact about 46% of cases of pancreatitis in this group of people were attributed by the researchers to smoking.\(^17\)

Note that pancreatitis may increase the risk of pancreatic cancer, which is one of the malignancies caused by smoking (see Section 3.5.2).\(^3\)
3.12.5 Other gastrointestinal disorders

Data from an Australian twin study suggest that smoking increases the risk of appendicitis by about 65%, but that this risk decreases by 15% every year after quitting. A retrospective survey of more than 6 000 male British construction workers who underwent appendicectomy over a 33-year period found that smoking increased the risk of perforated appendix and also increased the risk of post-operative complications in non-perforated appendicitis (see Section 3.15).

For information about anal fistula, see Section 3.17.2.

References


3.13 Musculoskeletal diseases

### 3.13.1 Bone density, osteoporosis and the risk of fractures

Osteoporosis is a skeletal disease characterised by low bone mass density (BMD) and deterioration of bone, with a consequent increase in bone fragility and risk of fractures. The diagnosis of osteoporosis is based on low BMD. The most common osteoporotic fractures are of the hip, lumbar spine and wrist. Hip fractures are the most severe.¹

Osteoporosis is common and is associated with older age and female gender. Women aged 50 have almost a 50% lifetime risk of an osteoporotic fracture.¹ In Australia, there were more than 16 000 osteoporotic hip fractures in 2006–2007; about three-quarters were in women whose average age was 83 years.²

Smoking decreases BMD in the middle and later years of life. The 2004 US Surgeon General's report concluded that smoking causes low BMD in postmenopausal women.³ BMD decreases by about an additional 2% per year in smokers compared with non-smokers, leading to a difference of about 6% by age 80.⁴

Although the Surgeon General concluded only that smoking 'may' cause low BMD in older men,³ a meta-analysis of the effect of smoking on BMD that included data from more than 40 000 subjects found that smoking has a more deleterious effect on bone mass for men than women.⁵ This meta-analysis also found that the effect of smoking on BMD is dose dependent. The more that people smoked—reflected in higher pack-years, cigarettes per day or number of years smoked—the lower their bone mass.

Both the meta-analysis and the Surgeon General's report found insufficient evidence that smoking lowers BMD in younger women and younger men.³, ⁵ The meta-analysis suggested that this is because the total exposure to smoking in young adults is insufficient to produce discernible decrements in BMD, and cited several studies that demonstrate a significant negative impact of smoking on BMD in young adults who are heavy smokers.⁵

Smoking cessation may slow or even partially reverse bone mass loss, but more research is needed to evaluate this issue.⁵

Because smoking lowers BMD and increases the risk of osteoporosis, it would be expected to increase the risk of bone fractures. Projections from the meta-analysis of the effect of cigarette smoking on BMD suggest that smoking will increase the lifetime risk of a vertebral fracture by 13% in women and 32% in men. For hip fractures, smoking-attributable increases in risk were projected to be 31% in women and 40% in men.⁵ The Surgeon General³ and two meta-analyses of studies investigating fracture risk in smokers have confirmed this effect.⁴, ⁶ The 2004 Surgeon General's report concluded that smoking increases the risk of hip fractures,
but found that there was inadequate evidence at that time to reach the same conclusion about smoking and fractures of other bones.\(^3\) The first meta-analysis of fracture risk, published in 1997, found that female smokers had a 41% increase in hip fractures at age 70 years.\(^4\) The most recent fracture meta-analysis, published in 2005, included data for almost 60,000 people and reported a 25% increase in risk of any fracture, and an 84% increase in the risk of hip fracture, associated with smoking.\(^6\)

A number of mechanisms may contribute to the loss of BMD in later life consequential to smoking. Nicotine and cadmium in cigarette smoke may have a direct effect on bone cells, and smokers' bone density could also be impaired by lower absorption of calcium and vitamin D, and altered metabolism of some other hormones. Smoking also affects oestrogen levels and the effectiveness of hormonal replacement therapy. Smokers tend to have a lower body weight and be less physically active than non-smokers. Both of these factors adversely affect BMD. Smokers also reach menopause earlier, on average, thereby extending the postmenopausal period of accelerated bone mineral loss.\(^3\)

Data from the 2005 meta-analysis of smoking and fracture risk suggest that smoking may also increase the risk of fractures independently of its effect on BMD.\(^8\) In this meta-analysis, there was still a 12% increase in the risk of any fracture associated with smoking, after adjustment for age, BMD and body mass index, and this increase was just statistically significant. The authors suggested that this effect could be due to the poorer balance and poorer physical function that has been reported in smokers (see Chapter 3, Section 3.13.4).\(^7\)

### 3.13.2 Delayed bone union

There is increasing evidence from both observational and experimental studies that smoking delays bone healing (union) after fracture or surgery.\(^8-11\) After elective foot surgery, for example, a 42% increase in the time to bone healing has been reported.\(^12\) Smoking has been found to increase the chance of non-union after spinal fusion surgery\(^13\) and to worsen outcomes.\(^14\)

### 3.13.3 Back pain

There have been suggestions in the medical and health economic literature that smoking causes low back pain and increases the incidence of sick leave due to back pain.\(^15, 16\) A meta-analysis of all studies published until February 2009 has confirmed an increase in low back pain in current and former smokers.\(^17\) The association was stronger in adolescents than adults and was more pronounced for chronic back pain and severe back pain. The authors of the meta-analysis speculated that the effect could be due to reduced perfusion of intervertebral discs.

An analysis of the link between smoking and low back pain in more than 70,000 Canadians was published in 2010.\(^18\) Smoking increased the likelihood that survey participants of all ages would have lower back pain, after adjustment for body mass index, level of activity and other factors that could have explained the association. The risk of having lower back pain was about 80% higher in daily smokers aged 20 to 29 years. The excess risk decreased with age but was still statistically significant at all ages.

### 3.13.4 Other musculoskeletal problems

Smoking might increase joint cartilage loss,\(^19\) and has been reported to increase the risk of tears of the rotator cuff (the muscles and tendons that stabilise the shoulder),\(^20\) but further research is needed before these adverse effects could be regarded as confirmed.

A study of almost 10,000 women aged 65 years and over in the US found that smokers had poorer physical function than non-smokers, as measured by tests of muscle strength, agility, co-ordination, gait and balance, and self-reported physical status.\(^7\) The researchers likened the poorer physical function to a hastening of ageing by about five years, and suggested that the effect may be due to the poorer vascular function
consequential to smoking.

A 2011 meta-analysis of 48 studies, including more than 500,000 participants, investigated the association between smoking and osteoarthritis. The analysis found that the protective effect of smoking observed in some studies, but not others, is likely to be false and may be caused by selection bias. The effect was seen in hospital-based case-control studies (where the control subjects are more likely to have smoking-related conditions), but not in community-based case-control studies, cohort studies or cross-sectional studies.

References


3.14 Skin

Smoking adversely affects the skin. Delayed wound healing is discussed in Chapter 3, Section 3.15.1.2 and other smoking-associated skin conditions are detailed in this section.

3.14.1 Facial appearance and premature skin ageing

Smoking affects facial appearance in men and women, independent of sun exposure and age. Increased wrinkling\(^1\)\(^-\)\(^5\) and altered complexion colour\(^1\) have been attributed to smoking, as have elastosis (loss of elasticity in the skin resulting from degeneration of connective tissue) and, in men, telangiectasia (dilatation of fine blood vessels in the skin visible as fine red lines).\(^6\) One recent study found that smokers appear up to 4.7 years older than non-smokers, and in the majority of cases smokers and non-smokers could be correctly distinguished by examining photographs of the face and temple region.\(^7\) Two studies of twins have confirmed that smokers tend to look older.\(^8\),\(^9\)

Visible wrinkling is most evident in older smokers, but even smokers aged in their 20s and 30s may show evidence of microscopic superficial wrinkling.\(^4\) Combined exposure to both sunlight and tobacco smoke causes a greater degree of damage than exposure to one agent alone,\(^10\) possibly through the phototoxic effects of tobacco smoke condensate, which increase the skin’s vulnerability to UV radiation.\(^11\) Even non-facial, non-sun-exposed skin may be more wrinkled in smokers than non-smokers.\(^12\)

A possible mechanism for premature wrinkling is that smoke affects the function of human skin fibroblasts (cells present in connective tissue that form collagen and elastin), thereby accelerating the appearance of ageing.\(^3\) Recent research has suggested a connection between wrinkling in smokers and the development of chronic obstructive pulmonary disease (COPD). Smokers with severe facial wrinkling may also have a higher susceptibility to developing COPD; possible mechanisms being damage to collagen and elastin, which are important to both skin and lung function.\(^13\)

3.14.2 Acne and other sebaceous conditions

A review of smoking-associated skin conditions published in 2010 noted that the evidence linking acne to smoking is conflicting. Only two of the five studies reviewed reported an association.\(^14\) Another study, published after the review, found a higher prevalence of comedonal postadolescent acne in women who were smokers compared with non-smokers.\(^15\)

The evidence linking some other sebaceous conditions with smoking is stronger. Smoking appears to cause
hidradenitis suppurativa (clusters of chronic abscesses or cysts in areas of sweat or sebaceous glands); up to 98% of patients with this condition are smokers.\textsuperscript{14, 16, 17} A case–control study found smoking was a risk factor for epidermal inclusion cysts in men, but not in women.\textsuperscript{18}

### 3.14.3 Dermatitis

The 2010 review of smoking and the skin concluded that the evidence linking smoking to dermatitis and eczema remains controversial.\textsuperscript{14} A small case–control study published after the review found that smoking increased the risk of adult-onset atopic dermatitis in smokers and members of their family who were exposed to environmental tobacco smoke.\textsuperscript{19} Cigarettes themselves can cause allergic contact dermatitis in both occupational and non-occupational settings.\textsuperscript{14, 20}

### 3.14.4 Psoriasis

Smoking is a well-established risk factor for psoriasis, a chronic autoimmune disease, the most common type being plaque psoriasis, which is characterised by scaly patches on the top layer of the skin.\textsuperscript{14, 21} Higher intensity smoking is associated with clinically severe disease,\textsuperscript{14, 22, 23} and psoriasis is less responsive to treatment in smokers.\textsuperscript{14, 24} The risk of developing psoriasis decreases progressively with increased time since smoking cessation.\textsuperscript{24}

Multiple mechanisms are thought to explain the association between smoking and psoriasis. Smoking enhances the expression of genes known to confer an increased risk of psoriasis. It increases oxidative damage—the free radicals in cigarette smoke, for example, trigger a cascade of systemic reactions. Smoking also promotes inflammatory changes by suppressing immune cell processes and nicotine depletes calcium stores in T lymphocytes, probably impairing their function.\textsuperscript{24}

The form of psoriasis known as palmoplantar pustulosis (which is confined to the hands and soles and is also known as ‘pustular psoriasis of the extremities’) is strikingly correlated with smoking; up to 95% of patients are smokers when the disease is diagnosed.\textsuperscript{14, 25}

### 3.14.5 Lupus erythematosus

Lupus erythematosus is an autoimmune condition that can manifest as a systemic disease, involving many different organs, or as a cutaneous disease, involving only the skin. The systemic form is referred to as systemic lupus erythematosus (SLE). It often involves a rash, and sometimes involves scaly patches or ulcers.

A meta-analysis of seven case–control studies and two cohort studies has confirmed that smoking is associated with SLE. Current smokers have around a 50% increased risk of SLE compared with never smokers.\textsuperscript{26} The risk is not elevated for ex-smokers. Smokers also have increased SLE disease activity,\textsuperscript{27} and poorer health-related quality of life has been reported.\textsuperscript{28} In 2014, the US Surgeon General’s report found that there is mixed and therefore inadequate evidence that smoking causes SLE, or affects its severity or treatment.\textsuperscript{29}

Smoking also increases the risk of developing some of the cutaneous forms of lupus\textsuperscript{14, 30} and has been reported to decrease the effectiveness of the antimalarial drugs that are sometimes prescribed for cutaneous lupus.\textsuperscript{31} The 2014 US Surgeon General’s report concluded that smoking is a risk factor for cutaneous lupus, but the evidence is too limited to determine if it is a cause.\textsuperscript{29}

### 3.14.6 Other skin conditions

Smoking is a risk factor for the development of alopecia (hair loss, usually from the scalp) and may increase
the likelihood of premature grey hair.\textsuperscript{14}

A retrospective survey of more than 58,000 women in Denmark found an increased risk of genital warts in smokers, which the authors concluded could be due to immunosuppressive effects or uncontrolled confounding.\textsuperscript{32}

For information about anal fistula, see Section 3.17.2.

References


3.15 The impact of smoking on treatment of disease

Last updated: March 2015

3.15.1 Surgery
Smoking increases the risk of postoperative complications. Smokers' higher prevalence of chronic diseases, impaired pulmonary reserve, altered immune responses and impaired wound healing are thought to cause such complications. Poorer surgical outcomes result.¹

3.15.1.1 Anaesthesia
The effectiveness of a number of commonly used anaesthetic drugs is reduced in smokers. Higher doses are therefore required. These drugs include opioids,² neuromuscular blocking agents and some of the volatile agents that are administered by inhalation (via a mask or tracheal tube). The polycyclic aromatic hydrocarbons in cigarette smoke induce the liver enzymes that metabolise anaesthetics, at least partly accounting for these effects.³ Smoking does however decrease postoperative nausea and vomiting, possibly because of the increased metabolism of volatile anaesthetics.³

Smoking increases the risk of intraoperative and postoperative respiratory complications, including bronchospasm, aspiration, hypoventilation and hypoxaemia.⁴⁻⁶ An increased risk for smokers of admission to intensive care after general or orthopaedic surgery has been reported,⁴ and was attributed to smokers' higher perioperative pulmonary complication rate.

The Australian and New Zealand College of Anaesthetists recommends that patients who smoke be encouraged to quit at any time before surgery.⁷ The optimal timing of smoking cessation has been a source of controversy, though there is agreement that longer quitting is best.⁷ Research suggests that recent quitters are no worse off than continuing smokers in terms of pulmonary complications. (see Section 3.15.1.3).⁸,⁹

3.15.1.2 Postoperative complications
Smoking delays wound healing after surgery. Complications such as infection,¹⁰ dehiscence (bursting of sutures)¹¹ and erosions (destruction of tissue surfaces) are increased.¹²,¹³

Such smoking-associated complications are particularly problematic after plastic and reconstructive surgery, orthopaedic surgery (see Section 3.13.2), bowel surgery, dental surgery (see Section 3.11.4), microsurgery
and organ transplantation.\textsuperscript{14–15}

For example, after breast reconstruction, smoking has been associated with a doubling of the risk of complications (such as mastectomy flap necrosis or infection) and a five-fold increase in the risk of implant failure.\textsuperscript{17} Such poor surgical outcomes have led to a call for caution when undertaking breast reconstruction in smokers.\textsuperscript{18} Similarly, impaired wound healing and wound infection in smokers undergoing breast reduction surgery\textsuperscript{19, 20} have led to a suggestion that perioperative smoking cessation be an essential eligibility criterion for this surgery.\textsuperscript{20}

Specific post-surgical complications linked with smoking include: a higher failure rate for oral mucosa graft urethroplasty;\textsuperscript{21} worse hearing and the need for repeat operations after ear surgery;\textsuperscript{22} increased complications post appendicectomy;\textsuperscript{23} increased mortality after liver transplantation;\textsuperscript{15} increased kidney transplant rejection;\textsuperscript{24} and poorer survival after heart transplantation if either the donor was, or the recipient is, a smoker.\textsuperscript{16, 25} The poorer organ transplantation outcomes in smokers, combined with the high demand for donated organs, have led to suggestions that smokers be given lower priority for organ transplants and debate about the ethics of such a policy.\textsuperscript{26-28}

Smoking has also been implicated (in a report of four cases) as a risk factor for late-onset infection and other complications after facial injection of a filler substance for cosmetic purposes.\textsuperscript{29}

The magnitude of the impact of smoking on perioperative outcomes was studied in a retrospective review of data from more than 500,000 patients in the US who had non-cardiac surgery.\textsuperscript{30, 31} Information on the 30-day period following surgery was compared for 82,304 current smokers and 82,304 control patients. Current smokers were 40\% more likely to die than never smokers. Their risk of major morbidity also increased: the risk of pneumonia doubled, the risk of unplanned intubation almost doubled, and the odds of postoperative ventilation increased by 50\%, cardiac arrest by 60\%, myocardial infarction by 80\%, and stroke by 70\%. The risk of superficial and deep infections increased by 30\% and 40\%, respectively, and sepsis, organ space infections and septic shock were 30\% to 50\% more likely. The increased perioperative mortality and morbidity were confined to patients who had smoked more than 11 pack-years.

### 3.15.1.3 Impact of smoking cessation

A meta-analysis published in 2011 reviewed data from randomised trials and observational studies that had compared postoperative complications in smokers and people who quit smoking before surgery. The analysis found that smoking cessation decreases postoperative complications. In the randomised trials, complications were reduced by about 40\%. The review found that the longer the period of preoperative smoking cessation, the greater the reduction in complications.\textsuperscript{32}

Another meta-analysis, also published in 2011, investigated the possibility that smoking cessation just before surgery may be harmful (see Section 3.15.1.1).\textsuperscript{9} Data from nine studies that had studied the impact of quitting within eight weeks of surgery were combined. The analysis found no increase (or decrease) in overall postoperative complications in recent quitters compared with smokers. Although the study authors suggested that concern about possible adverse effects associated with stopping smoking just before surgery might therefore be unfounded, an accompanying 'Invited Commentary' pointed out that there was significant heterogeneity in the results of the studies that were combined, which was not surprising because some included patients who had quit two to three days before surgery whereas others included patients who had quit eight weeks prior to surgery.\textsuperscript{8} The commentators affirmed the wisdom of encouraging patients to quit smoking several months prior to surgery, but queried whether clinicians should be reassured that the timing of smoking cessation in anticipation of surgery is immaterial.

A systematic review published in 2012 explored the relationship between short-term preoperative smoking cessation and postoperative complications, and concluded that at least four weeks of abstinence from smoking reduces respiratory complications, and abstinence of at least three to four weeks reduces wound-healing complications. Short-term (less than four weeks) smoking cessation did not appear to increase or reduce the risk of postoperative respiratory complications.\textsuperscript{33} The Australian and New Zealand College of Anaesthetists recommend that, based on the current available evidence, anaesthetists and surgeons should
not be dissuaded from advising patients to quit at any time before surgery.\textsuperscript{8}

### 3.15.2 Drug interactions

Smoking alters the effects of a number of medications (see also Section 3.15.1.1 on interactions with anaesthetics). Doctors and other healthcare workers need to be aware of these interactions when medications are prescribed and also when patients quit smoking, as drug dosages may need to be adjusted.\textsuperscript{34}

Drug interactions fall into two categories: (i) pharmacokinetic interactions, which occur when cigarette smoke alters a drug’s metabolism; or (ii) pharmacodynamic interactions, which occur when the physiological effects of cigarette smoke modify the physiological effects of the drug.\textsuperscript{35, 36}

Pharmacokinetic interactions include increased metabolism of caffeine, heparin, warfarin, theophylline, a number of antipsychotic drugs and a number of benzodiazepines. A meta-analysis of the interaction between smoking and warfarin, for example, found that smoking increased warfarin dosage requirements by about 12%.\textsuperscript{37} Although it is difficult to know which of the estimated 4800 compounds in cigarette smoke cause these interactions, the polycyclic aromatic hydrocarbons are suspected. These hydrocarbons induce liver enzymes (see Chapter 13, Section 13.15.1.1) and thereby hasten the clearance of any drug (or substance) whose metabolism requires the enzymes.\textsuperscript{35, 36}

Pharmacodynamic interactions include: reduced response to corticosteroids in smokers who are asthmatic,\textsuperscript{39, 40} decreased sedation with benzodiazepines (possibly due to the stimulant effects of nicotine), slowed absorption of sub-cutaneous insulin (possibly due to reduced blood flow to the skin, mediated by nicotine), and an increased risk of cardiovascular adverse effects in women taking oral contraceptives.\textsuperscript{35, 36}

In the above examples, smoking modifies the effects of particular drugs. It has also been hypothesised that bronchodilator drugs (mainly beta-2-agonists), prescribed for people with chronic obstructive pulmonary disease (COPD), may worsen the effects of cigarette smoke. The theory is that bronchodilation improves smoke inhalation, and may increase the deposition of cigarette smoke on the lungs, thereby increasing cardiovascular disease morbidity and mortality. This hypothesis is yet to be tested.\textsuperscript{41}

### 3.15.3 Cardiovascular disease

As detailed in Section 3.1, smoking causes cardiovascular disease, and generally, if a person continues to smoke after developing cardiovascular disease their prognosis is worse than if they had quit.

For example, a study of more than 18 000 patients with coronary disease who were receiving a statin drug for coronary disease found that over a five-year period those who continued to smoke had about a 50% higher chance of a major cardiovascular event (death, myocardial infarction, stroke or cardiac arrest) than patients who quit.\textsuperscript{42} Similarly, poorer treatment outcomes have been reported for people who continue to smoke after coronary artery bypass grafting (CABG)\textsuperscript{43} or a diagnosis of heart failure,\textsuperscript{44} compared with people who quit.

### 3.15.4 Cancer

As detailed in Section 3.5, smoking causes numerous cancers. A review has concluded that overall survival is poorer in smokers with cancer. Studies of lung cancer, prostate cancer, cervical cancer, and head and neck cancer were cited.\textsuperscript{45} All these studies involved radiotherapy, leading to suggestions that radiotherapy is less effective in smokers. Higher irradiation complication rates for smokers in the studies covered by the review, and other studies,\textsuperscript{46, 47} support this suggestion. The review also found that the risk of secondary primary tumours is increased in smokers, for malignancies that are related to cancer and also for those that are not.\textsuperscript{45}
The review found little information about the impact of smoking on chemotherapy for cancer, but suggested that treatment outcomes are likely to be worse because of the effects of cigarette smoke on immune function, appetite and basal metabolic rate.\textsuperscript{45}

More severe pain has been associated with smoking in patients with cancer,\textsuperscript{48} and specifically for lung cancer\textsuperscript{49} and head and neck cancer.\textsuperscript{50} This may be because of the decreased effectiveness of opioids (due to induction of liver enzymes by components of cigarette smoke) described in Chapter 13, Section 13.15.1.1, and failure to increase the dose in response.

The 2014 Surgeon General’s report was the first in its series to review the associations between cigarette smoking and health outcomes in cancer patients and survivors. It concluded that smoking causes adverse health outcomes in people with cancer, while cessation improves their prognosis. Smoking increases all-cause mortality and cancer-specific mortality in cancer patients and survivors, and increases the risk for second primary cancers that are caused by cigarette smoking, such as lung cancer. Smoking is also associated with an increased risk of recurrence, poorer response to treatment, and increased treatment-related toxicity.\textsuperscript{51}

A review of smoking cessation interventions in patients with cancer found that interventions increase quit rates but the difference was not statistically significant. Given the benefits of quitting on cancer outcomes, the authors called for research to identify more effective interventions for cancer patients.\textsuperscript{48}

### 3.15.5 Treatment of infertility including assisted reproduction

As detailed in Section 3.2, women who smoke have reduced fertility and there is emerging evidence that fertility may also be reduced in male smokers. Smoking also has a negative impact on the outcomes of infertility treatment.\textsuperscript{53} In women participating in assisted reproduction programmes, a meta-analysis found that smoking is associated with lower pregnancy rates, higher chances of miscarriage and of ectopic pregnancy, and a lower probability of a live birth.\textsuperscript{54}

One study found that for couples who smoked (either female, male or both), the risk of not achieving a pregnancy was about twice as high as for non-smokers.\textsuperscript{55} Female smoking during the period of infertility treatment has been associated with a decreased number of retrieved ova\textsuperscript{56} and a higher risk of repeated tubal ectopic pregnancies;\textsuperscript{57} male smoking has been associated with decreased live birth rates.\textsuperscript{56} Researchers have estimated that women who smoke need up to twice the number of in vitro fertilisation (IVF) cycles to conceive and suggest there is a correlation between the number of smoking years and the risk of not conceiving through IVF.\textsuperscript{53} Smoking cessation for both women and men is recommended for couples aiming to become pregnant\textsuperscript{56} and it has been suggested that access to fertility treatment should be conditional on quitting smoking.\textsuperscript{53}

### 3.15.6 Contraception

As detailed in Section 3.2, smoking causes coronary heart disease, increasing the risk two- to four-fold.\textsuperscript{1} The ‘combined’ oral contraceptive pill (which contains the hormone oestrogen) also increases the risk of myocardial infarction two-fold.\textsuperscript{59} Women who both take the oral contraceptive pill and smoke have a 20-fold increase in the risk of coronary heart disease, compared with non-smokers who are not taking ‘the pill’.\textsuperscript{60} The impact of smoking and the contraceptive pill is therefore ‘synergistic’, meaning that the risk of disease is multiplicative rather than additive. Heavier smokers have an even higher risk of coronary heart disease.\textsuperscript{61}

Although the newer ‘lower dose’ versions of the pill may be associated with a lesser risk of developing coronary heart disease, risk is still elevated in smokers. There is insufficient evidence to evaluate the risk profile of the ‘third-generation’ pills (containing 30 ÕŒg or less of ethynyl estradiol and either gestodene or desogestrel) combined with smoking, but clinicians are advised to be wary when prescribing oral contraceptives to smokers aged in their mid-30s and to exercise extreme caution or avoid using them altogether in smokers aged over 40 years.\textsuperscript{51}
In past decades the risk of stroke, particularly subarachnoid haemorrhage, has been significantly higher among smokers using the contraceptive pill. However research published since the 1990s following up women using lower dose pills is conflicting; some studies show increased risk, other studies have shown no significant effect.\textsuperscript{61}

There is some evidence to suggest that the combined contraceptive pill has a higher failure rate in smokers than in non-smokers.\textsuperscript{60}

### 3.15.7 Other conditions

A meta-analysis published in 2011 found that in patients receiving long-term haemodialysis or peritoneal dialysis, smoking increased the death rate (all-cause mortality) by 65%.\textsuperscript{62}

### References


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Diabetes mellitus (diabetes) is an umbrella term for a number of metabolic diseases which affect the body's ability to control blood glucose levels; it is a disease marked by high blood glucose levels resulting from defective insulin production, insulin action or both. The hormone insulin is produced in the pancreas, and helps the body use glucose for energy. If insulin production or the effectiveness of an individual's insulin is impaired, then diabetes may result.¹

There are three major types of diabetes: type 1 (sometimes referred to as 'insulin dependent diabetes'), type 2 (sometimes referred to as 'non-insulin dependent' or 'adult onset' diabetes); and gestational diabetes. Type 1 diabetes most often occurs in childhood or young adulthood (though it can occur at any age), and is the result of low levels of or the inability to produce insulin. People with type 1 diabetes need insulin replacement for survival.¹ Based on data from the 2007–08 National Health Survey, 10% of people with diabetes reported that they had type 1, while the majority (88%) of people with diabetes reported type 2. Another 2% of people reported diabetes, but did not know which type.² These figures correspond to an estimated 818 200 persons (4% of the population) in 2007–08 with diabetes mellitus who had been medically diagnosed (excluding those with gestational diabetes).²

As noted, type 2 is the most common form of diabetes; it occurs mostly in people aged 40 years and over and is marked by reduced or less effective insulin. Although uncommon in childhood, it is becoming increasingly recognised in that younger age group.¹ Gestational diabetes, the onset of diabetes in pregnancy, occurs in a small proportion of otherwise unaffected women and is usually transient, although women who develop gestational diabetes have a higher risk of developing type 2 diabetes later in life. The Australasian Diabetes in Pregnancy Society estimates that about 5% of pregnant women are affected by gestational diabetes.³

Some population groups are at much higher risk for diabetes, notably Indigenous Australians, people born overseas, and those subject to the poorest socio-economic circumstances. Aboriginal and Torres Strait Islander peoples are three times as likely as non-Indigenous people to have diabetes and have much greater hospitalisation and death rates than other Australians. Rates of diabetes, hospitalisations and/or mortality are more common among overseas-born people from the South Pacific Islands, Southern Europe, Middle East, North Africa and Southern Asia. Diabetes prevalence and death rates for the worst-off fifth of the population are nearly twice as high as for the best-off fifth of the population.¹

Many factors contribute to the onset and development of diabetes. Type 1 diabetes is believed to be caused by particular biological interactions and exposure to environmental agents among people genetically predisposed to diabetes. Obesity, physical inactivity and unhealthy diet play a role in the onset of type 2 diabetes, as well as genetic predisposition such as family history, ethnic background and age. There is some evidence that depression can increase the risk of developing type 2 diabetes and diabetes complications. It
is thought that the increased risk of type 2 may be due to elevated stress levels and weight gain. Poor foetal nutrition leading to low birthweight for gestational age may predispose some individuals to type 2 diabetes. If these individuals are exposed to other risk factors (such as obesity and physical inactivity) the likelihood of developing type 2 diabetes becomes greater.1

The risk factors for gestational diabetes are similar to those for type 2 diabetes: women are at higher risk if they are of relatively advanced age or obese when pregnant. There are also a number of additional risk factors for diabetes complications, including high blood pressure, high blood cholesterol and tobacco smoking. The "metabolic syndrome"—the clustering of a number of risk factors including abdominal obesity, impaired fasting blood glucose, raised blood pressure, raised blood triglycerides and reduced blood HDL-cholesterol—substantially increases the risk of type 2 diabetes.1

As well as being life threatening in its own right, diabetes can lead to a range of other serious health problems, including coronary heart disease, stroke, peripheral vascular disease, kidney disease, eye disease, and complications in pregnancy and childbirth.4 Smoking greatly increases the risk of pancreatic cancer in patients with diabetes mellitus and there is evidence that a combined risk of family history of pancreatic cancer, current smoking and current diabetes mellitus confers a 10-fold increase in risk of being diagnosed with this cancer.5, 6 Among male cancer survivors there is evidence that a history of smoking before diagnosis, obesity and insulin resistance increase the risk for several second primary cancers, indicating the need for screening for second primary cancers among cancer survivors with these risk factors.5 Smoking is related to low bone mass and increased risk of fracture risk in postmenopausal women in the general population, but recent evidence suggests that women with diabetes who are current smokers have more than a three-fold increase in risk (3.47; 95% CI, 1.82–6.62) of non-vertebral fractures than diabetic women who were never smokers.8

Among the lifestyle-related factors, smoking makes the largest contribution to the absolute risk of macrovascular complications for people with diabetes. The added risk from smoking is greater than in people without diabetes.9 Smokers with type 1 and type 2 diabetes are at increased risk of illness and premature death, mostly through development of cardiovascular disease, but other disease processes associated with diabetes may also be made worse by smoking. Smokers with type 1 diabetes in particular may have a higher risk of developing kidney disease, and possibly eye and nerve damage as well, whereas smokers with type 2 diabetes are more likely to increase their risk of coronary heart disease, stroke and peripheral vascular disease. Studies of individuals with diabetes consistently demonstrate that smokers have a heightened risk of cardiovascular disease, premature death and increased rate of microvascular complications of diabetes.4

The 2014 US Surgeon General’s report concluded that smoking causes type 2 diabetes, with the risk of developing diabetes 30–40% higher for active smokers than nonsmokers. Further, there is a positive dose-response relationship between the number of cigarettes smoked and the risk of developing diabetes. The report highlights that reducing tobacco use should be promoted as a key public health strategy to prevent and control the increasing worldwide epidemic of diabetes.10

Plausible biological mechanisms for this association include increase central obesity in smokers, increased inflammation and oxidative stress,10 increased insulin resistance, altered insulin secretion and other impairments to pancreatic function noted in smokers.11 Further corroboration for the Surgeon General's finding that smokers are more likely to develop type 2 diabetes than non-smokers has been provided in a 2007 systematic review11 and recent studies conducted in Japan,12 Korea,13,14 Taiwan,15 China16 and the US.11

There is also evidence that exposure to secondhand smoke is positively and independently associated with the risk of type 2 diabetes, from several observational studies recently conducted in the US,17,18 Germany19 and Japan.20 Some recent evidence suggests that smoking cessation among people with diabetes can lead to short-term increased risk of diabetes (probably because of weight gain) and that this may deter smokers with diabetes from attempting to quit.21,22 The research indicates that any temporary increase in risk may occur in the first three years after quitting, thereafter gradually decreasing to zero.21 Other studies also report that the risk of type 2 diabetes in former smokers returns to that of non-smokers after a number of years.10 A 2015 retrospective cohort study of more than 10,000 adults found that smoking cessation is associated with
deterioration in glycaemic control in smokers with type 2 diabetes, which lasts for 3 years and is unrelated to weight gain.\textsuperscript{23} This evidence underscores the need for smoking cessation to be accompanied by other strategies for diabetes prevention and early detection, as recommended in current clinical guidelines in the US and in Australia.\textsuperscript{4,9}

In conclusion, cigarette smoking produces insulin resistance and chronic inflammation, which can accelerate macrovascular and microvascular complications, including nephropathy. Many clinical and experimental studies have found significant associations between cigarette smoking and development of diabetes, impaired glycaemic control, and diabetic complications (microvascular and macrovascular). A different lifestyle of smokers, in contrast to that maintained by non-smokers, may also contribute to these effects. The development of type 2 diabetes is yet another harmful consequence of cigarette smoking, and one that adds to the heightened risks of CVD; smoking cessation is crucial to facilitating glycaemic control and limiting development of complications.\textsuperscript{24}

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3.17 Inflammatory conditions and autoimmune disease

The role of smoking in cancer, lung diseases and cardiovascular diseases is now widely recognised. The fact that smoking also affects the immune system is less well known. In fact, smoking has wide ranging and severe impacts on the immune system: it increases the risk of a number of allergic conditions, increases the incidence of autoimmune diseases, decreases innate and acquired immunity and increases infection rates. The 2014 US Surgeon General’s report concluded that components of cigarette smoke have both immune activating and immune-suppressive effects. Smoking compromises the immune system and immune homeostasis, which relates to a heightened risk for pulmonary infections and several disorders with an underlying immune predisposition.

Cigarette smoke triggers a systemic inflammatory response, through the release and inhibition of pro-inflammatory and anti-inflammatory molecules. These molecules are often referred to as cytokines or mediators or immunomodulating agents. For example, cigarette smoke induces the release of inflammatory cytokines such as TNF-α, interleukin (IL)-1, IL-6, IL-8 and granulocyte-macrophage colony-stimulating factor (GM-CSF). It increases production of endotoxin, one of the most potent inflammatory agents. It increases polymorphonuclear neutrophils (PMNs) with consequential adverse effects on the respiratory passage. A review of evidence about possible molecular pathways for smoking’s impact on inflammation concluded that activation of the nuclear factor kappa B (NF-kB) family is the main mechanism.

3.17.1 Rheumatoid arthritis

Rheumatoid arthritis is a systemic autoimmune disease characterised by disabling and painful destruction of the joints, and sometimes inflammation of the lungs and other organs. The incidence is about three-fold higher in women than men. A large proportion of patients (but not all) have the rheumatoid factor (RF) antibody.

Smoking causes rheumatoid arthritis. The risk is most markedly elevated in men who are RF-positive (RF is a type of antibody). There have been suggestions that smoking only increases susceptibility to rheumatoid arthritis in individuals who have specific genetic profiles.

A number of studies have reported a poorer response to drug treatments in patients with rheumatoid arthritis who continue to smoke. Smoking reduces the effectiveness of tumour necrosis factor-alpha (TNF-a) inhibitors, a type of immunomodulatory drug that is currently used to treat rheumatoid arthritis.
arthritis sufferers have a higher mortality rate than the general population. Recent cohort studies have found this excess mortality confined to patients who are RF-positive.\textsuperscript{7,8} It has therefore been suggested that this excess mortality could be due to smoking.\textsuperscript{8}

3.17.2 Anal abscess and fistula

Anal abscess is an inflammatory, fistulising cutaneous disease. A small case-control study in the US of 74 patients with anal abscess/fistula found that smoking within the previous year doubled the risk of this condition.\textsuperscript{9}

3.17.3 Graves' ophthalmology

(See Eye 3.10.3)

3.17.4 Psoriasis

(See Skin 3.14.4)

3.17.5 Lupus erythematosus

(See Skin 3.14.5)

References


3.18 Other conditions with possible links to smoking

This section provides information about the many other conditions (in addition to those discussed in Sections 3.1 to 3.17) that have been linked to smoking. The list of conditions discussed in this section is comprehensive but not exhaustive; because cigarette smoke can adversely affect most, if not all, organs of the body, the list of diseases that may be caused by tobacco is still growing.

Generally, causality between smoking and the conditions discussed in this section has not been definitively established. Before a causal link is confirmed by expert bodies such as the US Surgeon General's office, a plausible biological mechanism and multiple studies reporting the association (with large numbers of subjects, unbiased design and confounding controlled) are needed.

3.18.1 Mental illnesses

People with mood disorders or mental illness have a higher prevalence of smoking than the general population, and account for a large proportion of smokers. More than 32% of current smokers report some sort of mental health problem in the last 12 months, compared to about 18% of ex-smokers and 16% of people who have never smoked.

Smokers often report that smoking allays anxiety and has an antidepressant effect, but many studies suggest the reverse association, i.e. that smoking leads to anxiety, bruxism (teeth clenching and grinding), panic attacks, depression, suicide attempts, and schizophrenia. The difficulties involved in establishing the causal direction of the association between mental disorders and smoking have been well summarised (specifically for depression) by Munafò and Araya, who comment:

> Depression may cause people to smoke (perhaps to self-medicate their symptoms), or smoking may cause increased risk of depression (via alterations to neurotransmitter pathways following chronic exposure). The relationship may even be bidirectional (acute or infrequent tobacco use may reduce negative affect, but chronic use may exacerbate it), or be caused by shared risk factors (possibly genetic) so that the relationship is not causal at all.

Large longitudinal studies of smoking and mental health in Norway, New Zealand, Copenhagen and Finland have enabled researchers to account for previous mental illness in their analyses. These studies support the claim that smoking increases the risk of anxiety, depression and suicidality, especially in nicotine-dependent (i.e. heavy) smokers. Munafò and Araya point out that this creates a paradox, namely, that
smoking may cause depression, but smokers say they smoke to alleviate depression. Munafò and Araya suggest that because of nicotine's short half-life, and the consequential speed with which withdrawal symptoms appear in heavy smokers who stop smoking, acute abstinence is associated with anxiety. Smoking alleviates this anxiety. There is evidence that after a few weeks of smoking cessation the withdrawal syndrome ends and mood elevates above that reached when the individual was smoking.

The association between smoking and schizophrenia is different from the link between smoking and mood disorders. A meta-analysis of studies from 20 countries found consistent evidence that schizophrenia patients have a biological predisposition to smoke; genetic factors increase the risk of both becoming a smoker and developing schizophrenia. Schizophrenia is also associated with greater frequencies of heavy smoking and high nicotine dependence.

3.18.2 Neurological diseases

Smoking may be a precipitating factor for migraine and smokers may be at increased risk of developing cranial autonomic symptoms (for example, facial sweating) during an attack.

An association between smoking and hearing loss has been suggested. A case–control study in the US, which included more than 3 000 cases, found only a very small, marginally statistically significant increase in risk associated with smoking.

An analysis of the Nurses Health Study II in the US reported an increased risk of seizures associated with smoking.

Amyotrophic lateral sclerosis (ALS) is a progressive neurodegenerative condition; it is a type of motor neurone disease. A 2010 meta-analysis of 15 case–control and five cohort studies found that smoking increases the risk of ALS in women, but not in men. However a 2011 pooled analysis of data from more than half a million men and more than half a million women enrolled in five prospective cohort studies in the US found that smoking increases the risk of ALS by about 40% for both men and women. This large study therefore strongly supports the existence of a link between smoking and ALS. The risk in smokers increased with decreasing age at smoking initiation. Smoking has also been reported to decrease survival rates in female smokers.

Multiple sclerosis (MS) is a disease in which the myelin sheaths surrounding nerves in the brain and spinal cord become damaged and are eventually destroyed through an autoimmune process. A 2011 meta-analysis investigating the possible link between smoking and MS pooled data on more than 3 000 cases from 14 studies. The study found that smoking increases the risk of MS by about 50%. Smoking has also been reported to accelerate the clinical progression of MS, and the progression of the typical disease lesions visible on magnetic resonance images. However, a review article in 2011 looking at smoking and the onset and progression of MS found that while most of the studies on onset supported a positive association, the evidence on progression was more limited and mixed. The meta-analysis also analysed four studies that addressed this issue. Smoking did appear to increase the risk of progression from relapsing-remitting MS to secondary progressive disease, but the magnitude of the effect varied between studies and the pooled result was not statistically significant. Evidence on a potential mechanism is also limited.

3.18.3 Kidney disease

Smoking has physiological effects on the kidney. It has been reported to increase the glomerular filtration rate, possibly by relaxing renal arteries. There is evidence that smoking increases the risk of developing chronic kidney disease. For example, a 10-year follow-up study of more than 100 000 Japanese people found that smoking increased the risk of developing proteinuria and renal dysfunction. A case–control study in Syria found that smokers had a higher risk of hypertensive nephropathy and diabetic nephropathy, but the risk of kidney disease with other aetiologies was not increased by smoking.
3.18.4 Other conditions

Recent studies have suggested that smoking may worsen quality of life for patients with hepatitis C and increase the risk of moderate to severe hepatic lesions, which in turn hastens progression to cirrhosis. Impaired liver metabolism of nicotine and smoking-induced hypoxia are possible mechanisms.

Cystic fibrosis (CF) is a heritable disorder that severely affects the lungs and digestive system. Studies have shown that smoking worsens CF, and predisposes people with CF to infection. Children with CF exposed to secondhand smoke experience higher rates of hospital admissions and increased use of antibiotics. The mechanism underlying these associations is likely the suppression of antimicrobial host defenses, which is compromised already in people with CF.

References


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3.19 Smoking and accidents

This section covers two sorts of accidents: accidents caused by the distraction of smoking while engaged in driving or other activities, and burns caused by cigarettes or sustained in fires caused by discarded cigarettes. For further information about measures to reduce cigarette-caused fires, see Chapter 12, Attachment A12.2 concerning reduced fire risk cigarettes.

3.19.1 Smoking, motor vehicle crashes and other injuries

Driver distraction is an important cause of motor vehicle crashes. Loss of concentration (thinking about other things or daydreaming), adjusting controls for accessories in the car, and being distracted by passengers or people or events outside the car are common causes of driver distraction. In research from New South Wales and Western Australia examining driver distraction and road safety, 10% of drivers reported that they had smoked during their most recent driving trip of five minutes or more duration, ahead of 9% who had used mobile telephones and 6% who had eaten while driving. A study using video analysis of people driving while smoking suggests an average of measured driving distraction time to be about 12 seconds, or enough to cover a distance of 160 m at a speed of 50 km/h. The authors suggest that distraction of drivers through smoking may be greater in the case of mobile phone use and that it constitutes a remarkable risk for road safety. A small study conducted among Italian adolescents compared those who had not experienced any motor vehicle accidents with those having one or more crash; it reported that the latter were more likely to be tobacco users and the adjusted analyses found that tobacco use was independently predictive of a motor vehicle accident (OR 3.2, \( p < 0.0001 \)). Similarly, North American research among teenagers and young adults found that being a current smoker was associated with having been in a crash, while Canadian researchers found that smokers are more likely to have a car crash than non-smokers, whether or not they are actually smoking at the time of the incident. The Canadian study speculates that as well as the distraction factor, smokers may suffer physiological impairment due to smoking, or that there may be underlying behavioural differences between smokers and non-smokers that contribute to the difference in crash data. Whatever the explanation, Australian reviews have concluded that smoking while driving increases the risk of having a motor vehicle crash. This is consistent with findings from an earlier review by North American researchers that smokers are 1.5 times more likely than non-smokers to have a motor vehicle crash. Being a waterpipe and/or cigarette smoker was found to predict the number of traffic crashes in an adjusted analysis within a recent study of drivers in Iran. Other Italian research has estimated that about 7% of car injuries in that country may involve a subject who smokes while driving, while analysis of US motor vehicle crash data concluded that distraction caused by smoking may be responsible for almost 1% of car crashes over the five-year period 1995–1999, or about 12 780 crashes. Smokers are also more likely to be die from injury in motor vehicle crashes and other types of accidents,
including those involving falls, fires and other unintentional injuries.\textsuperscript{12-14} Possible reasons for this include the effects of smoking on physical performance (such as strength, agility, balance and speed) and recovery from physical trauma (such as post-operative complications and wound healing).\textsuperscript{14} A meta-analysis of randomised, controlled trials was conducted to examine whether cigarette smoking causes, and smoking cessation prevents, excessive injury burden. Intervention (cessation) was associated with pooled estimated injury risk reduction of 35\% within the trials (RR 0.65; 95\% CI, 0.36–1.19) and of 32\% (RR 0.68; 95\% CI, 0.43–1.09) with additional follow-up in two of the three studies; it should be noted that these associations were only of borderline statistical significance.\textsuperscript{15}

### 3.19.2 Burns and fires caused by tobacco use

Cigarettes and cigarette lighters have been shown to be a major cause of burn injury; globally they are responsible for one million fires per year.\textsuperscript{16} In the US, fires and burns are among the top 10 leading causes of unintentional death, with thousands of deaths occurring annually; the majority of these deaths and injuries occur in residential fires, and smoking has been identified as the leading cause of home fire deaths in the US.\textsuperscript{17, 18} Some data suggest that the rate of injuries is higher for fires that were started by smoking, heating equipment, or children playing with fire (relative risk, 2.6).\textsuperscript{19} There is evidence that reductions in smoking and increases in cigarette prices are associated with fewer fires.\textsuperscript{18} Smokers engage in behaviours such as smoking in bed and leaving lit cigarettes unattended that may place them at an increased risk of cigarette-caused fires; in a Canadian study 1 in 4 smokers admitted to leaving lit cigarettes unattended in the previous month, while 15\% admitted to smoking while in bed.\textsuperscript{20} It is a sad irony that smoking also compromises the prognosis of patients with severe burn injury.\textsuperscript{21}

Smoking is conservatively estimated to be the direct cause of at least 4574 fires in Australia each year, the real number probably being much higher.\textsuperscript{22} It is estimated that in 2004–05, 24 people died in Australia due to fires caused by cigarettes, and that nearly a quarter (23\%) of all deaths caused by fire are due to cigarette use.\textsuperscript{23} The National Coroners’ Information System has reported that between the financial years 2000–01 and 2005–06, 67 deaths were caused in Australia by cigarette-related fires.\textsuperscript{24} The authors of this report emphasise that this is highly likely to be an under-representation of the true number of deaths, particularly for the more recent years reported, since cases not concluded or as yet uncoded in their national database are not accounted for in the data. In a recent New Zealand study (conducted among callers to the national smoking cessation service) 6.8\% reported one or more fires caused by cigarettes, 60\% described at least one cigarette-caused burn and 5.2\% reported burns which required medical attention.\textsuperscript{25} Studies have emphasised the particular fire risk associated with smoking in long-term care settings such as nursing homes,\textsuperscript{26, 27} and in relation to the usage of certain highly flammable products such as liquid petroleum gas (LPG),\textsuperscript{28} automatic air-fresheners,\textsuperscript{29} or equipment used for home oxygen therapy.\textsuperscript{30, 31} In-car cigarette lighters have also been reported as a cause of burn injuries.\textsuperscript{32}

The role of smoking-related materials in causing fires has led to demands for tobacco manufacturers to introduce ‘reduced ignition propensity’ (RIP) cigarettes, which only burn while being actively inhaled upon, as opposed to when they are left idling between puffs, or after they have been discarded.\textsuperscript{22} Research into the ignition propensity of cigarettes has grown, notably during the past decade.\textsuperscript{22, 33-45} Among the research results are the important findings that RIP cigarettes do not adversely impact public perceptions about the need for safety,\textsuperscript{33} appear to reduce consumption although resulting in small increases in smoker exposure to the compound phenanthrene,\textsuperscript{35} may have little change in the carcinogenic aspects of particulate matter\textsuperscript{44} and tend to reduce risk behaviours such as leaving a cigarette burning unattended and smoking in bed.\textsuperscript{36} Recently a systematic review of the public health, scientific, technological, trade literatures and internal industry information has been made available following the Master Settlement Agreement between US states and tobacco companies. It reveals that the industry has made advancements in understanding the key parameters involved in cigarette smouldering combustion and ignition of substrates, developing new cigarette and paper wrapper designs to reduce ignition propensity, including banded and non-banded cigarette paper approaches, assessing toxicology, and measuring performance. It is possible that this technical knowledge, now in the public domain, will in the future allow further improvements in the fire safety aspects of cigarettes.\textsuperscript{38}
For further discussion about regulation of tobacco products, see Chapter 12.

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3.20 Tobacco poisoning

Smokers subject their bodies to continued exposures to low amounts of nicotine, leading to tolerance. However dizziness, nausea and vomiting may occur in response to tobacco use before tolerance is established. These symptoms also accompany cases of acute nicotine poisoning, which may occur through ingestion of tobacco or other products containing nicotine (such as pesticides or nicotine-replacement medications) or through absorption of nicotine through the skin, either from exposure to pesticides, unprocessed tobacco leaves (see ‘green tobacco sickness’ below), or nicotine replacement medications.

Symptoms of mild nicotine poisoning may include nausea and vomiting, progressing with increased exposure to cholinergic syndrome, which includes diarrhoea, increased salivation, increased respiratory secretions, and bradycardia (slow heart rate). Severe poisonings can lead to seizures and respiratory depression. Death may occur through respiratory failure. Although highly toxic, death due to ingested tobacco is extremely rare due to the unpleasant flavour of tobacco, the vomit response and early metabolism of the nicotine.

3.20.1 Ingestion

A study of cigarette or cigarette butt ingestion by children as reported to a state Poison Control Centre in the United States has shown that vomiting is the most common response, and that significant toxicity is rare. Cases have also been reported in which children have ingested or had transdermal exposure to nicotine replacement therapy patches, causing symptoms of nicotine poisoning and, in more severe cases, requiring hospitalisation. Reports of ingestion of novel smokeless nicotine products have also increased since the availability of these on the market in the US.

Solutions containing nicotine from cigarettes have also been reported in suicide attempts.

Nicotine is a scheduled poison in Australia, its distribution being controlled by state and territory drugs and poisons legislation, all of which refer to a nationally-accepted Standard for Uniform Scheduling of Drugs and Poisons devised by the Therapeutic Goods Administration of the Australian Government. In an exemption considered by many public health interests to be anomalous, tobacco prepared and packed for smoking is excluded from the standard. See Chapter 12 for further information.

3.20.1 Green tobacco sickness

Green tobacco sickness (GTS) affects individuals involved in tobacco farming, especially during the harvesting season. GTS occurs when nicotine is absorbed through the skin from direct contact with...
tobacco leaves and enters the bloodstream. Sufferers commonly experience dizziness, nausea, headache and vomiting; less frequent symptoms include abdominal pain, shortness of breath, diarrhoea, altered heart rate and blood pressure, sweating and increased salivation.\textsuperscript{10, 11} Non-smokers are more likely to be affected by GTS than smokers,\textsuperscript{12} which has in some cases lead to tobacco growers encouraging workers to take up smoking.\textsuperscript{11} GTS is treated with rest and rehydration, and treatment of additional symptoms if required. The incidence of GTS is reduced by provision of appropriate protective clothing and other workplace safety measures.\textsuperscript{10 11}

GTS is common, a recent international review reporting that 8–89\% of tobacco harvesters may be affected in the course of a season, this wide variation probably being due to differences between study methodologies as well as a range of working conditions.\textsuperscript{11} There are an estimated 33 million tobacco farm workers in the world, a substantial proportion living in developing countries. Long-term health outcomes for individuals exposed to nicotine transdermally for extended periods of time are not known.\textsuperscript{11}

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3.21 Health effects for younger smokers

Most of the risk of dying prematurely due to smoking is reversed if people quit smoking before the age of 30.\(^1\) However smoking during childhood and adolescence also causes a range of immediate health problems, as well as laying the foundation for the development of serious disease in adulthood.\(^2\)

3.21.1 Early signs of addiction

There is contention around how best to characterise nicotine dependence in young people, with increasing recognition that it may be inappropriate to extend adult criteria to adolescent smokers. Recent research has also highlighted qualitative differences between adolescents and adults in their experiences of withdrawal; craving tends to be the predominant symptom that is experienced by young people during abstinence, while withdrawal symptoms are minimal.\(^2\) Nonetheless, evidence shows that nicotine addiction can be developed rapidly by young people, with adolescent smokers reporting some symptoms of dependence at even low levels of cigarette consumption.\(^3\) The majority of people who begin to use tobacco products regularly have great trouble breaking this addiction.\(^3\) Factors influencing smoking behaviour in young people are discussed in Chapter 5. Research in Victoria tracking the smoking career of a large cohort of teenagers over a 10-year period has found a greater likelihood of substance abuse (especially cannabis dependence) and psychiatric illness in continuing smokers.\(^4\)

3.21.2 Respiratory infections and exacerbation of asthma

Active smoking is associated with an increased risk for developing asthma and for exacerbating existing asthma in adolescents.\(^5\) Smoking also causes wheezing severe enough to be diagnosed as asthma in children and adolescents.\(^2\) Surveys among adolescent smokers (12–14 year olds) have found active smoking to be associated with asthma/wheezing and rhinitis,\(^6,7\) particularly in girls,\(^7\) and with asthma-related wheezing symptoms in 15–16 year old adolescents.\(^8\)

Active smoking causes respiratory symptoms including shortness of breath, coughing, phlegm production and wheezing in children and adolescents.\(^19\) Even occasional smoking (on at least 5 days in the prior 30 days) has been found to be associated with shortness of breath/fatigue following regular activity in 18–24 year old college students,\(^9\) while regular smokers among this group were more likely than non-smokers to report having any cough or sore throat in the past 30 days.\(^8\) The prevalence of self-reported bronchitis symptoms (chronic cough and sputum production) among a cohort of 18–21 year old Finnish males was
significantly higher among daily smokers than occasional smokers, and symptoms were significantly associated with smoking history.10

3.21.3 General health of young smokers

Young smokers are more likely to report suffering an overall diminished level of health compared with non-smokers.11 Recurrent headache has been associated with current smoking (daily and occasional) in Norwegian students aged 13–18 years,12 while among US students (grades 6 to 10), adolescent daily and experimental smokers were more likely than never smokers to report recurrent subjective health complaints such as headache and backache.13 Among a cohort of young US Navy recruits (average age 19.7 years at baseline), cigarette smoking was a prospective predictor of hospitalisation: data for more than 5000 young healthy female recruits from entry into the Navy for up to 7–8 years of service indicated that daily smokers had higher rates of hospitalisation for any reason and for musculoskeletal conditions.14 Daily smokers were also hospitalised for a significantly greater mean number of days compared with never smokers and other smokers (including experimental, occasional and former smokers), following adjustment for differences in time in service and socio-demographic variables.14

Evidence suggests other health effects among young people associated with current smoking; these include significantly reduced taste sensitivity and atrophic papillary structures compared with non-smokers (all male participants, mean age 24.9 years),15 as well as an association between an increase in some sleep disorders and current smoking in adolescents.16 For example, a study among about 29 000 Chinese adolescents aged 12–18 years found current smoking to be associated with increased snoring, difficulty breathing during sleep and difficulty maintaining sleep compared with never smokers.16

3.21.4 Fitness and lung function in young smokers

Young smokers tend to be less physically fit than their non-smoking peers, fitness declining with increasing levels of tobacco consumption.11 The cumulative effects of smoking from a young age on physical performance in midlife was assessed in a large British cohort study with data on smoking history from age 20 (median age at smoking initiation 16 years).17 Researchers found that at age 53, ever-smokers had significantly poorer overall physical performance, balance and chair rising than never smokers, with performance decreasing significantly for every 10 pack-years smoked.17

A study among a cohort of 18–21 year old Finnish males found levels of aerobic fitness to be significantly lower in regular smokers compared with non-smokers (after controlling for physical activity, education and body mass index) and fitness was associated with smoking history.10 A small study among a group of healthy young smokers (mean age 21.4 years) found that smoking was associated with resting and exercise tachycardia, demonstrating significant and acute negative effects on cardiac autonomic function due to smoking both at rest and during exercise. Results suggested that pre-exercise smoking of a single cigarette impaired physiological response to both peak and sub-maximal exercise; this may greatly increase vulnerability to myocardial electrical instability and therefore predispose to higher risk of cardiovascular events.18

3.21.5 Early signs of lung disease

Lung development is also altered by early tobacco use. Active smoking causes impaired lung growth during childhood and adolescence, and the early onset of lung function decline during late adolescence and early adulthood.19 Young smokers’ lungs stop growing earlier, they attain lower maximal lung function, they have a briefer plateau phase, and their lung function declines earlier. This reduced lung growth can increase the risk of chronic obstructive pulmonary disease later in life. Early quitting may therefore be particularly beneficial, to potentially avoid these effects on growing lungs.2
3.21.6 Early signs of cardiovascular disease

Cigarette smoking during adolescence and young adulthood begins the damaging processes that lead to cardiovascular disease. Damage to the circulatory system becomes evident in young smokers, and may become clinically significant in early adulthood. There is robust evidence demonstrating that smoking during adolescence and young adulthood increases the development of atherosclerosis. By early middle age, the more rapid progression of atherosclerosis and the rapid decline of lung function mentioned above lead to higher rates of coronary heart disease, stroke, and COPD. These diseases play a major role in the premature mortality of middle-aged and elderly smokers. While premenopausal women are typically relatively protected from heart disease compared with men and develop coronary heart disease 10 years later, smoking increases cardiovascular risk in young women and removes the protective effect of the premenopausal state; this is possibly caused by smoking disrupting the normal ovarian pattern of sympathetic nervous system activity.

A study assessing the influence of smoking on blood biochemistry in male Taiwanese university students (mean age 19.4 years) found young smokers had significantly increased risk of several conditions associated with cardiovascular and haematological disorders, including hypertriglyceridaemia, neutrophilia and hyperchromia, compared with non-smokers. There is also evidence suggesting that even occasional smoking on a regular basis (<1 pack per week for at least 1 year) is associated with both acute and chronic impairment of arterial function (related to the development of atherosclerosis and future cardiovascular complications) in otherwise healthy young people (20–26 year olds). Similarly, a study on the effect of chronic smoking on arterial stiffness among young smokers (mean age 24.3 years) compared with non-smokers (mean age 20.2 years) found significantly higher arterial stiffness among smokers, suggesting that the negative effect of cigarette smoking on the vascular system may be apparent even in young smokers who have been smoking for fewer than 10 years. Arterial stiffness is an important factor in the development of a range of pathophysiological processes including atherosclerosis, left ventricular hypertrophy and aneurysm.

Tobacco use among young men, particularly cigarette smoking, is strongly associated with thromboangiitis obliterans (Buerger's disease), a recurrent inflammatory, non-atherosclerotic vasoooclusive disease. Caused by vasculitis (inflammation of the blood vessels), Buerger's disease mostly affects males aged 20–40 (average age of symptom onset about 35 years) with a current or recent history of heavy smoking or chewing tobacco. Typically it involves progressive inflammation and thrombosis (clotting) of blood vessels of the hands and feet, and the development of ulcers and gangrene of extremities as a result of vascular ischaemia. Commonly necessitating amputation, major amputations (of limbs rather than fingers/toes) are almost twice as likely in patients who continue to smoke. Buerger's disease has also been documented to involve scrotal and penile necrosis resulting in partial penectomy and scrotal debridement. The only apparent therapeutic measure to slow or prevent disease progression is smoking cessation.

Smoking has been identified as one of the major risk factors for hospital admission for acute coronary syndrome and more severe acute myocardial infarction (AMI) among Middle Eastern and Swedish populations, particularly among younger patients (≤40 years old; 25–<65 years old). A prospective study to evaluate the impact of smoking habits on long-term outcome in individuals who sustained AMI at the age of ≤35 years found that the most common risk factor at initial presentation was smoking. During follow-up (for up to 10 years) most patients (55.6%) reported continuing to smoke. One-third (32.6%) presented cardiac events during the follow-up period, including readmission for acute coronary syndrome, cardiac death or coronary revascularisation because of clinical deterioration. Continuation of smoking was the most significant predictor of cardiac events during follow-up.

Smoking has also been found to be a determinant of isolated systolic hypertension among younger US adults (18–39 year olds); this is of concern because even small increases in systolic blood pressure in early adulthood increase risk of further cardiovascular disease (CVD) morbidity in later life. Studies of hypertension in young adults have found associations with structural changes in the heart including increases in left ventricular wall thickness, left ventricular mass and higher prevalence of left ventricular hypertrophy.
Particular populations of young smokers may be at greater risk of experiencing tobacco-related adverse health effects, such as youth with chronic conditions because their health is already compromised. For example, people with diabetes mellitus are at increased risk of developing health problems such as CVD: a large US study among young people (10–22 years old) with diabetes mellitus found that current and past smokers were significantly more likely than non-smokers with the same condition to display cardiometabolic risk factors such as high triglyceride levels and to be physically inactive.

Evidence suggests there is a strong dose–response relationship between cigarette smoking and ischemic stroke risk in younger women. For example, a population-based case–control study of risk factors in more than 1000 US women aged 15–49 years found that the OR for ischemic stroke risk comparing current smokers to never smokers was 2.6 ($p<0.0001$), after multivariable adjustment (including for age, race, education, hypertension, diabetes, body mass index, coronary heart disease, oral contraceptive use and elevated total cholesterol); the adjusted OR increased with increasing number of cigarettes smoked per day. No difference in stroke risk was observed between former smokers (those who had smoked more than 100 cigarettes in their lifetime, but had not smoked in the 30 days before their stroke) and never smokers. A prospective cohort study among more than 45 000 Swedish women aged 30–50 years (mean age 40 years) at time of enrolment found that, after an average of 11 years of follow-up, current smoking significantly increased the risk of stroke, particularly for ischemic stroke. There was a dose–response effect of current smoking on all strokes, with those smoking ≥10 cigarettes/day having a three-fold excess risk compared with never smokers. Similar risk patterns were observed using cumulative pack-years as an indicator of smoking exposure. Former smokers had a 60% increased risk for all strokes, which was of borderline statistical significance. The use of the oral contraceptive pill is a risk factor for stroke, and research among teenage girls (15–17 year olds) who have suffered stroke suggests its use has a negative synergistic effect with smoking, risk increasing with the number of cigarettes smoked.

### 3.21.7 Dental health problems in young people

Smoking is also a major risk factor for poor periodontal health and oral cavity diseases; about half of the periodontitis seen in those aged under 30 is thought to be linked to smoking. Daily smoking and infrequent tooth brushing (less than twice a day) among 14–18 year old Finnish adolescents have been found to be strongly associated. There is also evidence to suggest that among young males (20–25 years old), even moderate smoking of 10 cigarettes per day induces variations of salivary lipid pattern. The regulation of salivary lipid levels is important in the maintenance of oral cavity health: elevated lipid levels are associated with an increase of caries incidence, plaque development, calculus formation and periodontal disease.

### 3.21.8 Muscular skeletal problems in young people

While considerable evidence associates tobacco use with low bone mass and increased fracture risk in older people, research has emerged more recently linking smoking at a young age with unfavourable bone geometry and density, reduction in peak bone mass and increased fracture prevalence. A cross-sectional population-based study among 677 healthy male Belgian siblings at the age of peak bone mass (aged 25–45 years, mean age 33.4–35.7 years) found that those who took up smoking at an early age (16 years old or younger) had lower areal bone mineral density (aBMD), lower cortical bone area at the tibia and lower trabecular and cortical bone density at the radius compared with current and never smokers. There were significant negative associations between number of pack-years smoked and lumbar spine, hip and total body aBMD, as well as total body bone mineral content. In addition, self-reported fractures were significantly more prevalent in early and current smokers, after adjustment for age, weight, education and alcohol use, and exclusion of childhood fractures. It has been suggested this may be caused by smoking disrupting the acquisition of peak bone mass during puberty, possibly due to an interaction with sex steroid action.

Similarly, in a large population-based study of more than 1000 young Swedish men (mean age 18.9 years), significantly lower aBMD of the total body, lumbar spine, femoral neck, and trochanter was found among...
current smokers (at least one cigarette per day) compared with non-smokers. The magnitude of observed differences was considerable, including a mean difference of 3.3% in the spine and 5% in the trochanter after adjusting for age, height, weight, calcium intake and physical activity. Volumetric BMD (vBMD) and bone size were also measured: smokers had lower cortical thickness than non-smokers of both the radius and tibia. The authors suggest that the effects of smoking on bone mass may occur quite rapidly, because the mean duration of smoking in this study was 4.1 years.

References


3.22 Poorer quality of life and loss of function

This section is about impacts on health-related quality of life (HRQOL), activities of daily living (ADL) and general health caused by smoking. The discussion here goes beyond the burden of morbidity and mortality from specific diseases that are comprehensively described in other sections. The evidence presented here confirms that exposure to tobacco smoke should be considered an important contributing factor to wide-ranging non-specific morbidity and a diminished quality of life.

3.22.1 Poorer health-related quality of life

The concept of health-related quality of life (HRQOL) and its determinants have evolved since the 1980s to encompass those aspects of overall quality of life that can be clearly shown to affect health—either physical or mental. Several instruments have been used to assess HRQOL and related concepts of functional status. Among them are the Medical Outcomes Study Short Forms (SF-12 and SF-36), the Sickness Impact Profile, and the Quality of Well-Being Scale.1 The SF-36 is the measure most commonly used in the HRQOL studies cited in this chapter.

There is compelling evidence linking active smoking and poor HRQOL; this has been demonstrated in large longitudinal studies conducted in North America,3,4 Spain5 and Finland6; in cross-sectional studies conducted in Croatia,7 Finland8 and North America9; in a large study of Medicare beneficiaries in North America10 and in a British study conducted among female patients with atherosclerosis.11

3.22.2 Impaired activities of daily living (ADL) and instrumental ADL

‘Activities of daily living’ (ADL) are those skills needed in typical daily self-care, while ‘Instrumental activities of daily living’ (IADL) refer to skills beyond basic self-care that evaluate how individuals function within their homes, workplaces and social environments. IADL may include typical domestic tasks such as driving, cleaning, cooking and shopping, as well as other less physically demanding tasks such as operating electronic appliances and handling budgets.12 Researchers use various tools to measure these capacities, such as the Katz or Barthel scales for ADL13 and the Lawton IADL scale.14

A longitudinal study conducted in Japan examined the relationship between smoking in middle age and long-term risk of impaired activities of daily living (ADL) in more than 2000 men and women. The researchers reported more than double the risk of impaired ADL among current smokers compared with non-smokers (OR 2.11; 95% CI, 1.09–4.06). Risk of impaired ADL was higher as the number of cigarettes increased. The
study concluded that smoking in middle age increases future risks of impaired ADL and that smoking cessation may be important to prevent future impairment of ADL. Another, similar longitudinal study was conducted in North America involving more than 10 000 middle-aged people; the study found that smoking was significantly associated with deterioration in ADL status. Impairment in instrumental activities of daily living (IADL) was associated with smoking in a Japanese longitudinal study involving more than 1200 elderly people and a North American cross-sectional study involving more than 9500 subjects. There is also evidence that women with COPD are more than twice as likely to have impaired ADL (OR 2.63; 95% CI, 1.15–5.99), and more than four times as likely to have impaired IADL (OR 4.23; 95% CI, 1.92–9.29).

3.22.3 Smoking, low bone density and hip fracture

A causal relationship exists between smoking, low bone density and hip fractures. The causal relationship with low bone density was previously established for older women only; however there is now systematic review evidence that smoking is a risk factor for low bone mineral density/bone loss among men over 50 years of age as well. These findings linking smoking to the risk of low bone density and hip fracture are important; it is well established that there is a pronounced and long-term reduction in HRQOL as a result of hip fracture and independence in many of the normal activities of daily living is compromised. (See Chapter 3, Section 3.13 for more detailed discussion of musculoskeletal disease.)

3.22.4 Diminished general health, accelerated ageing, disturbed sleep

There is established scientific evidence of a causal relationship between smoking and diminished health status, and this evidence is consistent across studies and indicators. Smokers of different ages, genders, and locations experience poorer physical and mental health compared with people who have never smoked. Manifestations of this diminished overall health include smokers’ increased absenteeism at work, self-reported poorer health, and higher health care costs and utilisation, and these relationships remain after controlling for a broad range of potential confounders. This more general decrement in health may be the result of altered inflammatory/immune processes, oxidative stress and sub-clinical organ injury. However, there are many direct and indirect mechanisms that link smoking to poorer health.

Smoking modifies leukocyte telomere length (TL – or DNA sequencing), which is thought to accelerate biological ageing as well as development of smoking-induced chronic diseases. A range of recent studies support the contention that smoking is associated with accelerated biological ageing. There is evidence that tobacco smoking is a contributing factor in a wide range of skin diseases, and that it is a cause of premature skin ageing with estimates of almost four times the amount of facial wrinkling in ‘heavy’ smokers (>40 packs per year) compared with non-smokers. (This issue is discussed in more detail in Chapter 3, Section 3.14.1.)

A large cross-sectional study conducted in North America found that tobacco use and exposure to secondhand smoke were associated with increased odds of earlier age at menopause.

Cigarette smoking is associated with sleep disorders in the general population; smokers are also more likely to experience sleep disturbances, including taking longer to fall asleep, being less likely to stay asleep, and having less total sleep time than non-smokers. There is also emerging evidence of an association with obstructive sleep apnoea. Recent evidence from a very large cross-sectional study conducted in North America indicates an association between smokeless tobacco use, secondhand smoke exposure and insufficient rest/sleep. Current users of smokeless tobacco were more than 70% (OR 1.74; 95% CI, 1.37–2.22) more likely to report insufficient rest/sleep compared to never smokeless tobacco users. For those who were both current smokers and current smokeless tobacco users there was more than a doubling of this risk (OR 2.21; 95% CI, 1.66–2.94). Those with secondhand smoke exposure had an estimated 29% increased risk for insufficient rest/sleep than those without (OR 1.29; 95% CI, 1.02–1.63).
3.22.5 Other impairments

In the elderly, smoking is associated with accelerated declines in physical function, and increased levels of clinical illness and physical and cognitive impairment.\textsuperscript{20, 43} (See Chapter 3, Section 3.23).

Recent research into the association between back pain and other types of chronic pain and smoking has indicated the possibility of a causal link; this finding is supported by recent prospective studies.\textsuperscript{44} Smokers are also more likely to report pain during health examinations.\textsuperscript{45}

Other studies show that smoking is associated with hearing impairment,\textsuperscript{46-51} (also see Chapter 3, Section 3.25) and poorer sense of taste\textsuperscript{52-55} and smell.\textsuperscript{56, 57} Smoking increases the risk of both cataract and age-related macular degeneration (AMD), responsible for a large burden of vision impairment and blindness (also see Chapter 3, Section 3.10), which impose substantial costs on the Australian community.\textsuperscript{58}

Many other impacts associated with exposure to tobacco smoke have clear implications for HRQOL even if specific confirmatory research to quantify the impairment with precision may not yet be available. For example, dental diseases\textsuperscript{59} are described in Chapter 3, Section 3.11, Gastro-intestinal diseases in Section 3.12, and the impact of smoking on treatment of disease\textsuperscript{60, 61} in Section 3.15.

3.22.6 Smoking and absence from work due to illness

Smoking is associated with the amount and duration of sick leave and degree of productivity loss at work.\textsuperscript{62-64} Smokers are more likely to miss work due to ill-health, have longer duration of absence from work, and access all levels of medical care more frequently.\textsuperscript{65, 20} Level of consumption also plays a role, with heavier smokers having more absences than lighter smokers.\textsuperscript{25} Work absences are reportedly higher in smokers resulting from a broad range of symptoms, including problems with the digestive tract, neck, back and upper limbs.\textsuperscript{65} These effects are evident in younger smokers, before the effects of major tobacco-caused disease become apparent during middle age and later years.\textsuperscript{20} There is also evidence that smokers are more likely to suffer injury in the workplace than non-smokers.\textsuperscript{20}

Australian data show that men who smoke are 66\% more likely to be absent from work than male never smokers, and that female smokers are 23\% more likely to miss work than female never smokers.\textsuperscript{66} This in turn has a major quantifiable economic impact on the nation's productivity.\textsuperscript{67} (Also see Chapter 17, Section 17.2.2.2.)

\textit{i A recent cross-sectional study of 2500 never smokers in Switzerland found that exposure to secondhand smoke was also associated with reduced HRQOL, more significantly so in women. Exposure to secondhand smoke at home and high levels of exposure were associated with lower SF-36 scores, suggesting a dose–response relationship.}

References


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3.23 Smoking, dementia and cognitive decline

Several systematic reviews have concluded that there is a likely association between current smoking and an increased risk of incident dementia, especially Alzheimer's disease, and that smoking may be a risk factor for cognitive decline.\(^1\)\(^2\)\(^3\)

A review of comparable research published up to June 2005 concluded that older smokers may have a greater risk of developing dementia (including Alzheimer's disease and vascular dementia) and cognitive decline than non-smokers.\(^1\) This meta-analysis focused on those over the age of 65 years, and included only longitudinal studies. It showed that current smokers had a 40–80% increased likelihood of experiencing dementia and cognitive decline compared with never smokers. The authors of this review observed that individual lifestyle and physiological factors may also influence the association between smoking, dementia and cognitive decline, and that further research is needed.\(^1\)

A similar systematic review based on longitudinal studies published between 1995 and 2007 examined the relationship between smoking, dementia and cognitive decline in an elderly population (aged 65 years and over).\(^2\) Meta-analyses found current smokers compared with never or non-smokers had a significantly increased risk of Alzheimer's disease and a higher but not significantly increased risk of cognitive decline, vascular and unspecified dementia.\(^2\) Contrary to the findings of a number of early studies suggesting that smoking may be protective against Alzheimer's disease,\(^4\)\(^–\)\(^9\) a systematic review of all research published up to 2007 concluded that current or ever smoking is in fact a significant risk factor for Alzheimer's disease.\(^3\)

The review controlled for study design, quality, secular trend and tobacco industry affiliation of study authors. One-quarter of the 43 studies reviewed had tobacco-affiliated authors. Based on average quality cohort studies with no tobacco industry affiliation published in 2007, the average risk of Alzheimer's disease associated with smoking was estimated to be 1.72 +/− 0.19 (\(p<0.0005\)).\(^3\)

A review of the peer-reviewed literature on the neurocognitive and neurobiological implications of chronic smoking (not defined specifically) concluded there is increasing evidence that chronic cigarette smoking is associated with demonstrable abnormalities in brain neurobiology and neurocognition across the lifespan, and is related to abnormal rates of loss of brain volume in the elderly.\(^10\) Focusing on smoking among cohorts and population-based samples not seeking treatment for substance use or psychiatric disorders, the authors found a likely association between chronic smoking and diminished executive functions, cognitive flexibility, general intellectual abilities, learning and/or memory processing speed, and working memory.\(^10\)

Several prospective cohort studies not included in the above reviews have also examined the effect of smoking in middle age on cognitive decline and dementia.\(^11\)\(^,\)\(^12\)\(^,\)\(^13\) This is partly to address the selection bias caused by differential mortality among smokers when examining the effects of smoking among the elderly.\(^13\)
middle-aged smokers are more likely to be lost to follow-up by death or through non-participation in cognitive tests. These studies provide further evidence of associations between smoking and dementia, Alzheimer's disease, vascular dementia, loss of cognitive flexibility and global cognitive function.

Most recently, Alzheimer's Disease International conducted a large-scale review of the association between lifestyles and dementia risk, and reported robust evidence for a relationship between current smoking (vs. never smoking) and the incidence of Alzheimer's disease. Evidence also suggested (though the relationship was non-statistically significant) a similar association with vascular dementia, and a smaller association with any dementia. Conversely, ex-smokers were at a similar risk to those who have never smoked for all types of incident dementia. These findings are important for prevention and cessation efforts, as the increased risk of dementia might be avoided by quitting smoking.

References


3.24 Genetic influences on tobacco-caused disease

There is evidence that the development of tobacco-caused disease may be mediated by the influence of an individual's genetic make-up. If confirmed, this means that someone with a genetic susceptibility to heart disease (or lung cancer or any of the other many diseases caused by smoking) could be at higher risk of developing these diseases if they smoke, and conversely, that some individuals may theoretically be at less risk of developing certain diseases. These findings have given rise to some debate about whether genetic screening might usefully contribute to the prevention of tobacco-caused disease, but the concept of genetic screening to ascertain individual risk of developing tobacco-related disease is considered to be impractical for the following three reasons:

Firstly, it is likely that many different genes, each contributing only a small portion of the overall risk, influence a smoker's individual risk of developing a particular disease which is caused by smoking. As discussed throughout this chapter, it is already well understood that smokers have a much higher risk than non-smokers of developing many conditions. Given this, it may be that being able to assess an individual smoker's personal risk of developing disease is in fact not much more informative than predicting risk simply on the basis of being a smoker.

Secondly, smoking is the cause of many different diseases. Even to test smokers for susceptibility to the most commonly-caused diseases such as cardiovascular disease, lung cancer and chronic obstructive pulmonary disease, would mean screening for a large number of polymorphisms (common, naturally occurring variations in a gene, DNA sequence or chromosome).

Finally, even if all these processes are eventually elucidated and genetic pathways identified for each kind of disease, it is probable that virtually everybody will have at least one susceptibility gene for one or more smoking-related disease.

Screening for susceptibility to tobacco-caused diseases therefore cannot be regarded as a viable tool for smoking prevention, and from a public health perspective, the only workable message is that smoking is not safe for anyone.
References


A preliminary note of caution about tobacco industry interference is appropriate before embarking on a discussion of other pollutants and health. Tobacco companies have historically sought to distract the public from the issue of secondhand smoke by emphasising the dangers of other pollutants, including carpet glue fumes and car exhaust. A broader discussion of indoor air quality, ventilation and 'sick building syndrome' has served, in some cases, to drown out concerns about the harms of secondhand smoke. According to a Philip Morris publication developed for Europe, the range of pollutants found in offices which cause sick building syndrome include fumes and gases emitted from carpets, computer screens, photocopiers, etc., with the problem often augmented by bacteria, moulds and dusts from ventilation equipment. It has even been argued that tobacco smoke can be a useful visual marker of bad ventilation inside buildings. Tobacco companies have invested heavily in research on air quality issues. Substantial funds have been channelled to outside investigators through scientific organisations and companies focusing on indoor air research that were meant to appear independent and objective, but in fact were run by tobacco industry consultants.\(^1\)\(^-\)\(^3\) The tobacco industry and its interference tactics are discussed more fully in Chapter 10, Section 10.12.

Particulate matter (PM), also known as particle pollution, is the term commonly used in discussion of outdoor air pollution. PM is a complex mixture of extremely small particles and liquid droplets suspended in air. It is made up of a number of components, including acids (such as nitrates and sulphates), organic chemicals, metals, and soil or dust particles and allergens (such as fragments of pollen or mould spores). The size of particles is directly linked to their potential for causing health problems; it is now generally accepted that the very small particles emitted into the surrounding air by combustion and abrasion have a large effect on annual death rates. The evidence points to PM2.5 as the most satisfactory index of particulate air pollution for quantitative assessments of the effects of policy interventions.\(^4\) Fine particle pollution or PM2.5 describes particulate matter that is 2.5 micrometres in diameter and smaller (about 1/30th the diameter of a human hair).\(^5\) A 2008 systematic review of the relation between long-term exposure to ambient air pollution and chronic diseases was conducted by Chen and colleagues.\(^6\) The analysis showed that long-term exposure to PM2.5 increases the risk of non-accidental mortality by 6% per 10 microg/m³ increase, independent of age, gender and geographic region. Exposure to PM2.5 was found to be associated with an increased risk of mortality from lung cancer (range: 15% to 21% per 10 microg/ m³ increase) and total cardiovascular mortality (range: 12% to 14% per 10 microg/ m³ increase). Living close to busy traffic is associated with elevated risks of these three outcomes. Evidence suggested that exposure to PM2.5 is positively associated with mortality from coronary heart diseases and that exposure to sulphur dioxide (SO2) increases mortality from lung cancer. For other pollutants and health outcomes, there are insufficient data to make solid conclusions.\(^6\) Studies examined in the systematic review are available as an online appendix.\(^7\) Virtually all studies covering non-accidental mortality and exposure to PM2.5 have adjusted for the effects of active
smoking; in other words, researchers used statistical techniques that attempted to allow for the adverse health effects of this exposure when estimating the risk from air pollution. One of these studies adjusted for exposure to secondhand smoke; McDonnell et al provided a risk estimate of 1.09 (95% CI, 0.98–1.21) for men, which is only on the cusp of statistical significance while there was a non-significant finding for women. Lung cancer mortality was also examined in this secondhand smoke adjusted study whereby a (non-significant) risk estimate of 1.39 (95% CI, 0.79–2.46) was obtained.\(^8\)

A number of studies provide insights into road traffic-specific component of outdoor air pollution. As noted earlier, a systematic review has concluded that living close to busy traffic is associated with elevated risks of the adverse outcomes associated with exposure to PM2.5.\(^6\) Other recent studies have found that long-term exposure to traffic-related air pollution may contribute to the development of chronic obstructive pulmonary disease with possibly enhanced susceptibility in people with diabetes and asthma;\(^9\) that medium-term exposure to traffic-related air pollution may induce an increased inflammatory/endothelial response, especially among diabetics and those not using statins;\(^10\) and that traffic intensity near the home is associated with natural-cause mortality (highest relative risks for respiratory mortality).\(^11\)

The National Pollutant Inventory holds data for all sources of particulate matter emissions in Australia.\(^12\) Overall, air quality in Australia is relatively good, but for some places, such as large cities and mining areas, air quality can be an issue. The 'headline indicator' for atmosphere in Measures of Australia’s Progress has generally regressed compared with 10 years ago.\(^13\) In recognition of the effects of these pollutants on health and climate, a range of standards has been introduced across Australia over recent decades. The National Environment Protection Measures describe standards for six main pollutants, including PM10 (particulate matter, or inhalable particles, more than 10 microns in diameter). The maximum PM10 concentration allowed under the standard is 50 microg/m\(^3\) and for a maximum of five days a year. (In 2003 the National Environment Protection Measures was varied to add an advisory reporting standard for PM2.5; the advisory standard for PM2.5 is a maximum concentration of 25 microg/m\(^3\) in one day and 8 microg/m\(^3\) per year.) All capital cities except Hobart had PM10 concentrations above the standard between 1991 and 2001. In Melbourne the levels of PM10 remained above the standard from 2001 to 2006, with concentration peaks seen in 2003 and 2006. New South Wales also recorded PM10 levels above the standard from 2001 to 2006, with a peak in 2003. The peaks can be attributed to severe bushfires and dust storms in those years. Most capital cities have shown a fairly steady rate of sulphur dioxide emissions and met the National Environment Protection Measures standards for highest daily average and highest daily maximum between 1991 and 2001. This trend was maintained in Sydney and Melbourne from 2002 to 2006. Until 1996, Adelaide exceeded the standards but since then the levels have been below the standard.\(^14\)

Outdoor air pollution is a significant cause of sickness and death in Australia. It is estimated that in 2003, about 2000 deaths in Australia were attributable to long-term exposure to urban air pollution, and a further 1000 deaths were caused by short-term exposure through exacerbation of pre-existing illness.\(^15\) Most deaths were due to ischaemic heart disease, followed by stroke, lung cancer and chronic obstructive pulmonary disease. In total, urban air pollution caused 2.3% of all deaths in Australia in 2003. Tobacco caused almost five times that amount.\(^15\)

Tobacco smoke is, of course, also a source of environmental particulate matter. A study comparing the output of particulate matter from three single cigarettes burning consecutively over 30 minutes showed that the total delivery of particulate matter was up to 10 times greater than emissions of particulate matter from the exhaust of a modern turbo diesel motor car with the engine left idling for the same amount of time.\(^16\) Tobacco smoke is also a major source of indoor carbon monoxide pollution\(^17\)–see following section.

Policy action to reduce levels of air pollutants is important and it is reasonable to assume that a reduction in air pollution will lead to considerable health benefits.\(^18\) However, since the adverse effects of outdoor air pollution may also be used by the tobacco industry to distract attention from policy measures for secondhand smoke, it is worth noting the recent report by the World Health Organization, which provides risk estimates for exposure to secondhand smoke across a wide range of adverse health outcomes for both children and adults.\(^19\) These well quantified risks from secondhand smoke exposure,\(^19\) the relative ease and low cost of policy measures to reduce or eliminate the risks, and the immediacy of their impact\(^20\) are all powerful arguments that should help ensure a clearer focus in the attention of policy-makers and members of the
public alike.

3.25.2 Indoor pollution

3.25.2.1 Indoor pollution: generic

With Australians spending up to 90% of their time indoors,\(^{21}\) indoor air quality, whether it be domestic or workplace, is an important health consideration. Sources of indoor pollution in Australia include asbestos products, radon gas, secondhand tobacco smoke, house dust mite allergens, formaldehyde (used in production of pressed wood products such as particleboard, or insulation products), nitrogen dioxide emissions from unflued gas appliances, and pesticides applied under buildings.\(^{22}\) Moulds, dust, animal fur or dander (tiny flakes from fur, skin or feathers), and chemicals arising from paint, glues or other household solvents are also a cause of irritation. 'Off-gassing', the emission of toxic fumes from furniture, carpets, paints, glues and sealants in newer buildings and houses may remain at high levels for several months.\(^{21}\) Of course, outdoor pollutants may also infiltrate the indoor environment. Exposures to these agents may cause a variety of responses, from mild irritation through to asthma and disease.

Of all these pollutants, the three most significant in Australia are asbestos fibres, radon gas and secondhand tobacco smoke.\(^{22}\) As already noted, asbestos fibres are a cause of lung cancer and other respiratory disease, and although it is no longer mined and its use in building products has been phased out, asbestos remains present in many structures. Radon gas is also a known human carcinogen that occurs naturally in soil and rocks, collecting in buildings from the soil beneath. Radon levels in Australian buildings are, for the most part, well below internationally recommended indoor levels, and significantly lower than for buildings in the US and the UK, probably due to different soils, building practices and the coastal proximity for much housing.\(^{22}\) Secondhand tobacco smoke causes lung cancer, as well as a range of other respiratory symptoms and illnesses among non-smoking adults and children.\(^{23}\) In past decades, exposure to secondhand smoke has been ubiquitous. Restrictions on smoking in the workplace and in many public places have reduced exposure to secondhand smoke for many Australians over the past 20 years, although exposure in recreational facilities and establishments such as hotels and nightclubs has remained high. A significant number of adults and children also remain exposed in the home (see Chapter 4, Section 4.14). For further information on secondhand smoke refer to Chapter 4. Smokefree environments are discussed in Chapter 15.

3.25.2.2 Indoor pollution: workplace exposure

Workplace exposures to a range of substances can cause illness. For example, environments containing fine particulate matter from grains, flours, plants, coal dust, asbestos, silica, wood, feathers, insects and fungi, drugs and enzymes, chlorofluorocarbons, alcohols, metals and their salts and welding fumes can cause asthma, progressive lung damage and other serious respiratory disease.\(^{24, 25}\) Combining smoking with these exposures may greatly increase disease risk.\(^{25}\)

A well-documented example is the interaction between workplace exposure to asbestos and cigarette smoking. Among the population not exposed to asbestos, smoking increases the lung cancer rate approximately 10-fold. In non-smoking asbestos workers, the lung cancer rate is increased five-fold; but among asbestos workers who smoke, the lung cancer rate is increased 50-fold. In other words, for those workers who both smoke and are exposed to asbestos, the risk of developing and dying from lung cancer is 50 times greater than the risk for individuals who neither smoke nor are exposed to asbestos at work. The risk is also dose-responsive, varying with exposure to both contributing factors. Heavy smokers heavily exposed to asbestos will have a higher than 50-fold increase.\(^{25}\) In Australia, most exposure to asbestos now occurs through the removal of asbestos from buildings, but the long lag-time for development of asbestos-caused disease means that death rates will continue to rise for the next two decades.\(^{26}\) There is also ample evidence suggesting that exposures to petrochemicals, aromatic amines, ionising radiation and pesticides interact with tobacco smoke.\(^{25}\)
Evidence on occupational exposures and health effects has further expanded in recent years. Researchers have reported on occupational exposures and lung cancer; 4.9% (95% CI, 2.0 - 7.8) of lung cancers in men were attributable to occupation in known higher risk professions; past exposure to occupational carcinogens is judged an important determinant of lung cancer incidence. A follow up study of 15 million people in Denmark, Finland, Iceland, Norway and Sweden showed that male waiters and tobacco workers had the highest risk of lung cancer, probably attributable to active and passive smoking. Miners and quarry workers also had a high risk, which might be related to their exposure to silica dust and radon daughters. Among women, tobacco workers and engine operators had a more than four-fold risk as compared with the lung cancer risk among farmers, gardeners and teachers. Waiters had the highest risk of bladder cancer among men; tobacco workers had the highest risk among women; the low-risk categories were the same ones as for lung cancer. The researchers stated that all of this could be accounted for by smoking.

A systematic review found consistent evidence that exposure to benzene at work increases the risk of leukaemia with a dose-response pattern. Studies have also demonstrated negative consequences from inhaling fumes during welding, and the impact of silica exposure on the risk of developing rheumatoid arthritis and lung cancer, with greatly increased risk for these diseases among smokers. Current or previous occupational exposure to organic solvents has been found to double the smoking-related risk of chronic bronchitis. Cigarette smoking accompanied by exposure to workplace noise has also been associated with a five-fold increase in risk of noise induced hearing loss among smokers compared with non-smokers. The risk of infertility and spontaneous abortion has been found to be 30% higher among female hairdressers than among women in other occupations, thought to be due to their occupational chemical exposure and found primarily among never smokers. Being a current smoker is a risk factor for sensitisation to workplace allergens. Smokers are more than twice as likely as non-smokers to have positive tests of allergic reactions (OR 2.39; 95% CI, 1.01–5.65).

Inhalation exposure to particulates such as cigarette smoke and coal dust is known to contribute to the development of chronic lung disease and several studies of the health and wellbeing of coal miners have been reported. These studies provide evidence that:

(i) elemental carbon levels in the lungs and pack-years of cigarette smoking correlate significantly, as do elemental carbon levels and the severity of small airway disease;
(ii) cumulative exposure to coal dust is a significant risk factor for the development of emphysema and has an additive effect to smoking;
(iii) exposure to coal mine dust leads to increased mortality, even in the absence of smoking;
(iv) increased exposure to coal dust is associated with increased risk of death from chronic obstructive pulmonary disease; and
(v) that in newly employed coal miners, bronchitis symptoms are associated with a rapid decline in lung function within two years after starting work. Other studies of exposure to dust or fumes have reported associations with the incidence of chronic obstructive pulmonary disease, and asthma; chronic bronchitis occurs more frequently among individuals exposed to mineral dust, and smoking doubles this risk.

The US Surgeon General has concluded that 'for the majority of American workers who smoke, cigarette smoke represents a greater cause of death and disability than their workplace environment'. The Australian Burden of Disease Study confirms this. It is estimated that in 2003, occupational exposures and hazards accounted for 2% of the total disease and injury burden and 1.3% of all deaths, while tobacco use accounted for 7.8% of the total disease and injury burden and 11.7% of all deaths.

The accumulation of evidence about workplace health hazards over previous decades has led to the introduction of industrial health and safety standards, which have greatly reduced exposures to carcinogenic and other toxic substances in developed countries. However, the relocation of hazardous industry to less developed countries, where occupational safety may be less regulated (and where, incidentally, there is more likely to be a higher prevalence of smoking), is a major cause for concern.

3.25.2.3 Sick building syndrome

Over the past 15 to 20 years awareness has risen about 'sick building syndrome'. The term sick building syndrome is used to refer to a heterogeneous group of work-related symptoms, including irritation of the skin
and mucous membranes of the eyes, nose and throat; headache; fatigue; and difficulty concentrating. These are considered illnesses because of the occurrence of symptoms, even though affected workers do not have objective clinical or laboratory abnormalities and causative agents cannot be found. The clinical symptoms of sick building syndrome, although not life-threatening, are disruptive: they reduce productivity and increase absenteeism from work. It is likely that the cause of sick building syndrome is multifactorial, involving ventilation, airborne particulates from a wide range of sources (including chemicals, micro-organisms and non-organic matter) and other vectors in the indoor environment. A recent update review concluded that sick building syndrome is related to both personal and environmental risk factors and, in the office environment, may have important economic implications. United States Environmental Protection Agency studies have identified four distinct groups of symptoms, representing 'tiredness', 'mucosal irritation', 'neuropsychological' and 'lower respiratory' conditions. Rather than a dichotomy of healthy versus unhealthy ('sick') buildings the Environmental Protection Agency found that the prevalence of health problems related to buildings spans a continuum; the distribution of work-related symptoms and identification of symptom groups can help in identification of problems.

A recent review of the literature on ventilation rates and health found biological plausibility for an association of health outcomes with ventilation rates, but a lack of clear evidence on particular agent(s) for the effects. Higher ventilation rates in offices, up to about 25 litres per person, are associated with reduced prevalence of sick building syndrome symptoms.

The limited available data suggest that inflammation, respiratory infections, asthma symptoms and short-term sick leave increase with lower ventilation rates. Home ventilation rates above 0.5 air changes per hour (h⁻¹) have been associated with a reduced risk of allergic manifestations among children in a Nordic climate. Improved ventilation practices and reduction of pollutants are key factors in alleviating sick building syndrome, along with adherence to building and maintenance standards and improved controls over temperature, humidity and lighting. During the 1980s the tobacco industry took a particular interest in sick building syndrome, using it as a means of deflecting attention away from newly emerging evidence about the health consequences of exposure to environmental tobacco smoke. This is discussed further in Chapter 15, Section 15.3.1.

The measure used to express total burden of disease and injury is the 'disability-adjusted life year' (DALY). The DALY 'describes the amount of time lost due to both fatal and non-fatal events; that is, years of life lost due to premature death coupled with years of 'healthy' life lost due to disability'.

References


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3.26 Health effects of brands of tobacco products which claim or imply, delivery of lower levels of tar, nicotine and carbon monoxide

With reports published during the 1950s linking cigarette smoking with disease, tobacco companies experimented with various modifications to their products. Firstly, filters were added, and later ventilation holes in the form of tiny perforations were added around the mouthpiece, with the apparent intention of diluting the delivery of tar, nicotine, carbon monoxide (CO) and other compounds to the smoker. Tobacco leaf itself has also undergone various treatments to alter toxic delivery. While it was initially hoped by health interests that lower delivery cigarettes would prove to be a less harmful form of tobacco use, bringing about reductions in death and disease, this has proved not to be the case.\(^1,2\)

Low delivery cigarettes do not offer a health advantage. Two major, recent reports have reviewed the evidence regarding the impact on health of smoking cigarettes with lower levels of toxicity. The US Surgeon General’s report for 2010 concluded that “changing cigarette designs over the last five decades, including filtered, low-tar, and “light” variations, have not reduced overall disease risk among smokers and may have hindered prevention and cessation efforts.” p. 25\(^3\) The National Cancer Institute, part of the US National Institutes of Health, stated in its 2001 report that “epidemiological and other scientific evidence, including patterns of mortality from smoking- caused diseases, does not indicate a benefit to public health from changes in cigarette design and manufacturing over the last fifty years,” and that “widespread adoption of lower yield cigarettes in the United States has not prevented the sustained increase in lung cancer among older smokers.” p. 10\(^1\)

The reasons why ‘lower delivery’ cigarettes have not benefited health lie in the way a smoker typically smokes a cigarette, as opposed to how a machine designed to measure smoke output ‘smokes’ a cigarette under laboratory conditions. Because addicted smokers need to maintain their level of intake of nicotine, they tend to compensate for the delivery of lower levels of nicotine by adjusting puff frequency, rapidity or depth of inhalation, by increasing the numbers of cigarettes smoked daily, or by blocking with their fingers the ventilation holes around the filter intended to dilute the smoke. Because exposure to tar compared to nicotine appears to be similar among smokers of conventional cigarettes, this results in very little change in the actual intake of tar and other compounds. In contrast, of course, a machine simply “puffs” according to its calibration.\(^1,4,5\)

Wider public health interests were first alerted\(^2\) to the discrepancy between laboratory-measured and
smokers' actual exposure by the release of the US Surgeon General's Report of 1981, but for the following decade it was generally held that smokers choosing low delivery brands were reducing their intake to at least some degree. The tobacco industry operated under no such illusions. It is now known that the industry had long been aware of compensatory smoking and the limitations of machine measurements, and from the 1970s onwards, was using this knowledge to manipulate consumers and subvert the official laboratory testing systems. Tobacco companies engineered their products so that smokers could obtain as much nicotine as they needed by altering their smoking behaviour, but also ensured that when tested by standard smoking machine protocols, the same brand of cigarettes would return acceptably low yields. The tobacco industry also experimented with the composition of cigarettes and filters in order to minimise machine-measured noxious outputs, but took no steps to create a cigarette which truly delivered lower toxins to the smoker under normal conditions.

Beyond the phenomenon of compensation, the 2014 US Surgeon General’s report explored whether the changes in the design and composition of cigarettes since the 1950s that paralleled the reduction in machine-measured tar yields may have changed smokers’ risk of lung cancer. The report concluded that the increased risk of adenocarcinoma of the lung has been caused by these changes in the design and composition of cigarettes. There is currently insufficient evidence to identify which changes have caused this increase, but ventilated filters and increased levels of tobacco-specific nitrosamines may be partially responsible.

The production and vigorous marketing of 'light' and 'mild' brands allowed the tobacco industry, over several decades, to reassure smokers that there were less harmful ways of smoking. Two major tobacco companies in Australia have agreed to discontinue the use of misleading descriptors and information on tar, nicotine and CO levels on tobacco packages since they have been judged by the Australian Competition Consumer Commission to be misleading. For detailed discussion about the changes made to manufactured cigarettes in Australia, along with regulatory aspects, refer to Chapter 12, Section 12.4.

References


i See the ACCC website: http://www.accc.gov.au/content/index.phtml/itemId/683533

3.27 Health effects of smoking tobacco in other forms

Data from the 2013 National Drug Strategy Household Survey show that while the vast majority of Australian smokers (89%) use manufactured cigarettes, a variety of other tobacco products are also used either regularly or occasionally, either in conjunction with use of cigarettes or exclusively. About one-third (33%) of smokers report any use of roll-your-own, and about ten percent (10.4%) report use of cigarillos or cigars. Fewer than 2% report use of pipe tobacco. Unbranded tobacco (also known as chop-chop) is roughly processed loose tobacco that has been grown, distributed and sold without government intervention or taxation. In 2013, of smokers aged 14 years or older about one-third had seen or heard of unbranded loose tobacco. The proportion of smokers who had ever smoked and smoked unbranded loose tobacco at the time of the survey declined between 2010 and 2013 (from 24.0% down to 16.5%, and from 4.9% down to 3.6%, respectively). The overall proportion of smokers using it regularly remained low at 0.8%.

Smoking tobacco by means of a waterpipe (using a system by which smoke passes through water prior to inhalation) is widespread in other parts of the world and is also practised in Australia. One study reporting prevalence of 11% among Arab-speaking adults). Data from the National Drug Strategy Household Survey indicates that 2.4% of all Australians aged over 14 years, and 6.7% of current smokers, had used a waterpipe in the year prior to the survey. Usage was much higher among young adults (18-24 years) who were also current smokers, at 17%. For more information on the extent to which tobacco products other than manufactured cigarettes are used in Australia, see Chapter 1, Section 1.11.

3.27.1 Manufactured loose tobacco

Manufactured loose tobacco, hand-rolled into cigarette paper and smoked with or without a filter, causes the same range of diseases as smoking manufactured cigarettes. Variations in quantity of tobacco used per cigarette and filtration make measurements of individual exposure more difficult to assess, but the directly comparable exposure to harmful constituents and method of consumption means that smokers of these products have at least an equivalent risk of developing disease as do smokers of conventional cigarettes. Several decades of research on the health effects of tobacco use have enabled comparisons between products with and without filters, and with high and low nicotine and tar yields. Overall, neither has the incidence of lung cancer varied with tobacco product used, nor have other health benefits become apparent.

3.27.2 Unbranded loose tobacco ('chop-chop')
Chop-chop is used by some as an alternative to other manufactured tobaccos due to its comparative affordability, and common misapprehensions that it is less harmful to health since it is apparently more ‘natural’ and ‘unadulterated,’ not having been processed in the usual way. Research has shown that some batches of chop-chop contain bulking agents such as twigs, raw cotton and grass clippings. Fungal (mould) spores have also been detected. Fungal spores are of particular health concern since they give rise to mycotoxins, including aflatoxin, a known carcinogen. Inhalation of and contact with fungi and their mycotoxins can cause a range of adverse responses in the liver, kidneys and skin, and cause illnesses including allergic reactions, chronic bronchitis, asthma and lung diseases. Australian chop-chop users report significantly worse health than smokers of licit tobacco. In a recent study in which a comparison with licit-only tobacco smokers was undertaken, current users of chop-chop had significantly greater odds of reporting below-average social functioning (OR 1.61; 95% CI, 1.06–2.44), measurable disability (OR 1.95; 95% CI, 1.08–3.51), below-average mental health (OR 1.61; 95% CI, 1.22–2.13) and above-average bodily pain (OR 1.40; 95% CI, 1.06–1.85).

3.27.3 Cigar smoking

Cigar smoke is at least as toxic and carcinogenic as cigarette smoke and possibly more so. Cigars contain more tobacco per stick than cigarettes, take longer to smoke, and produce higher concentrations of a number of noxious compounds including carbon monoxide, nitrogen oxides, carcinogenic N-nitrosamines and ammonia. A recent systematic review found that cigar smoking carries many of the same health risks as cigarette smoking. Mortality risks from cigar smoking vary by number of cigars per day and inhalation level, but can be as high as or exceed those of cigarette smoking. Specifically, primary cigar smoking (current, exclusive cigar smoking with no history of previous cigarette or pipe smoking) was associated with all cause-mortality, oral cancer, esophageal cancer, pancreatic cancer, laryngeal cancer, lung cancer, coronary heart disease (CHD), and aortic aneurysm. Strong dose-response relationships were observed between the number of cigars smoked per day and inhalation level, and oral, esophageal, laryngeal, and lung cancers. Among primary cigar smokers who reported that they did not inhale, relative mortality risk was still highly elevated for oral, esophageal, and laryngeal cancers. A 2010 European longitudinal study involving more than 100,000 men found that, compared to never smokers, the risk of cancers of the lung, upper aerodigestive tract and bladder combined was more than doubled (HR 2.2; 95% CI, 1.3–3.8) for exclusive cigar smokers. Effects were stronger in current than in ex-smokers and in inhalers than in non-inhalers. For ever-smokers of both cigarettes and cigars there was more than a five-fold increase in the risk of these cancers (HR 5.7; 95% CI, 4.4–7.3) making the risk elevation as high as that among exclusive cigarette smokers. A case–control study focusing only on lung cancer found a five-fold increase in risk for smoking cigars only compared to never smokers (OR 5.6; 95% CI, 2.9–10.6) consistent with estimates from earlier research. Other research has shown that cigar-only smokers had a 60% increased risk of pancreatic cancer compared with those who had never used tobacco (OR 1.6; 95% CI, 1.2–2.3), the risk with increasing according to the amount of cigar smoked per day (OR 1.82 for ≥10 grams of tobacco), which is broadly consistent with risk estimates from a 2008 meta-analytic review.

Smoke drifting from the burning tip of a lit cigar contains most of the same toxic and carcinogenic compounds as cigarette smoke, and because they are larger, cigars generate smoke for a longer period of time—as long as 90 minutes for a single large cigar. This is a health concern for those constantly exposed to an indoor environment affected by cigar smoke; some researchers have concluded that high passive exposure to smoke from cigars and pipes may be associated with lung cancer risk. Cigar use has increased in popularity in the US, prompting concern among some tobacco control organisations; in Australia the proportion of smokers using cigars and pipes alone rose from an estimated 1.2% in 2004 to 1.6% in 2010. See Chapter 1, Section 1.11 and Chapter 10, Section 10.6 for further discussion.

3.27.4 Pipe smoking

Longitudinal research conducted in Norway has reported that pipe smoking is not safer than cigarette smoking. The study followed a cohort of more than 16 000 men for up to 13 years. Between pipe and
cigarette smokers, no or only minor differences were found in mortality from any cause and the specified smoking-related diseases.\textsuperscript{24} Pipe smoking is associated with decreased lung function and increased odds of airflow obstruction, even in participants who had never smoked cigarettes;\textsuperscript{25} it is associated with a significantly higher risk of dying from COPD, cerebrovascular disease and cardiovascular disease. Compared to never smokers, exclusive pipe smokers are estimated to have a three-fold increase in risk (HR 3.0; 95% CI, 2.1–4.5) for cancers of lung, upper aerodigestive tract and bladder combined,\textsuperscript{16} and an eight-fold in the risk (OR 8.7; 95% CI, 4.0–18.9) of all upper digestive tract cancers (including a 12-fold risk for oral and seven-fold risk for pharyngeal cancer). Pipe smokers who are also heavy alcohol drinkers have a massive 38-fold increased risk of these cancers (OR 38.8; 95% CI, 13.6–110.9) as compared to never smokers and light drinkers, strongly suggesting that pipe smoking and heavy alcohol drinking may interact in a way that greatly increases the risks.\textsuperscript{26}

As with cigarette smoking, the risk of developing tobacco-caused disease varies in a dose–response relationship, disease risk increasing with the amount smoked, the depth to which it is inhaled and the duration of smoking. For most disease entities, the relative risk of developing tobacco-related disease declines with quitting, increased length of time of cessation and younger age at quitting.\textsuperscript{27}

Disease patterns differ from those observed in cigarette smokers because pipe smokers tend to inhale the smoke less deeply, taking up nicotine through the mucous membranes lining of the mouth instead of predominantly via the lungs. Some earlier studies suggested the possibility of some harm reduction benefits in switching from cigarette to pipe smoking.\textsuperscript{16} It was suggested that the magnitude of the extra risk was smaller if people had switched to cigars or pipes only (i.e. quit cigarettes) and had not compensated with greater smoking intensity. However it should be noted that recent research has found that men who switched from cigarettes only to pipe only had a risk that was not significantly different from the risk in sustained smokers of cigarettes only,\textsuperscript{24} so that the overall main conclusion about pipe smoking is that it is very hazardous and is certainly not a safe alternative to cigarette smoking.

### 3.27.5 Waterpipe smoking

Using a waterpipe to smoke tobacco is not a safe alternative to cigarette smoking.\textsuperscript{26} Secondhand smoke from waterpipe tobacco use produces a similar level of air pollutants as cigarettes, and poses a serious health risk to those exposed.\textsuperscript{5,28,29} Names for waterpipe vary and include ‘narghile’, ‘arghile’, ‘shisha’, ‘goza’, ‘hubble bubble’ and ‘hookah’.\textsuperscript{30} Waterpipe smoking use spread through the Middle East and Asia, and waterpipes were widely used in Turkey during the Ottoman Empire (15th century), Iran, Lebanon, Syria, Jordan, Greece, India, Pakistan, Palestine, Egypt and Saudi Arabia. As people immigrated to Europe from India, Pakistan, Northern Africa and the Middle East, hookahs and hookah cafes began appearing in European cities. Today, hookah bars and cafés are popular in many parts of Britain, France, Spain, Russia, India, Asia and throughout the Middle East and are growing in popularity in the US,\textsuperscript{3} with some estimates that about one billion people worldwide are waterpipe users.\textsuperscript{31}

Waterpipe apparatus varies widely in design, but the method of use requires the heating with burning charcoal of moist tobacco (usually sweetened and flavoured) to produce smoke, which is passed through water before being inhaled via a mouthpiece on the end of a hose.\textsuperscript{4,5} Electronically heated systems have also been developed in recent years, but the effects of these on smokers and the environment have not been well-studied.\textsuperscript{32} Waterpipe smoking usually occurs in a social setting with a number of participants seated around the waterpipe, taking it in turns to inhale. The availability of pre-moistened, shaped and flavoured tobacco made especially for waterpipe use (‘Maassel’) since the 1990s is likely to have contributed to a resurgence in waterpipe smoking in the Eastern Mediterranean Region, and its increased popularity.\textsuperscript{5,33} At least in some cultures, women and girls are more likely to use a waterpipe than to use other forms of tobacco, and it is popular among younger smokers. Because the smoke passes through a reservoir of water, waterpipe smoking may erroneously\textsuperscript{34} be perceived as being less lethal than other methods of tobacco use.\textsuperscript{5,30,35} Although the moist smoke produced by waterpipe smoking may be more palatable than cigarette smoke,\textsuperscript{4} many of the harmful gases and chemicals found in cigarette smoke are present in equal or even greater amounts in waterpipe smoke, including carbon monoxide, nicotine and heavy metals.\textsuperscript{5} Waterpipe
smokers are typically exposed to smoke over a longer period than cigarette smokers, a session lasting somewhere between 45 minutes to an hour, but some sessions may continue for many hours. Although waterpipe smokers do not usually smoke as frequently as do cigarette smokers, it has been estimated that during a typical session, a waterpipe smoker inhales more than 100 times the volume of smoke produced by smoking a single cigarette.

A systematic review was conducted by Akl and colleagues to examine the effects of waterpipe tobacco smoking on health outcomes. While the quality of available studies was poor, the researchers found that waterpipe smoking of tobacco was significantly associated with a doubling, respectively, in the risks of lung cancer (OR 2.12; 95% CI, 1.32–3.42), respiratory illness (OR 2.3; 95% CI, 1.1–5.1), low birthweight (OR 2.12; 95% CI, 1.08–4.18), and at least a trebling in the risk of periodontal disease (OR = 3–5).

Another recent systematic review and meta-analysis of six cross-sectional studies was conducted to examine the effects of waterpipe tobacco smoking on lung function compared with no smoking.

Despite methodological limitations in the reviewed studies, the authors were able to conclude that waterpipe smoking of tobacco negatively affects lung function, may be as harmful as cigarette smoking and is likely to be a cause of COPD. Research into the acute effects of waterpipe smoking on the cardiorespiratory system has been reported. Forty-five similarly sized volunteers (including 30 men) were studied after a single 30-minute domestic open-air group-smoking session of waterpipe smoking. Carboxyhaemoglobin levels were significantly raised post-waterpipe smoking, especially in women. Three of 45 subjects demonstrated carboxyhaemoglobin concentrations varying between 20% and 26%, high enough levels for consideration of inpatient treatment in susceptible individuals. Blood pressure, heart rate, and respiratory rate were all significantly increased post-waterpipe smoking. The authors concluded that one session of waterpipe smoking causes acute biologic changes that might result in marked health problems.

Accumulating evidence of the health effects associated with waterpipe smoking now makes a compelling case that there are serious risks for those exposed, that is in no way a safe alternative to cigarette smoking and that its spread among young people represents a global problem. Associations with lung cancer, respiratory illness, low birthweight, periodontal disease, impaired lung function, and acute cardiorespiratory effects have been noted. There is evidence that waterpipe smoke contains many of the same toxicants as cigarette smoke, including those that cause cardiovascular disease (e.g. carbon monoxide), lung disease (e.g. volatile aldehydes), cancer (e.g. polycyclic aromatic hydrocarbons) and dependence (i.e. nicotine). Waterpipe smoking is an efficient means of delivering toxicants to the smoker; for example, recent research reveals that, relative to a single cigarette, a single waterpipe session exposes the smoker to 3–9 times the carbon monoxide and 1.7 times the nicotine. WPS is associated with features of dependence, such as drug-seeking behaviour, inability to quit despite repeated attempts, and abstinence-induced withdrawal that is suppressed by subsequent waterpipe use. Sharing the waterpipe, a popular practice among youth worldwide, can be associated with infectious disease risks, such as tuberculosis. Waterpipe smoking-related emissions can harm non-smokers; for example, studies have shown that waterpipe smoking generates high levels of toxicants/carcinogens (e.g. volatile organic compounds, polycyclic aromatic hydrocarbons, metals, carbon monoxide and particulate matter) in the surrounding air, putting non-smokers at risk. Finally, evidence suggests that waterpipe smoking can undermine tobacco control, because it can be used as a replacement for cigarettes among quitters or serves as a gateway to cigarette initiation.

**3.27.6 Kreteks**

Kreteks are cigarettes that contain a combination of cloves and clove oil, tobacco and other additives. Originating in Indonesia, where they account for about 90% of the market, a small number of brands are currently imported into Australia.

Although kreteks are smaller than typical cigarettes, they can deliver similar levels of nicotine and carbon monoxide to smokers. Gas chromatographic analysis of kreteks has revealed high levels of eugenol, anethole and coumarin compounds. The authors of one such study noted that compounds such as eugenol...
are known to be hazardous to humans when inhaled in high concentrations, and pose significant health concerns. The researchers concluded that usage of such compounds in smoking products, particularly at high levels, should be discouraged pending the availability of detailed toxicity information. Another analysis found that the levels of these compounds found in kreteks are significantly higher than those typically found in commercial cigarette brands. Long-term research on the health effects of smoking kreteks is scant, but it can be reasonably assumed that they pose at least the same dangers to health as conventional cigarettes. Popular use of these cigarettes in the US commenced in about 1980; by the mid-1980s warnings had begun to appear in the literature, notably so when 13 cases of severe illness with clove cigarette smoking were reported to the Centers for Disease Control and the California Department of Health Services; the clinical characteristics of these cases included haemorrhagic pulmonary oedema, pneumonia, bronchitis and hemoptysis (spitting up or coughing up of blood).

Research from Indonesia has shown that regular kretek smokers have 13‒20 times the risk of abnormal lung function than non-smokers. Kretek use is associated with a higher risk of acute lung injury, particularly in susceptible individuals such as those with asthma or respiratory infections. There is also evidence that clove cigarettes are linked with greatly increased risk of dental disease. In a longitudinal study of more than 1000 male bus drivers in Jakarta, 27% of those who had smoked for 10 years or less had dental caries. This proportion increased to 79.6% among those smoking for 11‒15 years and rose to 89.3% among those smoking for more than 15 years. People who smoked 7–12 cigarettes a day were more than twice as likely (RR 2.66, p<0.0001) to develop dental caries compared to those smoking 0‒6 cigarettes a day. Those categorised as smoking either 13‒18 cigarettes a day (RR 3.19, p<0.0001) or more than 18 cigarettes a day (RR 2.96, p<0.0001) were three times more likely to do so.

As previously noted, cloves contain a substance called eugenol, which when inhaled has the characteristics of a local anaesthetic. In the past this attribute has lent kreteks a reputation as soothing for sore throats and asthma, but in fact it can reduce the gag reflex, leading to pulmonary aspiration (when substances such as food or drink enter the lungs). The American Medical Association reviewed the medical evidence concerning clove cigarettes in 1988 and reached the following conclusions:

(i) clove cigarettes are tobacco products; therefore they possess all the harms associated with smoking tobacco cigarettes; and

(ii) inhaling clove cigarette smoke has been associated with severe lung injury in a few susceptible persons. People with asthma or with a throat or lung infection in its early stages may have an increased risk of harm from inhaling clove cigarette smoke.

Kreteks are used by 2.4% of high school and 1.2% of middle school students in the US. Consumption patterns in Australia are not known.

### 3.27.7 Bidis (beedis, beedies, biris)

For information about bidis, see section 3.32.3

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3.28 Health 'benefits' of smoking?

Important note: smoking may offer a limited degree of protection in some individuals against the development of a small number of diseases, outlined below. However this information is of little relevance to public health, given that the amount of disease that tobacco may be said to prevent is insignificant in comparison with the far greater incidence of disease caused by smoking. Smoking kills one in two of its users.¹

Tobacco use confers a small degree of protection against several diseases and conditions, described in the sections below. It is estimated that in 2004–05, tobacco use prevented about 148 deaths in Australia, a very low number compared to the 15,050 deaths caused by smoking in that same year.² On the basis of these figures, tobacco might be said to save about one life for every 100 deaths it causes. Moreover, there is nothing to suggest that possible protection conferred against one disease will stop a given smoker from developing another tobacco-caused disease. So, for example, an individual who may have avoided Parkinson's disease due to his or her smoking still runs a significant risk of dying from heart disease, lung cancer, or any of the multiplicity of other tobacco-caused diseases. Equally, smoking does not prevent Parkinson's disease in all smokers.

While tobacco use cannot in any way be recommended as a prophylactic for these diseases and conditions, research on the mechanisms by which smoking appears to confer a protective effect against development of certain disease processes may lead to therapeutic benefits.³

3.28.1 Ulcerative colitis

Ulcerative colitis is a serious bowel disease in which the inner lining of the colon and rectum becomes inflamed and permanently damaged.⁴ Current smokers have a lower risk of developing ulcerative colitis, compared to non-smokers and ex-smokers,⁵ and according to the US Surgeon General, the evidence suggests that this protective relationship may be causal. A dose-response relationship has also been found, such that greater pack-years or numbers of cigarettes smoked per day were associated with a decreased risk of ulcerative colitis.⁶ Nicotine in tobacco smoke is thought to be the component that is most likely to affect the course of the disease.⁷ However, smokers have a greater risk of developing Crohn's disease, another inflammatory disease of the bowel (see Section 3.12.2). Due to the devastating effects of tobacco use, smoking is not recommended as treatment for ulcerative colitis, even though one research study has canvassed this as an extreme possibility for ex-smokers with steroid-dependent and resistant ulcerative colitis.⁸ Various forms of nicotine therapy are undergoing research to evaluate any possible benefits for individuals with this bowel disease.⁷
3.28.2 Parkinson's disease

An association between smoking and a lower incidence of Parkinson's disease has been observed in a number of studies. An analysis of longitudinal studies found a protective effect against Parkinson's disease for current and former smokers compared with those who had never smoked; the risk of Parkinson's disease was reduced by about half among ever smokers (RR 0.51; 95% CI, 0.43–0.61) and this protective effect was more pronounced among current smokers, where the risk was about one-third that of never smokers (RR 0.35; 95% CI, 0.26–0.47). Similar findings of a protective effect for Parkinson's disease were also reported from a case–control study conducted in Japan. Nicotine is thought to be the chemical in tobacco smoke mostly likely to be implicated in this finding, but there may be other chemicals or compounds involved. Based on data from 2004–05 we can derive theoretical estimates that about 97 deaths from Parkinson's disease are prevented by smoking in Australia annually. Finally, recent research also suggests that nicotine can improve compromised semantic processing in Parkinson's disease, and also influence semantic processing in healthy older individuals; however, the 2014 US Surgeon General's report found that controlled trials of the effects of nicotine on cognitive function in patients with Parkinson's disease are limited, with inconsistent findings.

3.28.3 Endometrial cancer and uterine fibroids

Epidemiological studies have consistently reported that active cigarette smoking is inversely associated with developing cancer of the endometrium (the membrane lining of the uterus) in women who have reached menopause. A recent meta-analysis found that cigarette smoking was significantly associated with a reduced risk, especially so among postmenopausal women, where a 29% reduction in risk was found (RR 0.71; 95% CI, 0.65–0.78). Very similar results have been reported from recent studies conducted in Poland although the researchers are at pains to point out their important finding that in postmenopausal women, obesity is an important modifier of the association between cigarette smoking and the risk of endometrial cancer. The Polish researchers found that obese women showed the greatest risk reduction for current smoking (OR 0.47; 95% CI, 0.27–0.81), a finding that further underscores the need for caution in interpreting these 'favourable effects' of cigarette smoking, considering the toxic and carcinogenic effects of tobacco.

Women who smoke may also have a decreased risk for uterine fibroids and endometriosis, but the evidence for this is not conclusive. Development of endometrial cancer is predominantly influenced by exposure to the hormone oestrogen, and the protection conferred by smoking is likely to be due to the 'anti-oestrogenic' effect of chemicals in tobacco smoke. This same interaction works to increase the risk among smokers of developing osteoporosis, and reaching menopause earlier than non-smokers (see Sections 3.13 and 3.6.1).

Based on data from 2004–05 we can derive theoretical estimates that smoking may prevent the loss of about 52 lives from endometrial cancer in Australia. However the numbers of lives saved that can be statistically attributed to the prevention of endometrial cancer among smokers pales into insignificance compared with the numbers of deaths due to other diseases caused by tobacco use, particularly in the light of the evidence that has established that smoking causes cancer of the uterine cervix.

3.28.4 Pre-eclampsia (hypertension in pregnancy)

Pre-eclampsia is a potentially serious condition in pregnancy in which the mother develops high blood pressure, fluid retention and abnormal kidney function. Smokers are less likely to develop pre-eclampsia than non-smokers; recent research points to the impact of smoking on the ratio of soluble fms-like tyrosine kinase-1 (sFlt-1) to placental growth factor (PlGF) as one possible pathway, however the mechanism by which the observed protective effects occur remains poorly understood. A study using Swedish birth registry data on more than 600 000 births examined the effects of snuff and cigarette smoking on pre-eclampsia risk and whether changes in tobacco habits during pregnancy affected the risk of developing
term pre-eclampsia. Compared with non-tobacco users, light smokers experienced a one-third reduction in risk (OR 0.66; 95% CI, 0.61–0.71) and heavy smokers a halving of risk (OR 0.51; 95% CI, 0.44–0.58) with ORs lower for term than preterm pre-eclampsia. The study found that tobacco combustion products rather than nicotine are the probable protective ingredients against pre-eclampsia in cigarette smoke and further concluded that it is smoking behaviour in the middle or late rather than in the beginning of pregnancy that seems to have the greatest effect on the risk of pre-eclampsia.\textsuperscript{21} A potential mediator of these associations might be carbon monoxide (CO), as it has vasoprotective properties, and CO and CO-releasing molecules lower soluble fms-like tyrosine kinase-1 (sFlt-1; a protein that is higher in women who develop preeclampsia) and soluble endoglobin in in vitro cultures.\textsuperscript{5}

The US Surgeon General has concluded that ‘the decreased risk of pre-eclampsia among smokers compared with non-smokers does not outweigh the adverse outcomes that can result from prenatal smoking’ (p576).\textsuperscript{20} These conclusions are underscored by findings from a recent case–control study conducted in Canada where notwithstanding a (non-significant) reduction in the risk of pre-eclampsia, persistent smoking was also associated with a 10-fold increase in the risk of low birthweight (OR 10.2; 95% CI, 2.49–41.8) and a four-fold increase in the risk of preterm birth (OR 3.59; 95% CI, 1.06–12.1).\textsuperscript{22}

### 3.28.5 Cognitive performance?

A meta-analysis of research into the effects of nicotine and smoking on human performance found positive effects of nicotine or smoking on six domains: (i) fine motor, (ii) alerting attention-accuracy, (iii) response time (RT), (iv) orienting attention-RT, (v) short-term episodic memory-accuracy, and (vi) working memory-RT (effect size range = 0.16 to 0.44).\textsuperscript{23} There is evidence that nicotine may stimulate immediate and sustained improvements in working memory,\textsuperscript{24} that nicotine replacement in smokers avoids cognitive impairment through direct pharmacological effects on brain neuronal activity,\textsuperscript{25} and that nicotine may improve prospective memory (the retrieval and implementation of a previously encoded intention).\textsuperscript{26} Note however that smoking in the longer term has been associated with cognitive decline—see Section 3.23.

### 3.28.6 Psychiatric symptoms?

The prevalence of smoking is higher among people with psychiatric conditions.\textsuperscript{27-29} The reasons for this are complex and are discussed in greater detail elsewhere (Chapter 1, Section 1.10.2 and Chapter 9, Section 9.6.4), but one motivating factor for smoking is that tobacco may be regarded by some individuals as a way of relieving unpleasant symptoms of certain types of mental illness, and could therefore be seen as helpful.\textsuperscript{28}

There is evidence that the action of nicotine in enhancing mood and concentration is more pronounced in some individuals with depression and cognitive problems (issues relating to mental awareness and judgement), and also that nicotine can help relieve unwelcome side effects from medication, particularly among patients being treated with antipsychotic drugs.\textsuperscript{28} These effects may occur because of different actions of nicotine on the brain chemistry reward system, which have been observed in individuals with particular psychiatric conditions.\textsuperscript{28}

It has been suggested that nicotine transiently enhances sustained attention in schizophrenia patients, and that these research findings might provide insights for the development of new treatment strategies for attention deficit and sensory disruption which occur in schizophrenia.\textsuperscript{30,31} However, in a 2012 Cochrane Review update, the authors reviewed all randomised controlled trials examining nicotine as a treatment for people with schizophrenia, and found no studies that met their inclusion criteria. Hence, there is a need for high quality research that investigates the effects of nicotine for schizophrenia.\textsuperscript{32} Limited research also suggests that nicotine might help alleviate some of the symptoms of ADHD, such as impulsiveness and memory deficits, which may explain the higher prevalence of smoking in this group.\textsuperscript{6}

Higher smoking rates among the mentally ill mean that they bear a disproportionate burden of morbidity and mortality from tobacco.\textsuperscript{33} Clinical and epidemiological data indicate that cigarette smoking increases the risk for the development and maintenance of panic disorder and that cessation may be one of the relevant steps
in treatment, while a recent case–control study suggests that smoking may be a risk factor for late-onset major depression. Smokers with severe mental health illnesses have been identified in the Australian National Tobacco Strategy as requiring specialised strategies to assist in cessation. See also Chapter 7, Section 7.19.

3.28.7 Thyroid cancer?

Some studies have suggested that smoking may be associated with a reduced risk of developing thyroid cancer, particularly for women; however this protective effect has not been found in all studies and more research is required before a definitive statement can be made.

3.28.8 Skin cancer?

Early epidemiological studies suggested a protective effect of smoking for melanoma. More recent analyses from two large prospective cohort studies provides limited evidence to suggest that smoking may reduce melanoma risk; analyses by smoking status provided inconsistent data and no clear dose–response pattern was found. This weakens the argument for a cause–effect relationship between smoking and a protective effect for melanoma.

3.28.9 Other possible health 'benefits'

There is some evidence that smokers and users of smokeless tobacco are less likely to develop aphthous stomatitis (common mouth ulcers). One recent study found that the possible protective effect of smoking was only present when there was heavy cigarette smoking or smoking for long periods of time (>5 years) and no significant associations were found between intensity or duration of smoking and clinical severity of aphthous stomatitis lesions. Transient increased incidence of mouth ulcers is commonly reported by individuals on quitting smoking.

References


19. US Department of Health and Human Services. Women and smoking. A report of the Surgeon General. Atlanta, Georgia: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and


The relationship between smoking and reduced body weight is widely recognised by smokers, and generally overestimated.

### 3.29.1 Does smoking cause smokers to weigh less than non-smokers?

While it is true that smokers weigh, on average, less than people who have quit smoking and those who have never smoked, the effect is modest and accrues over decades of smoking. For example, some studies have found only a small effect for daily smoking on body weight among some groups of young smokers. A US longitudinal study examining the relationship between trajectories of cigarette smoking from early adolescence to young adulthood and obesity in the mid-30s has found that heavy/continuous smokers and those who started smoking in late adolescence had a significantly lower likelihood of obesity compared with nonsmokers, however starting smoking does not appear to be associated with short-term weight loss.

The relationship between smoking and body weight is likely to be due to a range of effects of nicotine on the metabolism. It has been suggested that the typically lower body mass index (BMI) of smokers is due to higher total energy expenditure compared with never smokers. However, evidence regarding the relative roles of energy intake and expenditure is scant. Some research suggests the involvement of plasma leptin, which plays a key role in energy intake and expenditure regulation. There is also evidence to suggest that increased cigarette consumption among smokers can be associated with higher BMI. For example, a case–control study of over 1000 18–65 year olds found that for those of normal weight, smoking prevalence decreased as BMI increased. This trend was reversed among overweight, obese or morbidly obese individuals: smoking prevalence was significantly higher among the morbidly obese compared with those who were overweight or obese. Morbidly obese individuals were twice as likely to smoke as those of normal weight.

It has been suggested that such findings are due to the contribution of other risk behaviours such as physical inactivity, poor nutrition and higher alcohol consumption among overweight individuals; for example, there is some evidence of a positive association between physical activity and smoking, significantly mediating the relationship between smoking and BMI.

Recent research has found that while smokers tend to have a lower BMI than non-smokers, their fat distribution is more likely to be in the abdominal region (central adiposity or ‘male pattern’ fat distribution).
Individuals with the pattern of central fat accumulation are at greater risk of developing a range of cardiovascular and metabolic problems related to obesity including coronary artery disease.

The association between smoking and abdominal adiposity may be dose dependent: a large cross-sectional population-based survey among Swiss people aged 35–75 years found that smokers' mean waist circumference and percentage body fat increased with cigarettes smoked per day. In addition, there is some evidence that adolescent smoking is associated with abdominal obesity in adulthood: Finnish research found that smoking at least 10 cigarettes daily when aged 16 to 18 years is a risk factor for adult abdominal obesity among both genders and for overweight in females.

### 3.29.2 Smoking cessation and weight gain

Sustained cessation is associated with a mean weight gain of about 5–6 kg in the first year of abstinence, an issue of concern to some smokers. However the relationship between smoking and body weight is not clear cut. A prospective study of the association between smoking and weight gain among an adult Spanish cohort found that active smokers as well as those who stopped smoking during follow-up (median period 4.2 years) experienced significantly greater weight gains than never smokers. For further discussion on implications for cessation, see Chapter 7, Section 7.1.2.

### 3.29.3 Relative contribution of smoking and obesity to morbidity

Over the last decade, obesity has come to have an equal or even greater contribution to the burden of disease than smoking, and is associated with a greater negative impact on quality of life. This is based on US estimates of quality-adjusted life-years (QALYs) lost due to obesity and smoking between 1993 and 2008 and is related to a population obesity increase of 85% and a smoking prevalence decrease of 18.5% among US adults over this time. Overall, obesity had a greater effect on morbidity than mortality, while smoking has a negative impact on mortality; this has been demonstrated among US and Western European populations. Western European data also indicate that people in higher BMI categories spend more years living with disability, whereas life expectancy with disability may not be related to smoking status. In Australasia, a report published in 2014 cited dietary risks (accounting for 11% of the total burden), high body mass index (9%) and smoking (8%) as the leading risk factors for disease.

Childhood and adolescent behaviours and cardiovascular (CVD) risk factor profiles are important determinants of CVD risk factor prevalence later in life. A Finnish study following a cohort of 1809 people aged 3–18 years found the six-year progression of carotid atherosclerosis increased significantly with the number of childhood cardiovascular risk factors, including lipid levels, high blood pressure, obesity, diabetes, smoking and physical inactivity. A large prospective cohort study following people aged 18–26 years between 1995 and 1996 and 2001 and 2002 found that overweight and obese adolescents were more likely than those of normal weight to report a diagnosis of high blood pressure and cholesterol by the time they were young adults, regardless of BMI in young adulthood. Adolescent smoking and physical activity levels did not independently predict these CVD risk factors.

There is some evidence of a dose–response relationship between pregnancy smoking exposure and overweight and obesity in children. A UK cross-sectional survey of 3038 children aged 5–11 years found that overweight and obesity prevalence estimates were higher for those with mothers who smoked heavily (10+ cigarettes daily) during pregnancy. See Section 3.8.5 for further discussion on the long-term effects of smoking during pregnancy.

### 3.29.4 Smoking compared to and in combination with obesity: contribution to mortality

Since being overweight is strongly associated with many disease entities, it has also given rise to the
question of whether it is better to be a leaner smoker or a heavier non-smoker. A large international study
has investigated the connection between smoking, body weight and mortality from coronary heart disease. The study concludes that although smokers have on average a lower BMI than non-smokers, it is of nowhere near sufficient magnitude to offset the risk of dying from CVD as a result of smoking. The authors conclude that 'it is unquestionably better to quit smoking and gain weight than to continue to smoke' (p. 834) Similarly, smoking appears to offset the CVD risk factor benefits associated with parental longevity such as trends towards lower BMI, weight and waist circumference observed among women in Swiss population-based research.

Studies covering tens of thousands of deaths provide reliable evidence on mortality from persistent smoking and from adult obesity. An increase of 10% in the prevalence of smoking and of 2 BMI points in overweight populations reduce the life span in men comparably, each by about one year. An increase in life expectancy of about 10 years can be gained through smoking cessation, much greater than a smoker could expect to gain from weight control.

While smoking and adiposity are independent predictors of mortality, the combination of current or recent smoking with a BMI ≥ 35 or a large waist circumference is related to an especially high mortality risk (5 to 8 times that of never smokers within normal weight range). US all-cause mortality data from 51–72 year olds indicates an increase in mortality rates with greater BMI or waist circumference, and an increase from never to former to current smoking. Obese smokers have a 14-year reduction in life expectancy at age 40 compared with lean non-smokers, as well as an increased risk of developing type 2 diabetes and cancer.

The evidence regarding how smoking and obesity might interact to reduce life expectancy and the degree of interaction is not clear. For example, overweight or obesity in late adolescence has been found to increase the risk of all-cause adult mortality regardless of smoking status: obesity and overweight in late adolescence were as hazardous as heavy (>10 cigarettes/day) and light (1–10/day) smoking, respectively, with no interaction between BMI and smoking status. US research comparing the burden of mortality due to smoking, education levels and obesity in those aged 55+ years found smoking and low education decreased population life expectancy more than did obesity, with highest life expectancy at age 55 found in highly educated non-smokers, slightly higher than normal BMI.

Among modifiable dietary, lifestyle and metabolic risk factors, smoking and high blood pressure were responsible for the largest number of deaths in the US in 2005, accounting for about 1 in 5 or 6 adult deaths. In comparison, overweight/obesity caused about 9% of deaths, physical inactivity and high blood glucose about 8%, and high LDL cholesterol almost 5%. Estimates based on Swedish national data forecast a continued decline in premature deaths among males due to reductions in smoking over the last four decades, despite large increases in overweight and obesity over this time. However, one-third of the survival benefit from reduced smoking was estimated to be offset by the parallel increase in obesity.

Australian longitudinal research has identified central obesity and smoking as key independent risk factors predicting mortality from coronary heart disease and cardiovascular disease, with no added influence of measured lipids or blood pressure. When combined with cigarette smoking, waist-to-hip ratio was as effective as the Framingham risk score (incorporating the contribution of blood pressure, total cholesterol, high density lipoprotein cholesterol and diabetes) in predicting coronary heart disease and cardiovascular disease deaths. Similarly, US longitudinal research examining modifiable risk factors for cardiovascular disease found a low-risk profile (not smoking, moderate or high cardiorespiratory fitness, and healthy waist circumference) among men to be associated with a reduced risk of coronary heart disease, cardiovascular disease mortality and all-cause death while among women five lifestyle factors independently and significantly predicted mortality: 55% of deaths during follow-up were attributed to the combination of smoking, overweight, physical inactivity and poor diet; 28% of deaths were attributed to smoking.

Some evidence suggests that natural killer (NK) cells may be a mechanism by which obesity and smoking are associated with an increased risk of malignancy and mortality. Crucial in mediating anti-tumour immunity, there is a marked reduction in the numbers and function of NK cells in obese individuals, and
increased susceptibility of the cells to the harmful effects of cigarette smoke.\(^\text{26}\)

There is a lack of agreement about how to adjust for potential confounders including smoking status in the association between body mass index (BMI) and mortality.\(^\text{33}\) Research using 50 cohort data sets from about 30 international studies to examine the relationship between BMI and mortality and any interaction with smoking status found that excluding smokers’ data or disregarding smoking status did not affect the association between BMI and mortality.\(^\text{33}\) Worldwide, mean adult BMI increased between 1980 and 2008, although trends differed significantly between countries.\(^\text{34}\) In Australia, the prevalence of overweight and obesity in adults had increased to 62.8\% in 2011-12, from 61.2\% in 2007-08 and 56.3\% in 1995.\(^\text{35}\) Obesity and smoking are significantly more common in the most disadvantaged areas. In 2007-08 almost one-fifth of people who were overweight or obese were also current smokers.

Overweight or obese smokers were twice as likely to have heart disease as people who were within the normal weight range and who had never smoked, 2.2 times as likely to have Type 2 diabetes, and 2.8 times as likely to have bronchitis.\(^\text{36}\)

References


3.30 Deaths attributable to tobacco by disease category

Three studies conducted in the mid-2000s have estimated the numbers of deaths caused by tobacco use in Australia;\(^1\)\(^3\)\(^1\) Some of the key findings are presented in this section.


The most recent estimates of deaths caused by tobacco use show that almost 15 000 people died due to tobacco use in the financial year 2004–05.\(^1\) These calculations are the most recent in a series,\(^2\)\(^,\)\(^4\)\(^,\)\(^5\) the first of which was published in 1990.\(^6\) The methodology used in these reports has calculated 'attributable fractions' for the proportion of deaths due to specific diseases which can be said to have been caused by tobacco, based on extensive literature reviews and developed progressively since the first publication.

By this methodology, it has been estimated that in 2004–05, tobacco use caused a total of 14 901 deaths.\(^1\) Of these deaths, 14 790 were attributable to active smoking (including 56 deaths in infants subjected to maternal smoking) and 113 occurred in adults due to exposure to secondhand smoke (Table 3.30.1).\(^1\) As discussed in Section 3.28, smoking also confers a protective effect against a small number of diseases. It is estimated that in 2004–05, smoking prevented death from these specific diseases in 148\(^2\) smokers. However it should be noted that prevention of one kind of disease in a certain individual does not confer immunity against other diseases caused by smoking in the same individual; smokers do still die from these specific diseases; and there is no sound medical basis for taking up or continuing smoking in order to prevent or ameliorate the process of any disease.

<table>
<thead>
<tr>
<th>Condition</th>
<th>Male deaths</th>
<th>Female deaths</th>
<th>All deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oropharyngeal cancer</td>
<td>214</td>
<td>81</td>
<td>295</td>
</tr>
</tbody>
</table>


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---
<table>
<thead>
<tr>
<th>Condition</th>
<th>Male deaths</th>
<th>Female deaths</th>
<th>All deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oesophageal cancer</td>
<td>360</td>
<td>152</td>
<td>512</td>
</tr>
<tr>
<td>Stomach cancer</td>
<td>39</td>
<td>15</td>
<td>54</td>
</tr>
<tr>
<td>Pancreatic cancer</td>
<td>208</td>
<td>198</td>
<td>406</td>
</tr>
<tr>
<td>Laryngeal cancer</td>
<td>125</td>
<td>19</td>
<td>144</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>4164</td>
<td>1892</td>
<td>6057</td>
</tr>
<tr>
<td>Cervical cancer</td>
<td>0</td>
<td>30</td>
<td>30</td>
</tr>
<tr>
<td>Endometrial cancer</td>
<td>0</td>
<td>-52</td>
<td>-52</td>
</tr>
<tr>
<td>Bladder cancer</td>
<td>214</td>
<td>108</td>
<td>321</td>
</tr>
<tr>
<td>Kidney cancer</td>
<td>237</td>
<td>169</td>
<td>407</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>1282</td>
<td>361</td>
<td>1643</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>2226</td>
<td>1644</td>
<td>3870</td>
</tr>
<tr>
<td>Tobacco abuse</td>
<td>51</td>
<td>40</td>
<td>91</td>
</tr>
<tr>
<td>Parkinson's disease</td>
<td>-91</td>
<td>-6</td>
<td>-97</td>
</tr>
<tr>
<td>Pulmonary circulation disease</td>
<td>72</td>
<td>97</td>
<td>169</td>
</tr>
<tr>
<td>Cardiac dysrhythmias</td>
<td>38</td>
<td>21</td>
<td>59</td>
</tr>
<tr>
<td>Heart failure</td>
<td>81</td>
<td>43</td>
<td>124</td>
</tr>
<tr>
<td>Stroke</td>
<td>338</td>
<td>215</td>
<td>554</td>
</tr>
<tr>
<td>Peripheral vascular disease</td>
<td>38</td>
<td>21</td>
<td>59</td>
</tr>
<tr>
<td>Lower respiratory tract infection</td>
<td>58</td>
<td>30</td>
<td>89</td>
</tr>
<tr>
<td>Crohn's disease</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Ulcerative colitis</td>
<td>0</td>
<td>1</td>
<td>1</td>
</tr>
<tr>
<td>Antepartum haemorrhage</td>
<td>3</td>
<td>2</td>
<td>5</td>
</tr>
<tr>
<td>Low birthweight</td>
<td>8</td>
<td>4</td>
<td>12</td>
</tr>
<tr>
<td>SIDS</td>
<td>8</td>
<td>3</td>
<td>11</td>
</tr>
<tr>
<td>Fire injuries</td>
<td>12</td>
<td>12</td>
<td>24</td>
</tr>
<tr>
<td>Asthma (under 15 years)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Macular degeneration</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Otitis media</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Total deaths from active smoking</td>
<td>9686</td>
<td>5101</td>
<td>14790</td>
</tr>
</tbody>
</table>

**Secondhand smoke (deaths in adults only)**

<table>
<thead>
<tr>
<th>Condition</th>
<th>Male deaths</th>
<th>Female deaths</th>
<th>All deaths</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung cancer</td>
<td>2</td>
<td>9</td>
<td>12</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>33</td>
<td>68</td>
<td>101</td>
</tr>
<tr>
<td>Total adult deaths from SHS</td>
<td>35</td>
<td>77</td>
<td>113</td>
</tr>
<tr>
<td>Total all deaths from active smoking and exposure in adulthood to secondhand smoke</td>
<td>9723</td>
<td>5178</td>
<td>14901</td>
</tr>
</tbody>
</table>

*Columns do not add up to totals due to rounding*

*Department of Health and Ageing*

Source: Unpublished data from research undertaken for Collins and Lapsley

The same study also quantified deaths caused by other drug use in Australia. Table 3.30.2 shows that most drug-caused deaths in tobacco are caused by tobacco. Almost 90% of deaths due to drugs in 2004–05 were caused by smoking compared with 6% from alcohol and 5% from illicit drug use.
Table 3.30.2
Deaths due to drugs in Australia, 2004–05

<table>
<thead>
<tr>
<th>Drug</th>
<th>Males</th>
<th>Females</th>
<th>Total</th>
<th>% of Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco</td>
<td>9723</td>
<td>5178</td>
<td>14 901</td>
<td>89</td>
</tr>
<tr>
<td>Alcohol</td>
<td>1206</td>
<td>-149^</td>
<td>1057</td>
<td>6</td>
</tr>
<tr>
<td>Illicit drugs</td>
<td>583</td>
<td>289</td>
<td>872</td>
<td>5</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>11 512</td>
<td>5318</td>
<td>16 830</td>
<td>100</td>
</tr>
</tbody>
</table>

*Deaths are net of protective effect conferred for some disease entities
^Alcohol was estimated to have caused 913 deaths in females, but to have prevented 1061 deaths.
Source: Collins and Lapsley


The Burden of Disease and Injury in Australia 2003,\(^2\) (discussed in Section 3.29.2 above) also quantified deaths due to tobacco use, estimating that smoking caused a total of 15 511 deaths in 2003, or more than 1 in every 10 deaths (11.7%) (Table 3.30.3). The methodology used in this study is similar to that used in the AIHW/DoHA studies described in Section 3.29.1.

Lung cancer was the leading cause of deaths due to smoking, followed by chronic obstructive pulmonary disease, coronary heart disease and stroke (Table 3.30.3). This study did not report separately on fatalities due to tobacco by age or gender. The calculations includes deaths attributable to secondhand smoke, but separate estimates for the numbers of deaths caused by active smoking and exposure to secondhand smoke are not provided.\(^2\)

Table 3.30.3
Deaths attributable to tobacco by specific cause, Australia, 2003 (Burden of Disease calculations)

<table>
<thead>
<tr>
<th>Specific cause</th>
<th>Number of deaths</th>
<th>Percentage of all tobacco-caused deaths (rounded)*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lung cancer</td>
<td>6309</td>
<td>41</td>
</tr>
<tr>
<td>COPD</td>
<td>4175</td>
<td>27</td>
</tr>
<tr>
<td>CHD</td>
<td>1962</td>
<td>13</td>
</tr>
<tr>
<td>Stroke</td>
<td>577</td>
<td>4</td>
</tr>
<tr>
<td>Oesophageal cancer</td>
<td>572</td>
<td>4</td>
</tr>
<tr>
<td>Other</td>
<td>1916</td>
<td>12</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>15 511</td>
<td></td>
</tr>
</tbody>
</table>

*Column does not add up to 100% due to rounding.
Source: Derived from Begg et al\(^2\)

3.30.3 Estimated mortality from tobacco use, 2000–Peto et al methodology

Estimates of deaths caused by smoking in Australia have been calculated by Peto et al for 2000,\(^3\) using a methodology first described in 1992.\(^7\) Extrapolating from WHO mortality for lung cancer and other diseases, and using UN population data, Peto et al estimate that a total of 19 184 deaths were caused by active tobacco use in Australia in 2000 (Table 3.30.4).\(^3\) These estimates are likely to be conservative, because they do not include any deaths in individuals aged under 35 (including infants). Just under one third (about 6,000)
of all deaths due to smoking occur in individuals aged between 35 and 69, who lose, on average, about 23 years of life.\textsuperscript{3}

<table>
<thead>
<tr>
<th>Cause</th>
<th>Males</th>
<th>Females</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of deaths caused by smoking</td>
<td>% of all deaths from this disease attributable to smoking</td>
<td>Number of deaths caused by smoking</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>4029</td>
<td>88</td>
<td>1720</td>
</tr>
<tr>
<td>Upper aero-digestive ca</td>
<td>683</td>
<td>50</td>
<td>225</td>
</tr>
<tr>
<td>Other cancers</td>
<td>1393</td>
<td>10</td>
<td>307</td>
</tr>
<tr>
<td>COPD</td>
<td>2384</td>
<td>67</td>
<td>1553</td>
</tr>
<tr>
<td>Other respiratory</td>
<td>251</td>
<td>10</td>
<td>173</td>
</tr>
<tr>
<td>Vascular disease</td>
<td>2657</td>
<td>11</td>
<td>1851</td>
</tr>
<tr>
<td>Other medical</td>
<td>1086</td>
<td>10</td>
<td>872</td>
</tr>
<tr>
<td>All causes</td>
<td>12 483</td>
<td>19</td>
<td>6701</td>
</tr>
</tbody>
</table>

Source: Peto et al\textsuperscript{3}

The robustness and wide applicability of this methodology has enabled Peto et al to expand their calculations to worldwide estimates of mortality due to tobacco.\textsuperscript{8} (See Section 3.36).

Figure 3.30.1 presents the final column from Peto et al's data shown in the table above. This pie chart shows that of all deaths due to tobacco, 44% are cancer deaths, 23% are due to heart and circulatory diseases, and a further 23% are caused by lung and other respiratory disease.

Figure 3.30.1
Deaths attributable to smoking in Australia by disease entity, as a proportion of all tobacco-caused deaths, 2000

Source: Peto et al, 2006\textsuperscript{3}
This section will be updated when estimates covering more recent years are published.

From Table 13, Collins and Lapsley. ¹

References


As is the case elsewhere in the world, ill-health and rates of premature death in Australia show a clear gradient across socio-economic status (SES) groups.\textsuperscript{1–3}

People who are poorer or disadvantaged in other ways generally suffer more illness and reduced quality of life and die earlier than people who are better off. The social gradient holds regardless of how socio-economic disadvantage is measured.\textsuperscript{1}

People who are disadvantaged are more likely to live with multiple risks to their health. Lower socio-economic status is associated with higher rates of obesity, lack of adequate physical activity and diabetes—especially so among Indigenous communities.\textsuperscript{1,2,4}

However, with or without such additional risk factors, current smokers are much less likely than non-smokers to be in good health and the incidence of numerous diseases is significantly higher among smokers and recent ex-smokers than among long-time ex-smokers and never smokers.\textsuperscript{5,6}

Social differentials in smoking during pregnancy, smoking prevalence, cigarette consumption, duration of smoking and exposure to environmental tobacco smoke must contribute substantially to socio-economic differentials in health status and mortality.

This section outlines data on relative rates of poor health, disease, mortality and life expectancy across SES groups, and also presents estimates of the contribution of smoking to these health disparities.

### 3.31.1 Socio-economic position, reported health status and smoking

People who live in disadvantaged areas are much less likely to assess their own health as excellent or good.\textsuperscript{1,2}

Data from Australian national surveys commonly report higher rates of arthritis, chronic respiratory disease, cardiovascular disease and depression in least advantaged groups in comparison to more advantaged groups in the population.\textsuperscript{2,7–9} The rates of profound disability and type 2 diabetes in low socio-economic areas are double that of those in the highest socio-economic areas.\textsuperscript{2}
In 2010, only 41% of smokers participating in the National Drug Strategy Household Survey reported their overall health as ‘very good’ or ‘excellent’, compared to 50% of ex-smokers and almost 60% of non-smokers. Ex-smokers were more likely to report diagnoses or treatment for heart disease and cancer than smokers and non-smokers. Smokers were more likely to report asthma, and twice as likely as non-smokers to have been diagnosed with, or treated for, mental illness.\textsuperscript{10}

### 3.31.2 Socio-economic position and illnesses known to be caused by smoking

Hospitalisations for cardiovascular disease show a clear socio-economic gradient. In 2003–04 rates of hospitalisations for males in the most disadvantaged socio-economic group were 1.3 times those of males of least socio-economic disadvantage. The hospitalisation rate for the most disadvantaged females was higher again, with rates 1.4 times that of females in the least disadvantaged socio-economic group. A socio-economic gradient is evident for other chronic diseases for which smoking is a risk factor, with hospitalisations for coronary heart disease and stroke among males and females increasing as socio-economic status decreases.\textsuperscript{11}

The Australian Institute of Health and Welfare has estimated that lung cancer was the fourth leading cause of disease among males and the seventh leading cause of disease among females in 2011. Lung cancer incidence is disproportionately high in those of lower socio-economic status in Australia, with increasing incidence of lung cancer associated with decreasing socio-economic status, across the five years 2003–2007. In the year 2008–09, the rate of hospitalisations for lung cancer was higher for those living in the lowest socio-economic areas of Australia. Those living in the lowest socio-economic areas were hospitalised for lung cancer at 1.5 times the rate of those living in areas of highest socio-economic advantage.\textsuperscript{12}

Chronic kidney disease has increasingly been shown to be connected with smoking and cardiovascular disease.\textsuperscript{13, 14} It is more common in low socio-economic groups and particularly so among Indigenous Australians.\textsuperscript{2, 14}

Between 2000–01 and 2007–08, hospitalisations for chronic kidney disease were highest for Australians living in the most disadvantaged areas. Hospitalisations for kidney dialysis among the lowest socio-economic group were 1.6 times the rate of those in the most advantaged group. After removing the rates for regular dialysis from all chronic kidney disease-related hospitalisations, the rates of hospitalisations among the lowest socio-economic group remained almost twice that of the most advantaged group.\textsuperscript{15}

The worsening of asthma symptoms is known to be associated with active smoking and/or exposure to secondhand smoke. Smoking and asthma are both more common in those living in low socio-economic areas. The Australian Centre for Asthma Monitoring (a collaborating unit of the Australian Institute of Health and Welfare) reported that in 2007–08, not only was asthma much more common among those living in the most deprived socio-economic areas in Australia, but that rates of smoking among asthmatics in low socio-economic areas were far higher than for asthmatic smokers living in areas of higher socio-economic status (37.8% and 12.9% respectively). The disparity between the lowest and highest socio-economic group in asthma prevalence was found to have widened between survey years 2004–05 and 2007–08.\textsuperscript{16}

### 3.31.3 Socio-economic disparities in death rates from diseases known to be caused by smoking

Australians from lower socio-economic groups have a greater proportion of chronic disease mortality burden than those living in more advantaged areas.\textsuperscript{17} This sub-section presents information on socio-economic disparities in mortality rates from diseases associated with smoking, however it is important to note the influence and interplay of other health risk factors and social and economic deprivation across a life-course, in the contribution to disease and premature mortality among the disadvantaged. Section 3.31.5 provides a detailed discussion on quantifying the contribution of smoking to socio-economic differentials in health status; associations between childhood circumstances and health outcomes, smoking and intergenerational poverty
are discussed further in Section 3.3.1.

The potential years of life lost (PYLL) due to cancer deaths in 2007 was greater among Australians living in the most disadvantaged areas (55%) compared with those living in the least disadvantaged areas (42%). A gradient across socio-economic groups was evident for cardiovascular disease, chronic respiratory disease, digestive diseases and diabetes, whereby the proportion of PYLL due to premature mortality from these diseases were represented more highly in those living in lower socio-economic areas.\(^{17}\)

According to data compiled by the Public Health Information Development Unit in South Australia, a strong economic gradient was evident for premature mortality associated with lung cancer, with more avoidable lung cancer deaths in the most disadvantaged areas (25.9 per 100 000) compared with those in the least disadvantaged areas (15.2 per 100 000) between 2003 and 2007.\(^{18}\)

Similar trends of a disproportionate level of mortality burden being borne among those of less socio-economic advantage have been observed in international studies.

A 24-year study of British men and women examined the relationship between socio-economic status and mortality, and the influence smoking, alcohol consumption, diet and physical activity have on mortality. In terms of all-cause mortality, those of lowest socio-economic position had 1.6 times the risk of death in comparison with those of higher socio-economic position. There was also a graded association for cardiovascular disease mortality and socio-economic position. Health risk behaviours, including smoking, were connected with mortality.\(^{19}\)

Studies of cancer mortality in the US show disparities related to socio-economic position and also to ethnicity.\(^{20,21}\)

### 3.3.1.4 Socio-economic disparities in health-adjusted life expectancy

As part of research on preventable causes of disease conducted for the Australian Government, researchers at the University of Queensland examined differentials in the burden of disease across socio-economic groups.\(^6\)

At birth, those in the lowest socio-economic quintile could expect to die at least three years earlier than those in the highest economic quintile (79.6 compared with 82.7 years). Adjusting for ill-health, those in the lowest quintile could expect four years less than those in the highest quintile. By the age of 60, those in the lowest quintile could expect 15% fewer years of health-adjusted life than those in the highest quintile (Table 3.3.1.1).

<table>
<thead>
<tr>
<th>Socioeconomic quintile</th>
<th>Life expectancy at birth (years)</th>
<th>Health-adjusted life expectancy at birth (years)</th>
<th>Health-adjusted life expectancy at age 60 (years)</th>
<th>Life expectancy at birth lost due to disability (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>79.6 (79.4–79.7)</td>
<td>71.2</td>
<td>17.9</td>
<td>10.6</td>
</tr>
<tr>
<td>Moderately low</td>
<td>80.0 (79.9–80.2)</td>
<td>72.0</td>
<td>18.2</td>
<td>10.1</td>
</tr>
<tr>
<td>Average</td>
<td>80.2 (80.0–80.3)</td>
<td>72.2</td>
<td>18.4</td>
<td>9.9</td>
</tr>
<tr>
<td>Moderately high</td>
<td>81.2 (81.1–81.4)</td>
<td>73.6</td>
<td>19.3</td>
<td>9.4</td>
</tr>
</tbody>
</table>
Researchers estimated that, for the year 2003, a total of 2,632,800 disability-adjusted life years (DALYS) were lost in Australia. DALYs were calculated for each of the five socio-economic quintiles (Table 13.3.2).

Table 3.31.2
Disability-adjusted life years lost, Australia, 2003, by socio-economic quintile, Australia, 2003

<table>
<thead>
<tr>
<th>Socioeconomic quintile</th>
<th>DALYs ('000)</th>
<th>% of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>562.5</td>
<td>21.4</td>
</tr>
<tr>
<td>Moderately low</td>
<td>564.2</td>
<td>21.4</td>
</tr>
<tr>
<td>Average</td>
<td>523.6</td>
<td>19.9</td>
</tr>
<tr>
<td>Moderately high</td>
<td>507.7</td>
<td>19.3</td>
</tr>
<tr>
<td>High</td>
<td>474.8</td>
<td>18.0</td>
</tr>
<tr>
<td>Total</td>
<td>2,632.8</td>
<td>100.0</td>
</tr>
</tbody>
</table>

After adjusting for age, loss rates were 31.7% higher in the lowest SES quintile than in the highest. Rates of burden were higher for most causes, but particularly for mental disorders and cardiovascular disease. Per head of population, rates of burden were 26.5% higher in remote areas than in major cities.

Life expectancy among Indigenous Australians is discussed in Chapter 8.

In the US, researchers examined the effects of a number of health risk factors, including smoking, on life expectancy and disparities in life expectancy in eight sub-groups of the population. Individually, smoking and high blood pressure had the most profound effect on life expectancy disparities. They found that variation of life expectancies in the eight sub-groups would decline by 18% in men and 21% in women if the health risks (smoking, blood pressure, elevated blood glucose, and adiposity or obesity) had been reduced to optimal levels.

The Whitehall study followed more than 18,000 English males over a period of 38 years to examine life expectancy in relation to cardiovascular risk factors, which were recorded at middle age. The study reported that the presence of all three risk factors (smoking, high blood pressure and high cholesterol) at baseline (middle-age) predicted a three-fold rate of vascular mortality and about a 10-year reduced life expectancy from age 50 years, when compared with men who had none of the risk factors present at the commencement of the study.

3.31.5 Quantifying the contribution of smoking to socio-economic differentials in health status

Estimates of the contribution of smoking to social inequality vary, likely due to differences in study methodology and datasets. Estimates may also be affected by declines in smoking prevalence in developed countries, changing social demographics, latency of disease and death associated with smoking, and the emergence of other risk factors and their contribution to disease and mortality. This section presents research across time and using differing methods to quantify the contribution of smoking to health...
inequalities. Section 3.3.6 explores whether the inequalities in health outcomes and life expectancy are widening.

In the UK, Jarvis and Wardle used an 'indirect method' to estimate that tobacco caused about two-thirds of the difference in risk of death across social class in men age 35–69 years. Prabhat Jha and colleagues reported in a four-country study (England, Wales, Poland and North America) that most social inequalities in adult male mortality during the 1990s were due to smoking.24

Bobak and colleagues reported similar results for Canada, Poland and the US, and contended that eliminating smoking would halve the social gradient in mortality among men. Professor Sir Michael Marmot, a public health epidemiologist and expert in health inequality, has been critical of these sorts of estimates, because some estimates have been derived by using lung cancer mortality as a proxy measure for smoking exposure, rather than using crude estimates to determine the contribution of smoking to socio-economic differences in mortality; hence they are likely to overestimate the importance of smoking. Authors of these studies have generally acknowledged the limits of indirect estimation.

Blakely and Wilson and colleagues used direct methods to estimate the contribution of smoking to socio-economic and ethnic inequalities in mortality in New Zealand. Between 1996 and 1999, smoking contributed 21% to the gap between men aged 45–74 years with post-school qualifications and those with none. The corresponding figure for women was 11%. But other work suggested that only 5–10% of the larger inequality in mortality between Māori and non-Māori individuals was due to smoking, despite large differences in smoking prevalence. This estimate contrasted with a much greater estimated contribution by the Ministry of Health using Jha and colleagues' indirect method.

A study by Siahpush, English and Powles estimated that in Australia, smoking could account for just over one-third of the excess deaths in the 1990s that would otherwise be attributed to lower levels of education. Data on deaths among men aged 40–69 years taking part in a prospective cohort study in Melbourne between 1990 and 1994 showed that the association between education and mortality was greatly weakened after adjustment for smoking and the aetiologic fraction for low level of education was reduced from 16.5% to 10.6%.

Vallejo and colleagues used data from the National Health Survey for England to estimate the contribution of lifestyle factors–obesity and smoking–to health inequalities across social classes (classified by level of income). Their findings, released in 2010, show income as a significant contributor to health inequalities, and that obesity and smoking contribute significantly, but less profoundly, to income-related inequalities in health. Obesity and smoking were estimated to contribute 1.2% and 3.2% to inequality respectively. Despite the prevalence of smoking declining over time, its effects on inequalities have slightly increased because of its over-representation among the lowest socio-economic groups and its effects on health.

It is likely that indirect estimates of the contribution of tobacco smoking overestimate the importance of smoking by failing to take account of higher-than-average prevalence of behavioural and other risk factors in low-SES populations. Direct methods, however, may underestimate the importance of smoking because they do not take into account the long-term impact of smoking during pregnancy and the impact of smoking and exposure to tobacco smoke on diseases other than the ones for which epidemiological data are readily available. They also may not take account of the effects of spending on tobacco products on financial security and intergenerational poverty, which may help to perpetuate continuing high smoking rates in the children of smokers. These issues are explored further in Sections 9.4 to 9.8.

Thun also discusses the difficulties in directly quantifying the contribution of smoking to disparities across social classes in a review of a study by Menvielle and colleagues, whose work estimated the degree to which smoking contributes to social class differences (classified by education level) in lung cancer incidences across a cohort of individuals from 10 European countries. Menvielle and colleagues concluded that smoking could account for about 50% of the inequalities in lung cancer risk due social group disparities in education. They noted these findings were unusual, and suggest residual confounding by smoking. They noted that in future studies, other risk factors in relation to smoking should be considered. Thun expressed the complexity in quantifying a direct relationship in this study because of changing demographics in Europe—the relationship between social class, smoking and lung cancer incidences have evolved and changed over
time—noting, ‘it is extremely difficult for Menvielle et al. to disentangle the historical and birth cohort effects of lifetime smoking on lung cancer risk from any other factors that may have contributed to risk.’

3.31.6 Are tobacco-related differentials in health status widening?

In the US the socio-economic gap in life expectancy appears to be worsening. In people who had more than 12 years of education, life expectancy in the 1990s was about a year and a half greater than it was in the 1980s. In less educated people, life expectancy increased by only half a year. Much of the growing mortality gap can be attributed to the higher levels of decline in smoking-related diseases such as lung cancer and chronic obstructive pulmonary disease in more advantaged groups. Study authors attribute this to the larger declines in smoking prevalence in more advantaged compared with less advantaged groups that have been evident for some time in the US. Irvin and colleagues reported in 2009 that great disparities among socio-economic groups as well as racial groups exist for tobacco-related cancer incidences and mortality in the US. Disparities also 'exist in access to, and quality of, cancer treatment'.

The situation for Australia is much less clear-cut.

A study published by the Australian Institute of Health and Welfare in 2006 indicated that death rates for cardiovascular disease reduced in all socio-economic groups between 1999 and 2003. There was a decrease in the size of the gap between the rates of death between upper and lower socio-economic groups for coronary heart disease and cardiovascular disease as a whole but an increase in the relative effect of disadvantage (the proportion by which the lowest socio-economic group was higher than the highest socio-economic group) for coronary heart disease, stroke and cardiovascular disease as a whole.

In 2011, the Australian Institute of Health and Welfare reported death rates from cardiovascular disease have continued falling (based on AIHW mortality data from 2007). However, those of lower socio-economic status, the Indigenous and those living in remote areas of Australia still had the highest rates of hospitalisations and death from cardiovascular disease.

Between 1982 and 2007, the age standardised mortality rates for lung cancer among Australian males decreased significantly, whereas mortality rates among females increased across this period. This trend is indicative of past smoking patterns. Lung cancer mortality rates for males peaked in the early 1980s, and since this time, have declined substantially; a reflection of declining smoking rates in males in the second half of the 20th century. In the case of women, females took up smoking later in the 20th century (increasing since the mid-1940s and reaching prevalence of about 33% in the mid–1970s), yet they smoked less than males. This pattern is reflected in female lung cancer mortality rates. These have been increasing over time, but more recently in the 1990s and 2000s, the increase has slowed compared with decades prior. Mortality rates from lung cancer show a clear social gradient. For the period 2003–2007, the highest mortality rates for all persons were among those living in the most disadvantaged areas in Australia. The mortality rate for males living in the least advantaged areas was 1.5 times the rate of mortality for males living in the most advantaged areas. Among females, the gap was slightly less, with 1.3 times the mortality rate in females living in the least advantaged areas compared with females living in the most advantaged areas. No data could be located on whether or not disparities in lung cancer mortality have widened.

Between 1979 and 2006, mortality rates between low-SES groups and high-SES groups have narrowed in absolute terms among females for ischaemic heart disease (27 to 23 per 100 000). However, absolute differences for ischaemic heart disease widened in males across this period (52 to 63 per 100 000). Absolute differences for stroke between low and high-SES groups declined in males and females (16 to 13 per 100 000 among males and 13 to 7 per 100 000 among females).

However relative declines were greater in high socio-economic groups compared with low socio-economic groups for both ischaemic heart disease (28% average five yearly decline in high socio-economic status males compared with 21% in low-SES males, and 30% and 21% for females respectively). For stroke, there was a 25% average five yearly decline in high-SES males compared with 21% in low-SES status males; 26% and 23% for females respectively.
Estimate projected from a 2003 baseline, derived from AIHW Burden of Disease database, see table 7.1. (p.70)

Potential years of life lost (PYLL): ‘an indicator of premature death. PYLL are determined by age at death and takes in to account only deaths that occur before a particular age.’

References


3.32 Health effects of smoking other substances

This section is not intended to provide a comprehensive overview of drug use other than tobacco, but to demonstrate that the deliberate inhalation of smoke from the combustion of any matter is injurious to the health. The health effects of smoking any substance will depend on various factors, including the age at which smoking commences, the duration for which the body is exposed to the smoke, and the concentration and nature of constituents of the material smoked.

The scientific literature describing the health effects of smoking tobacco is comprehensive but the same cannot generally be said of other smoked substances, particularly where use is illegal. For example, in the case of marijuana, its illegality has militated against broad-based population studies, although data gathering has improved with its growing public acceptance (see Chapter 1, Section 1.10). Lack of adequate funding for medical and epidemiological research is a factor in some countries where smoking of substances other than tobacco may be more widespread. Another reason that these other forms of smoking have been under-researched is that the relatively small numbers of users and the scattered pattern of their geographical distribution make systematic study more challenging. Despite these issues the evidence base is sufficient to allow important conclusions on the adverse health effects of smoking these other substances on their own or in various combinations with tobacco. Here we cover the findings with regard to the smoking of herbal and other non-tobacco cigarettes, cannabis, kreteks and bidis.

3.32.1 Herbal and other non-tobacco cigarettes

Herbal and other non-tobacco cigarettes may erroneously be considered as a safer alternative to smoking, or an aid to quitting smoking, and are actively promoted as such by some manufacturers. However, even cigarettes that do not contain tobacco or nicotine may still produce toxic substances including carcinogens. Recent research has examined DNA damage response arising from exposure of human lung cells to smoke from tobacco- and nicotine-free cigarettes (made from lettuce and herbal extracts). This exposure led to formation of double-strand DNA breaks that are potentially carcinogenic; there was a dose–response relationship between exposure to the smoke and the severity of ensuing DNA damage response. The study concluded that smoking tobacco and nicotine-free cigarettes is at least as hazardous as smoking cigarettes containing tobacco and nicotine.

A 2009 study conducted in China tested the claim by the tobacco industry in that country that herbal cigarettes are less harmful than regular cigarettes. Four discriminating biomarkers were analysed from urine samples provided by 135 herbal cigarette smokers and 143 regular smokers. Importantly, the researchers found a concern about their health to be one of the main reasons that smokers switched from regular to herbal cigarettes and they reported increased consumption after doing so. The researchers found no
significant difference in the levels of the four biomarkers between smokers of herbal cigarettes and smokers of regular cigarettes, concluding that herbal cigarettes did not deliver less carcinogens than regular cigarettes and that the Chinese tobacco industry should avoid misleading the public by promoting herbal cigarettes as safer products. An analysis of ‘vegetable-based cigarettes’ manufactured in France and sold in Austria were found to yield levels of carbon monoxide at least as high as those produced by conventional cigarettes. Analysis of other constituents of the smoke was not made, although initial studies had shown that combustion may have produced carcinogens and other noxious chemicals. Investigations of a different brand of ‘non-nicotine, non-tar’ herbal cigarettes popular in the Philippines has also shown significant yields of tar and carbon monoxide. Smokers attempting to use herbal cigarettes as an adjunct to quitting are therefore exposing themselves to dangerous tar and carbon monoxide levels, without actually increasing their chance of quitting.

Cigarettes combining herbs and tobacco have been produced in China since 1959, and they are now manufactured in South Korea, Taiwan and Thailand. These cigarettes are commonly promoted with messages implying that they are aids in quitting, are less harmful than conventional cigarettes, or with claims for active health benefits such as raising immunity or protecting the kidneys. There is no reliable published literature to support any health claims for herbal-tobacco cigarettes, and at least one report (from the tobacco industry) has indicated that the tar and nicotine level of Chinese herbal-tobacco cigarettes is very high.

3.32.2 Cannabis (marijuana, hash, ganja)

The main forms of cannabis are marijuana, hashish and hashish oil. Marijuana comprises the dried leaves, flowering tops and stems of the hemp plant Cannabis sativa. The more concentrated resin from the flowers is called hashish. Cannabis is usually smoked as a cigarette (joint) or in a pipe (bong). The substance in cannabis that causes the user to experience a ‘high’ is THC (tetrahydrocannabinol), which binds to receptors in the brain. Recent studies have reported the emergence of synthetic cannabinoids; these compounds are more potent than traditional cannabis and have been widely used to deliver products with psychoactive properties while circumventing drug legislation. As a result, authorities around the world are now beginning to exert control by either naming individual compounds or using generic legislation. Some early research indicates that synthetic forms of cannabis may be more likely to provoke psychosis in vulnerable individuals.

Cannabis is the most frequently used illegal drug in Australia, with data published by the Australian Institute of Health and Welfare in 2011 indicating that about 1.9 million people had used the drug in a 12-month period. Between 2007 and 2010 the proportion of people in Australia who had used cannabis in the previous 12 months increased from 9.1% to 10.3%; among cannabis users, 20.9% said they used it once a week or more. More than half of people in Australia aged 30–39 years had used cannabis at some time in their lives, a proportion that was higher than in any other age group and was similar for both males and females. The highest proportion of males who had used cannabis in the last 12 months was for those aged 20–29 years (25%), and for females for those aged 18–19 years (19.3%). Fewer than 1 in 10 (8.8%) teenagers aged 12–17 years had used cannabis in the previous 12 months, but this proportion more than doubled to 1 in 5 (21.3%) among those aged 18–29 years. Continued use may lead to both physical and behavioural addiction, especially among regular, heavy users, and those who start using the substance at an earlier age.

A comprehensive review of the adverse effects of cannabis use was recently undertaken. The authors of the review concluded that the effects of cannabis are dose dependent; adverse effects most frequently reported in the literature are mental slowness, impaired reaction times, and accentuation of anxiety. Users can feel dependent on cannabis, but this dependence is usually psychological. Withdrawal symptoms tend to occur within 48 hours following cessation of regular cannabis use, and include increased irritability, anxiety, nervousness, restlessness, sleep difficulties and aggression. Symptoms subside within two to 12 weeks. Serious psychological disorders have been reported with high levels of intoxication. The relationship between poor school performance and early, regular and frequent cannabis use seems to be a vicious circle, in which each sustains the other. Many research studies have examined the possible long-term effects of cannabis on...
memory, but results to date are inconclusive. Longitudinal studies of the influence of cannabis on depressive thoughts or suicidal ideation have yielded conflicting results and are also inconclusive. Several longitudinal studies have shown a statistical association between psychotic illness and self-reported cannabis use, but methodological problems (particularly the unknown reliability of self-reported data) make it difficult to draw definitive conclusions about causation. Therefore the question as to whether cannabis use causes psychosis cannot yet be answered conclusively because of the limitations in the current evidence. This latter finding is supported by another robust review of cohort studies into cannabis and psychosis, conducted by McLaren and colleagues. Consistent with these study findings, a 2009 systematic review by Le Bec and colleagues noted that cannabis use may be an independent risk factor for the development of psychotic disorders, with the level of risk increasing by an estimated factor of 1.2–2.8. Hall and Degenhardt's review, published in The Lancet in 2009, is also consistent with the studies cited above; with a focus on adverse health effects of greatest potential public health impact (those most likely to occur and to affect a large number of cannabis users) these researchers concluded that the most probable adverse effects were: (i) a dependence syndrome, (ii) increased risk of motor vehicle crashes, (iii) impaired respiratory function, (iv) cardiovascular disease, and (v) adverse effects of regular use on adolescent psychosocial development and mental health. Studies have concluded that the provision of advice to vulnerable individuals that cannabis may cause acute psychotic effects (especially at high doses) is appropriate. There is evidence supporting an association between cannabis use and the development of major depression, anxiety and panic disorders. Cannabis use is linked to a 50% increased risk of a later depression spell (age ≥17 years) from early-onset use of cannabis (age <17 years). Recent Australian Institute of Health and Welfare population surveillance data provide corroborative support for these findings, noting the apparent relationship between a person's cannabis use and his or her mental health. For people in Australia aged 18 years or older, those who had reported using cannabis in the previous 12 months (18.7%) or in the previous month (20.5%) were more likely to have been diagnosed or treated for a mental illness than people who had not used it in the previous 12 month (11.3%); those who had used cannabis in the previous month (19.1%) or previous 12 months (16.3%) were more likely to report high or very high levels of psychological distress compared with those who had not recently used cannabis (9.1%).

A systematic review conducted to assess risk of cannabis-related mortality examined the scientific literature published between 1990 and 2008; it found that evidence published to date is insufficient to assess whether the all-cause mortality rate is elevated among cannabis users in the general population. Nonetheless, case–control study evidence does suggest that some adverse health outcomes may be elevated among heavy cannabis users, namely, fatal motor vehicle accidents, and possibly respiratory and brain cancers. The evidence as to whether regular cannabis use increases the risk of suicide is unclear. There is evidence that babies born to women who have smoked marijuana during pregnancy have a greater probability of experiencing developmental problems, evident from early schooling through to adolescence and that prenatal exposure may have long-term effects, specifically on attentional skills.

Mixing tobacco with cannabis elevates the amount of THC administered while smoking; a recent study found that tobacco increases the amount of THC inhaled per gram of cannabis from 32.70 +/- 2.29 mg/g for a 100% cannabis cigarette to 58.90 +/- 2.30 mg/g for a 25% cannabis cigarette. This indicates that tobacco increases the vaporisation efficiency of THC by as much as 45%. Recent laboratory studies have compared mainstream and sidestream cannabis and tobacco smoke condensates for their genotoxicity (level of damage or mutation caused to DNA). The cannabis condensates were all found to be more toxic to cells and more mutagenic than the matched tobacco condensates. For tobacco, the resulting genetic damage appeared to be dose dependent, whereas for marijuana it did not. Cannabis smoke contains a higher concentration of carcinogens than tobacco smoke, and because of a user's tendency to inhale deeply and hold the smoke in for longer, exposure of the lung and airways to toxic chemicals may be far greater per joint of cannabis than per typical tobacco cigarette. Long-term, heavy users of cannabis show a higher frequency of inflammatory and pre-cancerous changes to the bronchial tubes than non-users. A recent review of the evidence concludes that there are good grounds for believing that chronic smoking of cannabis carries a significant risk of cancer. There is some evidence of a possible link between cannabis use and testicular cancer; in a hospital-based case–control study, patients with nonseminoma (testicular germ cell cancer) were three times more likely than controls to be frequent cannabis users (OR 3.1; 95% CI, 1.2–8.2). Recent research examining the possible link between cannabis use and head and neck cancers
did not rule out completely the possibility of moderately increased risk; however large increases in risk levels for these specific cancers have not as yet been detected.24

Cannabis use has been linked to the development of cardiovascular symptoms, particularly in the predisposed.12 Smoking cannabis has a significant impact on lung structure and function; it causes airflow obstruction, impaired large airways function and hyperinflation (overexpansion of the lungs due to reduced elasticity), the effects worsening as the amount smoked increases. Research has shown one joint of cannabis to be equivalent to between 2.5 and 5 tobacco cigarettes in terms of airflow obstruction, probably for the most part due to differences in the way cannabis is smoked (deeper and longer inhalations), as well as the fact that a joint is generally smoked without a filter and down to a smaller butt length, and that the smoke is generated at a higher temperature.22 Cannabis smokers also experience respiratory symptoms including wheezing, coughing, phlegm production and chest tightness.22 Over the longer term, using both cannabis and tobacco has an additive effect on respiratory symptoms and lung function. This means that in users of both substances, respiratory symptoms are increased and lung function is worsened to a greater extent than if just one of the two drugs is used.25 A recent study found that concurrent use of cannabis and tobacco was associated with a two-fold risk of respiratory symptoms (OR 2.39; 95% CI, 1.58–3.62) and an almost three-fold risk of chronic obstructive pulmonary disease (OR 2.90; 95% CI, 1.53–5.51) if the lifetime dose exceeded 50 cannabis cigarettes. The research concluded that these increased risks of respiratory symptoms and of chronic obstructive pulmonary disease were related to a synergistic interaction between cannabis and tobacco.26

3.32.3 Bidis (beedis, beedies, biris)

Bidis are small, thin, hand-rolled cigarettes consisting of sun-dried and cured tobacco flakes rolled up in a piece of dried tendu or temburni leaf (from plants native to Asia). They may be flavoured with a variety of sweet or fruit essences (e.g. chocolate, cherry and mango) or unflavoured, and are secured at either end with colourful threads.36 Bidi use is most prevalent in India, Bangladesh, Nepal, Sri Lanka, Pakistan and the Maldives.37, 38 Up to 56% of men in South Asian countries smoke bidis.39 Smoke from a bidi contains 3–5 times the amount of nicotine as a regular cigarette and places users at risk for nicotine addiction.40 The compounds added to provide flavouring (such as clove, cinnamon, vanilla, cardamom, strawberry, mango, grape, lemon-lime and chocolate) are also present in high levels and may contribute to long-term damage to health, particularly in the case of cloves.41 Bidi smoking increases the risk of oral cancer,42-44 lung cancer,45-47 stomach cancer, and oesophageal cancer;48 is associated with a more than three-fold increased risk for coronary heart disease and acute myocardial infarction (heart attack);39, 49 is associated with a nearly four-fold increased risk for chronic bronchitis;39 and is also associated with emphysema.50 Bidi use in pregnancy is associated with perinatal mortality.51 Tobacco factory employees who hand-roll bidis are chronically exposed to potentially toxic levels of tobacco via inhalation of dust and flakes, and through the skin.39

Bidis are smoked by 2.4% of high school and 1.6% of middle school students in the US.35 Consumption patterns in Australia are not known.

References


2. Jorgensen ED, Zhao H, Traganos F, Albino AP and Darzynkiewicz Z. DNA damage response induced by exposure of human lung adenocarcinoma cells to smoke from tobacco- and nicotine-free cigarettes. Cell


50. Gupta PC and Asma S. Bidi smoking and public health. New Delhi: Ministry of Health and Family

3.33 Health effects of chewing tobacco, and of other smokeless tobacco products

See Chapter 12, Section 12A.3.
3.34 Public perceptions of tobacco as a drug, and knowledge and beliefs about the health consequences of smoking

Last updated: March 2015


3.34.1 Australian attitudes to tobacco as a drug

Findings from the National Drug Strategy Household Survey in 2013 show that as smoking rates continue to decline, fewer people think that tobacco is the drug that causes the most deaths (decreasing from 36% in 2010 to 32% in 2013). The drug perceived to be associated with the most deaths was alcohol (34.0%), followed by tobacco (32.0%) and heroin (14.1%)—see Table 3.34.1.¹

<table>
<thead>
<tr>
<th>Drug</th>
<th>2010</th>
<th>2013</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco</td>
<td>36</td>
<td>32</td>
</tr>
<tr>
<td>Alcohol</td>
<td>30</td>
<td>33.6</td>
</tr>
<tr>
<td>Heroin</td>
<td>16</td>
<td>14.1</td>
</tr>
<tr>
<td>Ecstasy/designer drugs</td>
<td>4</td>
<td>4.6</td>
</tr>
<tr>
<td>Cocaine</td>
<td>5</td>
<td>3.7</td>
</tr>
<tr>
<td>Meth/amphetamine</td>
<td>4.7</td>
<td>8.7</td>
</tr>
<tr>
<td>Other illicit drugs</td>
<td>9</td>
<td>0.5</td>
</tr>
</tbody>
</table>

Source: NDSHS 2013¹

Tobacco does not rank highly as ‘the drug thought to be of most serious concern for the community,’ presumably reflecting the greater social disruption caused by alcohol and illegal drugs (Table 3.34.2). In 2013, among people aged 14 years or older, 42.5% thought that excessive alcohol drinking was the most concerning form of drug use for the general community. This was followed by meth/amphetamines, identified by 16.1% of people.¹ Tobacco smoking ranked third, at 14.5%.
Males and females had similar perceptions about which drugs they thought were the most concerning for the community, but males were more concerned about smoking (16% compared with 13%), and females were more concerned about excessive alcohol use (44% compared with 41%).

Different age groups were concerned about different drugs, with older people more concerned with excessive alcohol use (45% for those aged 50–59 years compared with 39% for those aged 20-29 years), and younger people more concerned with tobacco use (18% for those aged 14–19 compared with about 13% for those aged 50-59).

Table 3.34.2
Form of drug use thought to be of most serious concern for the general community, population aged 14 and over, Australia 2013

<table>
<thead>
<tr>
<th>Drug</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco</td>
<td>14.5</td>
</tr>
<tr>
<td>Alcohol</td>
<td>42.5</td>
</tr>
<tr>
<td>Heroin</td>
<td>10.7</td>
</tr>
<tr>
<td>Ecstasy</td>
<td>5.2</td>
</tr>
<tr>
<td>Meth/amphetamines</td>
<td>16.1</td>
</tr>
<tr>
<td>Cannabis</td>
<td>3.8</td>
</tr>
<tr>
<td>Cocaine</td>
<td>3.6</td>
</tr>
<tr>
<td>Other</td>
<td></td>
</tr>
</tbody>
</table>

Source: Derived from NDSHS 2013

Again presumably reflecting the social disruption caused by other drug use, tobacco is not a drug most likely to be associated with a 'drug problem' by most people (Table 3.34.3).

Table 3.34.3
Drugs most likely to be associated with a 'drug problem', population aged 14 and over, Australia, 2013

<table>
<thead>
<tr>
<th>Drug</th>
<th>% (rounded)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco</td>
<td>3 2</td>
</tr>
<tr>
<td>Alcohol</td>
<td>8 7</td>
</tr>
<tr>
<td>Heroin</td>
<td>25 26</td>
</tr>
<tr>
<td>Ecstasy</td>
<td>4 3</td>
</tr>
<tr>
<td>Methamphetamine</td>
<td>23 21</td>
</tr>
<tr>
<td>Cannabis</td>
<td>23 23</td>
</tr>
<tr>
<td>Cocaine</td>
<td>10 12</td>
</tr>
</tbody>
</table>

Source: Derived from NDSHS 2013

Of all drugs used in Australia, alcohol has the greatest degree of personal approval, followed by pharmaceuticals (used for non-medical purposes) and tobacco (Table 3.34.4). Not surprisingly, individuals who have used a particular drug recently are more likely to approve of its regular usage than those who have not used the drug in the preceding year.

In 2013, people aged 14 years and over in the lowest socioeconomic group approved of regular tobacco use by adults more often than those in the highest socioeconomic group (19% compared with 11%, respectively), but were less likely to approve of regular adult alcohol use than those in the highest socioeconomic group (38% compared with 51%). People in remote or very remote areas (16%), Indigenous Australians (24%),
and those who identify as homosexual or bisexual (24%) approved of regular tobacco use more than people in major cities (14%), non-Indigenous Australians (15%), and people who identify as heterosexual (14%).

### Table 3.34.4

<table>
<thead>
<tr>
<th>Drug</th>
<th>% (rounded)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Males</td>
</tr>
<tr>
<td>Tobacco</td>
<td>17</td>
</tr>
<tr>
<td>Alcohol</td>
<td>52</td>
</tr>
<tr>
<td>Cannabis</td>
<td>13</td>
</tr>
<tr>
<td>Prescription pain killers</td>
<td>13</td>
</tr>
<tr>
<td>Over the counter pain killers</td>
<td>13</td>
</tr>
<tr>
<td>Ecstasy</td>
<td>3</td>
</tr>
</tbody>
</table>

*Source: NDSHS 2013*

### 3.34.2 Awareness among smokers and recent quitters of damage to health caused by smoking

Most Australian smokers agree that smoking causes disease. As part of the International Tobacco Control Four Country Survey (ITC-4) in 2002, a representative sample of Australian smokers was asked whether smoking causes lung cancer, heart disease, stroke and impotence. Awareness of the first three conditions was high across groups with vary levels of education and income (Table 3.34.5).

### Table 3.34.5

<table>
<thead>
<tr>
<th>Percentage* agreeing that smoking is a cause of:</th>
<th>Lung cancer</th>
<th>Heart disease</th>
<th>Stroke</th>
<th>Impotence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Education level</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>94</td>
<td>88</td>
<td>80</td>
<td>34</td>
</tr>
<tr>
<td>Medium</td>
<td>96</td>
<td>90</td>
<td>81</td>
<td>35</td>
</tr>
<tr>
<td>High</td>
<td>96</td>
<td>89</td>
<td>83</td>
<td>43</td>
</tr>
<tr>
<td>Income level</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Low</td>
<td>92</td>
<td>86</td>
<td>78</td>
<td>36</td>
</tr>
<tr>
<td>Medium</td>
<td>94</td>
<td>89</td>
<td>81</td>
<td>35</td>
</tr>
<tr>
<td>High</td>
<td>97</td>
<td>91</td>
<td>84</td>
<td>37</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>94</strong></td>
<td><strong>89</strong></td>
<td><strong>81</strong></td>
<td><strong>36</strong></td>
</tr>
</tbody>
</table>

*Percentages rounded

*Source: Siahpush et al.*

In 2004, 94% of Australian smokers surveyed as part of the same study agreed that smoking caused lung cancer, 89% agreed it caused heart disease, 81% agreed it caused stroke, 36% agreed it caused impotence and 69% agreed smoking caused lung cancer in non smokers.

Overall, of the four diseases mentioned, smokers had the highest awareness of lung cancer, followed by
heart disease, stroke and impotence. Smokers with higher education and income levels tended to have a greater degree of knowledge than other smokers. Although it is widely accepted that smoking causes lung cancer, it is of concern that around 20% of smokers do not believe tobacco use causes stroke, and 10% do not think that smoking causes heart disease. Canadian smokers demonstrated the overall highest awareness of the health risks of smoking, ahead of smokers in Australia, the United Kingdom and the United States of America.²³

As part of ongoing evaluation of the National Tobacco Campaign staged between June 1997 and December 2000, a series of national annual surveys of public awareness about the health consequences of smoking have been undertaken.⁴ Because the target group for the campaign was smokers aged between 18 and 40 years, the survey group studied falls within this age-range. The findings reported in the tables below do not, therefore, represent the whole population. However there is strong evidence that the advertising and promotion associated National Tobacco Campaign has had an impact on people aged under 18,⁵ and it is highly probable that population groups aged over 40 were also educated and influenced by the Campaign to some extent.¹

Survey respondents in each year were asked whether, in their opinion, there were any illnesses caused by smoking (Table 3.34.6). If they thought that smoking did cause illness, they were asked to name the diseases. In every survey year, about 95% of respondents believed that smoking caused illness or damage to health. Highest awareness was of lung damage (especially lung cancer) and arterial illness or damage. Four out of five smokers or recent quitters spontaneously nominated smoking as a cause of lung illness or damage, and between a quarter and a third of smokers stated that smoking was a cause of arterial illness or damage. Nomination of particular disease entities varied for some of the years, probably due to timing of various campaign initiatives.⁴

| Table 3.34.6 Unprompted awareness of illness and damage caused by smoking among smokers and people who have quit in the last year, aged 18–40, Australia 1997 to 2000 |
|-----------------------------------------------|-----------------|-----------------|-----------------|-----------------|-----------------|
| Smokers and recent quitters (percentage)      | Bench-mark May 1997 (n=1192) | Follow-up 1 Nov 1997 (n=2981) | Follow-up 2 Nov 1998 (n=1647) | Follow-up 3 Nov 1999 (n=1612) | Follow-up 4 Nov 2000 (n=1675) |
| Believe there are illnesses or damage caused by smoking | 95 | 93 | 95 | 95 | 94 |
| **Specific illness mentioned**                |                 |                 |                 |                 |                 |
| Blocked blood arteries                       | 9 | 13 | 12 | 12 | 9 |
| Blocked blood vessels                        | 3 | 6 | 6 | 6 | 6 |
| Circulatory disease                          | 4 | 7 | 5 | 8 | 5 |
| Circulatory problems                         | 8 | 11 | 10 | 12 | 10 |
| Blood pressure                               | 6 | 7 | 6 | 7 | 6 |
| **Any artery illness/damage**                | 26 | 32 | 30 | 32 | 26 |
| Emphysema                                    | 37 | 34 | 36 | 35 | 34 |
| Lung damage                                  | 13 | 12 | 15 | 11 | 13 |
| Lung cancer                                  | 64 | 62 | 61 | 62 | 66 |
| **Any lung illness/damage**                  | 80 | 79 | 79 | 80 | 80 |
| Genetic/DNA damage                           | 1 | 2 | 2 | 2 | 1 |
| Heart disease                                | 37 | 30 | 34 | 32 | 39 |
| Cancer (unspecified)                         | 34 | 34 | 32 | 37 | 32 |
| Throat cancer                                | 16 | 17 | 16 | 17 | 20 |
| Clots in the brain                           | - | - | 4 | 2 | 1 |
Respondents were also asked whether or not they agreed or disagreed with particular statements made about smoking (Table 3.34.7). Compared to the previous table, which shows unprompted awareness, not surprisingly prompted awareness was much higher. About 90% of smokers and recent quitters agreed that smoking causes lung cancer, and almost as many smokers were aware that smoking causes emphysema. Agreement with statements regarding heart disease and environmental tobacco smoke increased significantly over the survey period. Statements were also offered for smokers and recent quitters to express disagreement with. Over the time period surveyed, smokers and recent quitters became significantly less likely to disagree with the notion that the health dangers of smoking have been exaggerated. Respondents were also more likely to resist key myths about smoking over the years surveyed. Increasing numbers of smokers and recent quitters expressed disagreement with the statements that 'smoking can't be all that bad because many people smoke all their lives and live to a ripe old age,' and 'smoking the occasional cigarette does not cause any damage to your health.'

<table>
<thead>
<tr>
<th>Smokers and recent quitters</th>
<th>Bench-mark May 1997 (n=1192)</th>
<th>Follow-up 1 Nov 1997 (n=2981)</th>
<th>Follow-up 2 Nov 1998 (n=1646)</th>
<th>Follow-up 3 Nov 1999 (n=1611)</th>
<th>Follow-up 4 Nov 2000 (n=1675)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Agree with opinion statements (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>'Smoking causes lung cancer'</td>
<td>88</td>
<td>87</td>
<td>89</td>
<td>90</td>
<td>*na</td>
</tr>
<tr>
<td>'Smoking causes heart disease'</td>
<td>83</td>
<td>84</td>
<td>85</td>
<td>88</td>
<td>na</td>
</tr>
<tr>
<td>'Your smoking can harm others'</td>
<td>82</td>
<td>83</td>
<td>80</td>
<td>85</td>
<td>88</td>
</tr>
<tr>
<td>'Smoking causes emphysema'</td>
<td>86</td>
<td>86</td>
<td>86</td>
<td>88</td>
<td>na</td>
</tr>
<tr>
<td>'It would improve my health if I quit smoking'</td>
<td>93</td>
<td>93</td>
<td>94</td>
<td>95</td>
<td>na</td>
</tr>
<tr>
<td>Disagree with opinion statements (%)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>'The dangers of smoking have been exaggerated'</td>
<td>59</td>
<td>64</td>
<td>61</td>
<td>64</td>
<td>68</td>
</tr>
<tr>
<td>'Smoking can't be all that bad because many people smoke all their lives and live to a ripe old age'</td>
<td>59</td>
<td>61</td>
<td>60</td>
<td>62</td>
<td>66</td>
</tr>
<tr>
<td>'Smoking the occasional cigarette doesn't cause any damage to your health'</td>
<td>50</td>
<td>57</td>
<td>55</td>
<td>57</td>
<td>60</td>
</tr>
</tbody>
</table>

* statements involving specific diseases that were almost universally agreed to be caused by smoking in earlier years were not repeated in the 2000 survey.
Source: Wakefield et al4

Smokers were also asked whether or not they thought they were likely to become ill from smoking if they continue to smoke. Both smokers and recent quitters were asked whether they thought they had already sustained some harm from smoking. Over the survey period, smokers became significantly more likely to agree that smoking would make them ill, and more than half of smokers and recent quitters felt that their health had already been damaged by smoking (Table 3.34.8).
Table 3.3.4.8
Beliefs about personal risk of experiencing illness or harm from smoking among smokers and people who have quit in the last year, aged 18–40, 1997 to 2000

<table>
<thead>
<tr>
<th>Smokers and recent quitters</th>
<th>Bench-mark May 1997 (n=1192)</th>
<th>Follow-up 1 Nov 1997 (n=2981)</th>
<th>Follow-up 2 Nov 1998 (n=1646)</th>
<th>Follow-up 3 Nov 1999 (n=1611)</th>
<th>Follow-up 4 Nov 2000 (n=1675)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Likelihood of personally becoming ill from smoking: % of smokers agreeing that it is likely</td>
<td>45</td>
<td>52</td>
<td>50</td>
<td>50</td>
<td>53</td>
</tr>
<tr>
<td>Has smoking already done any harm to your body? % of smokers and recent quitters agreeing that it probably has</td>
<td>51</td>
<td>57</td>
<td>57</td>
<td>53</td>
<td>57</td>
</tr>
</tbody>
</table>

Source Wakefield et al

A realistic appreciation of the health risk posed by smoking to the individual helps to shape attitudes to quitting. Smokers who have an unrealistic optimism about their personal risk of avoiding illness from smoking are less likely to quit smoking. Attitudes to smoking are further explored in Chapter 6 and Chapter 7.


References


3.35 Health and other benefits of quitting

A substantial body of research has established that quitting smoking has immediate as well as long-term health benefits for men and women of all ages, reducing risks for diseases caused by smoking and improving health in general.1–4

The strongest evidence for this comes from a landmark 50-year follow-up of 34 000 British male doctors first studied in 1951.5–7 Many participants quit as the evidence on smoking and health accumulated from the 1950s onwards, providing a natural experiment demonstrating the impact of number of years smoking on health and eventual mortality. The study showed just how hazardous tobacco is and estimated that almost two-thirds of persistent smokers were killed by their smoking. Among those who quit, the greatest benefit was seen in those who quit earliest in life.7 Quitting at age 50 halved the risk of smoking-related death, but cessation by age 30 avoided almost all of the excess risk. Stopping at age 60, 50, 40 or 30 resulted in gains, respectively, of about 3, 6, 9 or 10 years of life expectancy.7,8

Changes in disease risk following cessation can be measured in different ways.

A common measure is relative risk, where the likelihood of developing or dying of disease in a population of former smokers is compared to either current or never smokers. At a population level, relative risk represents the fraction of disease attributable to smoking. However this measure is influenced by the rates of disease in the reference population, which should be taken into account when examining the influence of cessation on disease risk.

Another measure is absolute risk, where the actual rates of disease in former smokers are compared to those of current or never smokers. Rates can be directly compared, or the excess rate of disease caused by smoking in smokers can be calculated as can the excess disease rate in former smokers. Another measure is cumulative risk of disease, which enables the cumulative risk for those who quit at different ages to be compared to that of continuing smokers.

A more complete discussion of changes in risk following cessation can be found in a handbook published in 2007 by the International Agency for Research in Cancer.3,4 In general, the risk of disease is lower in former smokers than in otherwise similar current smokers. Smoking results in both acute and chronic changes to the body and progression towards disease. Cessation results in reversal of acute changes and slowing of disease progression and provides the potential for damage reversal.3

Many harmful effects of smoking are arrested or begin to decline as soon as a person stops smoking.1,2 Many disease risks in former smokers continue to decrease with prolonged abstinence, compared to
continued smoking. The risk for some health effects decreases more rapidly than for others, and improvement may continue for years after quitting. Some disease risks return to the level of never smokers after a long period of abstinence, but others do not, even after 20 years of abstinence.3

The extent of damage to health and risk for smoking-related disease is related to how much the person has smoked and for how long.3 For some health effects, for example inflammation of the lung, the reversal process is not yet well understood.3

However, while some damage may be irreversible or less reversible, there are substantial benefits to be gained from quitting at any age, regardless of smoking history.1,2 Benefits accrue to persons both with and without smoking-related disease.1

3.35.1 Health problems that may be temporarily exacerbated by quitting

While there is no question of the overall long-term benefits of cessation, quitting is associated with a number of bothersome short-term problems such as mouth ulcers and cold symptoms, weight gain and constipation.9

Mouth ulcers and colds

There is evidence that smokers and users of smokeless tobacco are less likely to develop aphthous stomatitis (common mouth ulcers). Individuals commonly report a short-term increase in mouth ulcers and cold symptoms on quitting smoking.10

Depression

Many smokers appear to have an increase in depressed mood and associated negative affect as part of nicotine withdrawal, but for the majority of people who quit this is temporary.11,12 Smokers with a history of depression tend to report higher levels of nicotine dependence and experience more severe and prolonged withdrawal episodes, including greater negative mood.13–16 Among smokers with a history of depression, around 30% who stop smoking will develop a new episode of major depression. The risk remains high for at least six months.17,18

3.35.2 Quitting and weight gain

While smoking cessation usually results in some level of weight gain, there is disagreement about the extent and how long it lasts.

Smokers’ average weight is about 3 to 4 kg less than that of non-smokers.12,18 Smoking appears to attenuate weight gain over time, in part due to increasing metabolic rate.19,20 The difference in weight between smokers and non-smokers is more marked in older long-term smokers while the average weight of younger smokers is similar.20–23 The weight difference, however, is further complicated by the finding that despite their lower weight and body mass index (BMI), smokers have a greater waist-to-hip ratio than non-smokers. Increased waist circumference is a stronger predictor of cardiovascular disease than BMI.24

When smokers quit, the majority experience some weight gain.1 Estimates of weight gain associated with cessation vary depending on the sample, study design and follow-up period.25 Most excess weight gain occurs in the first year after cessation, after which the rate of weight gain slows.19,21,26–30 One study found that increase in body weight may continue for longer.31 Estimates of the mean weight gain in people continuously abstinent for a year are about 5 to 6 kg.27–30 Individual experience of weight change after quitting is quite broad, ranging from weight loss to a minority gaining over 10 kg.21,28,30–35 Increase in waist circumference per kilogram gained is smaller in people who quit than in continuing smokers, indicating that recent ex-smokers gain less visceral fat.33,36

Limited research suggests that some of the weight gained during the first few years after quitting may be lost
with continued abstinence, however more research is needed to resolve this issue. Large cross-sectional studies show that long-term former smokers have a mean waist-to-hip ratio and a mean BMI similar to or approaching that of people who have never smoked.

Reasons for the association between smoking cessation and weight gain are not fully understood. Predictors of weight gain include younger age, lower socio-economic status and heavier smoking, with some influence of underlying genetic factors. Weight gain after smoking cessation is related to a transient increase in food intake and to changes in metabolic rate. There is some evidence that smoking and obesity are independently associated with specific food cravings and mood states.

The health benefits of smoking cessation far outweigh the health risk from extra body weight, unless the weight gain is extraordinarily large. Despite this, fear of weight gain is a significant factor in discouraging quitting and provoking relapse in smokers.

(See Chapter 3, Section 3.29 for further information on the health effects of smoking in conjunction with and compared with those associated with obesity, and Section 7.8.3 for further information on managing weight gain.)

### 3.35.3 Immediate improvements in wellbeing and functioning

Upon cessation, the nicotine and carbon monoxide levels in the body decline rapidly. Nicotine levels drop to very low levels within a few hours, and the main metabolites of nicotine are largely eliminated within a week. After 24 hours the level of carbon monoxide in the blood has decreased substantially. After a year blood pressure returns to normal levels (this means it generally stabilises at whatever the person’s new blood pressure is) and small airway function improves, with further improvements after six months. After two months, improvements can be seen in blood viscosity, blood flow to the limbs and blood levels of high-density cholesterol. Within six months the immune system improves greatly. Within a few months the cilia in the lungs and airways improve at sweeping mucus and debris from the lungs (as long as irreversible damage has not taken place). Lung function improves and the presence and severity of respiratory symptoms reduces. Symptoms of chronic bronchitis, such as chronic cough, mucus production and wheeze, decrease rapidly, and lung function in asthmatic patients improves within a few months after stopping smoking. Rates of respiratory infections such as bronchitis and pneumonia also decrease, compared to continued smoking.

### 3.35.4 Short to medium-term reductions in health risks following quitting

#### 3.35.4.1 Problems during pregnancy

It is extremely dangerous for a woman to smoke during pregnancy. (Refer to Chapter 3, Section 3.7 for a more detailed discussion of health effects, and Section 7.11 for a more detailed discussion of interventions aimed at pregnant women and their partners.) The US Surgeon General has stated that ‘smoking is probably the most important modifiable cause of poor pregnancy outcome among women in the United States’. Stopping smoking before or during pregnancy is important and has benefits for both the baby and the mother. Encouraging women to quit before they become pregnant or early in pregnancy is important because the critical period may be quite early. Although the effect of cutting down on the numerous health risks to the foetus is not well studied, there is no solid evidence that cutting down significantly reduces the risks to the foetus.

Women who stop smoking either before becoming pregnant or in the first three to four months of pregnancy have infants with a similar birthweight to those born to women who have never smoked. Women who stop smoking any time up to the 30th week of pregnancy have infants with higher birthweights than those who smoke throughout pregnancy. Reducing the number of cigarettes smoked, instead of quitting completely, does not appear to benefit the birthweight of the foetus. Low birthweight infants have a higher
risk of illness, death and developing diseases in childhood and adulthood.1,2 Women who quit smoking before or during pregnancy reduce their risk of pregnancy complications, including preterm premature rupture of membranes and preterm delivery (birth at less than 37 weeks gestation).1,20 Smoking cessation reduces the risk of infant death.62

3.35.4.2 Diseases for which the risk quickly declines

Heart disease

Smoking cessation reduces the risk of cardiovascular disease and death for male and female smokers of all ages with or without heart disease.4 There are immediate and long-term benefits.63 After one year the increased risk halves and after 15 years the rate is similar to that of a non-smoker.1 Quitting helps to improve peripheral vascular tone64 and to prevent atherosclerosis (the narrowing and hardening of the arteries due to build-up of plaque on the artery walls) advancing to heart disease and stroke.2,3,65 Smoking is a known risk factor for sudden cardiac death (SCD),2 and quitting smoking results in a significant reduction in SCD risk.66

Stroke

There is a marked reduction in risk within two to five years of quitting.3 After 15 years the risk of stroke is the same as a non-smoker.1

Oral health

Stopping smoking can reduce the risk of oral diseases associated with smoking including cancer, and improve the health of the mouth, gums and teeth.2,67–69 Stopping smoking reduces the risk of leukoplakia, and after one to five years about half of leukoplakia disappears.70 Cessation reduces the risk of developing periodontitis, slows down the progress of existing disease, and quite quickly improves wound healing.2,67,71–73 Following cessation, gum colour gradually returns to normal68 and so-called 'smoker’s palate' can disappear.67,68 Stopping smoking improves the success rate of dental implants.69 Smoking cessation may be associated with relatively rapid improvement in periodontal health in young adults.73

3.35.5 Medium to long-term health benefits of quitting

Successful cessation appears to stop the progressive increase in the use of health services associated with continued smoking within a few years.74

Specific long-term health benefits include:

- Lung cancer. Quitting is beneficial for lung cancer risk.75 Quitting at age 30 reduces the risk of lung cancer by several times compared to a lifetime smoker. Even quitting at 50 more than halves the risk over the next 25 years compared to continued smoking.76 The absolute annual risk of developing or dying from lung cancer does not decrease, but by stopping smoking the much greater increase in risk that would result from continuing to smoke is avoided.3

- Chronic obstructive pulmonary disease (COPD). Smoking cessation remains the only proven strategy for reducing the disease-causing processes leading to COPD.4 Cessation reduces decline in lung function.77 In smokers without COPD, stopping smoking improves lung function by about 5% within a few months of cessation. The accelerated decline in lung function in smokers stops within five years of smoking cessation, returning to the far slower rates of decline that naturally occur with ageing.1,3 Existing emphysematous damage to lung tissue caused by smoking is permanent1 however cessation slows the progression of COPD.9 Symptoms of COPD will be less likely in the short and long term.1 In people diagnosed with COPD, stopping smoking reduces the rate of lung function decline, and decreases the risk of hospitalisation for COPD.3,56,78
Other cancers. Smoking cessation is the only proven strategy for reducing the disease-causing processes leading to cancer.\textsuperscript{4} The risks of cancers of the mouth, throat, larynx, oesophagus, stomach, bladder, kidneys, pancreas and cervix are reduced after quitting compared to continued smoking, and continue to decrease over time.\textsuperscript{1,3} The risk of pancreatic, oral and cervical cancers quite quickly become similar to people who have never smoked, but the risks for the other cancers remain higher than never-smokers even after 15 to 20 years.\textsuperscript{1,3}

Peripheral vascular disease. Quitting slows down the build-up of plaque on artery walls, so that the risk of the disease is substantially reduced. For those who already have the disease, amputations are less likely.\textsuperscript{1,79}

Blindness. Cataract development and macular degeneration risks and progression are reduced.\textsuperscript{2,53}

Male erectile dysfunction is reduced when smokers quit.\textsuperscript{2,53}

Female fertility. Missed and painful periods are reduced after quitting, as is the risk of delayed conception and early menopause.\textsuperscript{20,80} The higher risk of heart disease and stroke among women smokers who use the contraceptive pill is reduced.\textsuperscript{81}

Overall health and quality of life improve, with some evidence that heavier smokers report greater improvement in quality of life after quitting and report being happier now than when they were smoking.\textsuperscript{1,2,82–88}

3.35.6 Cutting down: are there health benefits?

Cutting down the number of cigarettes smoked each day is a common strategy used by smokers to reduce harm, to move towards quitting, or to save money.\textsuperscript{86–89} However, research shows no noticeable improvement in health outcomes or lifespan among smokers who are able to cut down on a long-term basis.\textsuperscript{4,90–92} This is largely because smokers primarily seek a consistent level of nicotine. Those who cut down therefore tend to smoke the remaining cigarettes harder by taking more and larger puffs, and holding each puff longer. Thus they do not reduce their intake of toxins as much as the reduction in the number of cigarettes suggests.\textsuperscript{87,93}

3.35.7 Other benefits of quitting

There are other benefits of quitting smoking. Financial savings for a pack-a-day smoker are about $5000 per year (2012 prices).\textsuperscript{94} Smokers who quit reduce their likelihood of financial stress and are likely to enhance their material wellbeing.\textsuperscript{95} As more public and private places become smokefree, ex-smokers avoid the inconvenience of having to find somewhere to smoke. Quitting avoids further smoking-related damage to skin, and slows the development of wrinkles.\textsuperscript{96} Life insurance is often cheaper,\textsuperscript{97} the risk of smoking-related fires is reduced\textsuperscript{98,99} and people who quit have fewer sick days.\textsuperscript{2}

3.35.8 Population beliefs about the benefits of quitting

There are limited data on population beliefs of the benefits of smoking cessation. There is a strong belief among smokers that stopping smoking will improve their health.\textsuperscript{100,101} Evidence from Victorian surveys show that smokers mention saving money (57%), feeling healthier (55%) and breathing and fitness (31%) as particular advantages of quitting.\textsuperscript{102} Quitting protects the health of pets and smokers do perceive this as a benefit of quitting.\textsuperscript{103}

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According to a recent review of mortality trends, world life expectancy is currently slightly above 70 years; most deaths earlier than that are avoidable, and are primarily caused by non-communicable diseases. Although communicable diseases, for example influenza and influenza-like illnesses, have a tendency to capture news headlines and possibly the attention of policy-makers more dramatically than many other health issues, an examination of the disease burden of influenza compared with the disease burden of tobacco leaves little doubt about the salience of tobacco as an issue in global public health. Death and disease caused by tobacco use now constitutes a pandemic; its use is the leading cause of preventable death and is estimated to kill more than five million people each year worldwide. This constitutes one death about every six seconds. In 2004, 12% of all deaths worldwide among adults over 30 were attributed to tobacco. In 2010, smoking caused about a quarter of all cancer deaths in Europe and America, and even greater numbers from other diseases. Smoking is also a major cause of death in China, India, and other countries throughout Asia. In several Asian and African countries, more than 25% of male deaths are now related to smoking, matching regions in Europe and North America that previously had the highest proportion of tobacco-related mortality. More than two thirds of tobacco deaths occur in low- and middle-income countries, with the gap in deaths between low and middle income countries and high income countries expected to widen further over the next several decades if effective prevention measures are not implemented. If current trends persist, tobacco will kill more than eight million people worldwide each year by the year 2030, and about 1 billion by the end of this century. By 2030, it is projected overall that there will be approximately 26 million new cancer cases and 17 million cancer deaths per year. This compares with about 12 million new cases and 7.6 million cancer deaths estimated to have occurred globally in 2007. The projected increase will be driven largely by growth and ageing of populations and will be largest in low- and medium-resource countries. Under current trends, increased longevity in developing countries will nearly triple the number of people who survive to age 65 by 2050. This demographic shift will be compounded by entrenched modifiable risk factors such as smoking, which is the leading risk factor for cancer mortality in countries of low, middle and high income. On the basis of current tobacco consumption patterns, it has been estimated that approximately 450 million adults will be killed by smoking between 2000 and 2050. At least half of these adults will die between 30 and 69 years of age, losing decades of productive life. Cancer and the total deaths due to smoking have fallen sharply in men in high income countries but will rise globally unless current smokers, most of whom live in low and middle income countries, stop smoking before or during middle age. Projections of global mortality and burden of disease from 2002 to 2030 have been undertaken by Mathers and Loncar using three scenarios—‘baseline’, ‘optimistic’ and ‘pessimistic’. The projections highlight tobacco-
related mortality and burden of disease as a major threat to public health and allow comparisons with other major threats to public health such as human immunodeficiency virus (HIV) infection or obesity. In these projections, global HIV/AIDS deaths rise from 2.8 million in 2002 to 6.5 million in 2030 under the 'baseline' scenario, which assumes that coverage with antiretroviral drugs reaches 80% by 2012. Under the 'optimistic' scenario, which also assumes increased prevention activity, HIV/AIDS deaths drop to 3.7 million in 2030. By contrast, total tobacco-attributable deaths rise from 5.4 million in 2005 to 6.4 million in 2015 and 8.3 million in 2030 under the 'baseline' scenario. Tobacco is projected to kill 50% more people in 2015 than HIV/AIDS, and to be responsible for 10% of all deaths globally.

Tuberculosis (TB), HIV and chronic obstructive pulmonary disease (COPD) are burgeoning epidemics in developing countries. The link between TB and HIV is well established. Less well recognised is the strong relationship between tobacco smoking and the development and natural history of TB. These associations are of considerable relevance to public health and disease outcomes in individuals with TB. Moreover, tobacco smoking, a modifiable risk factor, is associated with poorer outcomes in HIV-associated opportunistic infections, of which TB is the commonest in developing countries. It is now also becoming clear that TB, like tobacco smoke, besides its known consequences of bronchiectasis and other pulmonary morbidity, is also a significant risk factor for the development of COPD. Almost 90% of COPD deaths occur in low- and middle-income countries, and it has been estimated that COPD will be the third leading cause of death in 2030 globally. Thus, the harmful synergistic interaction between TB, HIV, tobacco smoking and COPD in a large proportion of the world’s population that deserves urgent attention in developing countries.

International research on current smoking prevalence and behaviours among youth aged 13–15 has reported disturbing trends for the future. The Global Youth Tobacco Survey, assessing data from more than 130 countries and principalities, has found that:

- the gap in smoking rates between school-aged girls and boys is decreasing, a finding of particular importance for those countries in which smoking has previously been negligible among the female population,
- use of tobacco products other than cigarettes is widespread,
- a sizeable proportion of children who currently do not smoke are contemplating adopting the behaviour, and
- children are widely exposed to secondhand smoke.

Each of these findings can be expected to have a significant impact on morbidity and mortality from tobacco use in forthcoming decades.

While the current burden of death is distributed evenly between developing and industrialised countries, most of the future burden of death will occur in low and middle income countries, where more than 80% of the world's smokers live. Smoking rates are for the most part well in decline in Western Europe, the UK, the US, Canada, New Zealand and Australia. However in some countries in Asia, South America and Africa, the prevalence of smoking is still increasing. In China, home to one-third of the world's population, the death toll from smoking currently stands at about 800 000 per year and it has been estimated that smoking will cause three million Chinese deaths annually by the middle of this century. Tobacco smoking rates vary; men usually smoke more than women in overall consumption and in prevalence. Current available estimates are 49% for men and 8% for women in low and middle income countries, and 37% for men and 21% for women in high income countries. This is reflected in the proportion of mortality attributable to tobacco, which is higher among men than women. A series of country profiles on non-communicable diseases made available by the World Health Organization points out that since prevalence varies greatly, these country profiles provide a useful way of examining this aspect of the pandemic and are illustrative of the importance of tobacco as a cross-cutting risk factor for non-communicable diseases.

The global tobacco pandemic is characterised by an escalating burden of death and disease that is increasingly being borne by developing countries; efforts to promote global health equity must therefore prioritise reductions in tobacco consumption. The scale of this global tobacco pandemic and the globalisation of tobacco use provide a clear rationale for a global response such as that set out in the
World Health Organization Framework Convention on Tobacco Control (FCTC)\(^3^3\) and the supporting MPOWER package.\(^3^3\) The World Health Organization’s Report on the Global Tobacco Epidemic describes some of the progress achieved as a result of the FCTC, such as 739 million people in 31 countries being afforded protected by comprehensive smoke-free laws;\(^3^4\) but an earlier World Health Organization report also implicitly acknowledges that much more needs to be done because it notes that less than 10% of the world's population is covered by any one of the MPOWER measures by the year 2008.\(^1^4\) Chapman, while acknowledging that an acceleration of policy development in tobacco-control policy has been ushered in by the FCTC, has also noted the optional nature of some aspects of the treaty and thus the particular importance of intensified strategies in harm reduction, demand reduction, denormalisation of tobacco use (especially among health workers in nations where use remains high) as well as further efforts to regulate the tobacco industry (especially plain packaging, under-the-counter retail sales and the regulation of tobacco products).\(^3^5\)

Chapter 1 provides international prevalence comparisons while Chapter 2 gives international comparisons on tobacco consumption. The global nature of the industry is covered in Chapter 10. The WHO Framework Convention on Tobacco Control is described in detail in Chapter 18.

References


