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Introduction

Much of this chapter is based on information in the reports of the US Surgeon General (The Health Consequences of Smoking. Nicotine Addiction, 1988) and of the Tobacco Advisory Group of the Royal College of Physicians (Nicotine Addiction in Britain, 2000), both of which provide extensive, fully referenced reviews of the chemical, physiological and psychoactive properties of nicotine. Readers requiring more detailed information are referred to these publications.

Nicotine is the drug in the tobacco plant that causes tobacco users to become addicted to cigarettes and other tobacco products. An essence of tobacco was first extracted in the early 1800s and named ‘Nicotianine’ to commemorate Jean Nicot, the French diplomat and scholar who introduced tobacco to the French court in the late 1500s. German scientists in Heidelberg isolated the pure form of Nicotianine in 1828, calling it ‘Nikotin,’ but it took another century before its addictive nature was first suspected. Research during the 1920s and 1930s linked nicotine with the dependency observed among tobacco users. In 1942, Johnston showed that injections of pure nicotine reduced his need to smoke tobacco and concluded: ‘Smoking tobacco is essentially a means of administering nicotine, just as smoking opium is a means of administering morphine.’

The publication of major reports on smoking and health in the 1960s confirmed the role of nicotine in perpetuating tobacco use. However the prevailing view at the time was that tobacco smoking was best understood as a socially learned behaviour, and that it reflected a psychological ‘habituation’ rather than physical addiction. Treatment options for smoking cessation primarily used psychological models of behaviour modification. This was to change over the next 20 to 30 years, as awareness increased of the physiological role of nicotine in tobacco addiction.

In a report of the US Surgeon General in 1979, persistent tobacco use was described as ‘the prototypical substance-abuse dependency.’ In 1980, the American Psychiatric Association for the first time included tobacco use in its Diagnostic and Statistical Manual of Mental Disorders as a substance abuse disorder, signalling a greater emphasis on the psychoactive effects of nicotine, and a move towards viewing tobacco addiction through a disease model. The US National Institute on Drug Abuse took a similar stance during the 1980s.

The report of the US Surgeon General in 1988 focused solely on nicotine and concluded that (p9):

1. Cigarettes and other forms of tobacco are addicting.
2. Nicotine is the drug in tobacco that causes addiction.
3. The pharmacologic and behavioural processes that determine tobacco addiction are similar to those that determine addiction to drugs such as heroin or cocaine.

The US Surgeon General’s report of 2000 argued: ‘Tobacco dependence is in fact best viewed as a chronic disease with remission and relapse.’ The Royal College of Physicians’ report in the same year observed: ‘Doctors, other health professionals and indeed society as a whole, need to acknowledge nicotine addiction as a major medical and social problem’ (pxvi).

Cigarettes are now commonly understood to be a highly efficient nicotine delivery system that both causes and sustains addiction. Nicotine is a drug that ‘captures’ more of its users than do ‘hard’ drugs such as heroin and cocaine. The tobacco industry has long recognised addiction to nicotine as the primary motivation for persistent smoking, and has worked since the 1960s to make cigarettes as efficient as possible in delivering doses of nicotine to the brain’s receptors. These aspects of product development are discussed further in Chapter 12.
References


Defining nicotine as a drug of addiction

Note: the terms 'addiction' and 'dependence' are used interchangeably in this and the following sections to refer to a situation in which a drug or stimulus has unreasonably come to control behaviour.¹

The most widely used criteria for assessing drug dependence² are those from the International Classification of Diseases (ICD) of the World Health Organization, and the Diagnostic and Statistical Manual of Mental Disorders (DSM), compiled by the American Psychiatric Association. Both of these classification schemes undergo major periodical revision, the most recent versions being ICD-10 (1990),² and DSM-IV (1994).³ The main features from the DSM-IV and ICD-10 are summarised in Table 6.1.1, and include:

- a strong desire to take the drug
- taking the substance in larger amounts or for longer than intended
- difficulty in controlling use
- spending a great deal of time in obtaining, using or recovering from the effects of the substance
- giving a higher priority to drug use than to other activities and obligations
- continued use despite harmful consequences
- tolerance
- withdrawal symptoms.

The criteria in these systems apply to legal and illicit substances. Because tobacco products are legal to use, easily obtained, comparatively inexpensive, and may be readily used while engaged in other activities, some of the criteria listed above are less applicable to tobacco than other drugs of addiction. For instance, the average smoker does not need to devote a great deal of time to obtaining cigarettes, and only in countries where smoking restrictions have been introduced do smokers need to forego important activities to smoke a cigarette. Otherwise, nicotine use easily meets the criteria listed in Table 6.1.1; nicotine is, in fact, among the most addictive of substances known.¹⁴ According to the Royal College of Physicians, 'although nicotine in the form of tobacco is a legal drug, it should not be regarded as pharmacologically benign. The classification of drugs as "legal", "soft" or "hard" reflects public perceptions and current law enforcement practice rather than their pharmacological classification. In terms of addictiveness, nicotine delivered in tobacco smoke is a "hard" drug on a par with heroin and cocaine' (p184).¹

Research on the epidemiology of drug dependence has shown that of all people who initiate tobacco use, almost one-third (32%) become addicted smokers. This is a much higher addiction capture rate than for users of heroin (23%), cocaine (17%), alcohol (15%) or cannabis (9%).¹⁴ Tobacco's status as a legal, and until recently, a socially acceptable product, with a long history of high-profile marketing and promotion, has contributed to much higher levels of tobacco than illicit drug dependence in the community.
References


6.2

The physiological effects of nicotine

More than 4000 compounds are found in tobacco smoke. Many of them are pharmacologically active and have toxic, mutagenic or carcinogenic effects.\(^1\) Nicotine appears to be the most important pharmacological agent for sustaining smoking.\(^1,2\) Its chemical name is (S)-3-(1-methylpyrrolidin-2-yl)pyridine, and its formula is C10H14N2.\(^3\)

In large quantities, nicotine is extremely toxic and a potentially lethal nerve poison. A dosage of 40–60 mg is sufficient to kill an adult human, but acute nicotine poisoning is rare. Poisoning most often occurs through accidental swallowing or skin contact with pesticides containing nicotine. Workers who harvest tobacco leaves can experience a low level of intoxication from handling the leaf, known as ‘green tobacco sickness’,\(^4\) which diminishes with increased exposure.\(^3\) Nicotine poisoning in children can occur by ingestion of cigarettes, cigarette butts, or nicotine gum or other replacement products (see also Chapter 3, Section 3.20).

Tobacco smoke delivers nicotine in much lower quantities to smokers because much of it is destroyed by burning the leaf. Even in low doses, nicotine is a potent chemical that causes a range of physiological changes and can create dependence, which is reinforced by the unpleasant withdrawal symptoms when smokers abruptly stop.\(^2\)

The uptake of nicotine into the body is dependent on the pH levels of its transport system (generally smoke but also saliva when sucking, chewing and inhaling tobaccos). Cigarette smoke is the most rapid and effective way of delivering nicotine to the brain.\(^2\) Nicotine in the acidic tobacco smoke is drawn into the lungs, where it is swiftly absorbed because of the lungs’ large surface area and rapid transfer across membranes. A large dose of nicotine is delivered via the bloodstream to the brain.\(^6\)

Smoke from pipes and cigars is more alkaline, allowing for much slower uptake of nicotine through the lining of the mouth.\(^6\) Smokeless tobacco products such as snus, and nicotine chewing gum (an aid in cessation), are manufactured to facilitate oral absorption, but nicotine chewing gum delivers nicotine at lower doses and/or slower rates than more addictive forms of tobacco.\(^6\)

New smokers may experience nausea and dizziness from nicotine, but they quickly develop tolerance to these effects with continued use.\(^1\) Once smokers become accustomed to functioning with a specific level of nicotine in their blood, they maintain this level by continued self-administration of the drug. The effects of nicotine diminish with repeated use, often leading to increased smoking\(^1\) or to smokers varying their puff frequency and depth to obtain the desired amount of nicotine.\(^6\)

In regular smokers, nicotine levels rise quickly after smoking a cigarette. They then fall slowly until they reach a level that increases the urge to smoke another cigarette. Nicotine has a half life of 6–8 hours so that it gradually accumulates in blood over the course of the day. Nicotine levels decrease slowly during sleep.\(^1\) One of the simplest and most commonly used measures of an individual’s nicotine addiction is how soon after waking he or she has their first cigarette.\(^1\)

Nicotine reaches the brain in 10–19 seconds after entering the bloodstream.\(^6\) There, it acts on specialised cell receptors located in the brain and other organs and muscles to produce a wide range of physical reactions. Heart rate and blood pressure increase. Blood flow decreases in the skin, producing a subjective drop in temperature, while increasing in skeletal muscle. The coronary arteries vasoconstrict, or narrow.\(^6\) Additionally, brain waves are altered, a number of endocrine changes occur, and skeletal muscle relaxes.\(^3\) Nicotine also increases the metabolic rate and suppresses appetite, with the result that smokers weigh an average of 4 kg less than non-smokers.\(^6\)

The primary sites for metabolism of nicotine are the liver and, to a much lesser extent, the lung and brain.\(^6\) The main metabolites of nicotine are cotinine and nicotine-N-oxide. Cotinine has a much longer half life than nicotine (up to 20 hours). Cotinine is further metabolised into trans-3'-hydroxycotinine, the main nicotine metabolite that is found in urine. Up to one-third of nicotine by-products are eventually excreted in the urine.\(^6\)

Nicotine is known to interact with a range of drugs used in medicine. These drugs may be processed by the body in a different way when nicotine is present, and nicotine may affect the way that some drugs act. These drugs include oral contraceptives; sedatives; analgesics; and drugs that treat a number of different conditions, including aspects of heart disease, mental health problems and breathing difficulties. These interactions can produce clinically significant outcomes, making it vital for health professionals to ask whether or not a patient smokes tobacco.\(^6,9\)
The substance nicotine is not in itself strongly associated with the chronic diseases caused by smoking. These effects primarily arise from prolonged exposure to the thousands of noxious and carcinogenic substances in tobacco smoke and tar. The Royal College of Physicians has concluded that ‘pure nicotine may be harmful to the fetus in pregnancy but is likely to be far less hazardous than the effects of smoking’. This has implications for the use of nicotine replacement therapy for cessation during pregnancy (see Chapter 7, Section 7.11.5).
References


Psychoactive effects of nicotine

In addition to the physical effects outlined in the previous section, nicotine also has acute effects on a smoker's mental state.

Tobacco provides the average daily smoker with an efficient, convenient and socially acceptable way of self-administering a potent psychoactive drug more than 100 times a day (about 140 puffs per day for the 14-a-day smoker).

Nicotine has biphasic brain effects, that is, it can improve alertness by stimulating brain function and also produce a feeling of relaxation by depressing functioning. The mental and physical state of the smoker, and the situation in which smoking occurs, influence the way in which a cigarette affects psychological states and physiological responses.

In order to provide a strong psychoactive reaction, nicotine must be delivered rapidly to the brain. The inhalation of tobacco smoke into the lungs is a highly optimised method of rapidly delivering nicotine via the bloodstream to the brain, where it acts upon neuronal nicotinic receptors.

Nicotine, like all addictive drugs, causes a complex range of biochemical changes that create dependence, which is reinforced by the aversive effects of withdrawal.

The neurobiology of the positive reinforcing properties of nicotine

The pleasant and rewarding effects of nicotine reinforce smoking and other forms of tobacco use. The neurobiology of nicotine's rewarding effects is only partially understood. The same is true of the biological basis of the abstinence syndrome that occurs after abrupt cessation of smoking; avoiding the abstinence syndrome encourages smokers to continue smoking.

Like many other addictive drugs, nicotine activates release of the neurotransmitter dopamine in the brain's 'reward system' (the mesocorticolimbic dopamine system). Repeated nicotine exposure stimulates dopamine release in the nucleus accumbens, a part of the brain that is believed to play an important role in all forms of addiction. This region of the brain is also involved in the regulation of emotions and the processing of rewards such as food and sex. It also has a role in the actions of other drugs of abuse, such as amphetamines and cocaine.

Nicotine exerts its effects by activating specific sites called receptor proteins. These in turn trigger the release of dopamine in the nucleus accumbens and the secretion of other nerve-stimulating chemicals such as acetylcholine and glutamate in the hippocampus and cerebral cortex. Their effects improve vigilance, attention and cognition, benefits that smokers often cite as reasons for continuing to smoke. Low doses of nicotine may improve memory, information processing and attentiveness, although the benefits are small and similar to the effects of drinking coffee or other caffeinated drinks.

Repeated administration of nicotine greatly increases the release of dopamine in a specific region of the nucleus accumbens called the accumbal core. The enhanced dopamine release is central to Pavlovian or classically conditioned learning, which associates the effects of nicotine with cues present in the environment and in tobacco smoke inhaled by the smoker. The linking of these responses to such cues is strongly linked to the transition to addiction, whereby addicted persons find it difficult to control their cravings for the drug.

According to smokers, nicotine use helps them when they are depressed, stressed, embarrassed, bored, irritable or in a bad mood. Alleviating any of these unpleasant feelings by smoking a cigarette reinforces the psychological aspects of tobacco addiction. There is no intrinsic reason why tobacco use should serve this purpose, other than that it helps to avert the physiological and psychological discomfort of withdrawal. For more information on tobacco withdrawal, see Section 6.9.
References


6.4 The role of genetic factors in addiction

Many factors contribute to the initiation of smoking, as discussed in Chapter 5. Experimentation with tobacco and the progression to regular smoking are influenced by environmental factors, but the risk of addiction may also be significantly influenced by physiological factors that have an underlying genetic basis. \(^1-^4\) Twin studies have reported that the heritability for smoking (i.e. the proportion of the variance in smoking that is attributable to genetic factors) ranges from 50–80%. This is similar to heritability estimates for alcoholism, asthma and hypertension. \(^1,^3\)

Genetic factors that influence initiation and dependence are likely to overlap but there is some evidence that different genetic factors may act in each stage of tobacco use. \(^6\)

So far, large-scale genetic studies have identified a limited number of the genes underlying addiction risk. \(^7\) It is most likely that multiple genes underlie this heritability, each having only a small effect on addiction risk. \(^3,^8\)

Studies of specific ‘candidate’ genes have produced inconsistent findings, probably because of technical difficulties in analysing a complex behaviour such as smoking in which genes and environment both play a role. \(^2,^5\) Plausible ways in which genetic differences might influence susceptibility to addiction include variations in the number and sensitivity of nicotine receptors, the speed and efficiency with which the body metabolises nicotine, and the physiological and behavioural responses to nicotine. \(^1,^3,^8\) For example, inherited variation within the human CYP2A6 gene, which influences the rate at which the body metabolises nicotine, \(^10\) may in turn affect an individual’s level of tobacco consumption. \(^11\)

Research into the genetics of nicotine addiction may eventually produce more targeted and effective pharmacological aids to assist with cessation but the role of the environment in long-term smoking should not be underestimated, given that up to 50% of the risk of addiction to smoking is due to environmental factors. \(^12\)

According to Hall and colleagues, ‘Improved understanding of the genetics of smoking are not likely to affect public health tobacco control policies. It is much simpler, cheaper and more efficient to discourage the whole population from smoking tobacco than it is to attempt to make smoking safer by identifying those at highest risk of nicotine addiction or smoking-related disease’ (p123). \(^2\)

Further issues relating to genetic predisposition to tobacco-caused disease are discussed in Chapter 3, Section 3.3. The possible contribution of genetic research to advances in smoking cessation is discussed in Chapter 7, Section 7.7.
References


8. Hall WD. A research agenda for assessing the potential contribution of genomic medicine to tobacco control. Tobacco Control 2007;16(1):53–8. Available from: http://tobaccocontrol.bmj.com/cgi/content/abstract/16/1/53


Measures of tobacco dependence

A smoker's degree of tobacco dependence may be assessed by a range of measures. These include the frequency and quantity of tobacco consumed, biochemical markers (such as levels of cotinine, a by-product of nicotine metabolism, in the saliva), and questionnaire measures of self-reported smoking behaviour.

Tests of psychological and physiological dependence, in the form of a questionnaire for smokers, include the Fagerström Test for Nicotine Dependence (which evolved from the earlier Fagerström Tolerance Questionnaire), the Cigarette Dependence Scales, the Nicotine Dependence Syndrome Scale, the Wisconsin Inventory of Smoking Dependence Motives, and instruments that assess whether a smoker satisfies the criteria for drug dependence in the Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association, described in Section 6.1.

The most commonly used measure is the Fagerström Test for Nicotine Dependence. This instrument asks the smoker a set of questions, the answers to which are scored as shown in Table 6.5.1 and added to give a total score: a score of six or more is seen as an indicator of high dependence.

Another, newer measure of levels of addiction is the ‘Hooked on Nicotine Checklist,’ or HONC, which assesses when the individual loses their control, or autonomy, over their use of tobacco use. Loss of autonomy is the point at which ‘the sequela of tobacco use, either physical or psychological, present a barrier to quitting’ (p399). The HONC was originally devised for use with younger smokers for whom some of the questions in the Fagerström Test were much less relevant. For example, most adolescents would not be able to smoke within minutes of waking in the morning, or if ill in bed, unless they were doing so with parental permission. Subsequent research suggests that the HONC is also useful for measuring loss of autonomy over smoking in adults.

The HONC asks 10 questions to assess degree of tobacco dependence. The HONC asks 10 questions to assess degree of tobacco dependence.

1. Have you ever tried to quit, but couldn’t?
2. Do you smoke now because it is really hard to quit?
3. Have you ever felt like you were addicted to tobacco?
4. Do you ever have strong cravings to smoke?
5. Have you ever felt like you really needed a cigarette?
6. Is it hard to keep from smoking in places where you are not supposed to, like school?
7. Did you find it hard to concentrate because you couldn’t smoke?
8. Did you feel more irritable because you couldn’t smoke?
9. Did you feel a strong need or urge to smoke?
10. Did you feel nervous, restless, or anxious because you couldn’t smoke?

Research on the HONC in young people has found that dependence may develop rapidly, even at low, sporadic levels of consumption. Some of these findings are discussed in Section 6.7.

Table 6.5.1
The Fagerström Test for Nicotine Dependence

<table>
<thead>
<tr>
<th>Question</th>
<th>Answer</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>How soon after you wake up do you smoke your first cigarette?</td>
<td>Within 5 minutes</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>6–30 minutes</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>31–60 minutes</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>After 60 minutes</td>
<td>0</td>
</tr>
<tr>
<td>Do you find it difficult to refrain from smoking in places where it is forbidden?</td>
<td>Yes</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>Which cigarette would you hate to give up most?</td>
<td>The first one in the morning</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>All others</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>10 or less</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td>11–20</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>21–30</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>31 or more</td>
<td>3</td>
</tr>
<tr>
<td>How many cigarettes per day do you smoke?</td>
<td>Yes</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>Do you smoke more frequently during the first hours after waking than during the rest of the day?</td>
<td>Yes</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>Do you smoke if you are so ill that you are in bed most of the day?</td>
<td>Yes</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>No</td>
<td>0</td>
</tr>
</tbody>
</table>

Source: Heatherton et al. 3
Section 6: Addiction

References


The association between addiction and socio-economic status

In developed countries with well-developed tobacco control policies, smoking prevalence is \(1,2\) and has for many years been \(3–7\) higher among those with lower socio-economic status (SES). International research strongly suggests that levels of tobacco addiction are also greater among lower SES smokers.\(^8,9\) Data from the 2010 National Drug Strategy Household Survey\(^2\) indicates substantially higher numbers of reported cigarettes smoked each day among those with more limited educational qualifications; the unemployed compared to those in the labour force; the divorced, separated or widowed compared to those married; those of Aboriginal or Torres Strait Islander backgrounds; those living in regional compared to cities; and those living in more socially disadvantaged areas: refer to Table 6.6.1.

Comparisons of nicotine dependence in smokers in Australia, the UK, Canada and the US\(^9\) showed that in all countries, smokers of lower socio-economic status had higher levels of nicotine dependence and were less likely to make a quit attempt.\(^10\)

These findings are consistent with evidence that social disadvantage is linked with higher levels of stress, relief of which is commonly given as a reason for smoking (see Chapter 9). Smokers with lower levels of education are also more likely to be less confident about their capacity to quit (lower levels of self-efficacy) than more highly educated smokers; they are also less likely to report an intention to quit.\(^9\) These findings have important implications for cessation programs, which are discussed in Chapters 7 and 9.

### Table 6.6.1

Mean number of cigarettes smoked per week — by social characteristics, by sex, 2010, current smokers aged 14 years or older, (number)

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
<th>Persons</th>
</tr>
</thead>
<tbody>
<tr>
<td>All persons</td>
<td>108.6</td>
<td>96.9</td>
<td>103.2</td>
</tr>
<tr>
<td>Education</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>With post-school qualifications</td>
<td>99.6</td>
<td>88.4</td>
<td>95.0</td>
</tr>
<tr>
<td>Without post-school qualifications</td>
<td>120.3</td>
<td>104.4</td>
<td>112.2</td>
</tr>
<tr>
<td>Labour force status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Currently employed</td>
<td>102.4</td>
<td>88.5</td>
<td>96.9</td>
</tr>
<tr>
<td>Student</td>
<td>50.5</td>
<td>56.1</td>
<td>53.3</td>
</tr>
<tr>
<td>Unemployed</td>
<td>140.0</td>
<td>104.6</td>
<td>125.3</td>
</tr>
<tr>
<td>Engaged in home duties</td>
<td>131.1</td>
<td>99.9</td>
<td>101.5</td>
</tr>
<tr>
<td>Retired or on a pension</td>
<td>121.0</td>
<td>121.7</td>
<td>121.3</td>
</tr>
<tr>
<td>Unable to work</td>
<td>167.8</td>
<td>125.5</td>
<td>145.8</td>
</tr>
<tr>
<td>Other</td>
<td>142.7</td>
<td>121.2</td>
<td>128.3</td>
</tr>
<tr>
<td>Main language spoken at home</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>English</td>
<td>112.4</td>
<td>96.9</td>
<td>105.1</td>
</tr>
<tr>
<td>Other</td>
<td>65.1</td>
<td>62.3</td>
<td>64.5</td>
</tr>
<tr>
<td>Socio-economic status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1st quintile (lowest status)</td>
<td>132.3</td>
<td>113.5</td>
<td>123.4</td>
</tr>
<tr>
<td>2nd quintile</td>
<td>113.0</td>
<td>107.0</td>
<td>110.4</td>
</tr>
<tr>
<td>3rd quintile</td>
<td>109.5</td>
<td>87.9</td>
<td>99.6</td>
</tr>
<tr>
<td>4th quintile</td>
<td>91.4</td>
<td>91.8</td>
<td>91.5</td>
</tr>
<tr>
<td>5th quintile (highest status)</td>
<td>74.0</td>
<td>66.1</td>
<td>70.3</td>
</tr>
<tr>
<td>Geography</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Major cities</td>
<td>97.0</td>
<td>91.2</td>
<td>94.5</td>
</tr>
<tr>
<td>Inner regional</td>
<td>126.5</td>
<td>100.8</td>
<td>113.5</td>
</tr>
<tr>
<td>Outer regional</td>
<td>124.6</td>
<td>116.6</td>
<td>120.9</td>
</tr>
<tr>
<td>Remote and very remote</td>
<td>161.3</td>
<td>111.8</td>
<td>140.7</td>
</tr>
<tr>
<td>Marital status</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never married</td>
<td>104.0</td>
<td>85.1</td>
<td>95.9</td>
</tr>
<tr>
<td>Divorced/separated/widowed</td>
<td>138.7</td>
<td>119.8</td>
<td>127.6</td>
</tr>
<tr>
<td>Married/de facto</td>
<td>104.3</td>
<td>92.9</td>
<td>99.2</td>
</tr>
<tr>
<td>Household composition</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single with dependent children</td>
<td>136.9</td>
<td>105.7</td>
<td>110.8</td>
</tr>
<tr>
<td>Couple with dependent children</td>
<td>99.2</td>
<td>90.7</td>
<td>95.4</td>
</tr>
<tr>
<td>Parent with non-dependent children</td>
<td>119.3</td>
<td>120.6</td>
<td>119.9</td>
</tr>
<tr>
<td>Single without children</td>
<td>114.6</td>
<td>104.0</td>
<td>110.5</td>
</tr>
<tr>
<td>Couple without children</td>
<td>107.9</td>
<td>94.5</td>
<td>102.2</td>
</tr>
<tr>
<td>Other</td>
<td>100.7</td>
<td>77.8</td>
<td>90.7</td>
</tr>
</tbody>
</table>

Note: Base is current (daily, weekly and less than weekly) smokers
Source: 2010 National Drug Strategy Household Survey\(^2\)
References


6.7 Addiction and the adolescent smoker

Most smokers begin smoking during their teens. A person who smokes cigarettes in adolescence is more likely to progress to daily smoking and become addicted than someone who experiments with cigarettes in adulthood. About three-quarters of teenagers who smoke regularly continue to smoke as adults. Those who start smoking as teenagers smoke for longer and more heavily, on average, than those who start smoking at a later age. These smoking patterns increase the risk of developing tobacco-related disease later in life.

Signs of tobacco addiction may occur more rapidly in adolescent smokers at lower or more intermittent levels of consumption. US research using the Hooked on Nicotine Checklist (‘HONC’—see Section 6.5) has shown that adolescents lose autonomy over their smoking on average within two months of the onset of smoking. The median amount of smoking at which symptoms of dependency were reported was two cigarettes per day, smoked on one day per week. Those adolescent smokers who smoked only occasionally were less likely to develop any HONC symptoms and were more easily able to quit smoking. The frequency of smoking increased after one or more symptoms of dependency were reported. Although smokers may have recognised the emergence of dependence symptoms and attempted to quit, they found quitting more difficult than those who did not smoke regularly.

In US research using the HONC, 40% of young smokers aged 12–13 (followed up over 30 months) reported symptoms of dependence. In those who reported inhaling tobacco smoke, 58% reported symptoms of dependence. In those who reported one or more symptoms of dependence, 18% did so soon after their first use, 33% did so when smoking once monthly, 49% when smoking weekly, and 70% did so before they started smoking daily. Girls were more likely to report symptoms of dependence than boys, and experienced them sooner after starting smoking (21 days for girls compared to 183 days for boys). Adolescents who experienced nausea, dizziness or relaxation when they initially smoked a cigarette were much more likely to develop a HONC symptom than those who did not. Other US research shows that younger smokers (aged 10–18) are twice as likely to report that ‘it’s really hard to quit’ than older smokers (aged 19–22) who smoke the same amount.

One hypothesis to explain the younger smokers’ increased susceptibility to tobacco addiction is the greater immaturity of the adolescent brain, which allows nicotine to have more disruptive effects on brain function. There is a significant gap between the reality of adolescent addiction to nicotine and young smokers’ beliefs about their ability to control their use of the substance. This is discussed in Section 6.11.
References


6.8 Tobacco ‘chippers’

A ‘chipper’ is someone who uses a drug with a high dependence potential infrequently and intermittently, and does not experience withdrawal or other symptoms of dependence. The term was first used in relation to infrequent opiate users in the 1970s, and later applied to smokers by Shiffman in 1989.

Tobacco chippers smoke small numbers of cigarettes (1–5) regularly but differ from addicted smokers in maintaining these low levels of consumption and can abstain for considerable periods with little difficulty. Only a very small proportion of smokers—perhaps about 5%—are thought to fit into this category. Chippers inhale tobacco smoke in the same way as regular smokers, and their bodies metabolise nicotine in similar ways but, unlike most smokers, chippers do not report withdrawal symptoms if they do without tobacco. Chippers start smoking later, with about 80% starting after leaving high school.

Chippers appear to be protected from becoming addicted smokers by a combination of environmental, social and genetic factors. Chippers are less likely to smoke to reduce stress or improve mood, have fewer relatives who are smokers, and have higher levels of education than heavy smokers. Chippers tend to smoke in situations associated with relaxation, eating and drinking, but, like heavy smokers, chippers smoke almost half of their cigarettes when alone. This makes it unlikely that chippers are simply ‘social smokers’. Chippers’ smoking behaviour is strongly correlated with situational stimuli, whereas in heavy smokers smoking is less influenced by the situation and more by dependence. Shiffman and colleagues observe that chippers continue to smoke in ways that regular smokers do early in their smoking careers. The difference is that chippers do not progress to addictive smoking patterns, like the vast majority of smokers do.

One possible explanation of these findings is that individuals who are able to use tobacco products in this way may be less sensitive to the effects of nicotine. For example, research has suggested that chippers’ initial responses to the effects of nicotine, including pleasurable or unpleasant sensations and dizziness, may be much less than that experienced by individuals who become tobacco dependent.
References


Nicotine withdrawal syndrome

As is true of other addictive drugs, the abrupt cessation of tobacco use produces a withdrawal syndrome with a spectrum of symptoms in most people. The severity of symptoms decreases over time, as the body adapts to the absence of nicotine and returns to its normal state.\(^1\)

The major symptoms of the nicotine withdrawal syndrome include mood changes, including irritability, aggression, anxiety and depression; restlessness; poor concentration; increased appetite; urges to smoke; disturbed sleep; decreased heart rate; and decreased levels of adrenaline and cortisol.\(^1\) Apart from slowing of the heart rate and increased appetite, these symptoms are similar to withdrawal syndrome from other stimulant drugs.\(^2\) It is unclear whether increased anxiety occurs as a physical response to nicotine withdrawal or as a psychological response to the quit attempt, especially if the individual is finding quitting difficult.\(^3\) Most symptoms abate by four weeks’ abstinence from smoking, with the exception of increased appetite and decreased heart rate, which may persist for longer than 10 weeks.\(^4\) The urge to smoke, especially when under stress, may persist for much longer.\(^5\) Most symptoms are reduced by using nicotine replacement therapy, the exceptions being night-time awakening and decreased adrenaline and cortisol levels, about which more research is needed.\(^3\)

Newborn infants of mothers who smoke heavily during pregnancy may experience withdrawal symptoms following birth, measured by neurological assessment after birth, and markers of nicotine exposure.\(^6\)

The unpleasant nicotine abstinence syndrome experienced by many smokers is mediated by neuroadaptive changes in dopaminergic neurons that act on the nucleus accumbens, a part of the brain which plays an important role in addiction (described in Section 6.3.1). The behavioural changes (which are similar to those experienced by many patients with depression), such as a diminished ability to respond to pleasurable stimuli, are thought to arise from reduced dopamine release in the shell of the nucleus accumbens.\(^3\)

Ways of ameliorating tobacco withdrawal are discussed in Chapter 7.
References


Nicotine and other drug use

Tobacco use commonly precedes the use of illicit drugs. This does not necessarily mean that tobacco use causes other drug use. Familiarity with smoking as a technique may facilitate experimentation with and ultimate addiction to other smoked substances, such as cannabis (the most commonly used illicit drug), heroin, cocaine, methamphetamine and phencyclidine. The clustering of smoking and other types of drug use may also reflect shared biological, social, educational and other environmental risk factors.

Australian surveys of tobacco and alcohol use among secondary school students in 2005 showed that first experience with alcohol occurs at an earlier age than tobacco. By the ages of 12 and 13, more than 20% of school children reported consuming alcohol in the past month but fewer than 5% of 12 and 13-year old reported smoking cigarettes. Across all age groups, alcohol is by far the most widely used illicit drug among school children of both sexes, followed by tobacco.

The prevalence of smoking among those who had used various illicit substances was not reported for the 2008 survey, but in 2005 of those who had used any one of the following illicit substances (cannabis, amphetamines, hallucinogens and ecstasy) 40–50% reported having smoked tobacco at the same time.

In the 2004 National Drug Strategy Household Survey tobacco smokers aged 14 and over were more likely to use all other drugs. The majority of both smokers and non-smokers drank alcohol (92% vs. 81%). One-third of tobacco smokers reported using cannabis in the past 12 months, compared to only 7% of non-tobacco users. Nineteen percent of tobacco smokers had used an illicit drug other than cannabis in the preceding 12 months, compared to only 6% of non-smokers. (Data on concurrent use of drugs has not been included in reports on the 2007 or 2010 surveys, but figures extracted from the data file on concurrent use among those 18 plus are provided in Chapter 1, Section 1.10.7)

Tobacco is often mixed with cannabis to make cannabis easier to smoke. With declining rates of cigarette smoking, it is now more common for cannabis dependence to lead to tobacco dependence than vice versa. The practice of combining cannabis and tobacco has been identified as a factor for co-existing addictions in Australian Aboriginal and Torres Strait Islander communities—see also Chapter 8, Section 8.11.
References


6.11 Smokers’ attitudes to and beliefs about addiction

Some smokers may use their ‘addiction’ as an excuse for continuing to smoke but others may be reluctant to acknowledge their addiction for fear of expressing weak self-control and self-determination. Smokers may be ambivalent about their addiction: they may describe feeling entrapped by their smoking, while insisting that whether or not to smoke is a free choice. In an Australian study, only a quarter of smokers agreed that they could quit anytime they want to, however 80% agreed that smokers who really want to quit will “just do it” and only 34% agreed that they were “too addicted to be able to quit” suggesting that most saw continuing to smoke as a choice. Smokers who believe that the health risks of smoking do not apply to them are less likely to make quit attempts.

Smokers and non-smokers both strongly agree that tobacco use is addictive, but smokers are more likely to deny that they themselves are addicted, and to believe that they could quit, if they decided to try. This is particularly marked in younger smokers. In US research, 71% of adolescent smokers and 81% of adult smokers agreed with the statement: ‘Most people who smoke for a few years become addicted and can’t stop’. But significant proportions of the same smokers (60% of adolescents and 48% of adults) also believed: ‘I could smoke for a few years and then quit if I wanted to’. As smoking has become more stigmatised, young people who smoke in social situations may see themselves as ‘not real smokers’ and therefore not at risk of addiction. These misconceptions are particularly dangerous in the light of evidence that symptoms of nicotine dependence may develop after even sporadic smoking in adolescence—see Section 6.7.

The propensity among young smokers to believe that they are in control of their smoking has also been found in British adolescents. A study of the attitudes and beliefs of teenage smokers aged 16–19 found that only 20% believed themselves to be addicted. A further 20% believed that they were not addicted, and the remainder categorised their smoking as a habit or a social behaviour over which they could exercise some degree of control. This finding is supported by another British study in which children perceived adult smoking as a way of coping with everyday life, while maintaining that children who smoked did so for purely social reasons—to fit in with a social group, or to convey an image. Children seem more likely to classify adult smoking as addictive while regarding smoking in their own age group as discretionary and within the smoker’s control.

Younger children are more fearful than adolescents about addiction, but also have misconceptions about how quickly one can become addicted. In a study of Western Australian children aged 9–10, those who believed that they would become instantly addicted to smoking were less likely to intend to smoke than those who thought they would be able to smoke several cigarettes or smoke over a period of time before becoming addicted. Those who defined addiction as ‘liking’ or ‘enjoying the taste of cigarettes’ thought that as long as they didn’t actually enjoy smoking, they would not become addicted. Fear of addiction may be a more salient message in preventing uptake in young people than disease risk, which is a far more distant threat.

Whatever their perception of addiction, once addicted, most young and adult smokers regret that they ever started smoking and wish to give up. Cessation is discussed in the following chapter.
References


