Evaluation

Supporting research evidence

There is indirect support for the role of neurochemistry in nicotine addiction from research with humans. For example, Joseph McEvoy *et al.* (1995) studied smoking behaviour in patients with **schizophrenia**. *Haloperidol* is a dopamine **antagonist** which blocks dopamine receptors in the brain (and is used as drug treatment for **schizophrenia** for this reason). *Haloperidol* treatment increased smoking in this sample of participants. It appears that this was a form of self-medication, an attempt to achieve the nicotine 'hit' by increasing dopamine release.

There is also more direct evidence for the importance of the dopamine reward system in the mesolimbic pathway from brain imaging studies (Ray *et al.* 2008).

Real-life applications

A greater understanding of neurochemistry can lead to development of new treatments for nicotine addiction, such as nicotine replacement therapy (NRT) in the form of patches and inhalers (see page 368). Research is even raising the possibility of nicotine immunisation.

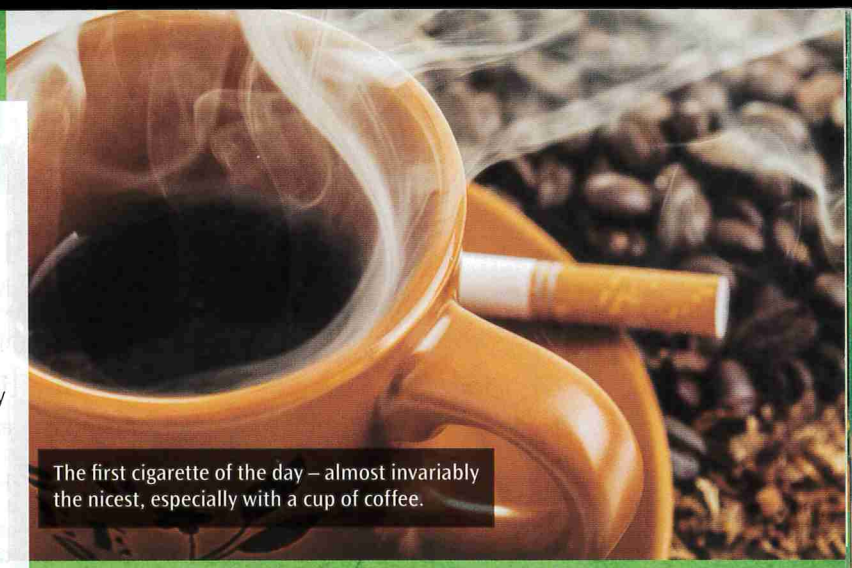
But the potential practical benefits of greater understanding go beyond nicotine addiction. This is because some diseases have high **co-morbidity** with nicotine use; that is they occur together frequently. Examples include schizophrenia, **depression** and alcoholism, all of which are strongly associated with continued smoking.

So further research which offers more effective treatments for nicotine addiction also holds out the prospect of greater advances in treatments for these co-morbid disorders.

Limited explanation

Any explanation of nicotine addiction which considers only the role of dopamine is limited because research increasingly shows that there are many other neurochemical mechanisms involved. The current research picture is one of a vastly complex interaction of several neurochemical systems, including neurotransmitter pathways such as **GABA** and **serotonin** (5-HT), plus other systems such as endogenous opioids (endorphins, the brain's natural painkillers).

However, as Fernando Berrendero *et al.* (2010) point out, the dopamine system is nevertheless central to the neurochemistry of nicotine addiction and these other systems all interact with it to have their effects.



The first cigarette of the day – almost invariably the nicest, especially with a cup of coffee.

Evaluation eXtra

A reductionist explanation

Neurochemical explanations are **reductionist** accounts of why people become addicted to nicotine. They explain addiction at the most fundamental level of the activity of neurotransmitter molecules, rather than at 'higher' levels (e.g. social and psychological influences). At most, only 50% of people who experiment with cigarette smoking become dependent on nicotine. Won Choi *et al.* (2003) found that adolescents who were most likely to become dependent were not committed to abstaining, had friends who smoked, and perceived themselves to be underachieving at school – all of these are psychological factors.

Consider: *Why is reductionism a problem for neurochemical theories? Can you think of any ways in which it could be a strength?*

Individual differences

Saul Shiffman *et al.* (1995) studied 'chippers', people who regularly smoke even for decades, but do not become dependent on nicotine. Those who smoked an average of five cigarettes a day showed no signs of a withdrawal syndrome when they abstained. They also performed better on **cognitive** tasks (such as mental arithmetic) than dependent smokers. It appears that there must be non-chemical factors that protect some smokers from addiction, although it is currently unclear what these factors are. One possibility the researchers suggest is that some people smoke because they have learned to do so through **modelling**. So their motivation has nothing to do with nicotine at all.

Consider: *Can this be explained by neurochemical theories? Is this a limitation of such theories? Explain your answer.*

Apply it — Concepts: The never-ending habit

Nick has smoked cigarettes for nearly two decades and has noticed that he is smoking more and more per day as the years have gone by. Despite this, he feels he doesn't get the same feeling from smoking that he used to and is worried that this means he'll just keep on increasing his intake more and more.

Question

Using your knowledge of the neurochemistry of addiction, explain why this is happening to Nick.

Apply it — Methods: A smoking correlation

As we saw in Evaluation extra, Shiffman *et al.* (1995) investigated several differences between 26 'chippers' and a **matched** group of 25 regular smokers. All participants smoked normally for two days and were deprived of nicotine for two days. These two-day periods were separated by a week. The order in which participants completed these periods was **counterbalanced**. Chippers suffered no apparent withdrawal effects. But regular smokers experienced more cravings, poorer mood, and sleep disturbances. They also performed more slowly on cognitive tasks.

Questions

1. Write an appropriate **aim** for this study. *(2 marks)*
2. What does it mean to say that the two groups were 'matched'? *(2 marks)*
3. What does it mean to say that the 'order in which participants completed these periods was counterbalanced'? *(2 marks)*
4. Identify *one* **dependent variable** in this study and explain how you would **operationalise** it. *(1 mark + 2 marks)*
5. Explain how the researchers could have obtained their samples of chippers and regular smokers. Identify the **sampling method** you have chosen. *(2 marks + 1 mark)*

CHECK IT

1. Outline the role of dopamine in nicotine addiction. *[4 marks]*
2. Outline the brain neurochemistry explanation of nicotine addiction. *[6 marks]*
3. Describe and evaluate **one or more** neurochemical explanations of nicotine addiction. *[16 marks]*