

Smoking and mental health

Action on Smoking and Health – September 2004

How smoking affects the brain

Within 10 seconds of inhaling tobacco smoke, nicotine reaches the brain and begins to act on a specific set of neurons, the working cells of the brain. On each of these neurons are receptors, which are like slots or keyholes onto which brain chemicals called neurotransmitters attach, causing the brain to transmit messages. Nicotine fits into one of the receptors acted upon by acetylcholine, one of several neurotransmitters in the brain. This causes the brain to release two other substances, noradrenaline and dopamine, that act as stimulants. [i\[1\]](#)

Smoking and Stress

Smokers often report that smoking tobacco helps to relieve feelings of anxiety and stress. However, smokers exhibit higher levels of stress in their lives than non-smokers. The high smoking prevalence among people facing social and economic deprivation suggests that smoking may be used as a stress coping mechanism. [ii\[2\]](#) However, the stress reducing properties of nicotine seem more illusory than real.

Nicotine stimulates the brain to release dopamine, which is associated with pleasurable feelings, and smokers quickly develop regular smoking patterns. Eventually, smokers need increasing levels of nicotine to feel 'normal'. As the nicotine content in their blood drops below a certain level, they begin to crave for a cigarette. This craving makes the smoker feel 'stressed' until the craving is relieved. The relief felt when this craving is finally satisfied is the feeling that smokers commonly mistake as 'relaxing'. (see also fact sheet no 9, [Nicotine and Addiction](#))

Depression

Cigarette smoking is linked with a wide range of psychiatric diagnoses including anxiety, agoraphobia and panic disorder but especially with depression. [iii\[3\]](#) Many epidemiological studies have reported an association between clinical depression and smoking. Some have concluded that the effects of long-term nicotine exposure on the brain may have a causal influence on major depression while others suggest that shared environmental or genetic factors may predispose to both smoking and major depression. A longitudinal study by Breslau et al found that a history of daily smoking increased significantly the risk of major depression. [iv\[4\]](#) This was consistent with earlier reports which suggested that previous smoking history increased the risk of depressive symptoms and increased the risk of attacks of major depression.

A study by Kendler et al suggested that the relationship between smoking and major depression results solely from genes that predispose to both conditions. [v\[5\]](#) Other potential shared aetiologies are factors in the social environment, personality (for example, low self-esteem), and coping styles. Nicotine may act as an anti-depressant in some smokers and could therefore be viewed as a form of self-medication.

When individuals with a history of depression stop smoking, depressive symptoms and, in some cases, serious major depression may ensue. [vi\[6\]](#) This accounts for the lower smoking cessation rates in depressed individuals as compared with smokers who do not have depressive symptoms. A study by Kinnunen et al showed that only 37% of the depressed smokers in their sample population were able to abstain for one week, whereas 56% of non-depressed were able to do so. [vii\[7\]](#)

The evidence so far is inconclusive and there is dispute among scientists as to whether smoking is the cause, or effect of mental illness. However, some researchers believe that smoking itself could act as a trigger for mental illness. In a review of the evidence to assess the links between tobacco smoking and mental disorder, two public health researchers concluded that nicotine dependence is indeed a mental disorder, from which most smokers suffer. They found that nicotine dependence was strongly associated with a variety of other mental disorders. Mental

disorder was linked with an increased propensity to smoke and a reduced likelihood of cessation. [viii\[8\]](#)

Schizophrenia

A link is thought to exist between smoking and schizophrenia. However, the key, relevant factors are the degree of the psychiatric disorder and whether the sufferer is institutionalised. As a consequence, the debate over the relationship between smoking behaviour and mechanisms underlying schizophrenia or its treatment, has been labelled “premature.” [ix\[9\]](#)

Patients with schizophrenia have an extremely high prevalence of smoking; a US study in 1986 found about 88% of patients were smokers compared with only 33% in the general population. [x\[10\]](#) The reason for this is unknown, but it is likely that smoking behaviour in schizophrenia is a complex process. The increase in dopamine release induced by smoking may be helpful in alleviating some schizophrenic symptoms. Therefore, schizophrenics may smoke in an attempt to self medicate. Smoking also interacts with neuroleptic treatment (drug treatment for schizophrenics), reducing neuroleptic plasma levels and possibly causing higher doses of neuroleptics to be prescribed. [xi\[11\]](#) One study has also shown that patients smoke more when treated with the neuroleptic Haloperidol than during a medication-free state. [xii\[12\]](#)

Alzheimer’s Disease and Dementia

AD is a common form of senile dementia, the other being vascular dementia. Loss of neurons (brain cells) that use acetylcholine as their neurotransmitter, and loss of memory are prominent features of AD. Studies conducted in the early 1990s suggested that smoking had a protective effect against AD. [xiii\[13\]](#) Although research on this subject has failed to be conclusive, it was thought that nicotine could delay the onset of familial AD. Acetylcholine binds to receptors, known as nicotinic receptors, to exert its effect. A loss of neurons leads to a loss of these receptors and this is associated with the aetiology of AD. It was hypothesised that nicotine from cigarettes may compensate for the loss of nicotinic receptors in AD and therefore postpone the onset of the disease.

Scientists at the Scripps Research Institute, California, have discovered that nornicotine, a by-product of nicotine, appears to prevent the abnormal build-up of amyloid protein plaques associated with Alzheimer’s disease. However, the research did not demonstrate that smoking had any protective effect for AD. Other research has shown that smoking increases the risk of AD and vascular dementia by increasing the amount of free radicals in the body, which impair brain and body cell functions and undermine immunity. [xiv\[14\]](#)

Recently, scientists have begun to challenge the protective role of smoking hypothesis. [xv\[15\]](#) [xvi\[16\]](#) They point out that earlier studies assumed that the genetic susceptibilities of a population of older surviving smokers was the same as that of the age matched non-smokers. However, it has been suggested that older surviving smokers must have relatively more effective DNA repair mechanisms than comparable non-smokers. Therefore, if AD is related to the accumulation of ageing-associated defects in DNA and DNA repair, older surviving smokers may be less susceptible to AD. This could explain the apparent inverse relationship found by many studies in the past.

A study involving 17, 600 people aged 65 and over, screened the participants for dementia. The survey, conducted in Britain, Denmark, France and the Netherlands looked at the effect of smoking on cognition in non-demented elderly. It concluded that smoking may indeed accelerate cognitive decline in non-demented elderly. [xvii\[17\]](#)

Even if smoking is “protective” against AD, smoking could never be advocated for this purpose. This is because the known health risks of smoking far outweigh any possible reduction in risk of getting AD in later life.

Parkinson's Disease

Parkinson's Disease is characterised by the symptoms of tremor, rigidity, bradykinesia (slowness of movement) and a lack of facial expression. Many studies have shown that smoking is protective against PD. [xviii\[18\]](#) PD occurs because there is a loss of dopaminergic neurons in the brain. These are neurons that release dopamine as their neurotransmitter and they are important in ensuring accurate movements of muscles as commanded by certain areas of the brain. It is thought that nicotine may have its effect by restoring dopamine to normal levels in the brain. [xix\[19\]](#) Again, the researchers emphasise that the possible benefits of smoking on PD risk would be small (the incidence rate of PD is only about 1-2%), and the health hazards associated with smoking would far outweigh any conceivable protection against PD. However, the findings should be viewed as potentially advancing the current understanding of the underlying pathology of PD.

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