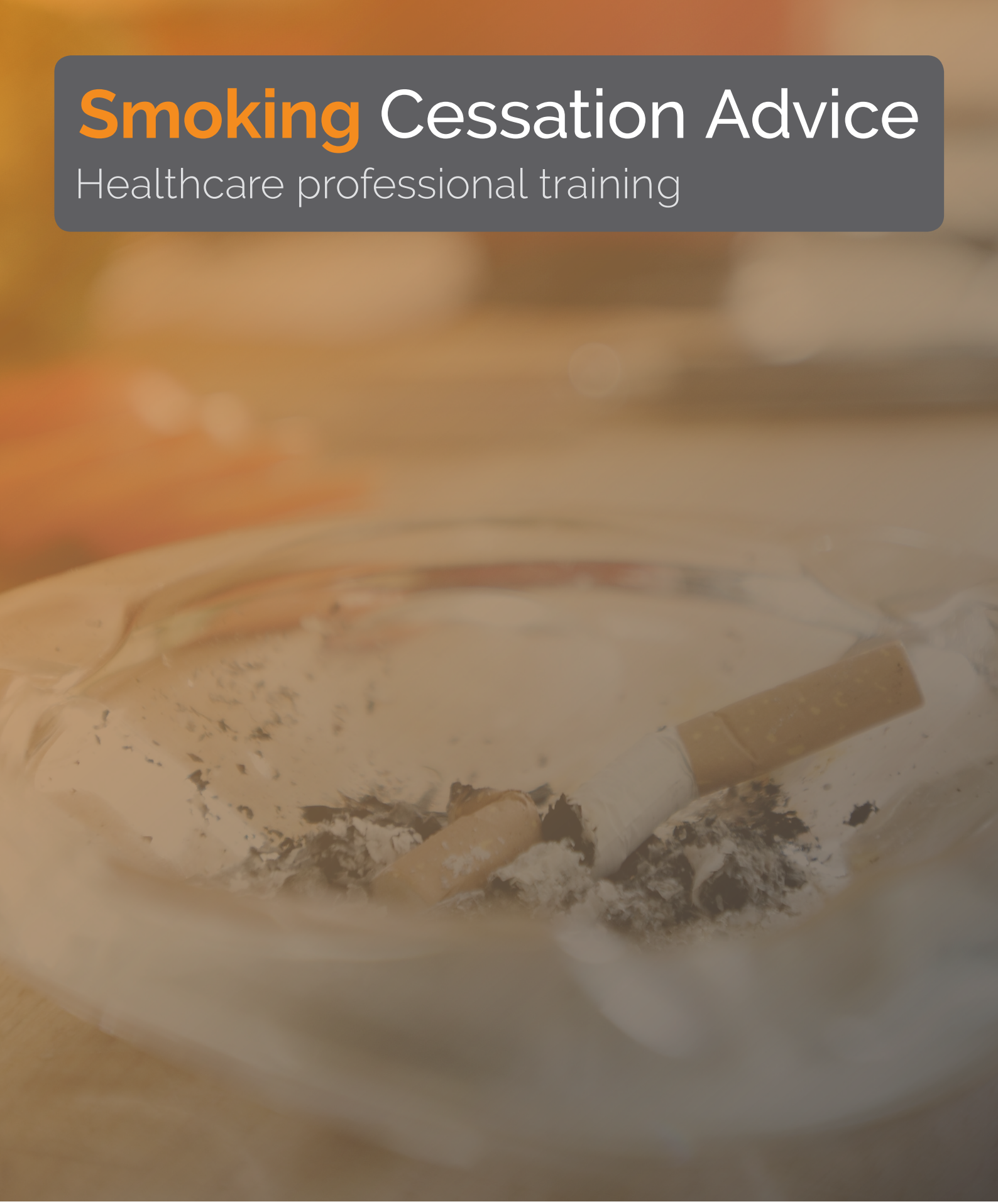
Smoking Cessation Advice

Healthcare professional training



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Barriers to Quitting

Introduction

For over 200 years, there has been an ever increasing body of evidence regarding the risks of smoking (Doll 1998). Links between smoking and malignancy of the lip and mouth were made as early as 1795 (Sömmering), with studies from the mid-20th century onwards linking smoking with lung cancer and vascular disease (Doll 1950, Doyle 1962). Smoking was initially an accepted 'social norm' in society, viewed simply as a glamourous habit, with little attention paid to tobacco's addictive nature (Cummings 2016). The concept of physical, and more so psychological, addiction in relation to cigarette smoking is a relatively new one (Jarvik 1970, Cosci 2012) and it is only in the past twenty years that the role of nicotine in sustaining smoking behaviour has become more widely accepted (Fagerstrom 1990).

The sequelae of smoking are now much better understood and discussed, but users choose to ignore these issues. Smoking is the largest cause of preventable disease and an important cause of premature death worldwide (Samet 2013, Fagerstrom 2002). Almost 90% of lung cancer diagnoses and 25-30% of all cancer related deaths are made in smokers (Anand 2008). 80% of COPD diagnoses are made in individuals who have a history of smoking (Kuempel 2009). The most damning indictment is made by the US Surgeon General in the report, 'The Health Consequences of Smoking – 50 Years of Progress' (2014). This publication states that the 'evidence is incontrovertible: inhaling combustion compounds in [tobacco] smoke is deadly,' and that 'cigarettes are a defective product - unreasonably dangerous, killing half its long-term users and addictive by design.' Smokeless tobaccos have also been implicated in causing pancreatic cancers (Boffetta 2005).

It is now understood that cigarette smoking is a manifestation of nicotine dependence in both physical and psychological forms (Breslau 1995, Ho 1989). Nicotine addicts will engage in smoking to relieve boredom and as a habitual act, manifestations of the psychological addiction. They will also regulate the draw and inhalation they take of a cigarette, titrating their nicotine dose to their perceived physical need (Ross 2016). A multifactorial addiction, we must also take the social, economic, personal, and political influences of smokers into account.

Why do people start smoking?

In the face of such open and targeted advice regarding the health effects of smoking, it poses the question 'why do people start smoking?' Smokers tend to begin using tobacco products before they leave school, with 90% of addicts commencing in regular tobacco use before their 19th birthday (USDHHS 1994). There are three separate factors to consider when assessing the likelihood of an individual to start smoking; environmental factors, socio-demographic factors and individual factors.

Environmentally, we are aware that parental and sibling smoking alongside contact with peers who are smokers promotes pro smoking ideology (Conrad 1992). Socio-demographically, those from a lower socio-economic background and individuals from an ethnic minority are more likely to smoke (Conrad 1992). Finally, individual factors, such as poor performing rebellious students, individuals with low self-esteem, and history of previous underage 'risky' habits (for example alcohol consumption) are more likely to smoke (Kearney-Cook 1999). There is also a strong link between perceived 'image' and smoking, with more popular individuals at school being more likely to engage in smoking habits as it is deemed to be rebellious and 'cool.' (Valente 2005). This strong evidence regarding at risk individuals allows us to explicitly target anti-tobacco advice to 'at risk,' adolescents, which has been shown to be effective in reducing desire to commence smoking in potential new smokers (Grandpre 2003). Interestingly, the rate at which individuals become daily smokers almost matches the quit rate, which means smoking prevalence is only declining very slowly (CDC 2009).

As healthcare providers, working within our care system engagement in and support of media campaigns deterring smoking uptake in youths is the best way to reduce smoking uptake in young people. Targeted media campaigns have helped reduce regular smoking to 3% of 11-15yr olds in England (Hawkins 2012).

Why is smoking addictive?

Nicotine is a natural ingredient within tobacco leaves, meaning it is present in cigarette tobacco and smokeless (oral) tobacco. Inhalation during the act of smoking a cigarette distils nicotine from the tobacco leaves (Benowitz 1996). The nicotine is transported into the lungs on tar droplets. When these droplets reach the alveoli of the lungs the nicotine is rapidly absorbed into the blood stream. Although some nicotine is absorbed through the oral and nasal mucosa, at the pH in cigarette smoke (5-6) nicotine is primarily ionised, meaning uptake is poor (Gori 1986). In the lungs, where the pH kept above 7 by the presence of lung fluid, nicotine is significantly less ionised, facilitating transfer across the mucosal barrier (Benowitz 2009). Smokeless tobacco can cross the oral mucosa more easily than inhaled smoke held in the mouth due to the natural pH of the mouth and absence of low pH smoke (Pickworth 2014).

Once within the bloodstream, the nicotine travels to the brain where it can bind to nicotinic cholinergic receptors on neurons. This facilitates the entry of calcium into the neuron upon which it has bound (Dajas-Baildor 2004). This movement of calcium into the neuron facilitates neurotransmitter release. The most important neurotransmitter in the dependence pathway is dopamine and this is released in the presence of nicotine. It is this neurotransmitter that gives the sensation of a pleasurable experience and is associated with reward motivated behaviour (Nestler 2005). Increased availability of dopamine in the brain is synonymous with drugs of abuse. Addictive substances, such as cocaine and MDMA, have heightened dopamine production strongly associated with their addictive characteristics (Ghodse 2010). Stimulating acetylcholine receptors in the brain is also associated with release of acetylcholine, noradrenaline, adrenaline and serotonin (Pomerlau 1989). This cocktail of neurotransmitters results in the user feeling more alert and more focussed alongside modulating their mood.

Why is quitting so hard?

Unfortunately, the pleasure of continued nicotine use is outweighed by the discomforts of quitting. Indeed, withdrawal symptoms are often cited as a reason to maintain, and relapse to, cigarette use (Baker 2004). Withdrawal symptoms begin within the first hour after the last cigarette (Gross 1997, Hendricks 2006), and this is concurrent with nicotine's terminal half-life of 2 hours (Benowitz 1982). Therefore, smokers must regularly engage in activity to keep a constant level of nicotine available within the brain (Bergen 1999). The neurotransmitters released and increased availability of nicotine within the brain of a smoker results in neuroadaptation occurring. Withdrawal symptoms typically include irritability, restlessness, depression, anxiety, issues with personal relationships, decreased concentration, increased hunger and eating and insomnia (ASA 1994). Indeed, the loss of mood regulation should not be overlooked, with untreated withdrawing nicotine users having been shown to have mood disturbances which are comparable to psychiatric outpatients (Hughes 2006).

Often the most cited reason reported by smokers for continuing smoking is the perceived effect on stress levels. This is difficult to quantify; however, smokers have self-reported lower anxiety when smoking compared to when not smoking (Jarvik 1989). In addition to this, studies have identified that smoking reduces perceived stress when in direct presence of a stressor, for example whilst watching a stressful film (Gilbert 1989). Those who are more prone to smoking (and indeed drug use in general) are often individuals who are more likely to be affected by anxiety, depression and addiction (Barlow 2002). Therefore, it is important to anticipate increased incidence of anxiety on initial cessation attempts in long term smokers, particularly during the height of nicotine withdrawal. This peak normally occurs at 1-2 days' post cessation (Jorenby 1996). Interestingly, smoking cessation has been shown to ultimately improve happiness and reduce stress levels in the long term (Taylor 2013).

The psychological addiction of nicotine is related to repeated actions and conditioning. There are often powerful environmental cues associated with the reward process of smoking, for example the act of having a cigarette whilst with friends (Benowitz 2008). The sensation and act of smoking then become linked with positive events, meaning there is a desire to engage in smoking as it is associated with positive experiences (Rose 1993). It is not even pleasant experiences which become conditioned to a smoker. The link between cessation and irritability alongside smoking and pleasure is often well understood by regular users of nicotine. This means that a smoker can link the desire of having a cigarette with any period where they feel irritable (Kobiella 2011). Individuals also regularly cite stressful scenarios as a major cause of post withdrawal relapse (Cohen 1990). Smokers who suffer from depression are 40% less likely to quit successfully than non-depressed smokers (Acton 2001).

Aids to cessation

Introduction

In England, 19% of the population identify themselves as smokers. Smoking and chewing tobacco are directly related to 30% of cancer diagnoses each year and estimated to be the cause of 120,000 deaths per annum in the UK (Britton 2004). The links between smoking and disease have been well documented over the past 50 years, with studies showing that over half of long-term smokers will die from smoking related disease (Doll 2004). There is hope, with 70% of smokers stating they 'intend,' to give up smoking (Lader 2003). 50% of this group intending to quit within the next 12 months. This means that there are roughly 6 million people who want to quit in England in the next year.

People have different requirements when they attempt full cessation, with some choosing to try and stop smoking without any assistance and opting for requiring focussed interpersonal support. Physical withdrawal symptoms can then be titrated through nicotine replacement therapy (NRT). For people who need face to face support to assist with cessation, there are NHS Stop smoking services. These services have been available to all UK residents since 2000 (Hiscock 2001). Unfortunately, despite consistent improvements throughout the service, there are currently only 230,000 people in the UK who have an NHS Stop Smoking 'Quit Date' recorded within the next 12 months. This proves that the service is currently underutilised (West 2013). Studies show that, unaided, the successful quit rate is 3-4% (Hughes 2004). For those who utilise 'Stop Smoking,' services, these rates can increase to 15-20% (Ferguson 2005, West 2000).

For individuals who do not require or want face to face support, nicotine replacement products have been shown to reduce the difficulties associated with nicotine withdrawal (Molyneux 2004). NRT is therefore a useful tool in remaining abstinent following cessation (Gourlay 1990). There are other medications which can be used alongside NRT to help maintain cessation in motivated individuals. Examples of these include Buproprion and Varenicline, however these have been linked with severe and undesirable side effects in some users (Gibbons 2013).

There is a growing number of smokers utilising electronic cigarettes or 'vaping,' as a method of improving cessation success (Brown 2014). This appeals to smokers because they help manage the physical craving by supplying nicotine into the lungs (where they can provide an arterial bolus to the brain). Additionally, they allow the user to engage in an act similar to smoking. This reduces the psychological impact to the user; the smoker can maintain their previous 'smoking habits' (Yeh 2016).

Stop Smoking Services

Stop smoking services can utilise multiple cessation aids, however in this section we will explore the role of interpersonal interaction in smoking cessation, as this is unique to Stop Smoking Services. It has been shown that individual counselling via the medium of a smoking cessation specialist may help make a successful attempt to stop smoking (Fiore 2000). The more intense this advice, the more likely an individual is to successfully stop smoking (Lancaster 2005). Indeed, it would seem that there is no difference in smoking cessation rates whether counseling is given by medically or non-medically qualified individuals. (Fiore 2008). The effect of individual counselling in isolation is confounded by NRT use.

There is also limited evidence which confirms that telephone support aids quitting. Three or more calls have been shown to increase effective cessation compared to self-help materials and other brief intervention (Stead 2006). However, it is unknown if this intervention would be successful in isolation, as studies show its efficacy as in tandem with NRT (An 2006).

Group based therapy is another way of delivering cessation via interpersonal means. Again, group cessation has been shown to be beneficial in improving cessation attempts (Wenig 2013). However, it is difficult to ascertain if group or individual therapy is more beneficial in stopping smoking (Stead 2005). Data is again frequently confounded by the use of NRT in cessation trials, however cessation with group therapy is more likely compared to NRT or cold turkey in isolation (Hollis 1993).

This shows that any form of targeted intervention by a specialist increases the chance of successful cessation. As stated, NRT is often used in conjunction with stop smoking services which together increases the likelihood of successfully remaining abstinent (Molyneux 2003). Depending on the health service of a country, decisions must be made on a cost-benefit basis. Whilst group intervention and telephone intervention are undoubtedly cheaper, they are less effective than a targeted individual intervention (Lancaster 2005). A decision must therefore be made; does the reduced cost of a less effective cessation modality outweigh the increased cost to society of an increased number of smokers?

Nicotine Replacement Therapy

All forms of nicotine replacement therapy have been shown to be beneficial in long term smoking cessation, almost doubling success rates (Kralikova 2009). NRT can take the form of gum, skin patches, inhalers, lozenges alongside nasal and oral sprays (Tool 2015). It is a frequent component of smoking cessation strategies as it reduces the physiological effects of cessation, providing nicotine to reduce the effects of withdrawal.

The first NRT to be developed and become widely available was nicotine gum. Normally this comes in two dosages, 2mg and 4mg, with the lower dosage not requiring a prescription (Ebbert 2007). Issues have been noted with nicotine gum however. Nicotine does not cross the oral mucosa well in acidic environments so food and drinks that may reduce salivary pH will reduce the amount of nicotinic absorption (Henningfield 1995). This reduces the efficacy of the gum as a cessation aid and can increase the risk of relapse. There is a similar issue with nicotine lozenges, as these require a suitable oral pH (7) to ensure nicotine is able to cross mucous membranes within the mouth (Santus 1996). The same is true for oral sprays. As such, patients utilising these NRTs may have to be advised of maintaining a non-acidic diet during their use.

Transdermal patches are another method of delivery which have been shown to be effective in delivering even high doses of nicotine (Fiore 1992). As the nicotine is absorbed through the dermis the user cannot impact the delivery, meaning nicotine transfer is much more predictable and consistent when compared to lozenges and gum (Stapleton 1995). It should be noted that gum and patches do not provide 100% nicotine replacement, meaning combinations of different NRT may help heavy smokers reduce their nicotine intake, for example gum and patches together (Dale 1995, Puska 1995).

Nasal sprays have had differing outcomes, with studies stating they are beneficial in high nicotine users (Stapleton 2011), but also that they are a poor cessation aid in adolescent smokers (Rubinstein 2008). It should be noted that NRT in isolation has been shown to be of limited use in adolescent smokers (Adelman 2004). Users of nicotine spray also complain of negative side effects, such as nasal irritation and nasal blockage. However, delivery via the nasal passage also negates the risk of any acidic foodstuffs impairing nicotinic absorption, so may therefore prove to be beneficial to some users.

The nicotine inhaler has seemingly been replaced by 'electronic-cigarettes,' in regards to patient perception of satisfaction and benefit (Steinberg 2013), however it still plays a role as an NRT (Bolliger 2000). Nicotine inhalers aid in the habitual element of smoking addiction (as the individual has something to hold and inhale), but they do not provide a similar arterial bolus of nicotine.Very little inhaled nicotinereaches the bronchioles of the lungs (Schneider 2001, Lunell 2000). Users of inhalers often choose this modality of nicotine delivery over an electronic cigarette because there is evidence medicinal nicotine inhalers are likely less dangerous than electronic cigarettes (WHO 2009).

Nicotine Replacement Therapy

A chart showing Pros and Cons of different over the counter nicotine replacement therapy and electronic cigarettes.

Product	Pros	Cons
Inhaler	• Gives psychological addiction relief • More effective than nicotine gum • Flexible dosing • Rapid nicotine absorption	• Reinforces psychological smoking addiction, which may ultimately be detrimental • Comparatively costly • Cannot be used discretely • Can irritate mouth and throat • Can't be used before eating/drinking as reduces efficacy
Oral Sprays	• Flexible dosing • More effective than nicotine gum • Rapid nicotine absorption	• Flavour isn't particularly nice • Must hold spray in mouth; difficult to talk whilst using • Can irritate mouth and throat • Can't be used before eating/drinking as reduces efficacy
Chewing gum	• Dosing more flexible than adhesive patches • Chewing can be a distraction from cravings • Discrete; people will assume chewing regular gum	• 'Gum' can be misleading - should be chewed then 'stored' in buccal sulcus, not chewed like normal gum • Must be used frequently to maintain nicotine levels • Can't be used before eating/drinking as reduces efficacy
Lozenges	• Easy to use • Flexible dosing • Delivers more nicotine than gum	• Lozenges must not be chewed or swallowed • Users commonly complain of nausea
Nasal spray	• More effective than nicotine gum • Flexible dosing • Rapid nicotine absorption • No effect of food or drink unlike oral NRT	• Can cause nasal and ocular irritation • Frequent use required to maintain nicotine levels
Adhesive patch	• Easy to use • Cheap • Only needs to be applied once a day • Limited side effects • Discrete	• Dosing not flexible • Nicotine uptake can be slow • Highest rates of relapse whilst using this NRT

All the products deliver nicotine, reducing physical craving and withdrawal symptoms.
All products may therefore perpetuate addiction to nicotine; users may become addicted to NRT.

Electronic cigarette n.b not officially recognised as an NRT	• Gives psychological addiction relief • Flexible dosing • Widely culturally accepted amongst smokers • 'Fun' to use • Limited side effects • Widely available and inexpensive	• Risks aren't known • Production is not regulated • 'Liquids,' used still contain known carcinogens • Viewed as a replacement, not a cessation aid • Cannot be viewed, currently, as a NRT • Risk reduction strategy only

Pharmacotherapy
(Varenicline, Buproprion, Clonidine & Nortriptyline)

Varenicline is first line systemic medication which acts as a partial nicotine receptor agonist useful for assisting in successful smoking cessation attempts (Gibbons 2013). Part of the mechanism of action of this drug is to block the nicotinic receptors in the brain, removing the pleasurable sensations experienced upon having a cigarette (Coe 2005). Compared to unassisted cessation attempts, users of varenicline are two to three times more likely to quit (Cahill 2012). Despite the overlap of mechanisms of action, it would appear that NRT (nicotine patch) in combination with varnicline increases the chances of successful cessation (Chang 2015). Any medicine which must be taken orally may result in side effects for the user. Varenicline's most common side effect is nausea, which is experienced by 33.5% of users (Tonstad 2006). There have been reports of more severe, neuropsychiatric adverse events such as depression and suicidal behaviour, however this is found to be inaccurate (Thomas 2015). Varenicline users do have less adverse neurological side effects, such as abnormal dreams and sleep disturbances (McKee 2009). These lesser side effects are documented and accepted throughout the literature, and are generally tolerated well by patients (Wang 2009).

Buproprion is another first line systemic medication which assists with smoking cessation when compared to willpower alone. Indeed, people attempting to quit through using buproprion were 20% more likely to cessate (Wilkes 2008). Bupropion exerts its effect primarily through the inhibition of dopamine reuptake, which attenuates withdrawal symptoms by prolonging the presence of this neurotransmitter within neuronal synaptic vesicles (Warner 2005). Buproprion has been shown to be slightly more effective combined with NRT (Jorenby 1999) Again, this medication has been associated with manageable side effects such as nausea, dizziness and vomiting (Boshier 2006). More serious side effects include hallucinations and seizures (Shepherd 2005), although these are very rare, with the risk of seizures being reported by GlaxoSmithKline (2006) at 0.1%. In relation to the two first line pharmacotherapy treatments, both have roughly the same amount of successful cessation after 2 years (Gonzales 2006).

Clondine and nortriptyline are given as a second line pharmacotherapy; it is given when the two rst line therapies don't work or are contraindicated (Nides 2008). These have both been shown to be as effective as the first line medications, but have an increased number of side effects (Fiore 2000, Hughes 1999). In isolation, clonidine has been shown to double the rate of quitting, and nortriptyline has been shown to triple success rates (Corelli 2002). Side effects are common in users and include Clonidine causing hypotension and drowsiness and nortriptyline causing sedation, nausea, dry mouth, constipation, and urinary retention (Hughes 2003).

Electronic Cigarettes

Electronic cigarettes, or e-cigarettes, are designed to facilitate 'smoking,' without the physical combustion of tobacco (Goniewicz 2014). Despite the novel method of delivery, electronic cigarettes allow an arterial bolus of nicotine to be transmitted through the lungs in much the same way as cigarettes, creating an arterial nicotine concentration similar to that of a smoker (Vansickel 2013). Such is the recent increase in popularity of electronic cigarettes that Google recorded a 5000% increase in searches for them between 2008-2010 (Yamin 2010). It has also been shown that non-nicotine containing e-cigarettes can help improve the success of quitting attempts, showing they are effective in maintaining the aspect of psychological addiction, whilst weaning off the physical addiction (Bullen 2013). The recent Cochrane review by Hartmann-Boyce (2016) showed that nicotine containing e-cigarettes are better than nicotine free e-cigarettes for promoting cessation. Their use can increase the likelihood of quitting, but often simply leads to a reduction of cigarette use opposed to complete cessation (Etter 2011).

Unfortunately, there may be some negative aspects to electronic cigarette use due to the constituents of the e-cigarette liquid which is 'vaped.' The glycerol and nicotine mix which is inhaled does contain toxic substances which are also found in cigarette smoke (Williams 2013). These toxic substances are at a significantly lower concentration than in traditional cigarette smoke, meaning it may be acceptable to view e-cigarettes as a 'harm reduction' device (Cahn 2011). At this stage NRT is better understood and should be recommended as the gold standard cessation aid by practitioners, however electronic cigarettes may be beneficial to smokers who are unwilling to quit (Hajek 2014). Ultimately, more research is needed into these as an adjunct to cessation, however until this point electronic cigarettes have not been shown to invoke any serious adverse events (Hartmann-Boyce 2016).

Referral to stop smoking services

Introduction

In the UK, the NHS stop smoking services were set up with the simple aim of helping people to stop smoking. These services implement different cessation methods which can be based around behaviour change techniques. Whilst these can be used in combination with nicotine replacement therapy (Stead 2008), it is these counselling services that are unique to the stop smoking services. Dependent on multiple factors, such as funding and service setup, behaviour change sessions can be via group therapy or individual support. It is shown that both of these methods in isolation have a positive outcome on cessation techniques (West 2010). However, we must note that not all individuals wish to utilise stop smoking services, meaning brief intervention from potential referral sources should always be undertaken in the first instance (Lichenstein 1992). When we consider it can take smokers up to 30 attempts to successfully quit, it is important for practitioners to reinforce simple cessation advice and offer stop smoking service referral at every opportunity (Chaiton 2016).

In 1998, the UK government outlined new policies to combat tobacco addiction in the White Paper 'Smoking Kills,' (DoH 1997). By 2001, this guidance had resulted in the nationwide introduction of the precursor to the current stop smoking service; the 'Smoking Cessation Services,' (McNeil 2005). These services were ultimately shown to be successful (Bauld 2003). Gradually, this service has evolved into the 'NHS Stop Smoking Service,' we currently utilise to assist individuals in their cessation attempts. For example, it was found initially that cessation attempts in poorer areas were less likely to be successful than those with lower levels of social deprivation (Nagelhout 2012). Increased advertising, alongside encouraging healthcare practitioners to refer these individuals to stop smoking services, means during the past 15 years there has been an increased amount of successful engagement with smokers from disadvantaged communities (Chesterman 2006). This highlights the learning culture present within the NHS.

Referral Sources

General Practice

General medical practitioners hold an important role in smoking cessation, with 70% of smokers visiting their GP at least once a year (Jamal 2012). There is also evidence to show that motivated physicians facilitate successful smoking cessation (Kottke 1988). If all physicians offered routine, standardised cessation advice the cumulative increase in quit rates could be substantial (Chapman 1993).

GPs may choose not to discuss smoking cessation as they believe it can damage the doctor patient relationship (Coleman 2000). For example, doctors are worried patients will perceive advice as being pushy. There is evidence that well managed cessation techniques can improve the doctor-patient relationship (Solberg 2001). GPs also have the authority to prescribe smoking cessation aids, which can enhance successful quit attempts two-fold (Stead 2008). Stop smoking service support further increases the chances of successful cessation, highlighting the benefit of referral to these services. We must view every GP appointment with a smoker where cessation is not discussed as a missed opportunity (Stead 2008).

Pharmacy

99% of the UK population live within 20 minutes of a pharmacy, either by foot or by public transport (Todd 2014). An estimated 1.6 million individuals visit a pharmacy every day (DoH 2008). The pharmacist is therefore in a privileged position as they have regular interactions with large numbers of individuals when they are both 'healthy' and 'sick' (HEA 1994). Whilst pharmacists are trained in nicotine replacement therapy use and behavioural support to assist in smoking cessation, there are issues identified by pharmacists in delivery of this information (RPS 1999, West 2000). For example, pharmacists believe they don't always have enough time due to dispensing duties to give adequate advice (Anderson 2003). It is shown, however, that cessation advice given by pharmacists is beneficial in increasing successful cessation (Maguire 2001). Again, the support offered by stop smoking services further increases the success of quit attempts compared to pharmacist cessation advice alone.

Referral Sources

Dentistry

59% of the UK population regularly visit a dentist (Nuttall 1998). Dentists are perfectly positioned to notice the impact of tobacco use in the mouth, in both smoked and smokeless forms (Monaghan 2002). Potential oral sequelae experienced by smokers include red and white patches, nicotinic stomatitis and periodontitis (BDA 2000). More threatening to patients than these is that tobacco exposes the oral cavity to carcinogens which initiate and promote oral cancers; these account for 2% of all cancer diagnoses in the UK (Chestnutt 2016). Dental teams are in the enviable position of being able to offer advice to a largely 'healthy' section of the population who engage in tobacco use (Chestnutt 1999). Brief 2-minute advice from dental practitioners can lead to a 2% increase in successful smoking cessation (Raw 1998). By referring to stop smoking services this increases to 15-20% (Ferguson 2005, West 2000).

Optometry

Smoking is the single greatest controllable risk factor in the development and proliferation of age related macular degeneration (Thornton 2005). It is also implicated in the development of nuclear cataract (Kelly 2005). It is documented that the use of optometrists as a healthcare professional to provide brief smoking cessation is underutilised (Thompson 2007). Despite this, it appears there is an appetite by optometrists to be involved in provision of cessation advice. We can conclude optometrists provide a further opportunity to deliver brief advice to those who utilise tobacco and offer referral to stop smoking services when appropriate.

Maternity Services

Smoking has numerous effects on a mother and her baby. It reduces the amount of available oxygen to mother and baby, it increases the heart rate of the child alongside increasing the incidence of premature birth, low birth weight, miscarriage and stillbirth (Cnattingius 2004). Smoking can also cause sudden infant death syndrome and increases the risk of the baby developing respiratory problems (Hunt 2001, Tager 1993). 12% of pregnant women smoke throughout their pregnancy term, with these infants more likely to become smokers themselves (Chan 2003, TAG 2000).

Referral Sources

As maternity services have the largest access to pregnant smokers, they can give brief cessation to these individuals alongside referral to NHS stop smoking services. Interestingly, NRT is found to be of limited use amongst pregnant women beyond their first trimester (Kapur 2001). Behaviour management strategies are shown to be effective for pregnant women, but not their partners (de Vries 2006). This is problematic, as second hand smoke can have similar deleterious health outcomes for the mother and her child (Leonardi-Bee 2011).

Secondary Care

Smokers have a significantly increased risk of cardiopulmonary and wound related post-operative complications than their non-smoking counterparts (Bluman 1998). A preoperative period of cessation of 6-8 weeks shows to greatly reduce the risks of post-operative complications (Møller 2002). In head and neck cancer patients, a period of just three weeks has shown to be beneficial (Kuri 2005). Therefore, a pre-operative assessment within a secondary care environment gives a perfect opportunity to recommend cessation and advise of potential post-operative risks to smokers.

Hospital admission can be useful in promoting smoking cessation; it imposes temporary smoking abstinence and the environment assists in making individuals aware of the health implications of smoking (Murray 2013). If initial cessation ideology is imparted during the inpatient stay, followed by one month of continued support (for example from stop smoking services) individuals are more likely to successfully stop smoking (Rigotti 2012). Cessation advice should be provided when a smoker has an interaction with any of the listed healthcare services.

Conclusions

In the UK, there are local stop smoking services, which individuals can contact through the 'NHS Smokefree,' website. It is these individuals, who are independently looking to stop smoking, that are more successful in their quit attempts; not ones recruited by medical professionals (Borland 2012). The NHS Smokefree website, which is often accessed by those proactively looking to stop smoking, helps individuals:

- Obtain details of the local stop smoking service through a web based search.
- Obtain a quit kit sent in the post (with associated smartphone app, email service or text service to assist with motivation).
- Access a Facebook page to offer quit assistance and support from other members of the quitting community.
- Obtain a Freephone number to talk to a stop smoking advisor, who can advise on quitting techniques and the local stop smoking services.
- Utilise a web chat for those who want the same advice but without making a phone call.
- View success stories from other successful quitters, alongside other pro-cessation content.

These services are extremely cost effective, costing £800 per life year saved on average (Jarvis 2002). It can be argued that stop smoking services are the most effective way that the NHS spends money (Stapleton 2001). Unfortunately, these services are undergoing a decrease in the number of users. 2015 was the third consecutive year where there had been a decrease in the number of smokers setting a quit date through the stop smoking services (Kmietowicz 2015). This may be due to the increasing number of individuals using electronic cigarettes as an aid to quitting without the use of stop smoking services as they are seen as a safer method of nicotine delivery by users (Hajek 2014). Despite this, there are still some concerns regarding their safety (Furber 2015). We should therefore consider e-cigarettes to be an interim to complete cessation and that e-cigarettes in combination with stop smoking services offer the greatest chance of quitting permanently (Fulton 2016). Long term use of electronic cigarettes may still have risks, but can be considered a harm reduction strategy (Polosa 2013).

Conclusions

Despite all the support that stop smoking services offer, it is those individuals who have high initial baseline motivation to quit who are usually successful (Kotz 2013). As smokers have varying levels of motivation towards cessation (for example, their financial situation may force them to consider quitting), healthcare professionals must ensure they are always proactive in offering cessation advice to smokers to ensure the highest possible impact on potential quitters (Wiltshire 2003). Using new technology, such as smartphone apps, can help maintain motivation and deliver cessation messages to individuals between appointments with the stop smoking services. As a new technology, we must accept that these have limitations and improvements to content and adherence are still to be made (Abroms 2013).

The Smoking Cessation Change Model

Introduction

Behavioural support is an important part of any smoking cessation attempt and should be recommended to all individuals who intend to stop smoking (Raw 2010). Utilising tobacco is a learned behaviour which causes a physical addiction in most of those who engage in its usage (RCP 2000). In the UK, evidence shows the stop smoking services are effective in increasing successful cessation and engaging with individuals from more disadvantaged backgrounds (McNeil 2005). Unfortunately, research shows that there has been no improvement in efficacy of behavioural support in both individual and group interventions within the last 20 years (Lancaster 2005, Stead 2005).

Smoking cessation can be achieved in two ways; with commitment to complete cessation on a targeted 'quit date,' or with gradual reduction in the amount of tobacco used. Both of these approaches result in comparable cessation success (Taylor 2005, Fiore 2008). A major challenge to success is maintaining self-motivation alongside lack of support and education for 'self-quitters,' (Uppal 2013). This results in a markedly low percentage of successful cessation. If we consider that 40% of nicotine users make a cessation attempt annually, but only 4-6% manage to maintain abstinence, a mere 2% of nicotine users reach full cessation per annum (CDCP 1998, Cohen 1989).

A misperception made by many is that willpower alone is enough to fuel a quit attempt, something which is refuted by nicotine addicts and academics in equal measure (Roddy 2006, Hughes 1999). One third of successful cessation attempts necessitate either medical or psychological therapy to result in a successful outcome (Hughes 2001). Even though smokers should always have brief cessation advice given to them by medical professionals, some choose to reject the conventional smoking cessation model, despite the knowledge there may be an increase in successful cessation (Morphett 2015). These reasons are often difficult to quantify and are more complex than misconceptions or correctable barriers to treatment (Smith 2015). With these individuals, practitioners must simply be supportive and understanding of the patient's wishes and understand that utilisation of the '5A's,' approach of brief cessation advice can offer some benefit (Cornuz 2008). For individuals who are amenable to cessation assistance, a 'stages of change,' approach can be of benefit (Zimmerman 2000). The suggestion that smoking cessation could be split into five different stages was first described by Prochaska and DiClemente (1983).

5 Stages of Change Model

Pre-contemplation

This is the stage where an individual has no intention to alter their habit in the near term, which is normally deemed to be within the next six months (Prochaska 2013). In this stage, individuals are often defensive, avoiding thinking or talking about their high risk behaviour. They may not be considering change because they are demoralised at previous quit attempts, do not understand the consequences of their actions or believe the consequences are insignificant (Levesque 2006). Simply put, at this stage the pros of smoking are outweighing the cons of smoking for the individual. To progress from this stage, an individual must accept their actions are problematic and potential harmful to themselves and others (Lenio 2006)

In order to transition individuals from the pre-contemplation to contemplation stage, education in the form of 'consciousness raising,' 'dramatic relief,' and 'environmental re-evaluation,' have been shown to be most effective.

- Consciousness raising is where the individual raises their own awareness about potential negative consequences (Prochaska 1992). Awareness can be increased through education, confrontation, feedback or media campaigns (Velicer 1998).
- Dramatic relief involves the individual expressing their emotions regarding their behaviour (Prochaska 1992). A dramatic event, such as the death of a family member from the at risk behaviour, is particularly effective at producing this form of transition (Patten 2000). Techniques such as roleplay can also be used to elicit a similar emotional response, but may be weaker (Tseng 2003).
- Environmental re-evaluation is how an individual may view their problem behaviour within their social environment (Patten 2000). It may be awareness of how their activity impacts on their position as a role model, for example to their children (Prochaska 2007). To elicit this response, a whole family intervention may be staged opposed to an intervention focussing on the individual performing the problem behaviour (Prochaska 2005).

Contemplation

In this state the individual intends to change their 'at risk,' behaviour within the next 6 months (Moore 2010). Whilst in this stage, the individuals are becoming more disillusioned with their current actions, and how it may be detrimental to their health. Whilst in this stage, the individual will be aware of the inherent risks of their actions, but is ultimately unwilling to change (Patten 2000). Whilst in this stage, the individual is in a state of flux, weighing up the pros and cons. Whilst the individual intends to change their habits, often they

5 Stages of Change Model

remain in a chronic contemplation phase as the force of motivation may not be great enough to enact change (Söderlund 2010).

To transition individuals from the contemplation to preparation stage of change, the most effective intervention is in the form of 'self-re-evaluation.'

- Self-re-evaluation is where the individual combines cognitive and affective assesments of their behaviour, comparing potential outcomes both with and without change. Techniques such as imagery can be used to allow the individual to identify a healthy role model, aspiring to be like them (Prochaska 2010).

Preparation

In this stage, the individual plans to make the change away from their harmful behaviour within one month (Patten 2000). This stage of change usually consists of individuals who have attempted to change from their 'at risk' behaviour in the last 12 months, but have relapsed and are still engaging in the activity (Patten 2000). The individual must decide on a strategy which results in the desired outcome. As many in this stage have attempted to change their behaviour once (or more times) before, they will often adopt a different method of progressing into the action phase (Velicer 1998).

To transition individuals from preparation to the action stage of change, the most useful intervention is 'self-liberation.'

- Self-liberation Is when the individual truly believes that they are committed, and prepared to take action, to change their behaviours (Patten 2000). Successful self-liberation strategies must have two options for the individual; they can continue to smoke if they wish to, however they are aware cessation is a better option for their health (Siqueira 2001). An individual who is committed to self-liberation will action upon to their decision to end their problem behaviour.

Action

In this stage, the individual has made the move to alter their harmful behaviour within the last 6 months (Patten 2000). During this stage the individual will both receive the greatest plaudits for their efforts from others, but also find this stage the most difficult due to withdrawal symptoms (Hughes 1986). The individual must remain in the action state until they move towards a state of 'self-efficacy,' and this is stronger than their temptation to revert to the problem behaviour.

To transition individuals through this final stage of change 'contingency management,' 'helping relationships,' 'counterconditioning,' and 'stimulus control,' are the most effective methods to prevent any relapse.

- Contingency management can be used to either reward the individual for continuing to remain abstinent from the problem behaviour, or to threaten punishment for relapse (Higgins 1999). Reward is for continued abstinence is preferable than any form of punishment. The individual is able to self-reward if they remain abstinent, or if working within a group can receive praise from their peers (Prochaska 1992).
- Helping relationships involve open, frank and trusting discussions between the individual and those who are helping them (Patten 2000). Such support can come from friends, family, self-help groups or phone services (Humphreys 1999, Orleans 1991).
- Stimulus control involves removing any cues which promote the risky behaviour, and adds prompts for healthy alternatives (Zimmerman 2000). For smokers, this may be being in a social situation with other smokers, particularly in combination with alcohol; this is particularly a cue in men (Ferguson 2015)

Maintenance

This stage of smoking cessation can simply be defined as that the individual has not engaged in their harmful behaviour for over 6 months (USDHSS 1989). Continued cessation focusses on the individual's satisfaction on cessation (Rothman 2000). If temptation to smoke is too great, or the pros of smoking begin to be outweighed, the individual will relapse from the maintenance state. It is shown that a majority of relapses from maintenance state happen in the presence of other smokers and to be successful individuals tend to avoid social situations with other smokers (Venters 1987, Foss 1973). Those who never smoke again remain in the maintenance stage; for an addict there is always a potential of relapse.

Relapse

Whilst not one of the five stages in the change model, it is important to note that not every individual manages to reach the maintenance stage, and those that do may not remain there. All individuals in either of these categories are said to have 'relapsed.' Those that relapse tend to be individuals who do not have good self-belief or conviction (Bandura 1982). To reverse relapse, it is important for the individual to remember why they quit in the first place. Relapses are a normal part of the change process and those that have relapsed should be reassured as such (Moore 2010). An individual may relapse 30 times during their attempt to stop smoking, for example (Chaiton 2016).

5 Stages of Change Model

Stage of Change	Patient's View	Interventions to move to next stage of change	Dental team involvement
Pre-Contemplation	No intention of quitting	'Consciousness Raising'	If patient wants to discuss media campaigns.
		'Dramatic Relief'	If patient wants to discuss media campaigns, or is amenable to receiving a stop smoking leaflet.
		'Environmental Re-evaluation'	May damage dentist-patient relationship.
Contemplation	Intending to quit in next 6 months	'Self-re-evaluation'	If patient wants to discuss reduction of oral and general health risks following cessation.
Preparation	Intending to quit in 30 days	'Self-liberation'	Encouragement and reassurance of patient's decision and health benefits.
Action	Has quit in last 6 months	'Contingency Management'	Suggest self-reward for patient quitting ('treating' themselves), and offer open praise for decision.
		'Helping relationships'	Offer support or reassurance based on patient's questions regarding smoking cessation.
		'Counter-conditioning'	Difficult to engage with, but can recommend referral if patient enquires.
		'Stimulus-control'	Difficult to engage with, but dentists can keep any treatment stress-free, avoiding creation of possible prompts associated with smoking.
Maintenance	Has quit for over 6 months	N/A	Congratulate and re-affirm decision.
Relapse	Has started smoking following a quit attempt	Identify where patient's location on 'stages of change,' model.	Remain non-judgemental. Reassure of normality of this - smokers can relapse 30+ times during cessation attempts! Engage in cessation attempts when patient is ready.

The Use of the 5 A's Protocol

The 5A's approach to smoking cessation is the internationally accepted approach to brief intervention in nicotine users (Dawson 2014). This 'brief,' advice in primary care settings has been shown to increase smoking cessation by 1-3% (Stead 2008). Before we discuss the stages of the 5A's, it is important to understand the local guidelines, adapted from WHO guidance, which this intervention in the UK is based upon:

NICE guidance - Gold standard guidelines for smoking cessation are issued by NICE.

The following is a summary of the key recommendations:

1. Everyone who smokes should be advised to quit, unless there are exceptional circumstances. People who are not ready to quit should be asked to consider the possibility and encouraged to seek help in the future. If an individual who smokes presents with a smoking-related disease, the cessation advice may be linked to their medical condition.
2. People who smoke should be asked how interested they are in quitting. Advice to stop smoking should be sensitive to the individual's preferences, needs and circumstances.
3. All other health professionals, such as hospital clinicians, pharmacists and dentists, should refer people who smoke to an intensive support service (for example, NHS Stop Smoking Services). Where possible, the smoking status of those who are not ready to stop should be recorded in clinical records and reviewed with the individual once a year, where possible.

These guidelines can be summarised by the '5 A's':

Ask about and record smoking status.

Advise smokers of the benefit of stopping in a personalised and appropriate way.

Assess motivation to quit (using stages of change model).

Assist smokers in their quit attempt.

Arrange follow up.

Healthcare practitioners often state that time constraints and lack of expertise are the biggest barriers in providing cessation advice (Richmond 2005). However, advice from the healthcare practitioner does not have to be focussed on the minutiae of cessation and needs to only last

The Use of the 5 A's Protocol

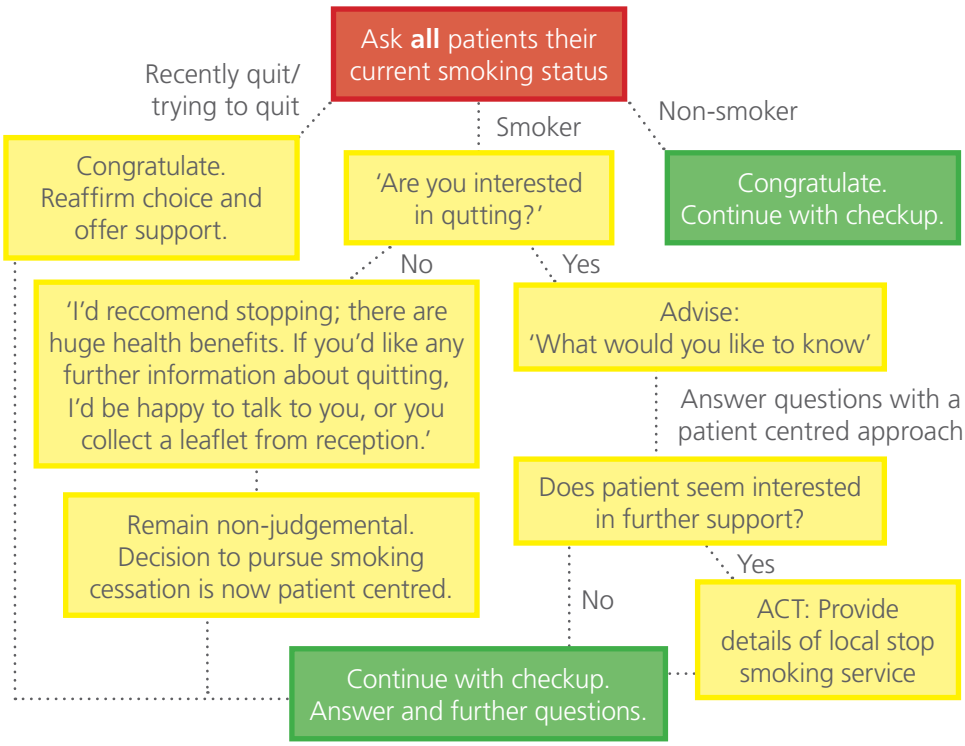
three minutes (Al-Doghether 2004). Whilst knowledge of the treatment modalities to result in cessation are useful, ultimately the specialised stop smoking services will be able to adequately discuss any potential issues with the individual. For practitioners who genuinely do not have time, a '3A's' approach may be acceptable. This can be summarised as follows:

Ask and record smoking status

Advise patient of personal health benefits

Act on patient's response

This very brief advice can be delivered in less than one minute(Aveyard 2009). There is an absence of conclusive evidence demonstrating the efficacy of 5A's intervention over the 3A's intervention, meaning either intervention is acceptable (Dawson 2014). As the 5A's intervention addresses WHO guidance more thoroughly, we should accept this brief intervention as the gold standard. However, we are aware of the need to be pragmatic in the approach to smoking cessation in the healthcare setting. 3A's intervention is better than no intervention and it is easier to complete and if done properly can still lead to cessation rate similar to that of the 5A's. An example of how healthcare professionals should interact with a smoking patient is shown below.



Withdrawal Symptoms and Withdrawal Orientated Approach

Cessation treatment based on controlling withdrawal symptoms assumes that individuals attempting to stop nicotine use are dependent on nicotine, and that the discomfort of withdrawal is a major issue in successful cessation (Russel 1984). Withdrawal orientated approach to smoking cessation therefore is therefore focussed on the use of nicotine replacement to reduce withdrawal discomfort and utilising group therapy to ensure abstinence through the period of greatest withdrawal (Hajek 1989). Withdrawal orientated approach to cessation can therefore be seen as an adjunct to other methods of smoking cessation, particularly when issues arise in self-efficacy and quitters become tempted to relapse (Hajek 1994). As an example, an individual who is in the maintenance stage of change may be tempted to smoke due to their social situation and recent cessation. Withdrawal orientated approach utilises nicotine replacement and behaviour management to control these urges.

Potential aids to cessation have been discussed in a different module but include (Chauhan 2016):

Inhaler

Nasal spray

Lozenges

Adhesive patch

Chewing gum

Electronic cigarette

These interventions can be remembered through the mnemonic **INLACE**.

The use of group behavioural therapy in the withdrawal orientated approach is discussed by Hajek (1989), and focusses on the following:

- A group structure is used because it allows individuals in similar situations to get to know each other, interact and discuss their personal experiences within a relevant forum. This also removes the focus from the cessation therapist to other members of the group, which removes any aspect of didacticism from the intervention. This shift in focus has been shown to reduce drop out from behavioural management in individuals attempting to quit (Hajek 1985).

Withdrawal Symptoms and Withdrawal Orientated Approach

- Running this intervention over a period of four weeks means that individuals receive the treatment they require during the acute withdrawal period (West 1987). Whilst some individuals would appreciate a longer course, this is not associated with any increase in successful cessation.
- Dissemination of withdrawal symptoms ensures individuals know what to expect during their cessation attempt, and that what they are experiencing is normal (West 1986). By normalising and discussing these symptoms, the intention is to influence those with more severe withdrawal symptoms (who are in turn are more likely to relapse) that their withdrawal is, in fact, manageable (West 1989).
- Group size is paramount to this approach. 12-15 members has been shown to be the optimum number (Hajek 1986). In this size group, there are sufficient numbers to create a 'bandwagon,' effect and boost morale, whilst being unaffected by a small number of dropouts. In a smaller group, dropouts are more noticeable and a larger group may impede on individual involvement and creation of interpersonal ties within the group.
- Intervention in the shape of educational material is limited. Individuals who have reached this stage are assumed to have a certain amount of cessation knowledge. By focussing brief education towards the end of the group of sessions it simply gives the individuals an educational boost, when novelty and drive to remain abstinent may be beginning to wane.
- Re-entry into such a programme is also limited to increase success rates. It has been shown that if individuals are told they can only enrol on such a scheme once a year they are more likely to be more determined to use the opportunity available to them and prevents populating the groups with individuals who repeatedly fail, which may result in unbalancing the group dynamic.

Withdrawal Symptoms and Withdrawal Orientated Approach

As healthcare practitioners, it is also important we understand and can identify withdrawal symptoms. These symptoms are strongest in the first few days, and dissipate over 4 weeks (Hughes 2007), which is concurrent with the length of time the withdrawal orientated approach is used. The most common withdrawal symptoms are (Hughes 1991):

Restlessness

Anxiety

Difficulty concentrating

Irritability/Anger

Sleep disturbance

Hunger or weight gain

These symptoms can be remembered through the mnemonic **RADISH**.

Communication and Counselling Skills

Effective communication during smoking cessation is one of the most important facets in encouraging reduction of potential harmful behaviours (Pommerenke 1992). Various modalities of communication can be utilised in the cessation pathway; didactic presentations, video demonstration, practice exercises and case studies in both individual and group sessions (Brunette 2016). Telephone based interventions have also been shown to be beneficial compared to a self-quitting approach (Stead 2006).

Individual counselling involves face-to-face appointments with a trained cessation therapist (Wu 2016). Intervention can vary in intensity, however there is no evidence that a more intense intervention actually increases cessation success (Lancaster 2005). Motivational interviewing is an integral part of this approach and is a patient-centred approach which is designed to modify the patient's approach to cessation (Hall 2004). The individual approach tends to include sessions over several weeks, with increased number and longer sessions shown to be more effective (Lai 2010).

Group counselling is shown to be helpful in cessation attempts compared to other less intensive interventions (Stead 2001). Group interventions can include skills training, mood management components and manipulation of group dynamics (Stead 2005). Group therapy allows interaction and peer support between users and is often combined with nicotine replacement therapy (Roberts 2013).

Telephone cessation advice appears to be dose dependent, with increased telephone intervention increasing the chance of successful cessation (Stead 2006). These phone lines may offer information, recorded messages or personal counselling (Anderson 2007). Telephone intervention is useful for smokers considering quitting and individuals who have just quit (Brandon 2000). It also provides intervention for individuals who may not have sufficient time or resources to attend group or individual sessions with stop smoking services (Zhu 2002).

When assisting with a quit attempt it is important to stress likely difficulties, for example dealing with withdrawal symptoms (Rigotti 2013). However, one of the most important things to stress to individuals who have recently quit or are going to quit is the 'not a puff' rule (Vangeli 2010). If an individual breaks a particular self-imposed rule even once, there is a tendency to abandon the behavioural aim (Schwarzer 2008). A significant number of quitters will smoke in the first few days of quitting. Those that lapse once are very unlikely to recover and remain abstinent (Brown 2005). It would appear that there is a strong link between alcohol consumption and lapse in cessation, meaning cessation advice for individuals should include advice regarding the avoidance of alcohol consumption (McKee 2006).

Smoking in Pregnancy

Risks

Men and women who smoke are less likely to conceive. Smoking reduces sperm count and motility in men and hormonal imbalance in women, reducing pregnancy rates in smoking couples (Zinaman 2000, Augood 1998). Additionally, 125,000 spontaneous miscarriages occur each year in the United Kingdom, with one fifth of these occurring in women who smoke (Jurkovic 2013). This makes smoking during pregnancy the single most preventable cause of foetal death alongside causing low birth weight and increased perinatal mortality (Foy 1988). Nicotine can freely cross the placenta along with toxic constituents of tobacco smoke, for example carbon monoxide (Harris 1996). Second-hand smoke can also result in reduced birthweight (Hegaard 2006).

Smoking damages the foetus in such a way that can affect a child through infancy, meaning infant sudden death syndrome is increased in the children of smokers. One estimate is that 24% of infant killed by sudden death syndrome are a result of mothers smoking during pregnancy (Pollack 2001). There are also links between smoking during pregnancy and intellectual and behavioural deficits through childhood and into adulthood (Faden 2000). Although the risks from smoking to the child appear to be dose dependent, light smokers still put their child at significant risk.

It should therefore constantly be reiterated by healthcare professionals to women who continue to smoke during pregnancy; smoking cessation is the best thing for both the mother and the baby through pregnancy and beyond. Cessation should occur prior to conceiving for the best possible outcome for the child (Das 1998).

Prevalence

In 2011, 11% of mothers were recorded as smoking at the time of delivery in 2015, with a range in all areas from 2% to 26% (HSCIC 2016). This is a reduction from 27% which was recorded in 1997 and had remained stationary at this figure for five years (Owen 1998). Most women who become pregnant and fail to quit smoking during the first trimester will not succeed in quitting through to term (Owen 1998). The prevalence of smoking through pregnancy is higher in those from lower socio-economic backgrounds, lower educational levels, those who started younger and those who have partners who smoker (West 2002).

Conversley, partners who are openly pro-cessation to the pregnant mother are more likely to be successful in succeeding to encourage complete cessation (Lindqvist 2001).

Worryingly, there appears to a perception amongst low income, teenage smokers that by smoking, and reducing the birth weight of the foetus, they will reduce the pain of delivery (Lawson 1994). Finally, we should note that, of the pregnant women who do manage to quit, 63% relapse within 6 months of birth (Mullen 1990).

Interventions

In the UK, the most effective intervention for reducing smoking in pregnant women has been increasing the price of cigarettes through taxation (Meier 1997). However, this has a downside; individuals from low socioeconomic backgrounds can be driven into poverty as they are unable to quit. It can therefore be argued taxing cigarettes is a tax on the poor (Bader 2011).

The most effective intervention in pregnant women centres around 'behavioural support' (Lumley 2002). This is similar to classical smoking cessation advice given and can involve individual, group and telephone intervention from stop smoking councillors. All these are effective at promoting smoking cessation in pregnant women, however they are underused (Taylor 2001).

Nicotine replacement therapies (NRTs) can also be used in assisting smoking cessation, however there is discussion as to whether these are completely safe or a harm reduction strategy. As previously stated, nicotine can cross the placenta and may affect the developing foetus. However studies have shown no adverse effects to the child from wearing a nicotine patch for up to four days (Ogburn 1999). The NHS currently advises that licenced NRTs are safe for pregnant women to use during pregnancy.

Electronic cigarettes are the newest smoking cessation aid which healthcare professionals must give advice about. Unfortunately, confusion and misconceptions about electronic cigarettes may prompt women who are pregnant to utilise an electronic cigarette without knowing all potential risks (Baeza-Loya 2014). It must be stressed that electronic cigarettes during pregnancy do not remove all risks and does still expose the unborn baby to nicotine and other inhalants from the electronic cigarette liquid. Some ingredients in the electronic cigarette liquid are toxic and liquid production is not regulated (Lerner 2015). Electronic cigarettes should therefore only be viewed as a risk reduction strategy, used in isolation with pregnant women who are unable to abstain from nicotine use due to addiction.

Smoking and Health

Smokers are often aware of health risks, but are often misinformed of the potential health consequences of their habit (Cummings 2004). There are also risks to those in close proximity to smokers through second-hand smoke; this includes exhaled smoke from the smoker and smoke from a lighted cigarette (Besaratinia 2008). Smokers should therefore be made aware that smoking in all forms carries severe health risks (even 'light' cigarettes) and there are risks to others who share space with them (for example their children). Such information can be used to encourage behavioural change within smoking individuals.

Smoking and Health

General health effects

Smoking most commonly causes lung cancer, with 90% of diagnoses being in smokers (Cancer Research UK 2010) and Chronic Obstructive Pulmonary Disease, with 80% of cases being diagnosed in smokers (NCGC 2010). Passive smoking also has been shown to increase diagnoses of Pneumonia and Tuberculosis, as well as asthma in young people (WHO 2014, HPACI 2010, Cabana 2005). Smoking has also been implicated in the following diseases (ACSH 2003):

Heart and circulation:

Angina, Buerger's Disease (severe circulatory disease), Peripheral and cerebro-vascular disease, stroke.

Respiratory:

Asthma, Chronic Bronchitis, Common cold, Chronic rhinitis (inflammation of nose), Influenza, Tuberculosis.

Stomach/digestive system:

Colon polyps, Crohn's disease (chronic inflamed bowel), ulcers, gastro-oesophageal reflux.

Ligaments, muscles and bones:

Ligament, tendon and muscle injuries, Neck and back pain, Osteoporosis (in both sexes), Rheumatoid arthritis (in heavy smokers)

Eyes:

Cataract, Macular degeneration, Nystagmus (abnormal eye movements), Optic neuropathy (loss of vision), Ocular histoplasmosis (fungal eye infection), Tobacco Amblyopia (loss of vision), Diabetic retinopathy, Optic neuritis.

Skin:

Psoriasis, Skin wrinkling, discolouration.

Reproductive functions:

Female fertility (30% lower), Menopause (onset 1.74 years earlier on average), HPV. Male fertility (Impotence, Reduced sperm count and motility). Increased miscarriage, low weight and pre-term birth. Cot death increased 25%

Other:

Depression, Hearing loss, Multiple sclerosis, Type 2 Diabetes.

What are the oral risks of smoking?

Oral Cancer

There is a great deal of evidence citing smoking as the main causative agent in oral cancer. Indeed, smokers have a three times greater chance of developing oral cancer, as shown by a meta-analysis of 254 publications reporting a relative risk 3.43 for oral cancer among current tobacco smokers compared with non-smokers (Gandini 2008).

Conditions commonly defined as cancer of the oral cavity comprise of those classified in the 'International Classification of Diseases' (IDC) as cancers of the lip, tongue, mouth and oropharynx (ICD10 codes: C00-08). The most common sites affected by cancer are the tongue, gingivae, floor of mouth and lip (NCIIDRG 2004). It has been identified that smokers often drink higher quantities of alcohol and the co-causative factor of alcohol and smoking in oral and pharyngeal cancer should not be overlooked. Whilst alcohol consumption has been linked with oral and pharyngeal cancer, it is the synergistic effect of alcohol with smoking that significantly increases the risks of such neoplasms (Bagnardi 2001).

In Europe, oral cancer accounts for 0.7% of all deaths from cancer. It is the 15th most common cancer with 61,400 diagnoses and 23,600 deaths across Europe in 2012 (Ferlay 2015). Of these diagnoses and deaths 42,600 and 17,600 respectively were reported in male patients.15 There was a 11% increase in 5-year survival following an oral cancer diagnosis from 2003 to 2013 when compared to 1993 to 2003. In the 2003-2013 group, patients typically presented older, with more advanced disease and more distant metastases. The reasons for increased survival therefore remain unclear, though likely involves improvement in surgical and medical therapies (Amit 2013).

Four years after stopping smoking, the risk of oral cancer is 35% lower than a 'current smoker.' It can take another 15 years for this to reach the level of someone who has never smoked (Marron 2010). In addition a meta-analysis based on nine studies, reported a non-significant risk among former smokers when compared with non-smokers (Gandini et al. 2008).

Oral Erythroplakia/Leukoplakia

They are both part of the group "potential malignant disorders" and both can present with dysplastic features.

Leukoplakia are defined as a 'white plaque of questionable risk having excluded known diseases or disorders that carry no increased risk of cancer' (Warnakulasuriya 2007). They are benign in 80%, dysplastic in 12%, carcinoma in situ (CIS) in 3%, and invasive carcinomas in 5% of cases.

Erythroplakia are defined as "A fiery red patch that cannot be characterized clinically or pathologically as any other definable disease". (Yardimici 2014). These present as carcinoma in 51% of cases, severe dysplasia or CIS in 40% of cases, and mild to moderate dysplasia in 9% of cases. When a single lesion contains both red and white patches, it is referred to as "erythroleukoplakia" (Yardimici 2014). These typically show the highest rates of advanced dysplasia on biopsy. It is important to understand the cellular changes present in dysplasia. Overleaf is a chart outlining histological changes present in mild, moderate, severe dysplasia and carcinoma-in-situ (Speight 2007).

Grade	Levels involved	Cytological changes	Architectural changes
Hyperplasia	N/A	None	Thickened epithelium Hyperkeratosis Normal maturation
Mild (I)	Lower third	Cell and nuclear pleomorphism	Basal cell hyperplasia
		Nuclear hyperchromatism	
Moderate (II)	Up to middle third	Cell and nuclear pleomorphism	Loss of polarity
		Anisocytosis and anisonucleosis	Disordered maturation from basal to squamous cell
		Nuclear hyperchromatism	Increased cellular density
		Increased and abnormal mitotic figures	Basal cell hyperplasia
			Bulbous drop shaped rete pegs
Severe (III)	Up to the middle third	Cell and nuclear pleomorphism	Disordered maturation from basal to squamous cell
		Anisocytosis and anisonucleosis	Increased cellular density
		Nuclear hyperchromatism	Basal cell hyperplasia
		Increased and abnormal mitotic figures	Dyskeratosis (premature keratinization and keratin pearls deep in epithelium)
		Enlarged nuclei and cells	Bulbous drop shaped rete pegs
		Hyperchromatic nuclei	Secondary extensions (nodules) on rete tips
		Increased number and size of nucleoli	Acantholysis
		Apoptotic bodies	
Carcinoma-in situ	Full thickness	All changes may be present	Top-to-bottom change
			Loss of stratification

(Speight 2007)

Oral Mucosal Conditions

Nicotinic Stomatitis: This typically presents as a greyish-white appearance of the palate, with a reddened, nodular appearance of inflamed minor salivary ducts throughout. The condition resolves following smoking cessation, however this resolution can take months to achieve. It is not considered potentially malignant.

Smoker's melanosis: 22% of smokers develop tobacco associated oral melanin deposits, with severity being dose dependent (Axell 1982). The condition can (but doesn't always) resolve following smoking cessation and has no long-term health consequences.

Oral Candidosis: Smokers have an increased risk of oral candidosis, but the mechanism is not fully understood (Soysa 2005). This could potentially be due to cigarette smoke which is nutritional for C. Albicans (the causative agent of candidal infection), or that the smoking results in localised epithelial alterations, resulting in candida colonisation (Arendorf 1980) Immunoglobulins, polymorphonuclear leukocytes and normal bacterial flora are important in preventing the proliferation of candida within the oral cavity (Samaranayake 1990). Smoking may have a role in supressing the oral immune response and disrupting normal bacterial flora. The condition can be treated with anti-fungal medications and typically causes no long-term consequences.

Smoking cessation and Recurrent Apthous Stomatitis: Recurrent Apthous Stomatitis (RAS), is one of the most common conditions to manifest within the oral cavity. It affects up to 20% of the population at some point in their lives and about 2% chronically. Most patients present with occasional ulceration which resolves rapidly without complications. Others have severe ulcers that interfere with diet and function. McRobbie et al 2004 showed that such ulcers are a common result of stopping smoking, affecting two in five quitters. Reassurance regarding oral aphtous ulcers should be given from primary care practitioners. In more severe or complex cases referral to an Oral Medicine Department may be indicated.

Periodontal Disease

There are clear, well documented links, between smoking and periodontal disease. Smokers have a 2 to 3-fold increased risk of clinically identifiable periodontitis. They also have fewer teeth and are more likely to be edentulous than non-smokers. The risk of alveolar bone loss is seven times greater amongst smokers than non-smokers, and the severity of periodontal disease in smokers appears to be dose dependent (Kinane 2000). There is evidence that smoking cessation reduces the risk of periodontal disease (Kinane 2000). Successful treatment of periodontal disease in smokers is lower than that in non-smokers (Kinane 2000). Increased prevalence of periodontal disease is linked to smoking dampening the body's immune response, meaning the smoker has a reduced ability to clear pathogens (Kinane 2000).

Smoking also increases the prevalence of acute necrotising ulcerative gingivitis and acts as a co-destructive factor for periodontal tissue damage alongside other predisposing factors (for example diabetes) (Kinane 2000). An example of an individual with chronic periodontal disease can be seen in figure C.

Wound Healing

Smoking is implicated in poor wound healing (Cardoso 2010). This is because smoking reduces blood flow to oral tissues alongside inhibiting the enzyme systems necessary in oxidative metabolism and oxygen transport.

The use of any tobacco product is associated with increasing risk of alveolar osteitis, or 'dry socket.' This has, in part, been related to the vasoconstrictive effects of nicotine on small blood vessels (Cardoso 2010). Abstaining from smoking following oral surgery/trauma has therefore been shown to reduce the risk of a 'dry socket' occurring.

Dental caries

Links have been established between smoking and dental caries, however a direct aetiological cause is missing. Smoking along with co-existing risk factors for caries can be associated with high caries rates.

Implants

Smoking is implicated in the failure of dental implants in all areas of the mouth (Strietzel 2007). Although there is a risk of dental implant failure in all patients, smoking can be considered a 'significant risk factor' in dental implant placement.

Aesthetics

All forms of smoking and chewing tobacco can discolour teeth, and smokers are more likely to perceive their teeth as discoloured compared to non-smokers (Alkhatib 2005). Smoking can also lead to increased incidence of tooth loss and has also been linked to increased skin ageing (Rexbye 2006).

Smoking also leads to an increased amount of calculus formation which can look unsightly and will normally require a dentist or hygienist to remove it.

Smoking products

Tobacco - What are the risks of use?

Smoking habits are deeply rooted in modern society and have been associated with an increasing number of diseases, including cancer. Cancer is a major cause of death worldwide, with lung, stomach, rectal, liver, oral and breast cancers being the most prevalent. Deaths from cancer worldwide are projected to continue to rise, with an estimated 12 million deaths in 2030.

According to data obtained by Richard Doll et al., half of regular smokers die as a result of their habit, a quarter of them in the age group between 25 and 69 years. Smokers lose an average of 10 years of life expectancy, which can be recovered if they stop smoking. Although smoking cessation has benefits at any age, the earlier smoking cessation occurs the greater the benefit and recovery on years of life. When initially suggested, a link between smoking and cancer was met with a degree of scepticism by the public and scientific community; testament to the marketing power of the tobacco industry at the time. However, with the presentation of a scientific report in 1964, relationship between smoking habits and some types of cancer, such as lung cancer, was confirmed (WHO 1992). Over time, more studies have been carried out on this subject, reporting some important data such as the risk of heart attack in people over sixty years of age, twice as high as in the non-smoker population and five times higher in people under fifty years of age (Edwards 2004).

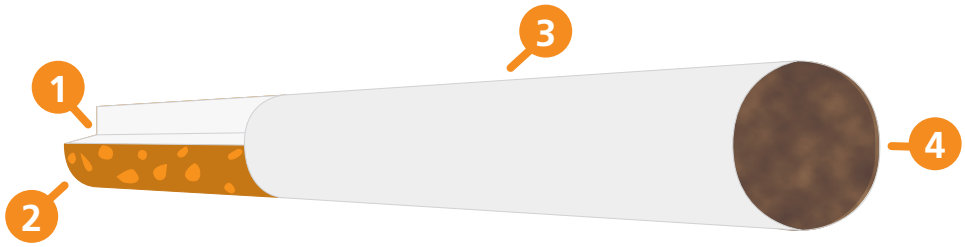
Health risks from tobacco use can be enhanced by the presence of other factors, such as alcohol consumption or occupational exposure to certain chemicals. We can identify more than 4500 substances in tobacco smoke, where about 81 of these have been identified as carcinogenic by the International Agency for Research on Cancer (IARC). These substances enter the body through the respiratory system, traversing through the blood, being later excreted, by the urine. As such, smoking increases the risk of cancer at multiple sites in the body beyond the lungs. These sites include the head and neck, bladder and kidneys, cervix, breast, pancreas, colon, among others.

Smoking products

Tobacco - A variety of choices!

Nowadays, the tobacco industry offers numerous options for smokers and several targeted products for younger users. Cigarettes, both unflavoured and flavoured, hookahs, pipes, rolling cigarettes, cigars and electronic cigarettes, offer a variety of choices to the consumer. For this reason, even with all the campaigns about the risks of smoking, there is normally an early age for first tobacco contact regardless of its type. This is usually between the ages of 13 and 14. Below is an overview of the types of tobacco commonly encountered;

'Common' Cigarette



Constituents of a common cigarette

1. Filter.
2. Tipping paper to cover the filter.
3. Rolling paper to cover the tobacco.
4. Tobacco blend.

The common cigarette consists of two essential elements, the filter and the paper:

Filter

At present, almost all cigarettes have filters, supposedly to minimize the absorption of toxic substances whilst smoking. The user has the false sense that he is protecting his health by receiving a lower dose of carcinogenic substances.

The filters are formed by holes. When the smoker sucks in the smoke during the 'draw' and compress the filter holes with his lips or fingers, they inhale nicotine and tar.

Smoking products

Paper

The paper is the tobacco 'shell.' Titanium oxide within the paper is responsible for the amount and density of the smoke produced, as well as the time the cigarette remains burning. During the act of smoking, the burn is faster to release a greater amount of nicotine; at rest, the burning is slower to increase the life of the cigarette. Titanium oxide can cause sneezing and coughing.

Menthol is used to disguise these side effects. It diminishes the cough reflex and disguises dry sensations in the throat (Santos).

Flavoured cigarettes

Flavoured cigarettes hold a strong attraction to the young population. The composition of these cigarettes is, in most aspects, the same as that of a common cigarette. The major difference is the inclusion of additives, which give the cigarettes a desirable flavour (Santos). It should be noted that in some countries, such as in Portugal, flavoured cigarettes are prohibited from being marketed.

Electronic Cigarettes

Electronic cigarettes produce inhalable vapour, with or without nicotine. It is a misconception to think that this alternative is completely harmless. Studies show that electronic cigarette vapour contains carcinogenic substances. These cigarettes have reduced the content of some substances, however, those that cause lung cancer, pulmonary emphysema, and other tobacco-related diseases remain present. The fact that these cigarettes may be lower in nicotine makes the smoker inhale even more to meet their perceived needs (Santos). There is a growing concern regarding individuals who have never consumed nicotine using electronic cigarettes as a gateway to actual smoking. They are perceived to be a desirable product to young people.

Smoking products

Rolling Cigarettes

Rolling cigarettes are potentially more harmful to health than classic cigarettes.

The smoker may choose not to, or not have access to, utilise a filter increasing the concentration of inhaled carcinogens. Even if a filter is placed, this is often not tightly adherent to the paper as in classic cigarettes, and allows increased numbers carcinogens to be inhaled. Secondly, their construction is not subject to the same regulations as classic cigarettes. There is no control over the tobacco utilised or how they are constructed by the user (Santos).

Shisha (Hookah/Water Pipe)

A hookah is a single or multi-stemmed instrument for vaporizing and smoking flavoured tobacco, popular with young adults. This form of consumption diffuses the smoke through an aqueous medium. Although less irritating to the mucosa of the airways, the amount of smoke inhaled can be as high as 10 litres an hour. When consuming a cigarette, intake does not reach even 0.5l/hr. In addition to the potentially toxic smoke, most people end up sharing this instrument. This increases the risk of transmission of infectious diseases such as herpes, tuberculosis or hepatitis.

Pipes/Cigar

Another widespread misconception that smoking pipes or cigars is significantly less harmful than smoking classic or rolled cigarettes. In some countries, such as the United States of America, there has been an increase in the use of cigars and pipes because of this. These tobacco containing products injure the lungs in the same way as other inhaled tobacco products, and due to the lack of filter can be extremely harmful to the lungs and oral cavity. It does not matter what the origin is, if tobacco smoke reaches the mucosa it can cause metastatic disease (Santos).

Tobacco - The Substances within a Cigarette

A cigarette contains about 4,000 substances with toxic and irritant effects. More than 70 of them are can be considered carcinogenic. Here are some examples:

- Nicotine, responsible for reducing blood supply to tissues and the central nervous system;
- Radioactive substances (such as Polonium 210 and Carbon 14);
- Heavy metals (such as lead and cadmium) that concentrate in the liver, kidneys and lungs;
- Carbon monoxide that takes the place of oxygen, leading to intoxication of the body;
- Tar (highly carcinogenic).

It is important to explore in more detail some of these constituents, to alert the general public of the potential harm of their inhalation:

Toxic gases

1. Carbon monoxide

Carbon monoxide is the same gas found in car exhaust fumes. It is a toxic, odourless gas, which has a high affinity for hemoglobin compared to oxygen. A person who smokes 20 cigarettes per day may have carbon monoxide levels in his or her body 5 to 10 times higher than normal when compared to a non-smoker. Carbon monoxide, when bound to hemoglobin, forms an element called carboxyhemoglobin, which hinders the oxygenation of tissues. Consequently, there is a reduction in the performance of the body for physical activity, making strenuous exercise and healing difficult.

2. Ammonia

The tobacco industry, noting that much of the cigarette's nicotine was not released during cigarette smoking, and utilised ammonia to overcome this problem. Ammonia is a chemical used in household cleaning and is corrosive to the nose and eyes. When attached to tobacco, it has two functions: it assists in the faster vaporization of nicotine during cigarette burning and facilitates its deposition within the lungs. As ammonia is deposited in the lung, this increases the likelihood of pulmonary emphysema and chronic bronchitis in the smoker.

Tobacco - The Substances within a Cigarette

This whole process aims to accelerate the path of nicotine into the brain, which leads to an almost immediate sense of well-being in the addicted user.

3. Toluene

Toluene is a toxic gas, once again present in the exhaust fumes from cars. It is used in the manufacture of rubbers, oils, resins, paints, glues, detergents. When inhaled, it is deposited in body fat, remaining for years. It causes central nervous system depression and long term, even with low levels of concentration in the human body, can cause headaches and loss of appetite.

4. Cyanide

Cyanide originates from the combustion of cigarettes, and is a recognised carcinogen. It is used in industry for the manufacture of fibers, plastics, paints, pesticides, and is also used as gas to kill rats. In World War II was used to murder prisoners in concentration camps. Inhaled in small amounts can cause such nefarious effects as dizziness, headache, nausea and vomiting.

5. Butane

A toxic, flammable gas that may be deadly. It is present in lighter fluid and also used as cooking gas. Inhalation causes problems such as breathing difficulties or visual changes.

6. Acetone

This is also a highly flammable chemical, with its primary use as a nail varnish remover. It is present in the smoke released by cigarettes. Inhalation in small quantities can cause irritation to the throat, dizziness and headaches. Ingestion of larger quantities can result in death.

7. Turpentine

This is a toxic substance obtained by extracting resin from pines. It is an oil paint thinner, also used for cleaning brushes. Inhalation causes eye irritation, dizziness and fainting.

Tobacco - The Substances within a Cigarette

8. Xylene

A flammable and carcinogenic chemical which can commonly be found in pen ink. When inhaled it can cause ocular irritation, dizziness, headache and even lead to loss of consciousness. Due to these risks to community health, the industries producing pens and derivatives are removing xylene from the composition of their products.

9. Levulinic acid

By adding levulinic acid to cigarettes, the strength of nicotine within cigarettes can be disguised, alongside reducing tar levels to keep them within legal limits. In addition, levulinic acid makes the upper respiratory system less sensitive to smoking.

Toxic Metals

1. Arsenic

Arsenic is a metal used in the manufacture of insect poisons, and because of this, many farmers use pesticides made of arsenic to combat insect pests. Arsenic can cause serious injuries to humans, affecting the liver, kidneys, heart, lungs, bones and teeth.

2. Cadmium

This is a heavy metal with toxic and carcinogenic power. It causes damage to the liver, kidneys, lungs and brain as well as can be a precursor of cancer at the lung, prostate, kidney and stomach. It is important to note that cadmium can remain in the human body for about 30 years.

3. Lead Acetate

This substance is present in the formula for hair dye, and can remain in the human body between 10 and 30 years. It causes anorexia and headaches, and has been implicated in lung cancer. Inhalation can also lead to shortness of breath and pulmonary emphysema. Inhalation or ingestion is very harmful to children or adolescents as it may alter their growth.

4. Phosphorus P4 P6

This chemical is present in fertilizers and cleaning products, as well as in rodenticides. It is extremely dangerous, and potentially fatal if ingested in a large enough quantity.

Tobacco - The Substances within a Cigarette

Carcinogenic substances present in cigarettes

1. Tar

Tar (in the context of cigarettes) is a set of solid, organic and inorganic particles which are absorbed by the smoker when a cigarette is lit. Among its compounds are 43 carcinogenic substances, such as Arsenic, Polonium 210, Carbon 14, DDT, Nickel, Lead, Benzopyrene and Cadmium. Most of these toxic substances are in the gaseous form and therefore not included in the 'components,' of the cigarette. It is also important to note that cigarettes with lower tar content are not any safer compared to non-'light' cigarettes, and still contain toxic and carcinogenic products. The tar present in smoking products causes stains on the teeth and fingers and, when deposited in the lungs, stains the organ a dark brown.

2. Polonium

This is a rare and radioactive element, with Polonium 210 being the most common form. Polonium produces extremely harmful radiation, called alpha-radiation. Several traces of polonium have been identified in cigarette smoke. These are deposited in the airways, emitting radiation to the cells around them. A smoker of 30 cigarettes per day is exposed to radiation equivalent to 300 x-rays to the chest over the course of 1 year.

3. Nickel

Inhaled nickel results in deposits forming in the liver, kidneys, heart, and lungs. Its inhalation triggers changes in the stomach and intestines, leading to an increase in metaplastic change. It is used in the production of stainless steel, alloys, coins and alkaline batteries.

4. Benzene

Benzene is used during the process of oil extraction and refinement as well as the manufacture of naphtha. It is also found in emissions from the burning of coal and oil, automobile exhaust pipes, and the evaporation of gasoline from service pumps using industrial solvents. It is considered a carcinogenic element and when absorbed within the lungs can cause long-term, irreversible damage. The chemical has also been implicated in causing leukaemia. Acute exposure to benzene may include eye and skin irritation, dizziness, nausea, headache, and irregular heartbeat.

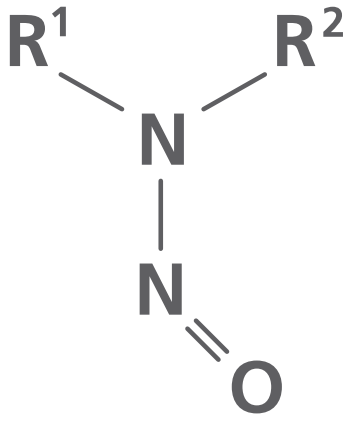
In women, prolonged exposure to benzene may lead to irregular menstrual patterns and decreased ovary size. Food or water contaminated with benzene may cause vomiting, irritation to the stomach lining and convulsions. As benzene is produced as a cigarette burns, there is a risk of second-hand inhalation of this harmful chemical by non-smokers. Non-smokers living with smokers will have a significantly greater exposure to benzene than a household of non-smokers.

5. N-Nitrosamines

Nitrosamines are produced from nitrites and amines, having a chemical structure R2N-N = O. Their formation occurs only under certain conditions, in particular a strongly acidic environment (which is present in cigarette smoke). N-nitrosamines have long been known to be organic compounds, but they have been the subject of further toxicological studies since 1956. Magee & Barnes reported the initiation of liver tumours in rats exposed to N-Nitrosodimethylamine. Since then, research has been done on animals, trying to assess the harmful effects these substances. Most of these compounds have been found to be carcinogenic with teratogenic and mutagenic action. Human exposure to nitrosamines may occur as a consequence of some habits, such as smoking. Other sources of note may be water, rubber, and cosmetic products.

Carcinogenic nitrosamines include, but are not limited to:

N-Nitrosodi-Butylamine, N-Nitrosodi-n-propylamine,
N-Nitrosoethylmethylamine, n-Nitrosodiethylethanolamide,
4- (N-Nitrosomethylamino) -1- (3-pyrindinyl) -1-butanone,
N-Nitrosonornicotine, N-Nitrosopiperidine and N-Nitrosopyrrolidine.
Below is a diagram of the chemical structure of nitrosamine.



Tobacco - The Substances within a Cigarette

6. Formaldehyde

A carcinogenic substance, used in the preservation of corpses and the manufacture of anti-bacterial compounds. It is also present in fertilizers and dyes. It causes respiratory diseases, as well as allergic reactions such as asthma, dizziness, decreased motor coordination, sore throat and disruption to sleeping patterns.

7. Acrolein

This gas has a strong smell and is present in large concentrations within a cigarette. It is responsible for the destruction of pulmonary cilia, which are essential in the defence of inhaled toxins. This is considered, alongside formaldehyde, to be the most carcinogenic product in cigarette smoker alongside formaldehyde.

Nicotine addiction

The majority of tobacco users continue their use because they are addicted to nicotine. By smoking, long term users modify their brain chemistry meaning it can be very difficult for individuals to stop (Hawkins 2004). Whilst nicotine plays a large part in individuals smoking, the process is undoubtedly multi-factorial. Pharmacology, genetics, learned habits and social/environmental factors all play their part (Carpenter 2007).

By smoking, users feel they are able to regulate their mood alongside their mental and physical abilities (Brandon 1994). We must also appreciate the psychological addiction that occurs in users of nicotine; these can include the rituals, feelings, or people associated with smoking (Crocq 2003). Combined, the physical and psychological addiction create a barrier to cessation which is too difficult for many to overcome.

Nicotinic Acetylcholine Receptors (nAChRs)

Nicotine distilled from smoking a cigarette travels from the mouth, to the lungs and finally to the brain, where it binds to nicotinic cholinergic receptors. This binding results in the uptake of sodium and calcium, resulting in neurotransmitter release. nAChRs are made up of five subunits, arranged symmetrically around the ion channel (Dani 2001).

The release of various neurotransmitters following nicotinic binding to the nAChRs plays a large part in the cycle of addiction associated with smoking.

Neurotransmitters

Dopamine: This neurotransmitter is linked with pleasurable experience and reward (Blood 2001). The release of dopamine in the nucleus accumbens is central to the addictive properties of smoking (Di Chiara 2002). Dopaminergic receptors in this part of the brain are central to drug induced reward (Dani 2001).

Glutamate: This is the major excitatory neurotransmitter within the mammalian brain, central to both memory and learning. Nicotine results in glutamate increasing dopamine release (Mansvelder 2000).

Gamma-Aminobutyric acid (GABA): This is the chief inhibitory neurotransmitter within the mammalian brain. Simply, it does the opposite of glutamate. By smoking, nicotine causes (initially an increased amount, but over the course of one hour) a reduced amount of GABA availability. This means dopamine released remains increased, and not inhibited by GABA (Mansvelder 2002).

Hypocretin 1 & 2: This neurotransmitter regulates wakefulness and appetite. Smoking causes attenuation of Hypocretin, increasing availability but also reducing the binding affinity of their receptors (Kane 2000). This promotes smoking behaviour; as there is reduced hypocretin uptake users can become tired and irritable if they do not replenish hypocretin levels.

Also of note is that products in cigarette smoke, such as acetaldehyde, can also increase the addictive nature of smoking. Condensation products of acetaldehyde reduce activity of monamine oxidases, responsible for the metabolism of neurotransmitters such as dopamine (Benowitz 2010). Inhibition on monamine oxidases therefore contributes to addictiveness by preventing metabolism of extra-neuronal dopamine (Fowler 2003).

Ultimately, prolonged smoking results in neuroadaptation. Withdrawal following prolonged exposure to nicotine results in an increase in the 'brain-reward threshold.' This demonstrates a central neuroadaption, and canexplainthe reduced perceived positiveperception towards pleasurable stimuli when a smoker first quits (Epping-Jordan 1998). Managing withdrawal is therefore paramount; fear of withdrawal can be enough to deter smokers from even attempting to quit.

Conditioned Stimuli (Psychological Addiction)

In experimental rats, nicotine's properties of reinforcement are considerably weaker when compared to other addictive substances. It is therefore hypothesised that habitual behaviours can only be developed in 'higher' animals, where more complex cognitive skills can be developed (Le Foll 2007). Indeed, it has been showed that smoking nicotine free cigarettes is almost as satisfying as their nicotine containing counterparts, simply due to the habitual enjoyment (Rose 2000), something that is not present in experimental rats. Repetition of smoking activity, for example with a certain friend or with an alcoholic drink, becomes a powerful cue for individuals to light up.

Genetics and Vulnerability

Studies in twins shows heritability of cigarette smoking (Lessove-Schlaggar 2008). Clearly this means that some individuals are simply more predisposed to engaging in smoking activity. Strong genetic associations with smoking activity have been discovered in relation to the genes within the α5/α3/β4 nicotinic cholinergic receptor gene complex on chromosome 15 (Saccone 2007). It is interesting to note that nicotinic cholinergic receptors are involved in the body's inflammatory response; alteration to the genetic makeup of these receptors could therefore increase susceptibility to diseases caused exclusively by nicotine as these receptors are activated and utilised more in smokers.

It is younger individuals who are more likely to initially engage in smoking behaviour; 80% of smokers are already doing so regularly by their 18th birthday (Lynch 1994). Two-thirds of young people will try a cigarette before their 18th birthday, with between a third and a half of these individuals continuing the habit regularly. There is evidence to show that early exposure to nicotine increases the severity of dependence (Dwyer 2009).

We must also acknowledge the strong links between smoking behaviours and mental illness (Lasser 2000). The association between smoking and feeling relaxed is particularly well understood by individuals who suffer from poor mental health, with these individuals smoking, to 'self-medicate,' their mental illness (Ratschen 2011, Khantzian 1997). Worryingly, individuals who are mentally healthy but smoke are increase their risk of developing mental illness (Cuijpers 2007).

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