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Tobacco smoking as a chronic disease: notes on prevention and treatment

Vikas Batra, MD[a], Ashwin A. Patkar, MD[b], Sandra Weibel, MD[a], Frank T. Leone, MD, MS, FCCP[a,c,*]

[a]*Division of Critical Care, Pulmonary, Allergic and Immunologic Diseases, Thomas Jefferson University, 1025 Walnut Street, 805 College Bldg, Philadelphia, PA 19107, USA*

[b]*Division of Substance Abuse Program, Thomas Jefferson University, 833 Chestnut Street, Suite 210, Philadelphia, PA 19107, USA*

[c]*Center for Tobacco Research and Treatment, Thomas Jefferson University, 1025 Walnut Street, 805 College Bldg, Philadelphia, PA 19107, USA*

Smoking is the leading preventable cause of illness and death in our society. Tobacco use is responsible for approximately 3 million deaths worldwide [1] with over 430,000 in the United States alone [2]. To put these figures in perspective, more lives are lost to tobacco than those caused by alcohol, recreational drugs, fires, automobile accidents, suicides, homicides and AIDS combined. Currently, more than one in five adult deaths in the United States is attributable to tobacco [2]. It has been projected that one in three adult deaths will be attributable to tobacco use worldwide by 2020 [3]. Despite the known health hazards of tobacco use, smoking remains highly prevalent, with an estimated 46.5 million adults, or nearly 24% of US adults over 18, currently smoking cigarettes [4]. The economic burden of tobacco related health care is enormous. An estimated $50 to $73 billion are spent on direct excess medical costs attributable to smoking annually, with an additional estimated $50 billion in indirect attributable costs [5,6]. However, these figures may grossly underestimate the total cost to the society, since they do not include loss of lives and property from smoking related fires, perinatal care for low birth weight infants of mothers who smoke, or medical care costs associated with diseases caused by second hand smoke.

* Corresponding author.
E-mail address: Frank.leone@mail.tju.edu (F.T. Leone).

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Smoking: a chronic disease

Smoking is not simply a bad habit that can be easily overcome by self-determination and will power. The physiologic basis for tobacco dependence is an addiction to nicotine, a critical constituent of tobacco smoke [7,8]. Nicotine is a colorless to pale yellow oily liquid with the formula $C_{10}H_{14}N_2$. Similar to other addictive substances like heroin and cocaine, it acts on the neural reward center in the limbic system of the brain [9,10]. It is well documented that as many as 90% of smokers identify tobacco as harmful and want to reduce or stop using it [11–13]. Unfortunately, less than 7% of those who try to quit on their own achieve more than one year of abstinence [14]. It is a testament to the power of nicotine addiction that millions of tobacco users are unable to overcome their nicotine dependence. Recognizing the addictiveness of nicotine, the American Psychiatric Association has included nicotine dependence in its Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) as a substance abuse disorder. Other factors that contribute to smoking besides nicotine addiction include its high level of availability, social acceptability and sophisticated marketing and advertising methods used by tobacco companies.

In many respects nicotine addiction is similar to a chronic disease [15]. Despite widespread experimentation and exposure, some people rapidly become nicotine dependent while others develop a pattern of occasional use, or discontinue use entirely. Individual differences in susceptibility to nicotine dependence and ability to quit smoking may suggest the presence of inheritable and/or acquired biological defects that then manifest as the tobacco use behavior. Only a minority of smokers are able to quit upon their initial quit attempt. The majority follows a course of multiple relapses and remissions and require sustained treatment rather than simple acute interventions. In this respect nicotine dependence is similar to hypertension, diabetes and asthma; chronic medical disorders requiring continuing care. Treatments of these illnesses are effective but heavily dependent on adherence to the medical regimen for effectiveness. Studies have shown that less than 60% of adults with type 1 diabetes and less than 40% of patients with hypertension and asthma fully adhere to their medications [16–18]. Adherence is even worse to behavioral and dietary changes that are so important in the management of these diseases. Not surprisingly, comparable to nicotine dependence, 30% to 50% of diabetics and 50% to 60% of patients with hypertension and asthma experience recurrence of symptoms every year that require additional medical care [19]. While we may expect hypertensive patients to be committed participants in their own care, we do not expect hypertension to be cured by self-determination alone. It may be similarly unfair to attribute failure to permanently quit smoking solely to a lack of will power and self-determination. The relapsing and remitting nature of nicotine dependence may be one reason that, despite the known health risks of smoking, a general desire among smokers to discontinue tobacco use,

and the availability of effective treatments, physicians often fail to address tobacco use consistently and effectively [20].

Development of nicotine addiction

Environmental and social factors

The process of becoming addicted to nicotine proceeds through several stages. The first step toward smoking initiation involves baseline *susceptibility*, defined as the absence of a firm decision not to smoke [21]. Next is *experimentation*, during which the person chooses to smoke a cigarette or takes a few puffs, ironically often as an expression of independence and self-determination. In some individuals continuing experimentation leads to a pattern of *regular use*, followed by progressive tolerance, the hallmark of *nicotine addiction*. Various studies have found that baseline susceptibility to smoking is the strongest independent predictor of experimentation [21–23]. Even infrequent experimentation in adolescence significantly increases the risk for adult smoking [24]. Among experimenters, susceptibility to smoking, having parents, friends or siblings who smoke, lower parental education level, and lack of parental concern about future smoking all predict continued smoking into adulthood [21,22,25,26].

Approximately 38% of middle school and 24% of high school students who currently use tobacco believe that cigarette smokers have more friends [27]. Thirty-four percent of all experimentation in adolescents between 1993 and 1996 has been attributed to tobacco promotional activities [28]. Furthermore, 86% of adolescent smokers buy the 3 most heavily advertised brands of cigarettes, compared to 46% of adult users [29]. These facts suggest that eminently modifiable social perceptions about cigarette use play a dramatic role in smoking initiation.

Genetic factors

Twin studies and animal studies

Although environmental and social factors are clearly important in the process of smoking uptake, several studies have highlighted the important role of genetic influences on various aspects of the smoking behavior [30]. Over 40 years ago, Fisher reported that concordance for smoking was higher in monozygotic (MZ) twins than in dizygotic (DZ) twin pairs [31,32]. Subsequent studies have also indicated that there is substantial genetic influence on smoking initiation, persistence of smoking, number of cigarettes smoked and ability to quit. In various studies heritability estimates (ie, proportion of phenotypic variation attributable to genetic variation) has ranged from 46% to 84% [33–40], comparable or higher than the heritability estimates for hypertension (0.25 to 0.50) [41,42], asthma (0.36 to 0.70) [43,44] or type I diabetes (0.33 to 0.55) [45,46]. Support for the notion of several

inheritable components of the smoking behavior comes from a series of experiments on inbred strains of animals. Members of an inbred strain are similar to MZ twins in that all members of a strain are genetically identical to all other members. Different strains of animals differ in their sensitivity to various and separate effects of nicotine, including tolerance to nicotine and sensitivity to aversive reactions like nicotine induced seizures [47–50].

Nicotine metabolism

In humans, 80% of nicotine is converted into the inactive metabolite cotinine by the enzyme CYP2A6 [51]. Two variant alleles of CYP2A6 have been identified (CYP2A6*2 and CYP2A6*3) and both are associated with reduced activity of the enzyme. Compared to a control population, the frequency of these alleles is lower in nicotine dependent subjects, [52] and individuals heterozygous for these alleles smoke fewer cigarettes, and are more likely to quit, than smokers with two normal alleles [53]. This suggests that impaired metabolism of nicotine may protect against becoming nicotine dependent. Another P450 enzyme, CYP2D6 is also involved in nicotine metabolism. Individuals are referred to as poor metabolizers (PM) or extensive metabolizers (EM) based on CYP2D6 enzyme activity. Smokers are less likely to have the PM genotype [54]. Interestingly, CYP2D6 status appears not to influence smoking initiation but predicts smoking behavior after initiation [55].

Dopaminergic neurotransmission

It has become apparent that nicotine increases dopaminergic neurotransmission in the limbic system of the brain, and that polymorphism of the genes involved in the metabolism of dopamine and dopaminergic neurotransmission have been linked to differences in behavioral phenotype [10]. Dopamine receptor D2 (DRD2) gene polymorphism and its association with smoking behavior has been a major area of interest. The DRD2 gene has two allelic variations: DRD2*A1 and DRD2*A2. DRD2*A1 has been associated with a reduced number of dopamine receptors [56]. A higher prevalence of the DRD2*A1 allele has been reported in smokers, compared to their ex-smoker and non-smoker counterparts [57,58]. There is an inverse relationship between the presence of a DRD2*A1 allele and the age of onset of smoking, as well as the maximum duration that a smoker has been able to quit on their own [57]. It has been suggested that individuals with DRD2*A1 allele have fewer dopamine receptors and require more nicotine to increase synaptic dopamine. However, some researchers have failed to find an association between DRD2 gene polymorphism and nicotine dependence [59].

Dopamine is removed from synapses by reuptake and metabolism. Dopamine reuptake is mediated through a transporter protein expressed by gene SLC6A3. Genetic variation in this transporter protein gene influences

amount of synaptic dopamine. Lerman et al found that smokers are less likely to have SLC6A3 gene allele associated with excess synaptic dopamine.

There are data that support several inheritable neurophysiologic circumstances that impact risk for nicotine dependence. In general, it may be said then that nicotine dependence, manifested most frequently as cigarette smoking, is a chronic disease impacted by a complex interaction between inheritable and environmental determinants, and not merely a bad habit. A fuller understanding of these genetic and environmental determinants of cigarette smoking will become helpful in clinical practice as we begin to stratify patients at high risk for nicotine dependence, and target emerging treatment schemes more effectively and precisely.

The prevention and treatment of nicotine dependence

Primary prevention

Findings from the National Youth Tobacco Survey and the state Youth Tobacco Survey show that current tobacco use ranges from 15.1% among middle school students to 34.5% among high school students [27]. Smoking prevalence among adolescents has risen dramatically since 1990, with more than 3000 additional children and adolescents becoming regular users of tobacco everyday [60–62]. Since approximately 80% of tobacco users start smoking before the age of eighteen, prevention of smoking initiation among youth is very important.

Many of the predictors of smoking uptake like genetic factors, socioeconomic status, family dynamics, etc are difficult to modify. One influence that can be modified is tobacco marketing. In 1998, leading US tobacco companies and the Attorneys General of 47 states entered into a settlement agreement, known as the Master Settlement Agreement (MSA), which sought to recover a portion of the states' expenses incurred because of tobacco use, and imposed restrictions on tobacco advertising, prohibiting advertising that targeted people younger than 18 years of age. A more recent analysis of trends in advertising expenditure for 15 specific brands of cigarettes and the exposure of young people to cigarette advertising in 38 magazines between 1995 and 2000 found that the tobacco industry did not significantly decrease advertising in magazines following the MSA, and exposure of young people to advertisements remained unchanged [63]. In addition to enforcement of laws that limit tobacco promotion, increasingly sophisticated counter-marketing activities promote smoking cessation and decrease the likelihood that adolescents will start smoking [64,65]. The type and target of the prevention message is very important. While advertising messages which raise concern over the long-term health effects of tobacco use can be effective in encouraging adult smokers to quit, they are generally ineffective at preventing experimentation among youth [66]. Strategies that emphasize industry manipulation (ie, tobacco industry uses deceitful, manipulative and

dishonest practices to hook new users, sell more cigarettes and make more money), or the effects of secondhand smoke (reminding people that, as smokers, their smoking endangers others), are the most effective prevention tactics. In addition, messages that emphasize the addictive properties of nicotine, and highlight the relative ease of youth access to cigarettes can also be effective [66].

Enforcement of tobacco-control policies is another method of reducing smoking in adolescents. On August 28, 1996, the Food and Drug Administration (FDA) issued regulations that prohibit the sale of tobacco products to persons aged <18 years, and require retailers to request photographic identification to verify the age of all persons aged <27 years [67]. Despite these federal regulations, approximately 70% of middle school and 60% of high school students who are current smokers report not being asked to show proof of age when they purchase cigarettes in convenience stores [27]. Of those who are asked for proof of age, approximately 65% are still sold tobacco products [27]. Enforcement of laws that restrict minors' access to tobacco is necessary to reduce smoking in youth.

Another effective public health intervention that reduces cigarette consumption and slows uptake by minors is an increase in the tobacco excise tax [68–71]. Research suggests that a 10% increase in cigarette price reduces the number of adolescents who smoke by 7%. However, most adolescents do not buy cigarettes in the experimentation phase. About 35% of middle school and 21% of high school children <18 years borrow cigarettes from someone [27]. Therefore, enforcement measures alone are not sufficient and they have to be combined with community wide education programs. Several studies have shown that enforceable public policy restricting access, combined with effective school-based tobacco prevention programs, can substantially reduce smoking prevalence [72–74]. School based anti-smoking programs that educate students regarding health consequences of smoking and teach refusal skills have been most effective [75].

Secondary prevention

The first step in treating tobacco use is to identify tobacco users. Simple interventions to identify smokers, like expanding the vital signs to include smoking status, can dramatically increase the rate at which clinicians intervene with their patients who smoke [76–79]. All physicians should advise their smoking patients to quit. Physician's advice to quit smoking increases abstinence rates approximately three-fold [80–83]. After the tobacco user is identified and advised to quit the physician should then assess willingness to quit. Patients unwilling to quit should be motivated to do so, generally by highlighting the relevance of tobacco use to their own personal condition, and expressing your concern about their continued health. Possible barriers to quitting cigarettes, such as lack of insight, poor social support, or limited access to treatment should be identified and addressed. Fear of, and

concerns about, quitting are common but often concealed. Physicians are in a particularly advantageous position to address these fears in a supportive way, minimizing reluctance to make a cessation attempt. Demoralization from previous failed quit attempts can be critical in a smoker's decision to continue smoking. Physicians can encourage their patients to try again by emphasizing the chronic disease aspects of tobacco use, highlighting the lessons learned during previous attempts, and offering ongoing support that is more problem focused and less outcome oriented.

Brief intervention promoting motivation to quit cigarettes should be built around the AHRQ's recommended "5R's": Relevance (Why is quitting cigarettes personally relevant to this patient?), Risks (What are the other possible harmful effects of smoking?), Rewards (What are the benefits this patient will derive from smoking cessation?), Roadblocks (Are there any obvious impediments or barriers to quitting that must first be addressed?), and Repetition (Why not try again?) [84].

Individuals willing to quit smoking should be assisted with every quit attempt. The most common, but least effective, method of smoking cessation is sudden, impulsive withdrawal of smoking, without any pharmacological support, also referred to as "cold turkey". The success rate with this method is low and the patient may experience considerable nicotine withdrawal symptoms. Almost all smokers trying to quit should receive pharmacotherapy. Exceptions include patients with absolute medical contraindications to therapy, and persons suspected of having very low level, or no nicotine dependence at all. Pharmacological approaches can be divided into two broad categories, nicotine replacement therapy and antidepressant therapy. Table 1 summarizes various pharmacological agents that can be used in smoking cessation.

Nicotine replacement therapy

Nicotine replacement therapy (NRT) provides an alternative source of nicotine to the smoker, and helps combat nicotine withdrawal symptoms during cessation. There are currently four formulations of nicotine available for replacement, the nicotine patch, gum, inhaler, and nasal spray. Using NRT increases the odds of smoking cessation 1.7 times over control and decreases severity of nicotine withdrawal symptoms [85–87]. When used properly, all forms of NRT are safe, and contrary to popular misconceptions, do not increase the risk of acute myocardial infarction even when used with cigarette smoking [88,89].

The transdermal nicotine patch delivers nicotine through skin at a relatively constant rate. In the Unites States, nicotine patches vary in strength from 7 to 22 mg/patch, and are worn for 16 to 24 hours daily. Adverse effects of nicotine patch are mild and rarely cause discontinuation of treatment. The most common side effect is local skin irritation, however some people experience significant sleep and dream disturbances, which can be

Table 1
Pharmacological agents for smoking cessation

Name	Dose	Side effects	Precautions	Availability
Nicotine patch	21 mg/24 hrs × 4 wks 14 mg/24 hrs × 2 wks 7 mg/24 hrs × 2 wks Start at a lower dose if smoking less than half pack/d of cigarettes	1. Local skin reaction 2. Insomnia	1. Pregnancy: FDA class C 2. Caution in patients with serious arrhythmias and worsening angina pectoris	Nicoderm CQ:OTC Nicotrol: OTC Generic: OTC
Nicotine gum	2–4 mg as needed for cravings (up to 24 pieces/d)	1. Mouth sourness 2. Hiccups 3. Dyspepsia 4. Jaw ache	1. Pregnancy: FDA class D 2. Caution in patients with serious arrhythmias and worsening angina pectoris	Nicorette, Nicorette mint: OTC
Nicotine inhaler	6–16 cartridges/d each cartridge delivers 4 mg of nicotine over 80 inhalations	1. Local irritation of mouth and throat 2. Cough 3. Rhinitis	1. Pregnancy: FDA class D 2. Caution in patients with serious arrhythmias and worsening angina pectoris	Nicotrol inhaler: Prescription
Nicotine nasal spray	0.5–1 mg/nostril/h (max 40 doses/d)	1. Nasal irritation 2. Nasal congestion	1. Pregnancy: FDA class D 2. Caution in patients with serious arrhythmias and worsening angina pectoris	Nicotrol NS: Prescription
Bupropion SR	150 mg q am × 3 d then 150 mg b.i.d. till 7–12 w after quitting Can also be used for maintenance therapy (150 mg b.i.d. × 6 mon)	1. Insomnia 2. Dry mouth	1. Pregnancy: FDA class B 2. Contraindications: Seizure disorder Eating disorder Use of MAO inhibitor	Zyban: Prescription

Clonidine	0.15–0.75 mg/d PO OR 0.10–0.20 mg/d transdermal (TTS)	1. Dry mouth 2. Drowsiness 3. Dizziness 4. Constipation 5. Hypotension	1. Pregnancy: FDA class C 2. Rebound hypertension if stopped suddenly	Catapress, Clonidine generic: Prescription
Nortriptyline	75–100 mg/d	1. Dry mouth 2. Drowsiness 3. Dizziness 4. Urinary retention 5. Arrhythmias	1. Pregnancy: FDA class D 2. Contraindication: Use of MAO inhibitor	Pamelor, Aventyl HCL, Nortriptyline generic: Prescription

prevented by removing the patch before going to bed. Use of a nicotine patch as a cessation aid more than doubles the likelihood of quitting cigarettes as compared to placebo [84,90,91].

Nicotine Polacrilex Gum is the oldest form of NRT and is available in two strengths, 2 mg or 4 mg per piece. Absorption of the nicotine in the gum is through the buccal mucosa. Since it undergoes extensive first pass metabolism by the liver, any swallowed nicotine causes gastrointestinal side effects without increasing blood nicotine level. To maximize the effectiveness of nicotine gum, patients should be instructed to "activate" the gum by briefly chewing, then "parking" the gum against the buccal mucosa. A Meta-analysis of 13 studies found that odds of quitting smoking with nicotine gum were 1.5 times that with placebo [84]. Nicotine gum will increase abstinence rates when used either alone [92–97], or as an adjunct to nicotine patch to help control sudden cravings [98]. The 4-mg gum may be more efficacious than 2-mg strength when used as the sole NRT device, especially in heavily dependant smokers [99,100].

The nicotine inhaler is an effective smoking cessation aid with very few side effects [101–104]. It may be used alone or as an adjunct to nicotine patch [105]. The nicotine inhaler apparatus consists of a small white plastic tube, vaguely reminiscent of a cigarette, within which is housed a clear plastic canister of deliverable nicotine. Each cartridge of the inhaler contains 4 mg of deliverable nicotine that is absorbed through the oropharyngeal mucosa. The manufacturers tout the benefit of simulating the hand to mouth behavior of cigarette smoking, though it remains unclear precisely how important this effect is. More likely, the benefits of the inhaler may be related to their ease of use, flexibility and rapid delivery of absorbable nicotine. As a safe, self-titratable delivery mechanism for nicotine, the inhalers may provide the smoker with "quick relief" of sudden urges, and act as a reasonable alternative to cigarette smoking.

Nicotine delivered via nasal spray is an effective method of NRT and significantly increases smoking abstinence rate [106–109]. It also can be used either alone or in conjunction with nicotine patch [110]. Nicotine from the nasal spray reaches the brain faster than other forms of NRT and is thus very effective at relieving cravings. Unfortunately, this rapid delivery also increases its dependency potential [106,110]. Each spray delivers 0.5 mg of nicotine, and the recommended starting dose is 1 spray each nostril every 1 hour as needed to control cravings [84]. The most common side effects are nasal irritation and congestion. These symptoms are frequently severe and, combined with the elevated dependency potential, limit the clinical utility of nicotine nasal spray.

Anti-depressant medications

Bupropion SR (Zyban) is the first non-nicotine containing medication approved by the FDA for smoking cessation. It is a non-tricyclic antidepressant

that acts by inhibiting uptake of dopamine and norepinephrine from the neuronal synapse. Sustained release Bupropion is effective for smoking cessation and is accompanied by a reduction in associated weight gain [111–113]. Unlike NRT, patients should begin Bupropion SR about 7 to 10 days before the quit date. Bupropion SR is more effective than NRT alone, and may be most effective when combined with NRT [114]. It is contraindicated in patients with seizure disorder, eating disorders, uncontrolled hypertension, and in patients taking MAO inhibitors. When used properly, it is a safe medication, with the most common side effects including insomnia, mild agitation, and dry mouth. More severe effects such as major depression and psychosis have been reported but are rare. Several studies have shown that the tri-cyclic antidepressant nortriptyline (Pamelor, Aventyl HCL) may be effective in smoking cessation [115,116]. It is not approved by FDA for this purpose and, because of the limited number of studies supporting its use, should only be used as a second line medication for smoking cessation. The use of other antidepressants such as selective serotonin reuptake inhibitors and other tricyclic antidepressants as smoking cessation aids cannot be supported by currently available evidence [117].

Other medications

Clonidine (Catapress) is used as an antihypertensive medication and has not been approved by the FDA for use in smoking cessation. Various studies have looked at the role of oral [118–120] and transdermal clonidine [121–123] in smoking cessation. Meta-analysis of these studies shows that use of clonidine approximately doubles abstinence rates when compared to placebo [84,124]. Some patients find the side effects of clonidine troublesome, particularly dry mouth, sedation and constipation. Nevertheless, it may be an alternative for smokers who do not prefer or have failed NRT. Mecamylamine is a nicotine receptor antagonist. The rationale for its use in smoking cessation is based on the theory that mecamylamine may block the rewarding effects of nicotine and thus decrease the urge to smoke. Data from two small studies by the same investigators suggest that combination of NRT and mecamylamine may be superior to NRT alone in promoting smoking cessation [125,126]. However, large studies are necessary before it can be recommended for general use. A few trials have evaluated anxiolytics as a treatment for smoking cessation, however none of these trials showed any significant benefit of using these drugs for smoking cessation [127].

Non-pharmacologic interventions

Counseling and behavioral therapy play an important role in smoking cessation. The key components of an effective behavioral program include the assessment of behavior change stage, the identification of barriers to quitting, and the development of cessation and relapse prevention plans.

Studies have shown that three specific types of counseling and behavioral therapy are particularly effective [84]. Practical counseling involves teaching smokers to identify situations that increase their risk of smoking or relapse, and developing skills to cope with these situations. Excellent written material is available through voluntary health organizations such as the American Lung Association that can help busy clinicians educate patients in practical cessation techniques. Intra-treatment social support, provided during a smoker's direct contact with their physician, is also effective at preventing relapse. Creating an atmosphere that allows free, non-judgmental communication between the patient and the physician, and encourages the patient to talk about their concerns, difficulties, and even relapses, facilitates the quit process. Finally, extra-treatment social support should also be arranged. Helping the smoker identify culturally appropriate social support within his or her own community, and facilitating a connection to available community resources like hospital-based group sessions, state-wide help lines, internet cessation groups, etc. can help promote long-term abstinence. In addition to behavioral treatments, self-help materials have been found to increase motivation and impart cessation skills. Written manuals are the most common forms although video and computer versions have been developed recently.

Outside the realm of the typical physician office visit, several other forms of behavioral modification have been advocated in special circumstances. Aversive smoking, a monitored smoking sessions in which patients smokes intensively to a point of developing adverse effects like nausea, dizziness or vomiting, may increase smoking cessation rates [128,129]. The rationale is to pair smoking with an unpleasant stimulus, decreasing urge to smoke. AHCPR guidelines recommend that aversion therapy should be conducted only in selected patients under medical supervision [84]. Hypnotherapy and acupuncture, widely promoted strategies to aid smoking cessation, lack sufficient scientific data to be recommended as effective treatment for smoking; though individual smokers may benefit from these treatments [130,131].

Tertiary prevention

Since nicotine dependence has a chronic relapsing nature, it is important for physicians to prevent relapse following successful treatment. Relapse prevention interventions are especially important in the immediate post cessation period, and can be delivered during a scheduled clinic visit or via telephone. All ex-smokers should be congratulated on their success and advised to remain abstinent. Patients should be encouraged to communicate their concerns and potential factors that threaten abstinence should be identified and addressed. Patients having strong negative feelings or depression, prolonged or severe nicotine withdrawal, or excessive weight gain may benefit from counseling and/or specific pharmacotherapy. Hays et al [132] evaluated the efficacy of Bupropion SR to prevent smoking relapse. They reported that sustained release Bupropion for 12 months delayed relapse and resulted in

less weight gain. Although most relapses occur soon after quitting, some patients relapse months or even years after their quit date. These relapses are the very nature of the nicotine dependence and do not reflect a lack of patient determination or failure on the part of the physician.

Analysis of data from the National Ambulatory Medical Care Survey showed that physicians failed to identify smoking status of 33% of their patients. Only 21% of smokers received smoking cessation counseling and only 1.3% of smokers were prescribed nicotine replacement therapy [133]. In the same survey physicians provided smoking cessation counseling at only 1.6% of all adolescent visits and 16.9% of visits by adolescents that smoke [134]. Another population based survey of adult cigarette smokers found that only 45.5% of smokers were advised to quit, 14.9% were offered help and only 8.5% were prescribed medications [135]. These discouraging statistics exist despite substantial evidence of effective treatment for nicotine addiction. There are several possible reasons for reluctance to intervene on the part of physicians, including the belief that smoking is a lifestyle choice and not a true dependence, smoking cessation interventions are ineffective, pharmacotherapy should be reserved for heavily dependent patients, and that the doctor-patient relationship may be affected if physicians continue to advise smoking cessation to a patient who does not want to quit. Recent survey of Pennsylvania physicians showed that about 50% of physicians felt frustrated by smokers who do not want to quit smoking. Only 42% of physicians felt that it was possible to adequately counsel patients during regular office visits and 30% found smoking cessation counseling financially prohibitive. Although almost all physicians agreed that health care personnel are responsible for encouraging smoking cessation, only 25% of physicians were familiar with the AHCPR guidelines for smoking cessation [136]. Most medical schools in the US do not require training in smoking cessation techniques [137]. In fact, only one third of medical schools around the world teach about smoking cessation [138]. Several studies have shown that comprehensive training of physicians on nicotine dependence and smoking cessation have a positive and powerful effect on their tobacco intervention attitudes and behavior [139–144].

Summary

Tobacco use represents a rare confluence of interesting circumstances. Elements of inheritable risk combine with powerful neuropharmacology and a ubiquitous environmental exposure and result in an epidemic that claims over 430,000 lives and costs us over $100 billion annually. It is the single most important remediable public health problem in the United States. Most smokers want to quit smoking and a simple advice from a physician can increase the likelihood of doing so. Moreover, there are a number of pharmacologic and behavioral therapies that are proven to be effective in

smoking cessation. Yet, there is an apparent reluctance among physicians to address smoking cessation, perhaps due to a sense of frustration or low self-efficacy. Physicians play an important role in smoking cessation, and intensive interventions are necessary to improve their participation and efficacy. Teaching practical smoking cessation techniques within medical school curricula, with an opportunity for standardized practice and self-evaluation, may be an effective strategy to improve physician practice in this area.

Since most smokers try their first cigarette before the age of 18, and youth smoking is on the rise, targeted interventions aimed at preventing initiation and encouraging cessation of smoking among youth are needed. For all tobacco users, a better understanding of the pharmacology and physiology of nicotine addiction may translate into targeted and individualized treatment and prevention strategies, which may improve success rates dramatically. To better control this epidemic, and to meet the nation's public health goals for the year 2010 [145], local tobacco control interventions need to be multifaceted and well integrated into regional and national efforts [146]. Because of the physician's unique societal role with respect to tobacco, doctors may indeed find it possible to impact public opinion and significantly reduce the toll of tobacco by acting at the public health and public policy levels [147]. Those interested in engaging in the public health debate can do more than relay facts about tobacco and health. Involvement in tobacco-control issues provides the opportunity to impact the environmental influences promoting smoking among patients, and is likely to be synergistic with efforts to help smokers quit within the office. Physicians who take steps to engage in local public health initiatives are likely to magnify the effects of their efforts at the bedside [148,149].

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