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Smoking Cessation for the Primary Care Physician

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Editor's Note—*Cigarette smoking is the leading cause of preventable death in the United States and around the world. Smoking cessation is highly effective in reducing the death and disability from smoking. However, while approximately 50% of the 50 million smokers in the United States attempt to quit each year, less than 5% are successful without assistance from their physician. Primary care physicians have a major opportunity to reduce the death and disability from smoking because nearly three quarters of smokers visit a primary care physician each year and because physician advice at least doubles smoking cessation success rates. Effective, evidence-based guidelines exist for smoking cessation that, when implemented, substantially improve smoking cessation treatment outcomes. Primary care physicians should use a system-wide approach in their clinics to identify all patients who smoke and provide brief interventions that can take as little as 3 minutes. Brief interventions should include 5 components: 1) Ask—systematically identify all smokers at each visit; 2) Advise—strongly urge all smokers to quit; 3) Assess—determine willingness to quit smoking; 4) Assist—develop a patient-centered plan to quit smoking including pharmacotherapy unless contraindicated; 5) Arrange—schedule follow-up visits to discuss smoking cessation. Physicians should provide a a brief motivational intervention to patients who lack motivation to quit smoking. Brief motivational interventions should use the "5 Rs": physicians should help patients to identify the Relevance of smoking to their lives and health, identify the Risks of smoking and the Rewards of quitting smoking, characterize the Roadblocks to sustained abstinence from tobacco, and Repeat these motivational interventions at each visit. There are 5 F.D.A.-approved medications for smoking cessation: sustained release bupropion hydrochloride, nicotine transdermal patch, nicotine gum, nicotine nasal spray, and nicotine inhaler. Specific instructions on how to prescribe, along with contraindications and adverse effects are described in Table 3. Current research is focusing on whether use of genetic tests will provide physicians with an empiric decision tool to appropriately match individual smokers with specific pharmacotherapies to maximize success rates.*

Introduction

In the United States alone, more than 430,000 people die each year due to smoking-related causes—more deaths than AIDS, alcohol, motor vehicle accidents, murders, suicides, drugs and fires combined.[1] One hundred and fifty million people worldwide and are expected to die prematurely due to cigarette smoking-related causes between 2000 and 2025.[2] While these numbers are indeed harrowing, primary care physicians are in a unique position to dramatically reduce the death and disability from smoking. The solution is smoking cessation. If primary care physicians could assist one half of all adult smokers to quit, approximately 50 million lives could be saved during this period.[3] Seventy percent of smok-

ers visit a physician each year and the majority of these visits are with primary care physicians.[4] Fifty percent of these individuals attempt to quit smoking each year but only about 3% are successful on their own. In the United States, approximately 17 million smokers attempt to quit each year but only 1.2 million are successful.[5] Appropriate identification, advice, assistance, and follow-up by the health care team can significantly improve success rates. However, primary care physicians need to be more vigilant in screening and treating their patients who smoke cigarettes. Primary care physicians identify less than 70% of smokers, offer counseling to about 30%, and offer nicotine replacement therapy to about 3% of smokers.[6] The good news is that effective, evidence-based smoking cessation therapies can substantially improve success rates,[7] and the benefits of quitting are great-smokers who quit as late as the sixth decade of life reduce the cumulative mortality of lung cancer by 90% and achieve an even greater risk reduction for coronary artery disease.[8]

Diseases Caused by Smoking

Medical interest in the effects of using tobacco date back more than 2000 years. Initially, it was believed that tobacco had healing powers and it was for this medicinal value that use gradually spread throughout Europe and North America. Data gradually emerged of the deleterious effects over the next 2 centuries but little attention in the medical field was paid until after World War II. Two case-control trials published in Germany during World War II,[9,10] one in the Netherlands in 1948,[11] five case-control studies published in the United Kingdom and the United States in 1950,[12-16] and more than 50 years of prospective follow-up studies[17] have built a powerful case for causation between smoking and lung cancer. In the decades that followed smoking has been positively associated with 40 diseases or causes of death (*see Table 1*). In nearly all cases, risk for development of cancers and other diseases increases with the amount smoked. The longer people smoke the higher the risk of death from lung cancer and other attributable diseases.[17] The risk of smoking is great. For example, approximately one-half of people who become regular smokers during early adulthood will die prematurely due to smoking with an average loss of life of 16 years.[2] Smoking has been negatively associated with Parkinson's disease, ulcerative colitis, uterine cancer, and other diseases for reasons not completely understood.[17]

Nicotine Dependence

Even though the disease and disability associated with smoking develop during adulthood, smoking almost invariably begins in childhood.[18] Every day, 3000 young people become regular smokers.[19] More than 90% of adult smokers tried their first cigarette and nearly 80% become regular smokers before the age of 20.[20] In fact, a person who has not started as a teenager is unlikely ever to become a smoker.[21] Unfortunately, once young people become regular smokers they are under the grips of a powerful addiction. Seven of 10 young people who smoke report that they regret ever having started, and 3 of 4 young smokers have tried to quit at least once and failed.[22]

There is overwhelming evidence that nicotine is an addictive drug and that chronic smokers meet several criteria for dependence including the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)* criteria[23] and those outlined by the Surgeon General's Report (*see Table 2*).[24] Understanding the pharmacological properties of nicotine sheds light on how to effectively manage smoking cessation and diminish withdrawal symptoms.

Nicotine addiction is a complex interplay of pharmacology, learned or conditioned factors, personality, social settings, mood, cognitive factors, and genetic factors. The pharmacologic effects of nicotine are involved with nicotine addiction in multiple ways. Smokers report positive, reinforcing properties such as pleasure, arousal, relaxation, improved attention, and improved performance on certain tasks. Smokers may also experience relief of aversive emotional states, including the reduction of anxiety, relief from hunger and prevention of weight gain, relief of negative affect, and relief of withdrawal symptoms from nicotine.[25]

Converging findings from animal and human studies are elucidating the complex neurobiochemical contribution to nicotine dependence. Once nicotine enters the blood through smoking (or other forms of nicotine consumption), the nicotine molecules cross the blood-brain barrier and bind stereotypically to nicotinic cholinergic receptors throughout the brain, with the greatest number of binding sites in the cortex, thalamus, and interpeduncular nucleus. Nicotine also binds to receptors in the amygdyla, septum, brain stem motor nuclei, and locus coeruleus.[26] Release of various neurotransmitters throughout the brain results in neurochemical effects believed to be

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In order to reveal any potential bias in this publication, we disclose that Dr. David (author) reports no consultant, stockholder, speaker's bureau, research, or other financial relationships with companies having ties to this field of study.

Table 1. Diseases Caused by Smoking

Cancers	Other Diseases
Cancer of the mouth and pharynx	Ischemic heart disease
Cancer of the esophagus	Hypertension
Cancer of the lung	Myocardial degeneration
Cancer of the pancreas	Aortic aneurysm
Cancer of the bladder	Peripheral vascular disease
Cancer of the lip	Arteriosclerosis
Cancer of the nose	Cerebral vascular disease
Cancer of the stomach	Chronic bronchitis and emphysema
Cancer of the kidney pelvis	Pulmonary tuberculosis
Cancer of the kidney body	Asthma
Myeloid leukemia	Pneumonia
	Peptic ulcer disease
	Cataracts
	Impotence
	Reduced production of sperm

Source: *Doll R. Fifty years of research on tobacco. J Epidemiol Biostat. 2000;5(6):321-329.*

rewarding or reinforcing (*see Figure 1*). For example, enhanced release of dopamine and norepinephrine may be associated with pleasure and appetite suppression. Release of acetylcholine may be associated with improved performance on behavioral tasks and improvement of memory. Release of beta-endorphin may be associated with reduction of anxiety and tension.[25]

An important neural pathway in nicotine addiction is the nigrostriatal system. Nicotine stimulates neurons in the ventral tegmental area of the brain leading to release of dopamine in the outer shell of the nucleus accumbens (the brain's pleasure center).[27] Dopamine release in the nucleus accumbens is characteristic of the effects of many addictive drugs (eg, heroin, cocaine, alcohol) and is thought to be an important site for nicotine-mediated reinforcement.[28] Converging evidence supports a "Self-Medication" hypothesis[29] posing that individuals smoke to relieve negative affect, boredom, or other aversive cognitive-behavioral states and that many individuals smoke because of chemical imbalances in the brain leading to a "Reward-Deficiency Syndrome."[30] Central deficiencies in dopamine or norepinephrine transmission may explain the efficacy of the mixed dopamine/norepinephrine reuptake inhibitor bupropion hydrochloride and tricyclic antidepressants like nortriptyline.

Smoking Cessation Therapies

In June 2000, an expert panel of scientists, clinicians, consumers and others selected by the US Agency for Healthcare Research and Quality (AHRQ) performed an extensive systematic review of the tobacco use literature and published the US Public Health Service Report *Treating Tobacco Use and Dependence: A Clinical Practice Guideline*. The Clinical Practice Guideline provides recommendations for brief clinical interventions, intensive clinical interventions, and system changes to promote the treatment of tobacco dependence. This report underscored the observation that tobacco dependence has many features of a chronic disease as the majority of smokers persistently use tobacco for many years and typically cycle through multiple periods of relapse and remission.[31] While only a small number of patients successfully quit on their own (about 7%) there is strong evidence that even brief advice from a physician can double long-term quit rates and that use of intensive counseling and pharmacotherapies can increase success rates to 15% to 30% or higher.[7] The Clinical Practice Guideline recommends a clinic-wide approach to

Table 2. Surgeon General's Criteria for Drug Dependence

Primary Criteria

Highly controlled or compulsive use

Psychoactive effects

Drug-reinforced behavior

Additional Criteria

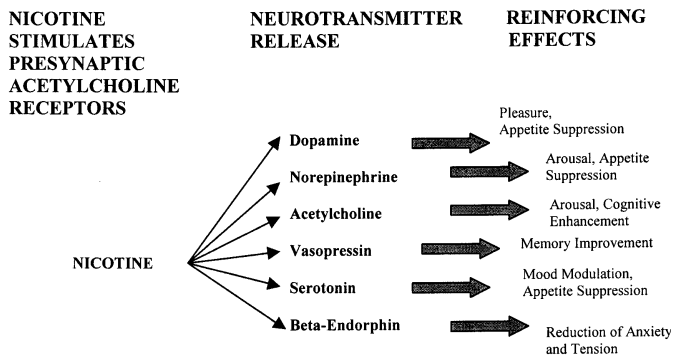
Addictive behavior often involves:

- Stereotypic patterns of use
- Use despite harmful effects
- Relapse following abstinence
- Recurrent drug cravings

Dependence-producing drugs often produce:

- Tolerance
- Physical dependence
- Pleasant (euphoric) effects

Figure 1. Neurochemical Effects of Nicotine on the Brain



identifying smokers at every visit and offering a brief clinical intervention to all smokers. Brief clinical interventions were designed to be as brief as 3 minutes and have been shown to increase smoking cessation success rates significantly.[31] A brief clinical intervention consists of 5 major steps (the "5 As"). These steps are as follows: 1) *Ask* every patient if he or she uses tobacco; 2) *Advise* him or her to quit; 3) *Assess* willingness to make a quit attempt; 4) *Assist* those who are willing to make a quit attempt; and 5) *Arrange* for follow-up contact to prevent relapse.

STEP 1—*Ask*: Physicians should implement an office-wide approach to ensures that tobacco use status is queried and documented at every visit. One effective office-wide approach is to expand the vital signs to include tobacco use. This can be facilitated by using a vital signs stamp in the medical record for every patient visit to your clinic (*see Figure 2*). Alternatives to the vital signs stamp include stickers on the outside of charts indicating tobacco use status or identifiers in the electronic medical record that highlight or flag physicians to prompt them to counsel smoking cessation. Physicians should verbally ask every patient if they smoke if this has not already been documented using a system-wide approach. Smoking status should be queried at every visit.

STEP 2—*Advise*: Once physicians have identified their patient as a smoker the next sequential step in a brief clinical intervention is to advise him or her to quit. Advice should be given in a clear, strong, and personalized fashion. Primary care physicians who have established continuity with their patients are able to leverage this advice with a direct connection to patients' concerns, family systems, and chronic or acute illnesses. An example of a clear, strong, and personalized statement of advice is: "As your primary care physician, I need to tell you that quitting smoking is the best thing you can do to prevent a second heart attack and may add years to your life." or "As your family doctor, quitting smoking may be the most important thing you can do to prevent your child from developing new ear infections and asthma attacks."

STEP 3—*Assess*: Assessment of willingness to quit is essential as it provides an evidence-based decision point for treatment. Tobacco users cycle through a number of stages of willingness to quit and the type of intervention the physician offers should be tailored to the individual patient's stage of change.[32] The first question to ask is: "Do you wish to quit smoking?" If the answer is "no", the smoker is in the *Precontemplative Stage of Change*. Patients in the precontemplative stage have no interest in quitting smoking and do not believe that the negative consequences of smoking outweigh the positive rewards. These patients require a brief motivational intervention discussed below. If the patient states that they would like to quit smoking but are not willing to commit to quitting in the next 30 days they are in the *Contemplative Stage of Change*. Patients in the contemplative stage are ambivalent about quitting and lack motivation. These patients should also be given a brief motivational intervention. The patient who is willing to set a date to quit (quit date) within the next 30 days is in the *Preparation Stage of Change*. Physicians should assist these patients in developing a quit plan as discussed below. Patients who have quit recently are in the *Action Stage of Change*. Patients in the action stage are at high risk of relapse and should receive a brief relapse prevention intervention also discussed below. Ex-smokers who have not smoked for several years are at very low risk for relapse and the Clinical Practice Guideline does not recommend interventions for these patients. However, it is important to query about tobacco use even in these patients and to ascertain whether or not they smoke cigars or use other delivery systems for nicotine.

STEP 4—*Assist*: Once willingness to quit has been assessed, physicians should follow the algorithm in Figure 3. Patients who are not willing to quit at all or are not willing to quit in the next 30 days should be given a brief motivational intervention.

Figure 2. Expanded Vital Signs Stamp Including Tobacco Use Status

VITAL SIGNS

Blood Pressure: _____ Pulse _____ Weight _____

Temperature: _____Respiratory Rate: _____

Tobacco Use: Current Former Never

(Circle One)

Patients who are willing to quit now should be offered a patient-centered treatment plan. Patients who have recently quit should be given a brief relapse prevention intervention.

For patients who wish to quit in the next 30 days, the physician should congratulate the patient and assist him or her in making a quit plan. A quit plan prepares the patient for quitting and should include 4 major pieces: 1) The patient should *Set* a quit date, preferably in the next 2 weeks. The physician should document this quit date in the medical record; 2) The patient should *Tell* family, friends, and coworkers about quitting and request understanding and support; 3) The physician should help the patient to *Anticipate* challenges to the planned quit attempt including withdrawal symptoms and avoiding social triggers; 4) The patient should *Remove* tobacco products from his or her environment. Also, during the weeks prior to quitting, patients should be instructed to avoid smoking in places where they spend a lot of time. A useful mnemonic is S.T.A.R. for *Set, Tell, Avoid, and Remove*.

In addition to a quit plan, the physician should provide practical counseling including problem solving and skills training. Patients should identify events, internal events, or activities that increase craving for nicotine and increase the risk of relapse (eg, negative affect, being around smokers, drinking alcohol, experiencing urges, or experiencing stress). The physician and patient should then identify and encourage the patient to practice coping and problem-solving skills to deal with these triggers (eg, learning to avoid tempting situations, using other, healthy methods to relieve stress like exercise, etc). Finally, the physician should provide basic information about successful quitting. For example, patients should know that smoking even a single puff will increase the likelihood of a full relapse; that withdrawal typically consists of negative mood, urges to smoke and other aversive symptoms; and that withdrawal typically peaks 1-3 weeks after quitting.

The physician should make the patient aware of resources available in the clinic to assist in successful cessation such as educational materials, follow-up calls from practice nurses, support groups, and group counseling if available. It is also important to highlight and suggest ways for the patient to seek support outside of the clinic (eg, spouse, partner, co-workers, Nicotine Anonymous, etc).

Unless contraindicated, the physician should offer pharmacotherapies to assist in smoking cessation. The only FDA approved and first-line medications for smoking cessation are sustained release bupropion and the nicotine transdermal patch, nicotine gum, nicotine nasal spray, and nicotine inhaler. Nortriptyline and Clonidine are 2 second-line medications that have been efficacious in clinical trials and may be considered for patients who have contraindications to bupropion (ie, history of seizure or history of eating disorder) or cannot tolerate nicotine replacement therapy. None of these medications have been well studied in pregnant patients or children and therefore these medications should be used in these patients if nonpharmacological interventions are ineffective or when the physician feels that the consequences of smoking are greater than the risk of using the medication. Nicotine replacement therapy has been studied in one large randomized controlled trial of pregnant smokers in Denmark, which found no evidence of efficacy of the 15 mg/d nicotine patches vs. placebo and no evidence of adverse effects in patch users.[33] Two small clinical trials of pregnant smokers found that short-term use of nicotine patches or nicotine gum resulted in serum nicotine concentrations comparable to or lower than those obtained smoking 10-20 cigarettes per day.[34-36] Nicotine is neurotoxic and crosses the placenta during pregnancy so theoretically there is no 'safe' level of nicotine exposure to the fetus. For this reason, physicians might consider intermittent forms of nicotine replacement therapy and at low-end dosages (eg, nicotine gum at 2 mg dose). To date, there are no published data on the safety or efficacy of bupropion, nortriptyline, or clonidine in pregnancy. Specific information on use of these medications, contraindications, adverse effects, and cost based on the Clinical Practice Guideline are in Table 3.

Little research has been performed on pharmacological aids to relapse prevention after cessation. Most studies of bupropion have used a relatively short duration of treatment (7-8 weeks). However, a recent randomized, clinical trial demonstrated that, among patients who were abstinent for 7 weeks on bupropion, those who continued to use bupropion had significantly higher success rates at one year when compared to those who switched to placebo (55.1% bupropion vs 42.3% placebo).[37]

Patients in either the Precontemplative or Contemplative Stages of Change lack motivation to quit. Physicians should provide a brief motivational intervention tailored to the individual patient centered on the "5 Rs": *R*elevance, *R*isks, *R*ewards, *R*oadblocks, and *R*epetition.

Physicians should start by encouraging the patient to express why quitting is personally *Relevant* and ask for specific examples. Motivational interventions are most effective when patients identify how smoking is personally relevant to disease status risk, family or social situations, health concerns, age, sex, and other important patient characteristics. Next, the physician should ask the patient to identify negative consequences of smoking. The physician can highlight or add *Risks* that the patient does not identify. These negative consequences can be expressed as short-term risks, long-term risks, and environmental risks. For example, acute risks could include asthma exascerbations, harm to pregnancy (eg, abruption), impotence, infertility, respiratory infections, etc. Long-term risks include myocardial infarction, stroke, COPD, and multiple cancers (lung, larynx, oral cavity, pharynx, esophagus, pancreas, etc). Finally, the physician should ask the patient to identify environmental risks of smoking. These risks include otitis media, asthma, and respiratory infections in children, lung cancer in exposed partners, sudden infant death syndrome, fires, and increased risk of smoking among children.

Next, the physician should ask the patient to identify *Rewards* of quitting smoking. The patient can identify those rewards that are most relevant personally but the physician should highlight additional rewards of quitting smoking. Rewards commonly include improved health status, saving money, breath smells better, reduced aging/wrinkling of skin, healthier babies, enhanced athletic performance, less shortness of breath, and additional years of life.

The patient should also identify *Roadblocks* to quitting.

Identifying roadblocks or barriers to quitting or making a quit attempt is crucial in developing an effective quit plan and will be important in relapse prevention. These barriers to treatment will also direct the type of treatment required (eg, problem solving, pharmacotherapy). Common roadblocks include fear of weight gain, lack of social support, depression, withdrawal symptoms, and enjoyment of smoking.

Primary care physicians are uniquely qualified through the longitudinal nature of the patient-physician relationship to provide *Repetition* for all smokers who lack motivation to quit. The motivational intervention should be repeated every time an unmotivated smoker presents to the clinic or is admitted to the hospital. Smokers who have made several quit attempts unsuccessfully should be told that most smokers make several quit attempts before they are successful.

STEP 5—*Arrange*: Physicians should arrange follow-up for all patients who smoke regardless of stage of change for cessation. Follow-up could be either in person or by telephone. For individuals in the preparation stage of change, follow-up should ideally be arranged during the first week following the quit date with a second follow-up within one month of the quit date. Physicians should offer follow-up to patients in the Precontemplative and Contemplative Stages of Change to discuss smoking cessation. These follow-ups

Table 3. Suggested Use of Pharmacological Therapies for Smoking Cessation

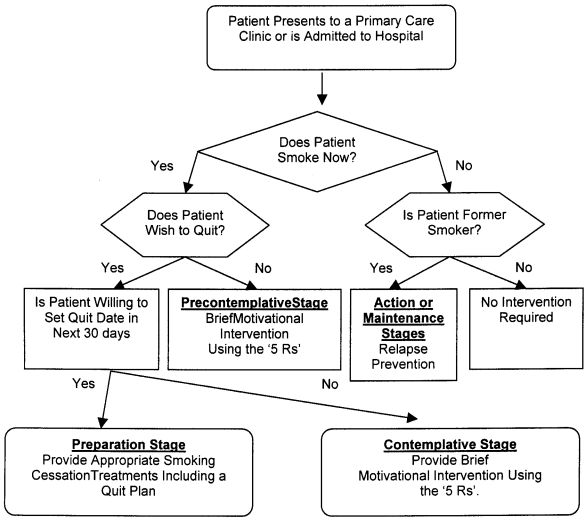
Pharmacotherapy	Precautions/ Contraindications	Adverse Effects	Dosage*	Duration	Cost per Day**
First-line					
Sustained-release bupropion hydrochloride	History of seizure History of eating disorders	Insomnia dry mouth	150 mg every morning for 3 days, then 150 mg twice daily (begin treatment 1-2 weeks before quitting)	7-12 weeks maintenance up to 12 months	$3.33
Nicotine gum		Mouth soreness dyspepsia	1-24 cigarettes/d: 2 mg gum (up to 24 pieces/d); ≥ 25 cigarettes/d: 4 mg gum (up to 24 pieces/d)	Up to 12 weeks	$6.25 for 10 2-mg pieces; $6.87 for 10 4-mg pieces
Nicotine inhaler		Local irritation of mouth and throat	6-16 cartridges/d	Up to 6 months	$10.94 for 10 cartridges
Nicotine nasal spray		Nasal irritation	8-40 doses/d	3-6 months	$5.40 for 12 doses
Nicotine patch		Local skin reaction insomnia	21 mg/24 hours 14 mg/24 hours 7 mg/24 hours 15 mg/16 hours	4 weeks then 2 weeks then 2 weeks 8 weeks	$4.22 $4.51
Second-line					
Clonidine	Rebound hypertension	dry mouth drowsiness dizziness sedation	0.15-0.75 mg/d	3-10 weeks	Oral: $0.24 for 0.2 mg Patch: $3.50
Nortriptyline	Risk of arrhythmias	Sedation dry mouth	75-100 mg/d	12 weeks	$0.74 for 75 mg

* Dosage recommendations are based on pharmacological preparations available in the United States at the time of publication and may differ from preparations available in other countries.

**Cost per day based on USPHS Practice Guideline estimates June 2000, and does not reflect the cost of generic brands.

Adapted from: *The Tobacco Use and Dependence Clinical Practice Guideline Panel, Staff, and Consortium Representatives. A clinical practice guideline for treating tobacco use and dependence: A US Public Health Service Report. The Tobacco Use and Dependence Clinical Practice Guideline Panel, Staff, and Consortium Representatives. JAMA. 2000;283(24):3244-3254.*

Figure 3. Algorithm for Treating Tobacco Use



could be linked to routine health care visits including complete physical examinations, or chronic illness care (eg, diabetes or hypertension). Follow-up contact should include congratulations for success. If smoking has occurred, the physician and patient should review the circumstances and the physician should again query willingness to make another quit attempt. Relapses should be considered as learning experiences. Follow-up visits for patients who have recently quit should include relapse prevention counseling and an assessment of pharmacotherapy use and problems. Physicians should also consider whether patients would benefit from more intensive treatment.

Current and Future Research

Twin studies suggest that there is a major genetic component to nicotine dependence and there may also be a major genetic contribution to smoking initiation.[38,39]

Current research is exploring the role of genetic factors on neurobiological mechanisms that contribute to dependence and gene-environment interactions.

Research using animal models suggests that the dopaminergic mesolimbic system is the primary neural pathway involved in dependence to many drugs of abuse including alcohol, cocaine, and nicotine.[28,40] Nicotine stimulates release and inhibits reuptake of dopamine in the nucleus accumbens (the brain's pleasure center), thereby increasing levels of synaptic dopamine and satisfying the reward mechanism.[41] Increased extracellular dopamine in the nucleus accumbens is believed to contribute to the development and maintenance of nicotine dependence through acute physiological effects and conditioned reinforcement.[42] Ablation of the mesolimbic system appears to decrease the rewarding effects of IV nicotine in the animal model.[43] Dopamine release is attenuated by mecamylamine, a CNS nicotine antagonist.[44] Therefore, polymorphisms associated with dopamine transmission are logical candidates for tobacco use research. A number of dopamine-related genes are under study for their relationship with smoking (*see Table 4*). One example of a dopaminergic candidate gene is the dopamine transporter. The dopamine transporter gene regulates synaptic dopamine by coding for a reuptake protein called the dopamine transporter.[45] This gene has 2 common polymorphisms or variants (*SLC6A3-9* and *10* alleles). *SLC6A-9* has been associated with diseases attributed to excess dopamine (eg, cocaine-induced paranoia).[46] *SLC6A3-10* repeat allele, has been associated with attention deficit disorder,[47] Tourette's syndrome,[48] and Parkinson's disease,[49] conditions attributed to insufficient dopamine. In aggregate, 4 published case-control studies have shown significant associations between smoking cessation and the dopamine transporter *SLC6A3-9* polymorphism.[50-53] This evidence suggests that the *SLC6A3-9* polymorphism is associated with diminished reuptake and increased synaptic dopamine and that, therefore, individuals with *SLC6A3-9* polymorphisms may have less of a need to use nicotine to stimulate dopamine transmission.[30] Other published work has shown associations between polymorphisms associated with diminished synaptic dopamine and various aspects of smoking. These studies are described in Table 4. Recent research[54,55] suggests that bupropion significantly reduced withdrawal symptoms when compared to placebo but that these symptoms were significantly reduced only among smokers with a specific dopamine receptor 2 polymorphism (*DRD2-Taq1A2*). The same dopamine receptor polymorphism (and other genes) was studied in a randomized control trial of the nicotine patch. This study demonstrated that the rate ratio (quit rate in nicotine group/quit rate in placebo group) was higher in smokers with the other form of this polymorphism *DRD2-Taq1A1*.[56] While such research is preliminary, these observations illustrate the potential for primary care physicians to use genetic testing and counseling in the future to individually tailor smoking cessation treatment plans.

A growing body of genetic research is focusing on the P450 enzymes involved with metabolism of nicotine in the liver. The CYP2A6 enzyme, responsible for more than 80% of the oxidative metabolism of nicotine,[57] is also involved with the metabolism of procarcinogens[58] and the metabolism of several pharmaceutical drugs.[59] Evidence from in vivo studies suggests that there is wide inter-individual variation in CYP2A6 activity, with enzyme activity ranging from "poor" to "rapid."[60] The poor metabolizing phenotype may be more common in Asians[61] and African Americans[60] than in Caucasians. Pharmacological inhibitors of this enzyme reduce smoking and it is thought that an inherited defect in this enzyme would have the same effect.[62] A substantial portion of the variability in CYP2A6 activity appears to reflect genetic polymorphisms.[63,64] While early studies of genetic associations between CYP2A6 and smoking have yielded conflicting results,[65,66] researchers are focusing more on polymorphisms in this enzyme and implications for tailoring pharmacological treatments involving nicotine replacement therapy and nicotine antagonists for smoking cessation.

Although a limited number of clinical trials have evaluated the efficacy of combining bupropion with various forms of nicotine replacement therapy,[31] further studies are needed to

examine the efficacy and safety of combining 2 or more pharmacotherapies on smoking cessation.

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3. Peto R. (August, 2000). The Hazards of Smoking and the Benefits of Stopping. Address to the 11th World Conference on Tobacco OR Health, Chicago.
4. American Academy of Family Physicians (1999). 1999 Facts About Family Practice; Table 23-Percentage Of Office Visits (in thousands) By Preventive and Therapeutic Service Ordered or

Table 4. Commonly Studied Candidate Genes for Tobacco Use

Polymorphism (gene product)	Associations with other related conditions	Associations with smoking behaviors
Dopamine Transporter *[SLC6A3-9/10]* (Protein that modulates dopamine transport and reuptake)	Tourette's syndrome[48] Parkinson's syndrome[49] Cocaine-induced paranoia[46] Novelty-seeking behavior[51] Protein modulates	4 case-control studies found association of dopamine transporter (SLC6A3-9) polymorphism with cessation[51-53,67] Meta-analysis of 3 studies demonstrates association with nicotine dependence[51-53]
Dopamine Receptor 2 *[DRD2-A1/A2]* (Type 2 dopamine receptor)	Severe alcohol dependence and polysubstance abuse,[68] obesity[69]	1 randomized controlled trial: group with polymorphism demonstrated higher quit rates on the nicotine patch than group without polymorphism;[56] 5 case-control studies found association with nicotine dependence.
Dopamine beta hydroxylase *[DBH 1368-G/A]* (Enzyme involved in dopamine degradation)	Lower plasma levels of this enzyme are related to drug dependence[70]	1 randomized controlled trial: group with polymorphism demonstrated higher quit rates on the nicotine patch than group without polymorphism.[56] Polymorphism associated with daily consumption of nicotine.[71]
Dopamine receptor 4 *[DRD4-L/S]* (Dopamine receptor 4)	Attention deficit hyperactivity disorder,[71] novelty-seeking behavior[72]	2 case-control studies found association with nicotine dependence,[73,74] age of initiation, and duration of cessation.
Monoamine Oxidase A *[MAO-A 1460 T/C]* (Dopamine degradation enzyme)	Inhibitors of monoamine oxidase are present in tobacco smoke and have been shown to contribute to nicotine dependence[75]	1 randomized controlled trial: group with polymorphism demonstrated higher quit rates on the nicotine patch than group without polymorphism;[56] Polymorphism associated with daily consumption of nicotine.[71]
Catechol O-Methyl Transferase *[COMT 1947 A/G]* (Dopamine degradation)	Polysubstance abuse,[76] alcoholism,[77] bipolar depression,[78-80] unipolar depression[81]	No association with consumption in one study[71]
Tyrsosine Hydroxylase *[TH]* (Dopamine synthesis)	Gene associated with altered transcription of enzyme[82]	1 case-control study found association with tobacco consumption but not with smoking persistence[83]
Serotonin Transporter *[ST-L/S]* (Serotonin reuptake and transport)	Associated with anxiety-related personality traits[84]	No association with nicotine dependence in one case-control study[85] Modifies effects of neuroticism on smoking motivation and nicotine dependence[86]
Coumarin Hydroxylase *[CYP2A6 deletion/duplication]* (nicotine metabolism)	Enzyme responsible for 80% of metabolism of nicotine to cotinine, ultra-rapid form more prevalent in Caucasians than in African Americans[60]	Severity of dependence and amount of daily consumption in 2 prospective studies

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Physician CME Questions

50. Smoking is positively associated with all of the following diseases *except*:
    a. Parkinson's disease
    b. Cancer of the bladder
    c. Cataracts
    d. Myeloid leukemia
    e. Cancer of the pancreas

51. Quitting smoking as late as the 6th decade of life eliminates what percent of the cumulative mortality from lung cancer?
    a. 10
    b. 30
    c. 50
    d. 65
    e. 90

52. Each of the following medications is FDA-approved for smoking cessation *except*:
    a. Nicotine gum
    b. Sustained release bupropion hydrochloride
    c. Nicotine nasal spray
    d. Nortriptyline
    e. Nicotine inhaler

53. Which type of assistance should be offered to a patient in the Precontemplative Stage of Change for smoking cessation?
    a. Pharmacotherapy
    b. Intensive counseling
    c. Offer to set a quit date
    d. Brief motivational intervention
    e. Relapse prevention counseling

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