

# Treating tobacco dependence as a chronic illness and a key modifiable predictor of disease

 Editor's  
Choice

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Tobacco smoking is unique among modifiable risk factors in that it has a causal effect on serious diseases affecting every organ system. Yet clinicians frequently fail to assess and treat tobacco dependence. Now that we have effective treatments, clinicians should refocus on aggressively treating tobacco dependence as a serious life-threatening chronic disease.

When a male patient in his late 60s walks into his doctor's office with a blood pressure of 200/100, a total cholesterol of 280 mg/dl, smoking 40 cigarettes a day and complains of chest pain, the doctor will quickly realise that this patient is at very high risk of having a myocardial infarction. Cases with these characteristics may have a 50% chance of having a heart attack in the next 12 months unless immediate steps are taken to reduce modifiable risk factors.

But even if this patient's cholesterol and blood pressure are brought down to within the normal range, he will remain at increased risk of a heart attack and stroke if he continues to smoke. In addition, he will have six times the risk of death from an aortic aneurysm, 15 times the risk of cancer of the larynx, 23 times the risk of lung cancer, 17 times the risk of death from emphysema and will remain at increased risk for a host of other serious diseases compared with having quit smoking before the age of 30 (1).

It is the magnitude and comprehensive nature of the health impact of smoking that makes it unique amongst predictors of disease (1). But there are aspects of this risk factor that unfortunately inhibit clinicians from intervening in the way they do routinely with other risk factors, which have much smaller impacts on patients' health. Perhaps the primary problem is that unlike blood pressure or C-reactive protein (CRP), smoking is perceived as a matter of patient choice, and not a medical sign, symptom or illness requiring treatment. In addition, specialists tend to focus on the risks for their specific disease interest, rather than thinking of the overall health risk. And finally, clinicians are often unsure of the best way to treat tobacco dependence, having advised many patients to quit, but seeing most continue to smoke at follow up. An 'acute treatment' model is applied to tobacco that would not be considered acceptable for other risk factors or chronic diseases (2). Some take the view that, 'I advised him to quit last year and he

continues to smoke. Oh well.' If the patient continued to have very high blood pressure after initiating a blood pressure medication, few clinicians would give up and say 'oh well.' They would continue to investigate and treat until all avenues had been explored to control the blood pressure.

It is quite correct for practicing clinicians and medical researchers to continue to seek to discover and understand new and potentially useful modifiable predictors of disease (3,4). But it is important not to focus excessive amounts of attention and resources on measures of unproven reliability and specificity, while largely ignoring an easily measurable risk factor that has a strong, proven, dose-dependent, causal and modifiable effect on numerous life-threatening diseases in clinical practice. In the United States, despite the existence of national treatment guidelines stating that all smokers should be offered a smoking cessation medication (5,6), only 2.4% of doctor visits by smokers result in a prescription for a smoking cessation medication (7). There was a decrease in rates of U.S. doctors smoking cessation counselling over the decade 1993–2003 (8). Reasons given by family doctors for not counselling smokers include lack of time (42%), lack of confidence in their own ability (38%) and lack of knowledge (22%) (9).

This paper will briefly summarise the health effects of smoking and smoking cessation, and recommend that tobacco dependence should itself be routinely assessed and aggressively treated like any other serious chronic illness.

## Effects of smoking on premature death

Half of all long-term smokers will be killed by their smoking, and on average, a continuing smoker will lose 10 healthy years of life (10) as smokers spend more years in sickness (11). The smoker loses an average of 3 months of life for each year quitting smoking is delayed after age 35 (12). The approximate doubling of mortality risks for smokers is evident at age 50 (6% of smokers already died vs. 3% of non-smokers) and continues past age 70 (42% of smokers have died vs. 19% of never smokers) (10).

**The magnitude of the health impact of smoking makes it unique amongst predictors of disease**

## Effects of smoking on specific diseases

Tobacco smoking causes serious damage to practically every organ of the body (1). However, smoking is particularly harmful to the lungs and cardiovascular system. Depending on the diagnostic criteria used, 25% to 50% of smokers will develop chronic obstructive pulmonary disease (COPD) (13), and 16% of those who continue to smoke until age 75 will develop lung cancer (compared with < 1% of never smokers) (14).

For most cardiovascular diseases (e.g. ischaemic heart disease or stroke), the risks of death are tripled for smokers aged under 65, and then just less than doubled at older ages (where these cardiovascular deaths become common for non-smokers). For some specific causes of death, such as aortic aneurysm, the risks are greater than five-fold for smokers (compared with never smokers). All of these risks are dose-related (1).

## The timescale for risk reduction

The pattern of risk reduction after smoking cessation varies with each disease and stage of disease. Thus, the excess risk of death from coronary heart disease will be cut in half within a year of quitting smoking, but the same level of risk reduction may take 10–15 years for lung cancer. The cumulative risks of death from lung cancer by age 75 for British men who quit smoking at age 30, 40, 50 and 60 are around 2%, 3%, 6% and 10% respectively (14).

The U.S. Lung Health Study (15) showed that amongst middle-aged smokers with early signs of COPD, quitting smoking results in an absolute improvement in lung function during the first year, followed by a rate of decline in lung function (FEV1) half of that of continuing smokers (31 ml/year vs. 62 ml/year).

This brief summary does not include numerous additional health benefits of smoking cessation, including benefits to the pregnant woman and unborn child of quitting prior to or early in pregnancy, and avoidance of numerous other cancers and non-fatal illnesses (e.g. respiratory infections) that are reduced after smoking cessation (1). Many of these benefits of cessation are particularly apparent amongst people with additional risk factors.

## Treating tobacco dependence as a life-threatening chronic illness

This and other medical journals have discussed the potential of various risk predictors and screening options (3,16). Spiral CT for early detection of lung

cancer and CRP as a marker of cardiovascular risk are examples of promising but unproven methods for estimating and reducing risk. As one reviewer put it, 'not ready for prime time' appears to be the current verdict on many of these promising technologies (17). This means that clinicians should focus on assessing and treating proven risk factors such as hypertension, hyperlipidemia and tobacco dependence. Of these, it is tobacco dependence that requires increased attention, partly because it has such a large influence on so many disease risks, and partly because we now have relatively effective (and highly cost-effective) assessments and treatments available (6).

## What should clinicians do to assess and treat tobacco dependence?

### • Ask every patient if they use tobacco

### • Advise to quit

Before assessing willingness to quit, deliver a positive statement emphasising the importance of treatment (do you typically ask your hypertensive patients in a neutral manner if they are interested in reducing their blood pressure?).

*"As your clinician, I want you to know that the single best thing you can do for your health right now is to quit smoking. Every year that you continue smoking takes off 3 months from your life. I understand that it may not be an easy thing to do, but there are now treatments that have been shown to work, and I believe it is very important for your health that we get started on this."*

### • Assess motivation to quit and level of tobacco dependence

Motivation can then be assessed by asking, "Are you interested in quitting smoking?"

Tobacco dependence can be assessed by a few simple questions:

*"How many cigarettes per day do you smoke?"*  
(< 5 = low dependence, > 14 = high dependence)

*"How soon after you wake in the morning do you smoke?"*  
(< 31 min = high dependence, > 120 min = low dependence).

Waking at night to smoke (18), previous unsuccessful quit attempts and experience of withdrawal symptoms (moodiness, poor concentration, insomnia) and cravings during prior periods of abstinence are also indicators of high dependence.

### • Assist in the quit attempt

Counselling is effective in helping smokers to quit. Simple brief advice and arranging a target quit date with a doctor or other healthcare professional will increase the quit rate (6).

**Treat tobacco dependence as a chronic illness**

Many countries now have free telephone quitlines and internet-based assistance, and clinicians should routinely advise patients to use these.

Smoking cessation medicines are safe and effective (6). Smokers of less than five cigarettes per day will have a decent chance of success in quitting by choosing a quit date, getting rid of tobacco and using freely available counselling/support services. Smokers of 6–14 cigarettes per day are likely moderately dependent and will benefit from an approved smoking cessation aid (nicotine patch, gum, lozenge, inhaler or nasal spray, bupropion, varenicline). Smokers of 15 or more cigarettes per day are likely highly dependent and will benefit from more intensive counselling and possibly combination pharmacotherapy (typically nicotine patch plus at least one other medicine other than varenicline). Combination pharmacotherapy is commonly used in treating other common risk factors such as hypertension and diabetes, and longer-term

combination smoking cessation pharmacotherapy has been shown to be safe and effective for smokers with existing medical illnesses (19). Table 1 shows the results of the U.S. Public Health Service Guideline meta analysis of medication effects, with longer-term patch plus another nicotine replacement therapy appearing to be the most potent combination therapy and 2 mg varenicline the most potent monotherapy (6).

#### • Arrange a follow up

Patients willing to make a quit attempt should be followed up by a clinician (either in person or via telephone) within a week of their target quit date. This will increase the chances that they follow through with the plan and allows monitoring of withdrawal symptoms and medication use. Clinicians would not typically initiate a new hypertension medication without arranging a follow up to monitor the response. The same degree of follow up is

**The single best thing you can do for your health is to quit smoking**

**Table 1** Meta analysis: effectiveness and abstinence rates for various medications and medication combinations compared with placebo at 6 months postquit ( $n = 83$  studies)\*

Medication	Number of arms	Estimated odds ratio (95% CI)	Estimated abstinence rate (95% CI)
Placebo	80	1.0	13.8
<b>Monotherapies</b>			
Varenicline (2 mg/day)	5	3.1 (2.5–3.8)	33.2 (28.9–37.8)
Nicotine nasal spray	4	2.3 (1.7–3.0)	26.7 (21.5–32.7)
High-dose nicotine patch (> 25 mg) (these included both standard or long-term duration)	4	2.3 (1.7–3.0)	26.5 (21.3–32.5)
Long-term nicotine gum (> 14 weeks)	6	2.2 (1.5–3.2)	26.1 (19.7–33.6)
Varenicline (1 mg/day)	3	2.1 (1.5–3.0)	25.4 (19.6–32.2)
Nicotine inhaler	6	2.1 (1.5–2.9)	24.8 (19.1–31.6)
Clonidine	3	2.1 (1.2–3.7)	25.0 (15.7–37.3)
Bupropion SR	26	2.0 (1.8–2.2)	24.2 (22.2–26.4)
Nicotine patch (6–14 weeks)	32	1.9 (1.7–2.2)	23.4 (21.3–25.8)
Long-term nicotine patch (> 14 weeks)	10	1.9 (1.7–2.3)	23.7 (21.0–26.6)
Nortriptyline	5	1.8 (1.3–2.6)	22.5 (16.8–29.4)
Nicotine gum (6–14 weeks)	15	1.5 (1.2–1.7)	19.0 (16.5–21.9)
<b>Combination therapies</b>			
Patch (long-term; > 14 weeks) + ad lib NRT (gum or spray)	3	3.6 (2.5–5.2)	36.5 (28.6–45.3)
Patch + bupropion SR	3	2.5 (1.9–3.4)	28.9 (23.5–35.1)
Patch + nortriptyline	2	2.3 (1.3–4.2)	27.3 (17.2–40.4)
Patch + inhaler	2	2.2 (1.3–3.6)	25.8 (17.4–36.5)
Patch + second generation antidepressants (paroxetine, venlafaxine)	3	2.0 (1.2–3.4)	24.3 (16.1–35.0)
<b>Medications not shown to be effective</b>			
Selective serotonin re-uptake inhibitors (SSRIs)	3	1.0 (0.7–1.4)	13.7 (10.2–18.0)
Naltrexone	2	0.5 (0.2–1.2)	7.3 (3.1–16.2)

\*Go to <http://www.surgeongeneral.gov/tobacco/gdlhrefs.htm> for the articles used in this meta analysis from Fiore et al. (6).

**Table 2** Meta analysis: effectiveness of and estimated abstinence rates for the number of person-to-person treatment sessions ( $n = 46$  studies)\*

Number of sessions	Number of arms	Estimated odds ratio (95% CI)	Estimated abstinence rate (95% CI)
0–1 Session	43	1.0	12.4
2–3 Sessions	17	1.4 (1.1–1.7)	16.3 (13.7–19.0)
4–8 Sessions	23	1.9 (1.6–2.2)	20.9 (18.1–23.6)
> 8 Sessions	51	2.3 (2.1–3.0)	24.7 (21.0–28.4)

\*Go to <http://www.surgeongeneral.gov/tobacco/gdlnrefs.htm> for the articles used in this meta analysis, from Fiore et al. (5).

indicated for tobacco treatment. Additional counseling sessions with the doctor, nurse or a smoking cessation specialist will likely increase quit rates, as shown in Table 2, above.

#### • Re-treat or refer

If the patient does not successfully quit smoking, continue or increase the treatment (e.g. the dose of medication/support) and/or refer for a more intensive intervention (e.g. to a specialist smoking cessation service). Smoking is such a potent risk factor for future disease, and it is not appropriate to give up if initial treatment is unsuccessful (2,20). Rather, try a different treatment or get specialist involvement, as would happen for other serious chronic illnesses.

### Conclusion

Tobacco smoking is unique amongst established health risk factors (e.g. hypertension, hyperlipidemia) in that it is causally related to diseases of virtually every organ in the body. While other predictors (e.g. lipoprotein a, CRP) show promise and are worthy of research, tobacco dependence is easy to assess, and effective treatments are available. Rather than only treating the downstream effects of the results of continued tobacco use, healthcare professionals should take a proactive approach and seek to eliminate the cause of the disease by aggressively treating, and if necessary re-treating, the patient's tobacco dependence.

### Disclosures

JF has carried out consulting work and speaking for pharmaceutical companies with tobacco treatment medicines (e.g. Pfizer, Novartis and GSK) and writes

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**Effective  
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### References

- 1 U.S. Department of Health and Human Services. *The Consequences of Smoking: A Report of the Surgeon General*. Washington, DC: U.S. Government Printing Office, 2004.
- 2 Steinberg MB, Schmelzer AC, Richardson DL, Foulds J. The case for treating tobacco dependence as a chronic disease. *Ann Intern Med* 2008; **148**: 554–6.
- 3 Tonstad S, Cowan JL. C-reactive protein as a predictor of disease in smokers and former smokers: a review. *Int J Clin Pract* 2009; **63**: 1634–41.
- 4 Yusuf S, Hawken S, Ounpuu S et al. INTERHEART Study Investigators. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet* 2004; **364**: 937–52.
- 5 Fiore MC, Bailey WC, Cohen SJ et al. *Treating Tobacco Use and Dependence: Clinical Practice Guideline*. Rockville, MD: US Department of Health and Human Services. Public Health Service, 2000.
- 6 Fiore MC, Jaén CR, Baker TB et al. *Treating Tobacco Use and Dependence: 2008 Update*. Clinical Practice Guideline. Rockville, MD: U.S. Department of Health and Human Services. Public Health Service, 2008.
- 7 Steinberg MB, Akinciquil A, Delnevo C et al. Gender and age disparities for smoking cessation treatment. *Am J Prev Med* 2006; **30**: 405–12.
- 8 Thorndike AN, Regan S, Rigotti NA. The treatment of smoking by US physicians during ambulatory visits: 1994–2003. *Am J Public Health* 2007; **97**: 1878–83.
- 9 Vogt F, Hall S, Marteau TM. General practitioners' and family physicians' negative beliefs and attitudes towards discussing smoking cessation with patients: a systematic review. *Addiction* 2005; **100**: 1423–31.
- 10 Doll R, Peto R, Boreham J, Sutherland I. Mortality in relation to smoking: 50 years' observations on male British doctors. *BMJ* 2004; **328** (7455): 1519.
- 11 Nusselder WJ, Looman CW, Marang-van de Mheen PJ, Van e Mheen H, Mackenbach JP. Smoking and the compression of morbidity. *J Epidemiol Community Health* 2000; **54**: 566–74.
- 12 Aveyard P, West R. Managing smoking cessation. *BMJ* 2007; **335**: 37–41.
- 13 Lundbäck B, Lindberg A, Lindström M et al. Obstructive lung disease in Northern Sweden studies. Not 15 but 50% of

- smokers develop COPD? – report from the obstructive lung disease in Northern Sweden studies. *Respir Med* 2003; **97**: 115–22.
- 14 Peto R, Darby S, Dep H, Silcocks P, Whiteley E, Doll R. Smoking, smoking cessation, and lung cancer in the UK since 1950: combination of national statistics with two case-control studies. *BMJ* 2000; **321**: 323–9.
  - 15 Scanlon PD, Connett JE, Waller LA, Altose MD, Bailey AS. Smoking cessation and lung function in mild-to-moderate chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 2000; **161**(2 Pt 1):381–90.
  - 16 Stanbrook MB, Flegel K. A pause for thought on lung cancer screening. *CMAJ* 2009; **180**: 793.
  - 17 Patz EF Jr, Goodman PC. Low-dose spiral computed tomography screening for lung cancer: not ready for prime time. *Am J Respir Crit Care Med* 2001; **163**: 813–4.
  - 18 Bover MT, Foulds J, Steinberg MB, Richardson D, Marcella SW. Waking at night to smoke as a marker for tobacco dependence: patient characteristics and relationship to treatment outcome. *Int J Clin Pract* 2008; **62**: 182–90.
  - 19 Steinberg MB, Greenhaus S, Schmelzer AC et al. Triple-combination pharmacotherapy for medically ill smokers: a randomized trial. *Ann Intern Med* 2009; **150**: 447–54.
  - 20 Han ES, Foulds J, Steinberg MB et al. Characteristics and smoking cessation outcomes of patients returning for repeat tobacco dependence treatment. *Int J Clin Pract* 2006; **60**: 1068–74.