Thorax

Editorials

Journal impact factors for 2000: *Thorax* flying yet higher

The journal impact factors for 2000 have recently been released and the good news for *Thorax* is that it has again increased its impact factor from 3.44 in 1999[1] to 3.98, and it has narrowed the gap considerably on its main two competitors—the American Thoracic Society publications. The position of *Thorax* in terms of impact factor relative to other selected specialist journals of similar complementary interests is shown in table 1. What is the reason for this further improvement? We think that it is due to a number of factors. Our fast review process[2] and rapid publication undoubtedly helps, but the most important factor is the quality of the contributions that we receive from you, the contributors. We have been very fortunate to receive a number of excellent original papers for the last two years. In addition, we have benefited from having several authoritative guidelines in the journal,[3] a number of excellent review series,[4–7] good contributions from symposia,[8] and the well received *Year in Review* publication.[9] *eThorax* may also have contributed.[10]

*Table 1 Journal impact factors 2000**

American Journal of Respiratory and Critical Care Medicine	5.44
American Journal of Respiratory Cell and Molecular Biology	4.35
Thorax	**3.98**
American Journal of Physiology—Lung Cellular and Molecular Physiology	3.30
Journal of Thoracic and Cardiovascular Surgery	3.06
Clinical and Experimental Allergy	2.95
European Respiratory Journal	2.59
Journal of Heart and Lung Transplantation	2.53
Chest	2.45

*Source: Institute for Scientific Information Journal Citation Reports, 2000. http://jcrweb.com

We hope that the journal can continue to go from strength to strength, but it can only do so by continuing to receive high quality papers. We thank all the Associate Editors, Editorial Board, Technical Editor, and Editorial Assistants and referees for their efforts and hope that you the readers and contributors will continue to think of *Thorax* for your best work. Perhaps the drivers of the two American Thoracic Society publications will not be looking in their rear view mirrors to see *Thorax* for much longer!

A J KNOX
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Executive Editors

1 Britton J, Knox A. Journal impact factors for 1999. *Thorax* 2000;**55**:725.
2 Knox AJ, Britton J. Rising impact, fast review: Annual report October 1997 to September 1998. *Thorax* 1999;**54**:5–6.
3 Britton J, Knox AJ. Helping people to stop smoking: the new smoking cessation guidelines. *Thorax* 1999;**54**:1.
4 Tattersfield AE, DuBois RM. Rare diseases: Introduction. *Thorax* 1999;**54**:253.
5 Sly PD. Paediatric origins of adult lung disease. *Thorax* 2000;**55**:585.
6 Holmes WF, Macfarlane J. Issues at the interface between primary and secondary care in the management of respiratory disease: Introduction. *Thorax* 1999;**54**:538–9.
7 Barnes PJ. Genetics and pulmonary medicine—9: Molecular genetics of chronic obstructive pulmonary disease. *Thorax* 1999;**54**:245.
8 Vane J. Aspirin and other inflammatory drugs. *Thorax* 2000;**55**(Suppl 2):51.
9 Mitchell DM, Woodcock A. *2000 Year in Review*: Introduction. *Thorax* 2000;**55**(Suppl 1):S1.
10 Orme R. Thorax with extra "e". *Thorax* 1999;**54**:189.

Antidepressants in the treatment of patients with COPD: possible associations between smoking cigarettes, COPD and depression

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The antidepressants bupropion and nortriptyline are effective agents in stimulating smoking cessation.[1–4] The mechanism by which bupropion and nortriptyline act as smoking cessation aids is unknown, as is its exact mechanism of antidepressant activity. What we do know is that the effects on smoking cessation are not produced through the treatment of depression itself.[1–4] Bupropion and nortriptyline are thought to produce their therapeutic antidepressant effects via inhibition of the neuronal uptake of noradrenaline and/or dopamine. Furthermore, the effects of nicotine are supposed to occur through the stimulation of dopaminergic pathways. Because (1) smoking cigarettes is the major risk factor for chronic obstructive pulmonary disease (COPD), (2) smoking cessation is the main preventive measure in reducing the decline in lung function in patients at all stages of COPD, and (3) patients with COPD can be characterised as a population of chronically ill patients with a higher than normal prevalence of psychiatric disorders, the use of antidepressants in the treatment of these patients needs further attention.

Smoking cigarettes is the main cause of COPD in 80–90% of cases (fig 1, arrow 1). It is the single most important way of affecting outcome in patients at all stages of COPD,[5] and is the only evidence based treatment (as confirmed in the Lung Health Study[6]) which has been proved to slow down the development of the disease by preventing further deterioration in lung function. It is the only intervention at this time to affect long term decline in forced expiratory volume in one second (FEV_1). However, for most people stopping smoking is difficult, especially

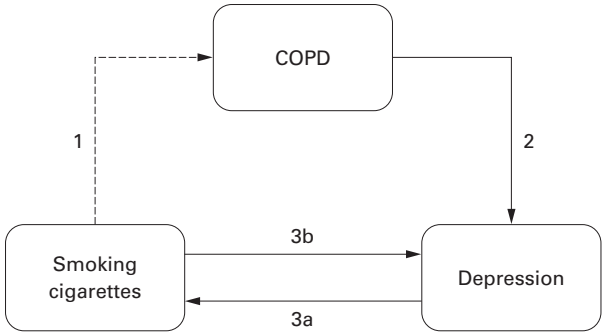

Figure 1 Association between smoking cigarettes, COPD, and depression.

when the attempt to stop is being hampered by the co-existence of a psychiatric disorder. The effectiveness of smoking cessation programmes can be influenced by the co-existence of depression or other psychiatric disorders. Both depression and depressive symptoms are independently associated with failure to quit smoking and relapse.

Patients with COPD have repeatedly been characterised as a population of chronically ill patients with a higher than normal prevalence of psychiatric disorders such as depression.[7 8] Psychiatric disorders complicating COPD may be overlooked.[9] Many clinicians remain uncertain about the exact status of depressive moods and associated symptoms in patients with chronic diseases.[10] The physical illness itself is often considered reason enough for patients to be depressed.[9] This could lead to an underestimation of the prevalence of psychiatric disorders in the physically ill because symptoms of a psychiatric disorder such as severe fatigue or insomnia may be attributed to physical illness when they are, in fact, caused by psychological illness, and these increased scores may be related to an understandable and expected emotional reaction to the physical illness itself.[7 10] However, distinguishing depression from COPD can be difficult because several of the same emotional and physical signs and symptoms are associated with both disorders.[9 11 12] Until now it has not been clear whether depression is more prevalent in patients with COPD than in those without COPD or those with other chronic illnesses. We hypothesise that COPD, as a chronic disease, causes patients to suffer from depressive symptoms which may lead to the development of depression (fig 1, arrow 2).

Furthermore, although a positive association between psychiatric disorders and cigarette smoking has been well established,[5 6] the association between smoking cigarettes, COPD, and depression is not yet clear. Several explanations for the association between psychiatric disorders and cigarette smoking have been proposed. According to the notion of self-medication, smokers use nicotine to medicate their depressed mood.[13] Alternative explanations include a causal influence of smoking on major depression based on the possible effects of long term nicotine exposure on neurobiological systems implicated in the aetiology of depression,[14] and the effects of shared environmental or genetic factors that predispose to both smoking and major depression.[14] The observed influences from depression to subsequent daily smoking (fig 1, arrow 3a) and smoking to depression (fig 1, arrow 3b) support the plausibility of shared aetiologies (or at least their close interrelationship).[13] However, separate causal mechanisms in each direction might also operate, including self-medication of depressed mood as a factor in smoking progression and neuropharmacological effects of nicotine on neurotransmitter systems linked to depression.[13] The association between smoking cigarettes and depression is certainly present but is not yet understood.

In daily practice it is quite common to treat the symptoms of COPD as separate disease entities. However, for continuity of the effect of individual treatments in patients with COPD, we recommend a holistic approach in which the possible co-existence of multiple problems is being integrated. For example, the attempt to quit smoking and maintain abstinence from smoking might be hampered by the co-existence of depression. The use of antidepressants such as bupropion or nortriptyline is therefore of particular interest in the treatment of patients with COPD.

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1 Hall SM, Reus VI, Munoz RF, *et al*. Nortriptyline and cognitive-behavioral therapy in the treatment of cigarette smoking. *Arch Gen Psychiatry* 1998;**55**:683–90.
2 Prochazka AV, Weaver MJ, Keller RT, *et al*. A randomized trial of nortriptyline for smoking cessation. *Arch Intern Med* 1998;**158**:2035–9.
3 Hurt RD, Sachs DP, Glover ED, *et al*. A comparison of sustained-release bupropion and placebo for smoking cessation. *N Engl J Med* 1997;**337**:1195–202.
4 Jorenby DE, Leischow SJ, Nides MA, *et al*. A controlled trial of sustained-release bupropion, a nicotine patch, or both for smoking cessation. *N Engl J Med* 1999;**340**:685–91.
5 Fletcher C, Peto R. The natural history of chronic airflow obstruction. *BMJ* 1977;1:1645–8.
6 Anthonisen NR, Connett JE, Kiley JP, *et al*. Effects of smoking intervention and the use of an inhaled anticholinergic bronchodilator on the rate of decline of FEV_1. The Lung Health Study. *JAMA* 1994;**272**:1497–505.
7 Dudley DL, Glaser EM, Jorgenson BN, *et al*. Psychosocial concomitants to rehabilitation in chronic obstructive pulmonary disease. Part I. Psychosocial and psychological considerations. *Chest* 1980;**77**:413–20.
8 Isoaho R, Keistinen T, Laippala P, *et al*. Chronic obstructive pulmonary disease and symptoms related to depression in elderly persons. *Psychol Rep* 1995;**76**:287–97.
9 Borson S, McDonald GJ, Gayle T, *et al*. Improvement in mood, physical symptoms, and function with nortriptyline for depression in patients with chronic obstructive pulmonary disease. *Psychosomatics* 1992;**33**:190–201.
10 Gill D, Hatcher S. A systematic review of the treatment of depression with antidepressant drugs in patients who also have a physical illness. *J Psychosom Res* 1999;**47**:131–43.
11 Gift AG, McCrone SH. Depression in patients with COPD. *Heart Lung* 1993;**22**:289–97.
12 McKinney B. COPD and depression. Treat them both. *Res Nurs* 1994;**57**:48–50.
13 Breslau N, Peterson EL, Schultz LR, *et al*. Major depression and stages of smoking. A longitudinal investigation. *Arch Gen Psychiatry* 1998;**55**:161–6.
14 Kendler KS, Neale MC, MacLean CJ, *et al*. Smoking and major depression. A causal analysis. *Arch Gen Psychiatry* 1993;**50**:36–43.