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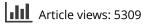
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Preventing chronic obstructive pulmonary disease

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"Prevention has long been considered better than cure. Unfortunately, for many patients suffering from COPD today it is too late to prevent the development of disability and they must rely on the effective treatments currently available."

The Global Initiative for Chronic Obstructive Lung Disease and the American Thoracic and European Respiratory Societies boldly define chronic obstructive pulmonary disease (COPD) as a 'preventable' as well as 'treatable' disease [1,2]; however, although there have been great advances in the treatment of COPD in the last 20 years, less attention has been paid to its prevention.

Disease prevention is conventionally divided into primary, secondary and tertiary prevention. Primary prevention aims to prevent the development of the disease, secondary prevention involves reducing the impact of disease by early identification and modification of risk factors, whilst tertiary prevention aims to prevent the development of complications of the disease. Primary, secondary, and tertiary prevention strategies exist for COPD and whilst they are all important, primary prevention is undoubtedly the most important long-term approach.

In the Western world, approximately 80% of COPD cases are attributable to cigarette smoking and primary prevention of COPD here depends on strategies to help people stop smoking and to dissuade them from starting smoking. Worldwide, it is estimated that 1.3 billion people currently smoke cigarettes or other products (over 1 billion men and 250 million women) [3]. Cigarette consumption has declined in several of the WHO regions in the past 30 years, most notably in the USA and Canada, where popular support for clean indoor air laws, increases in cigarette excise taxes and counter-advertising have discouraged smoking initiation by adolescents and reduced consumption among addicted smokers. However, over the last 10 years consumption has remained fairly stable in the Western Pacific, Eastern Mediterranean, Southeast Asian and African regions, and recent trends in Europe are mixed, with decreased consumption in some countries (Austria, Croatia, France, Greece, Iceland, Poland, Slovenia and the UK) but increases in others (Bulgaria, Italy, The Netherlands, Spain and the Russian Federation).

Globally, tobacco use is substantially higher in men than in women (reaching over 50% in some countries), reflecting the traditionally low prevalence of female smokers in many developing countries [3]. However, the situation is changing, and in many countries in Europe and North America the male and female smoking prevalence is converging [4]. Even in countries in Europe (e.g., Spain, Italy and Greece) and Asia (e.g., Cambodia, Malaysia and Bangladesh) where there have historically been cultural barriers to tobacco use, the proportion of women smoking has increased in recent years.

In most countries, the great majority of smokers begin to use tobacco before the age of 18 [5] and in many countries, including the UK, teenage girls are now more likely to have smoked than boys [6]. There is evidence that suggests that women are at greater risk of smoking-induced lung function impairment for the same level of tobacco exposure than men [7] and that female smokers have a significantly faster annual decline in forced expiratory volume in 1 s (as a percentage of their predicted value) with increasing age than men [8]. This makes it particularly important to target smoking cessation services at young women when trying to prevent COPD.

When asked, most smokers in Western countries, such as the UK, say that they want to quit and many make an attempt [9], but unfortunately only 2–3% have sustained success [9]. Over 50% of smokers are eventually successful in their attempt to quit [10] but it may take many efforts and it is often too late to prevent the development of diseases such as COPD. It is therefore vital to encourage people to stop at an early age, and certainly before the age of 40, as on average for every year that smoking cessation is delayed after this, life expectancy is reduced by 3 months [11].

At a population level, the most effective way of reducing smoking prevalence is to increase the price of the product [12,13]. In the UK, a 10% rise in the price of tobacco typically leads to a 4% decline in consumption [12], with higher quit rates among lower-income [14] and younger smokers [12] – the groups more difficult to reach by other approaches.

A variety of services and therapies are currently available to individual smokers to help them quit: specialist stop smoking services, including one-to-one consultation and group support; telephone helplines; advice from health professionals such as GPs, dentists and practice nurses; nicotine replacement therapy (NRT); and prescription-only stop smoking therapies (bupropion and varenicline).

"If smoking accounts for approximately 80% of the burden of COPD in Western countries, what accounts for the other 20% and can these cases be prevented?"

A meta-analysis of more than 100 randomized, controlled trials shows that all forms of NRT increase the rate of quitting by 50–70%, regardless of the duration of therapy, the intensity of additional support provided or the setting in which the NRT was offered [15]. Using bupropion approximately doubles cessation rates [16], and varenicline more than doubles quit rates compared with placebo and increases them by 30–50% compared with NRT and bupropion [17].

If smoking accounts for approximately 80% of the burden of COPD in Western countries, what accounts for the other 20% and can these cases be prevented? Occupational dust exposure has long been linked to symptoms of chronic bronchitis [18] and over 50 years ago was shown to be associated with airflow obstruction and emphysema [19]. A landmark review of epidemiological evidence concluded that there was a causal relationship [20] and more recent studies have shown that occupational exposure to vapours, dust or fumes is strongly associated with the development of COPD. In both smokers and nonsmokers, occupational exposure approximately doubles the risk of developing COPD and occupational exposure in nonsmokers is thought to account

for 15–20% of all cases of COPD [21]. Alongside smoking cessation, reduction in relevant workplace exposure by legislation and the use of personal protection, and identification of early disease is essential for the effective primary and secondary prevention of COPD [22].

"The WHO estimate that in low- and middleincome countries, exposure to smoke from wood, coal, straw and dung fuel accounts for approximately 35% of all cases of COPD."

Occupational or domestic exposure to second-hand smoke has been linked to the development of respiratory symptoms [23] and passive smoking at home appears to have a detrimental effect on lung function in children [24]. Workplace exposure to secondhand smoke after working just one shift in a smoky bar or restaurant has been shown to produce acute changes in lung function [25]. Emerging epidemiological and experimental evidence indicates that passive smoking is related to the development of COPD [26,27]. Reduction in passive exposure, by measures such as workplace smoking bans, leads to improvements in lung function [28] and has a role in the prevention of COPD.

Globally, exposure to indoor air pollution from biomass fuels is the second most important cause of COPD after smoking. Biomass fuels are extensively used for cooking and home heating in developing countries. Several studies have shown a relationship between the prevalence of COPD in women and exposure to biofuel smoke [29,30]. The WHO estimate that in low- and middle-income countries, exposure to smoke from wood, coal, straw and dung fuel accounts for approximately 35% of all cases of COPD. Up to 50% of the deaths from COPD in developing countries may be attributed to exposure to biomass fuel, and approximately 75% of these cases are in women [31,32].

Preventing the development of COPD as a result of biomass exposure is challenging as it involves education and cultural changes, as well as the introduction of better stoves and alternative fuels together with improvements in home ventilation [32]. There are no data as yet to show whether changing cooking methods affects the development of COPD; however, there is evidence that it can reduce respiratory symptoms in women and children [32] and it seems very likely that it will have a long-term impact on preventing COPD.

In developed countries, COPD is more common in individuals of lower socioeconomic status [33] and there is evidence that dietary factors [34] and factors involved *in utero* and early childhood may also be important in the development of COPD [35–37]. It is likely that these factors are also relevant in developing countries. Modification of these risk factors through better maternity care and nutrition may already have had a preventative effect and reduced the risk of developing COPD in future generations.

One of the biggest challenges facing those promoting the primary prevention of COPD is overcoming the lack of awareness of the existence and importance of the disease amongst the general population, as well as underestimation of the importance of COPD-related morbidity and mortality among healthcare professionals, particularly nonspecialists. The majority of the public are unfamiliar with the term COPD [38] and there is poor awareness in the general population of basic information, such as the size and location of the lungs, and the importance of lung health [39].

Secondary prevention of COPD depends on early identification of disease and reduction or avoidance of risk factors. Opportunistic spirometry in smokers at risk of developing COPD can identify people with undiagnosed COPD [40,41]; however, there is still uncertainty about whether it would be cost effective if introduced on a wide scale [42]. Paradoxically, the prevalence of smoking in people with COPD is higher than in the general population (34.9 vs 22.4%) and the prevalence of smoking is highest in patients with more severe COPD (40.5% in the most severe category versus 29.5% in the mildest category) [43]. This raises particular challenges when considering secondary prevention, but getting smokers with COPD to quit once a diagnosis has been made is the single most important secondary prevention intervention. There is some evidence that among people with COPD, quit rates can be improved by informing them of their abnormal lung function results but not all studies have confirmed this [44].

Reducing occupational exposure [22], reducing the risk of exacerbations, which are associated with faster decline in lung function [45], and introducing inhaled therapies, which have been

References

- Rabe KF, Hurd S, Anzueto A *et al.* Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. *Am. J. Respir. Crit. Care Med.* 176(6), 532–555 (2007).
- 2 Celli BR, MacNee W. Standards for the diagnosis and treatment of patients with COPD: a summary of the ATS/ERS position paper. *Eur. Respir. J.* 23(6), 932–946 (2004).
- 3 Guindon G, Boisclair D. Past, Current, and Future Trends in Tobacco Use. The World Bank, DC, USA (2003).
- 4 Forey B, Hamling J, Lee P, Wald N. International Smoking Statistics: A Collection of Historical Data from 30 Economically Developed countries (2nd Edition). Oxford University Press, London, UK (2002).
- 5 Global Youth Tabacco Survey Collaborative Group. Tobacco use among youth: a cross country comparison. *Tob. Control* 11(3), 252–270 (2002).
- 6 Swedish Council for Information on Alcohol and Other Drugs. *The 1999 ESPAD Report. Vol. 2003.* The Swedish Council for Information on Alcohol and Other Drugs, Stockholm, Sweden (2000).
- 7 Chapman KR. Chronic obstructive pulmonary disease: are women more susceptible than men? *Clin. Chest Med.* 25(2), 331–341 (2004).

- 8 Halbert RJ, Natoli JL, Gano A, Badamgarav E, Buist AS, Mannino DM. Global burden of COPD: systematic review and meta-analysis. *Eur. Respir. J.* 28(3), 523–532 (2006).
- 9 Lader D. Smoking-related Behaviour and Attitudes, 2006. Office for National Statistics, Newport, UK (2007).
- Stapleton J. Cigarette smoking prevalence, cessation and relapse. *Stat. Methods Med. Res.* 7, 187–203 (1998).
- 11 Doll R, Peto R, Wheatley K, Gray R, Sutherland I. Mortality in relation to smoking: 40 years' observations on male British doctors. *BMJ* 309(6959), 901–911 (1994).
- 12 Jha P, Chaloupka F. Curbing the Epidemic: Governments and the Economics of Tobacco Control. The World Bank, DC, USA (1999).
- 13 Hu T-W, Sung H-Y, Keeler T. Reducing cigarette consumption in California: tobacco taxes vs. an antismoking media campaign *Am. J. Public Health* 85(9), 1218–1222 (1995).
- 14 Townsend J, Roderick P, Cooper J. Cigarette smoking by socioeconomic group, sex, and age: effects of price, income, and health publicity. *BMJ* 309, 923–927 (1994).
- 15 Stead LF, Perera R, Bullen C, Mant D, Lancaster T. Nicotine replacement therapy for smoking cessation. *Cochrane Database Syst. Rev.* 1, CD000146 (2008).

shown to have an effect on the rate of loss of lung function [46,47], are also important secondary prevention strategies.

Tertiary prevention in COPD involves preventing the development of complications such as cor pulmonale or loss of skeletal muscle mass [48]. Effective interventions include long-term oxygen therapy and pulmonary rehabilitation.

Prevention has long been considered better than cure. Unfortunately, for many patients suffering from COPD today it is too late to prevent the development of disability and they must rely on the effective treatments currently available. However, there is real hope that reductions in smoking, improvements in working environments, changes in heating and cooking practices, and better nutrition and perinatal care will substantially reduce the burden of the disease for future generations around the world.

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- 16 Hughes JR, Stead LF, Lancaster T. Antidepressants for smoking cessation. *Cochrane Database Syst. Rev.* 1, CD000031 (2007).
- 17 Cahill K, Stead LF, Lancaster T. Nicotine receptor partial agonists for smoking cessation. *Cochrane Database Syst. Rev.* 3, CD006103 (2008).
- 18 Goodman N, Lane R, Rampling S. Chronic bronchitis: an introductory examination of exisiting data. *BMJ* 4830, 237–243 (1953).
- 19 Becklake M, Dupreez L, Lutz W. Lung function in silicosis of the Witwatersrand gold miner. Am. Rev. Tuberc. Pulm. Dis. 77, 400–412 (1958).
- 20 Becklake MR. Chronic airflow limitation: its relationship to work in dusty occupations. *Chest* 88(4), 608–617 (1985).
- 21 Blanc PD, Iribarren C, Trupin L *et al.* Occupational exposures and the risk of COPD: dusty trades revisited. *Thorax* 64(1), 6–12 (2009).
- 22 Harber P, Tashkin DP, Simmons M, Crawford L, Hnizdo E, Connett J. Effect of occupational exposures on decline of lung function in early chronic obstructive pulmonary disease. *Am. J. Respir. Crit. Care Med.* 176(10), 994–1000 (2007).
- 23 US Department of Health and Human Services. The health consequences of involuntary exposure to tobacco smoke: a report of the Surgeon General. Department of Health and Human Services, Atlanta, GA, USA (2006).

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- 24 Gold D, Wang X, Wypij D, Spizer F, Ware J, Dockery D. Effects of cigarette smoking on the pulmonary function in adolescent boys and girls. *N. Engl. J. Med.* 335, 931–937 (1996).
- 25 Skogstad M, Kjaerheim K, Fladseth G et al. Cross shift changes in lung function among bar and restaurant workers before and after implementation of a smoking ban. Occup. Environ. Med. 63(7), 482–487 (2006).
- 26 Eisner MD, Balmes J, Katz PP, Trupin L, Yelin EH, Blanc PD. Lifetime environmental tobacco smoke exposure and the risk of chronic obstructive pulmonary disease. *Environ. Health* 4(1), 7 (2005).
- 27 Eisner MD. Secondhand smoke and obstructive lung disease: a causal effect? *Am. J. Respir. Crit. Care Med.* 179(11), 973–974 (2009).
- 28 Goodman P, Agnew M, McCaffrey M, Paul G, Clancy L. Effects of the Irish smoking ban on respiratory health of bar workers and air quality in Dublin pubs. *Am. J. Respir. Crit. Care Med.* 175(8), 840–845 (2007).
- 29 Dennis R, Maldonado D, Norman S *et al.* Wood smoke exposure and risk for obstructive airways disease among women. *Chest* 109(1), 115–119 (1996).
- 30 Behera D, Jindal S. Respiratory symptoms in Indian women using domestic cooking fuels. *Chest* 100, 385–388 (1991).
- 31 Lopez AD, Mathers CD, Ezzati M, Jamison DT, Murray CJ. Global and regional burden of disease and risk factors: systematic analysis of population health data. *Lancet* 367(9524), 1747–1757 (2006) (2001).
- 32 Torres-Duque C, Maldonado D, Perez-Padilla R, Ezzati M, Viegi G. Biomass fuels and respiratory diseases: a review of the evidence. *Proc. Am. Thorac. Soc.* 5, 577–590 (2008).

- 33 Mannino DM, Buist AS. Global burden of COPD: risk factors, prevalence, and future trends. *Lancet* 370(9589), 765–773 (2007).
- 34 Anto JM, Vermeire P, Vestbo J, Sunyer J. Epidemiology of chronic obstructive pulmonary disease. *Eur. Respir. J.* 17(5), 982–994 (2001).
- 35 Barker DJ, Godfrey KM, Fall C, Osmond C, Winter PD, Shaheen SO. Relation of birth weight and childhood respiratory infection to adult lung function and death from chronic obstructive airways disease. *BMJ* 303, 671–675 (1991).
- 36 Shaheen S, Barker D, Holgate S. Do lower respiratory tract infections in early childhoo cause chronic obstructive pulmonary disease? *Am. J. Respir. Crit. Care Med.* 151, 1649–1652 (1995).
- 37 Lawlor D, Ebrahim S, Davey Smith G. Association of birth weight with adult lung function: findings from the British Women's Heart and Health Study and a meta-analysis. *Thorax* 60, 851–858 (2005).
- 38 Halpin DMG, Fehrenbach C, Bellamy D, Rudolf M. What does the general public know about COPD ? *Thorax* 57(Suppl. 3), iii45 (2002).
- 39 Weinman J, Yusuf G, Berks R, Rayner S, Petrie KJ. How accurate is patients' anatomical knowledge: a cross-sectional, questionnaire study of six patient groups and a general public sample. *BMC Fam. Pract.* 10, 43 (2009).
- 40 Seamark DA, Williams S, Timon S et al. Home or surgery based screening for chronic obstructive pulmonary disease (COPD)? Prim. Care Respir. J. 10(2), 30–33 (2001).
- 41 Van Schayck CP, Loozen JM, Wagena E, Akkermans RP, Wesseling GJ. Detecting patients at a high risk of developing chronic

obstructive pulmonary disease in general practice: cross sectional case finding study. *BMJ* 324(7350), 1370 (2002).

- 42 Lin K, Watkins B, Johnson T, Rodriguez JA, Barton MB. Screening for chronic obstructive pulmonary disease using spirometry: summary of the evidence for the U.S. Preventive Services Task Force. *Ann. Intern. Med.* 148, 535–543 (2008).
- 43 Shahab L, Jarvis MJ, Britton J, West R. Prevalence, diagnosis and relation to tobacco dependence of chronic obstructive pulmonary disease in a nationally representative population sample. *Thorax* 61(12), 1043–1047 (2006).
- 44 Bize R, Burnand B, Mueller Y, Rege Walther M, Cornuz J. Biomedical risk assessment as an aid for smoking cessation. *Cochrane Database Syst. Rev.* 2, CD004705 (2009).
- 45 Donaldson GC, Seemungal TA, Bhowmik A, Wedzicha JA. Relationship between exacerbation frequency and lung function decline in chronic obstructive pulmonary disease. *Thorax* 57(10), 847–852 (2002).
- 46 Celli BR, Thomas NE, Anderson JA *et al.* Effect of pharmacotherapy on rate of decline of lung function in chronic obstructive pulmonary disease: results from the TORCH study. *Am. J. Respir. Crit. Care Med.* 178(4), 332–338 (2008).
- 47 Tashkin D, Celli B, Senn S, Burkhart D, Kesten S, Menjoge S. A 4-year trial of tiotropium in chronic obstructive pulmonary disease. *N. Engl. J. Med.* 359, 1543–1554 (2008).
- 48 Decramer M, Rennard S, Troosters T et al. COPD as a lung disease with systemic consequences – clinical impact, mechanisms, and potential for early intervention. COPD 5(4), 235–256 (2008).