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Detecting mild COPD is not a waste of resources

Dear Sirs,

We thank Professor Enright and Dr White for their linked editorial[1] on our paper on early diagnosis and early treatment of COPD published in the March issue of the *PCRJ*.[2] We would like to examine their concerns.

As most patients with COPD are diagnosed late in the disease, we suggested that patients would be advantaged by earlier diagnosis – in terms of lung function severity, symptoms and exacerbations – rather than clinicians simply focusing on those patients who are already diagnosed (as Enright and White suggest).

We disagree that establishing borderline spirometric abnormality following case finding – i.e. identification of symptomatic and clinically impacting disease – is of little value in terms of clinical importance and as a means of enhancing smoking cessation. Evidence from three studies[3-5] suggesting that COPD diagnosis can be effective in increasing smoking cessation rates has been ignored.[1] In addition, any opportunity for further discussion with our patients to try to move them one step further in their behaviour change towards smoking cessation is valuable; but this is hard to assess in short-term limited-funding classical randomised trials in potentially biased sub-populations who are willing to undertake such studies. We believe this speaks to the ennui and the frustration of clinicians in providing smoking cessation counselling.

We agree that there is limited proven value in treating GOLD I COPD, but patients need assessment to diagnose more severe disease and to establish asthma differential diagnoses accurately. Asthma misdiagnosis due to the overlap of symptoms is common.[6,7] Leading questions about the presence of dyspnoea, cough, sputum, wheeze, or frequent respiratory infections in at-risk patients, can assist in identifying those patients requiring spirometry who have probable COPD.[8-10]

In addition, patients with undiagnosed GOLD I COPD may already show signs of impaired daily activities (HRQoL),[11] resulting in deconditioning and an increased risk of social isolation and depression[12,13] which necessitates intervention. COPD exacerbations occur in patients with milder COPD as well, and can present before COPD is diagnosed;[14] their prevention and appropriate treatment is essential.

Enright and White claim that GOLD II patients are based only on low FEV_1 values of 65-80% predicted, and should be described as either 'probably normal' or "mild restriction with a low FEV_1 and low FVC".[1] To our minds the diagnosis of COPD is based first on the FEV_1/FVC ratio being less than 0.7,[15] with the severity being dependent (as previously mentioned) on the FEV_1. Patients with mild restriction should not be labelled as COPD, because they do not have COPD…

Early identification is stated to be of no value because there is no treatment or good biochemical or clinical markers to allow measurement of COPD activity or disease progression.[1] Whilst we do not yet have any biomarkers predicting disease progression, there are clinical predictors (of disease progression) through increased frequency of exacerbations in those with the clinical phenotype of cough and sputum.[16]

Evidence for short-acting bronchodilator (SABD) use in symptomatic mild COPD is extrapolated from patients with more severe disease, but they continue to be included in all world guidelines.[15] The widespread use of SABD in these guidelines belies the lack of evidence. The use of long-acting bronchodilators (LABD) in GOLD stage II showed a small 6 ml improvement in FEV_1 annually on treatment with tiotropium, but this was additive to other therapies and not in a population without treatment.[17] Also, FEV_1 improvement does not capture all of the benefit of bronchodilators as evidenced by health status improvement and exacerbation reduction. There is no data to support the use of LABD in GOLD stage I disease.

The suggestion that there is no value in treating milder patients as they are more likely to die of a cardiac problem[1] seems very utilitarian especially since treating their lung disease may reduce cardiac events which are often associated with COPD exacerbations. In addition, since primary care practitioners take a holistic approach of the entire patient, we realise the importance of linked co-morbidities and the value of treating the whole patient.

In summary, it should be a part of all physicians' care to proactively assess symptomatic patients who are at risk of COPD, thereby hoping to make a diagnosis earlier; this allows earlier institution of both pharmacologic and non-pharmacologic therapies, education, potential interruption of the progressive nature of the illness, prevention of exacerbations, prevention of the development of co-morbidities, and improved patient quality of life. The more good reasons we can give patients to quit smoking, and the more we can relate these to them, the better our chance of assisting our patients in this goal. We feel that the evidence, as well as a common sense approach, clearly supports our position. The IPCRG has created a position statement[18] on early diagnosis of COPD which provides more information on this topic.

*Alan Kaplan[a], Daryl Freeman[b], Jennifer Cleland[b], Frank Cerasoli[c], David Price[b]

[a] Chair, Family Physician Airways Group of Canada, Richmond Hill, Ontario, Canada
[b] Centre of Academic Primary Care, University of Aberdeen, Aberdeen, UK
[c] Pfizer Inc, New York, NY USA

*Correspondence: E-mail: for4kids@gmail.com

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Conflicts of interest

For the full list of authors' conflicts of interest see original article (reference 2 below).

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Authors' reply

Wasteful wishful thinking

Dear Sirs,

We share the desire of Price, Kaplan and colleagues to optimise the treatment of patients with clinically-important COPD (with FEV_1 below 60% predicted), and we note the response from Jones,[1] and Kaplan and colleagues (above), to our editorial[2] which accompanied their original paper.[3] However, the fact that many patients with COPD are diagnosed late in the disease is not in itself a reason to diagnose the disease when it is mild. We disagree that we "must do something" to prevent mild COPD from becoming severe COPD. It is simply wishful thinking that we can confidently detect mild COPD or that any drug treatments for mild COPD are effective. Where is the evidence?

Can we use "abnormal" spirometry results in mild disease to convince smokers to quit? We and others have reviewed the studies of Bednarek *et al*.[4], Gorecka *et al*.[5], and Parkes *et al*.[6] in our editorial and elsewhere, and dispute the evidence.[2,7] If there is a positive effect, it must be very small.

The presence of respiratory symptoms or "exacerbations" (increased cough and phlegm) in patients with mild COPD does not "necessitate intervention" to prevent them. Where is the evidence that the benefits of daily drug therapy for mild COPD justify the costs and side-effects of these drugs?

We admit it was confusing to suggest that a patient with an FEV_1/FVC ratio above 0.7 might have COPD when clearly they do not have airway obstruction *per se*. Our intention was to point out that non-respiratory causes such as obesity and heart failure can reduce the FEV_1. We mentioned cardiac mortality to draw attention to the very point made by Kaplan and colleagues – which is that smokers with a low FEV_1 have all the other risks that smoking brings.

We all hope for new interventions that will improve the outlook of COPD, and that will do so early in the disease. But hope is not enough to justify prescribing drugs for "mild COPD" that have become the most costly in national prescribing budgets.[8] Time and money spent intervening in mild COPD (other than efforts to prompt smoking cessation) have no justification at present, a position confirmed in the recent joint guideline update of the American College of Physicians, American College of Chest Physicians, American Thoracic Society and the European Respiratory Society.[9] To do so is to engage in wasteful wishful thinking.

*Patrick White[a], Paul Enright[b]

[a] Department of Primary Care and Public Health Sciences, King's College London, London, UK.

[b] College of Public Health, The University of Arizona, Tucson, Arizona, USA

*Correspondence: E-mail: patrick.white@kcl.ac.uk

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