The National Human Rights Commission and the Nepal Medical Association—the umbrella organisation of Nepali medical doctors—showed strong solidarity, and on July 15, 2016, issued a press release showing concern over KC's deteriorating health and urging the government to honour their past agreements with KC.[4]

The increase in medical colleges—mostly private and urban-centric—has very little to do with the most remote and inaccessible communities. In a country where 80% of the population lives in villages,[1] medical training needs to focus on the rural population. Of the 19 medical colleges in Nepal in 2012, 14 (74%) were private and eight (43%) were in Kathmandu Valley alone, serving only 1·7 million—6% of the total population.[1,6] Of the 11 medical colleges outside the Valley, almost all were based in the cities, largely depriving health care from those living in rural regions of Nepal.[6]

The growth of urban-centric medical institutes does not come with a golden solution that could regulate norms and policies. The first step to revert this growing trend is to immediately consolidate the Medical Act of Nepal in line with the Mathema Committee report. The second step is to entirely depoliticise medical and academic institutions, enabling them to run under the principles of pure academia and humanitarian service. The third step is to immediately monitor the Nepalese Medical Council and university boards, and bring transparency to their decisions with stringent implementation of the rule of law.

We declare no competing interests.

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Chronic obstructive pulmonary disease: time to discuss new concepts

The adoption of precise concepts, decades ago, is one of the landmarks that has allowed enhancement of the knowledge about chronic obstructive pulmonary disease (COPD).[1,2] Chronic bronchitis is clinically characterised, emphysema is pathologically defined, and COPD is defined as a respiratory function abnormality. These classic definitions are still the foundations for appropriate clinical communication and scientific discussions.[3]

Although smoking remains the most important risk factor associated with COPD, exposure to biomass smoke in low-income countries carries an explosive potential to boost the incidence of the disease. Additionally, other risk factors have been associated with the condition, such as low socioeconomic status, ageing, respiratory infections, and genetic markers.[3]

Data from 2015–16 indicate that a substantial number of smokers and ex-smokers can experience respiratory symptoms, impaired quality of life, and episodes of exacerbation, despite spirometry results within the normal range.[4,5] These individuals can also display airway wall thickening and areas of emphysema on chest CT scans. Calling such individuals patients with COPD would be a serious conceptual mistake since the disease definition requires airflow limitation not completely reversible in spirometry.[3]

On the basis of this perspective, we propose to start a discussion with medical societies and experts in respiratory medicine about a novel definition for the state of lung disease associated with smoke inhalation of different causes.

We suggest a distinct concept named common smoke-related pulmonary disease (CSPD). CSPD would be defined as a preventable and treatable disease, associated with an enhanced inflammatory response in the airways and the lungs to long-term exposure to noxious particles or gases. The disease can be characterised by clinical symptoms, radiological abnormalities, or persistent airflow limitation and it is usually progressive. Exacerbations and comorbidities contribute to the overall severity in individual patients. The table shows a hypothetical classification of CSPD severity.

The suggested concept covers the definitions of COPD, chronic bronchitis, emphysema, and other

	FEV1/FVC	FEV1
I. Incipient*	≥70% or LLN	≥80% prediction
II. Mild	<70% or LLN	≥80% prediction
III. Moderate	<70% or LLN	<80% prediction ≥50% prediction
IV. Severe	<70% or LLN	<50% prediction ≥30% prediction
V. Very severe	<70% or LLN	<30% prediction

*Characterised by symptoms or radiological abnormalities. FEV1=forced expiratory volume in 1 s. FVC=forced vital capacity. LLN=lower limit of normality.

Table: **Hypothetical classification for disease severity in common smoke-related pulmonary disease**

alternative terminologies such as obstructive chronic bronchitis or small airways disease. The word common limits the definition to the morbid conditions most often associated with smoke exposure, in opposition to other smoking-related diseases such as respiratory bronchiolitis, idiopathic pulmonary fibrosis, or lung cancer. The proposed terminology also allows for incorporating future developments, considering genotypes and endotypes of the disease.

Regardless of the work ahead, it is essential to recognise for now that, particularly in medicine, applying the right words really matters.

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Severe asthma and asthma-chronic obstructive pulmonary disease syndrome

We read the excellent work by Sally Wenzel and colleagues (July 2, p 31)[1] with great interest. Treatment with dupilumab, a human anti-interleukin-4 receptor α monoclonal antibody, resulted in elevated forced expiratory volume in 1 s (FEV1), improved asthma symptom control, and reduced annual rates of exacerbation in patients with uncontrolled persistent asthma.

Severe asthma remains a challenge for patients and clinicians, with substantial unmet clinical need worldwide. This study established the role of anti-interleukin-4 pathway in the targeted therapy of difficult-to-control asthma. Severe asthma is defined by the requirement of high-dose inhaled corticosteroids and a second controller (or systemic corticosteroids) to prevent asthma from becoming uncontrolled, or the disease remains uncontrolled despite therapy in European Respiratory Society/American Thoracic Society (ERS/ATS) guidelines.[2] The guidelines further clarified the conditions attributed to uncontrolled asthma, including poor symptom control, frequent severe exacerbation, serious exacerbation, and airflow limitation. 384 of 755 patients enrolled in the study received high-dose inhaled corticosteroids plus long-acting β_2 agonist with the overall mean FEV1 of 60·77.[1] These data suggested that many patients had treatment-resistant asthma characterised by persistent airflow limitation, which was a concern as the definition of this subgroup of patients in the ERS/ATS guidelines overlaps with asthma-chronic obstructive pulmonary disease (COPD) syndrome. The latter disorder is defined by persistent airflow limitation with several features shared with both asthma and COPD in the Global Initiative for Asthma report.[3] In this case, a portion of participants eligible for this severe asthma study might be asthma-COPD syndrome patients and hence, subgroup analysis is required. Evidence of specific therapeutic approaches for severe asthma on asthma-COPD syndrome is still scarce, including treatment with monoclonal anti-IgE antibody (omalizumab), anti-interleukin-5 monoclonal antibodies (mepolizumab and reslizumab), antifungal treatments, and bronchial thermoplasty. Dupilumab was effective in severe asthma patients irrespective of baseline eosinophil count, implying patients with asthma-COPD syndrome will probably benefit from this specific immune therapy, especially in those with the asthma component of asthma-COPD syndrome. In a few studies with small sample size, patients with asthma-COPD syndrome treated with omalizumab had significantly lower rates of exacerbation, less hospitalisation, and better asthma control test score than at baseline.[4,5] These results need to be supported by further studies.

Clinicians do not have the capacity to distinguish asthma-COPD syndrome from severe asthma using a phenotype-based definition. Clinical trials and mechanism studies are imperative to identify the biomarkers and endotypes of asthma and COPD, and to facilitate the precise management of the two diseases.

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