

- to 23 potentially modifiable risk factors in China: a comparative risk assessment. *Lancet Glob Health* 2019;7:e257–e269.
- Vieira AR, Abar L, Vingeliene S, Chan DSM, Aune D, Navarro-Rosenblatt D, et al. Fruits, vegetables and lung cancer risk: a systematic review and meta-analysis. *Ann Oncol* 2016;27:81–96.
 - Wang X, Ma K, Cui J, Chen X, Jin L, Li W. An individual risk prediction model for lung cancer based on a study in a Chinese population. *Tumori* 2015;101:16–23.
 - Kipnis V, Midthune D, Freedman LS, Bingham S, Schatzkin A, Subar A, et al. Empirical evidence of correlated biases in dietary assessment instruments and its implications. *Am J Epidemiol* 2001;153:394–403.
 - Schatzkin A, Kipnis V. Could exposure assessment problems give us wrong answers to nutrition and cancer questions? *J Natl Cancer Inst* 2004;96:1564–1565.
 - Schatzkin A, Kipnis V, Carroll RJ, Midthune D, Subar AF, Bingham S, et al. A comparison of a food frequency questionnaire with a 24-hour recall for use in an epidemiological cohort study: results from the biomarker-based Observing Protein and Energy Nutrition (OPEN) Study. *Int J Epidemiol* 2003;32:1054–1062.

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Definition of Chronic Obstructive Pulmonary Disease: Occupational Environmental Contribution

To the Editor:

The recent proposal to reclassify chronic obstructive pulmonary disease appropriately identifies environmentally related disease (1). However, in addition to biomass and pollution exposure, the critical role of occupation in the development (2) and worsening (3) of chronic obstructive pulmonary disease should be acknowledged. ■

Author disclosures are available with the text of this letter at www.atsjournals.org.

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References

- Celli B, Fabbri L, Criner G, Martinez FJ, Mannino D, Vogelmeier C, et al. Definition and nomenclature of chronic obstructive pulmonary disease: time for its revision. *Am J Respir Crit Care Med* 2022;206:1317–1325.
- Blanc PD, Annesi-Maesano I, Balmes JR, Cummings KJ, Fishwick D, Miedinger D, et al. The occupational burden of nonmalignant respiratory diseases. An official American Thoracic Society and European

- Respiratory Society statement. *Am J Respir Crit Care Med* 2019;199:1312–1334.
- Harber P, Tashkin DP, Simmons M, Crawford L, Hnizdo E, Connett J; Lung Health Study Group. Effect of occupational exposures on decline of lung function in early chronic obstructive pulmonary disease. *Am J Respir Crit Care Med* 2007;176:994–1000.

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Chronic Obstructive Pulmonary Disease Definition: Is It Time to Incorporate the Concept of Failure of Lung Regeneration?

To the Editor:

We applaud the proposal of Celli and colleagues (1) to provide an updated definition of chronic obstructive pulmonary disease (COPD), driven by the slow progress of therapeutic interventions to decrease morbidity and mortality. This authoritative group of experts focused on two main limitations of the previous COPD definition: 1) the lack of identification of the disorder at its early stages in the absence of flow limitation; and 2) the consideration of COPD as a single disease despite diverse causes other than cigarette smoking. The proposed solutions are aimed to encourage novel treatments and translational studies: 1) incorporating into the definition objective early computed tomography (CT) scan changes; and 2) describing the heterogeneity of COPD according to its recognizable causes. We noted that the revised definition of COPD addressed a clinician's typical point of view, probably with the same basic intentions of the Global Initiative for Chronic Obstructive Lung Disease guidelines. Will this be enough to describe the essential nature of COPD and particularly to stimulate more efficacious therapeutic interventions? We wish all the best for this attempt, but we argue that a change of paradigm (e.g., regenerative pathways) is desirable to drive novel therapeutic approaches. It was only in 2012 that the first demonstration of adult lung growth in humans by a multidisciplinary team of investigators focused on translational bench-to-bedside medicine (2). Now, we have abundant evidence that the lung, the organ of our body most widely exposed to the external environment, has extensive regenerative ability to respond to most injuries, rapidly regenerating damaged tissue (3, 4). COPD is characterized by both distal airways and parenchymal remodeling, which may be practically considered as due to failed regenerative processes. Recently, the highly talented interdisciplinary biomolecular investigators led by Ed Morrissey found that endothelial and mesenchymal cells in patients with COPD have different gene expression patterns from healthy individuals. In particular, they showed that the distal airway multipotent respiratory airway secretory (RAS) cells, usually able to regenerate alveoli in humans by differentiating into alveolar type II epithelial (ATII) pneumocytes cells, follow an aberrant differentiation trajectory leading to the accumulation of RAS-to-ATII transitioning

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