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References

1. Kahn JM, Andersson L, Karir V, Polissar NL, Neff MJ, Rubenfeld GD. Low tidal volume ventilation does not increase sedation use in patients with acute lung injury. *Crit Care Med* 2005;33:766–771.
2. Protti A, Andreis DT, Monti M, Santini A, Sparacino CC, Langer T, *et al.* Lung stress and strain during mechanical ventilation: any difference between statics and dynamics? *Crit Care Med* 2013;41:1046–1055.
3. Webb HH, Tierney DF. Experimental pulmonary edema due to intermittent positive pressure ventilation with high inflation pressures. Protection by positive end-expiratory pressure. *Am Rev Respir Dis* 1974;110:556–565.
4. Loring SH, O'Donnell CR, Behazin N, Malhotra A, Sarge T, Ritz R, *et al.* Esophageal pressures in acute lung injury: do they represent artifact or useful information about transpulmonary pressure, chest wall mechanics, and lung stress? *J Appl Physiol (1985)* 2010;108:515–522.
5. De Santis Santiago R, Teggia Droghi M, Fumagalli J, Marrazzo F, Florio G, Grassi LG, *et al.*; Lung Rescue Team Investigators. High pleural pressure prevents alveolar overdistension and hemodynamic collapse in ARDS with class III obesity. *Am J Respir Crit Care Med* 2020;203:575–584.
6. Terragni PP, Rosboch G, Tealdi A, Corno E, Menaldo E, Davini O, *et al.* Tidal hyperinflation during low tidal volume ventilation in acute respiratory distress syndrome. *Am J Respir Crit Care Med* 2007;175:160–166.
7. Goligher EC, Costa ELV, Yarnell CJ, Brochard LJ, Stewart TE, Tomlinson G, *et al.* Effect of lowering Vt on mortality in acute respiratory distress syndrome varies with respiratory system elastance. *Am J Respir Crit Care Med* 2021;203:1378–1385.
8. Amato MB, Meade MO, Slutsky AS, Brochard L, Costa EL, Schoenfeld DA, *et al.* Driving pressure and survival in the acute respiratory distress syndrome. *N Engl J Med* 2015;372:747–755.
9. Cavalcanti AB, Suzumura ÉA, Laranjeira LN, Paisani DM, Damiani LP, Guimarães HP, *et al.*; Writing Group for the Alveolar Recruitment for Acute Respiratory Distress Syndrome Trial (ART) Investigators. Effect of lung recruitment and titrated positive end-expiratory pressure (PEEP) vs low PEEP on mortality in patients with acute respiratory distress syndrome: a randomized clinical trial. *JAMA* 2017;318:1335–1345.

Asthma and Chronic Obstructive Pulmonary Disease: Just Old Friends or Relatives?

To the Editor:

There is broad consensus that asthma and tobacco smoke–related chronic obstructive pulmonary disease (COPD) are clinically, immunologically, and histopathologically distinct. However, recent evidence identified different trajectories that lead to chronic airflow limitation at different times of one's life, starting from childhood (1). It is now well established that prenatal, perinatal, and early childhood factors affect lung development and function (2). The more we learn about these common etiologies, the more studies aimed at digging into the first years of life are needed to shed light on the roots of the structural and functional changes associated with airflow limitation.

We have read with great interest the study from Izadi and colleagues. By further analyzing the Childhood Asthma Management Program, an initial drug trial turned to a well-defined, long-term, ongoing cohort study. Izadi and colleagues unravel some knowledge gaps in the nature of childhood factors associated with persistent severe asthma in adult life (3). These authors reported that only two determinants among more than 22 explored, namely, reduced lung growth in childhood and maternal smoking during pregnancy, were predictors of persistent severe asthma later in life. Interestingly, this association was not maintained if the subjects had normal lung growth in childhood even in the presence of early lung function decline, suggesting that lung function in the first years of life is a major determinant of chronic airflow limitation regardless of how the lung function is going to develop and/or decline. These findings shed light on the gray transition area in which patients with asthma become progressively irreversible until they meet the spirometric criteria for COPD. Large cohort studies had previously found irreversible airflow obstruction to be associated with low lung function at birth, especially when modifiable early-life exposure such as first- or second-hand smoke, including maternal exposures, are present (4). Also, irreversible airflow obstruction compatible with COPD is common in those with severe asthma (5). Thus, this study serves as a bridge between early origins of asthma and COPD.

From a pathobiological standpoint, inflammatory processes contribute to small airway obstruction in both asthma and COPD. The nature of these processes and the extent to which they overlap in both conditions or evolve into each other are not well understood (6). We have missed for years the opportunity to explore, especially by longitudinal imaging, the lungs from younger subjects before they develop any spirometric criteria for COPD and/or after they surpassed the critical age to be studied as patients with asthma.

Altogether, these studies aimed at profiling the causes and the evolution of airflow obstruction are slowly shifting the focus toward younger individuals who have been so far overlooked, as COPD was wrongly considered a disease of the elderly. These long-term (life-long?) cohorts are crucial to further understand the determinants of lung function from birth; how small airway obstruction shapes into asthma, COPD, or both; and the potential long-term implications of these factors for the development of COPD later in life.

Here and now, there is a brand new window of opportunity to tackle the multifaceted aspects of airflow limitation. Let's keep this window wide open to promote lung health starting from birth. ■

Supported by NIH/NHLBI grants HL149744 and HL132523 (F.P.).

Originally Published in Press as DOI: 10.1164/rccm.202106-1358LE on June 30, 2021

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

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References

1. Celli BR, Wedzicha JA. Update on clinical aspects of chronic obstructive pulmonary disease. *N Engl J Med* 2019;381:1257–1266.
2. Martinez FD. Early-life origins of chronic obstructive pulmonary disease. *N Engl J Med* 2016;375:871–878.
3. Izadi N, Baraghoshi D, Curran-Everett D, Zeiger RS, Szefler SJ, Covar RA; Childhood Asthma Management Program Research Group. Factors associated with persistence of severe asthma from late adolescence to early adulthood. *Am J Respir Crit Care Med* [online ahead of print] 2021 May 24; DOI: 10.1164/rccm.202010-3763OC.
4. Bui DS, Lodge CJ, Burgess JA, Lowe AJ, Perret J, Bui MQ, *et al.* Childhood predictors of lung function trajectories and future COPD risk: a prospective cohort study from the first to the sixth decade of life. *Lancet Respir Med* 2018;6:535–544.
5. Tai A, Tran H, Roberts M, Clarke N, Wilson J, Robertson CF. The association between childhood asthma and adult chronic obstructive pulmonary disease. *Thorax* 2014;69:805–810.
6. Soriano JB, Polverino F, Cosio BG. What is early COPD and why is it important? *Eur Respir J* 2018;52:1801448.