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Statement of Interest: None declared.

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From the authors:

We would like to thank M. Filippone and co-workers for their comments regarding our paper, “Lung-specific inactivation of CCAAT/enhancer binding protein α causes a pathological pattern characteristic of COPD” [1]. We share their concern regarding the lack of human studies addressing the adult phenotype of former bronchopulmonary dysplasia (BPD) patients, and hope to continue this discussion. M. Filippone and co-workers address the difference between “old” and “new” BPD phenotypes, which warrants further discussion with regards to the findings presented in our paper. Old BPD, characterised by intense bronchial inflammation and massive parenchymal disruption in relatively immature newborns with surfactant deficiency, is greatly believed to be a consequence of oxygen ventilator treatment [2]. In contrast, new BPD, as M. Filippone and co-workers state, involves extremely premature neonates that consequently demonstrate disrupted lung development resulting in immature lungs, vulnerable to multiple injuries. In our publication, when comparing the phenotype of mice with lung-specific inactivation of CCAAT/enhancer binding protein α (*Cebpa*^{ΔLE}) presenting BPD pathology, we were referring to new BPD that, like in *Cebpa*^{ΔLE} mice, is characterised by immature lung morphology. That is, a structural simplification with fewer, larger alveoli causing reduced surface available for gas exchange; a dysmorphic vascularity; and very limited, if any, inflammation [2].

One prominent difference between adult and neonatal or young *Cebpa*^{ΔLE} mice is the development of a spontaneous inflammation as the mice reaches adulthood. The origin of this inflammation remains unknown; however, the spontaneous inflammation in our animals presents an opportunity to study the sensitivity of a lung with a morphological resemblance of new BPD to inflammation. There is a possibility, as mentioned by M. Filippone and co-workers, that the pathology of long-term survivors of new BPD includes an inflammatory component

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not triggered by external factors, such as tobacco smoke, which could contribute to the development of an obstructive lung disease. Although, further investigations in long-term survivors of BPD need to be conducted to investigate the presence of an inflammation before such conclusions can be drawn. However, if there is a spontaneous inflammation in long-time survivors of BPD, this might provide a valuable opportunity for treatment, as it might prove beneficial to suppress this inflammation to prevent development of chronic obstructive pulmonary disease. Finally, these findings are from an animal model and, in agreement with M. Filippone and co-workers, we urge further clinical studies to address the link between respiratory problems early in life and respiratory complications, such as chronic obstructive pulmonary disease, later in life.

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