Reviewer 2 v.1

Comments to the Author

In this review, authors described the clinical manifestations of BPD, the pathophysiology of this disease and the arguments in favor of the role of BPD in further development of COPD in adults. This review summarizes the current knowledge on the subject.

Page 5 line 14-16 : I agree that BPD may be associated to pulmonary hypertension but it is important to detail that most of the time this is due to chronic hypoxemia leading to pulmonary vessels vasoconstriction in combination with a real vascular disease in the more preterm babies, and with a potential aggravating role of persistent ductus arteriosus that may add an overflow in pulmonary arteries. Nevertheless, I do not understand why the authors evoke the role of LV dysfunction, intracardiac shunts or pulmonary venous stenosis especially in this setting of preterm birth and BPD.

Page 5 line 30 : I would precise “the majority of infants on supplemental oxygen when discharged home are weaned to room air by 12 months of age and the median age of being weaned from home mechanical ventilation is 25 months.” If not, there is a risk for the reader to assume that this data relate to all preterm babies that develop BPD.

Page 6, line 50-53: “In general, it is not known whether any specific asthma or COPD risk genes are more likely to be present in children with BPD, particularly severe BPD”. The authors should replace “genes” by “genes variants” because all individuals have the same genes but with different possible variations.

Page 7 line 12-17: “Identification of SNPs in BPD will help to identify risk genes and provide mechanistic insights into disease severity and progression in BPD, and potentially have relevance for COPD.” Again gene is not the proper word and should be replace by “alleles” here.