association. Further, nearly all published studies uniformly recommend that the inclusion of atelectasis as a cause of postoperative fever be discarded as historical conjecture and unsupported dogma. Mavros and colleagues\(^1\) conclude that there is a need for additional large studies to precisely evaluate whether there is an association that the prior studies have not detected. We respectfully disagree, given the findings of our most recent study. From our perspective, based on our large cohort of patients and the outcome of our study, rather than performing additional studies, it would seem more prudent and appropriate to simply discard old dogma.

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Response
To the Editor:

We thank Drs Kane and Backer for their correspondence on our recent article, which contributed to the discussion of the potential association between postoperative fever and atelectasis.\(^1\) The authors conducted a retrospective cohort study, examining the potential association between postoperative fever and atelectasis in pediatric patients.\(^2\) They concluded that no such association existed, and, thus, postoperative fever should not be attributed to atelectasis.

To our knowledge, a total of three studies, including the one by Drs Kane and Backer,\(^2\) have specifically examined this association. In their correspondence, Drs Kane and Backer suggested that no further studies are necessary to confirm the lack of association between atelectasis and postoperative fever and that this old idea should simply be discarded. Unfortunately, it is not so rare in clinical practice for clinicians to choose to follow their own rationale rather than what is suggested by the published evidence; in fact, several such cases have been published so far.\(^3,4\)

Regarding the potential association of atelectasis and postoperative fever, however, all the published studies have certain limitations, including methodologic concerns and a relatively small sample size. This fact does not allow, at least in our point of view, for firm conclusions to be drawn. Although the rather limited evidence implies no association between atelectasis and postoperative fever, we believe that future well-designed and well-conducted studies will be useful in reaching a safe conclusion, and convincing even the most doubtful minds.

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Montelukast for Bronchiolitis Obliterans Syndrome After Lung Transplantation

To the Editor:

We read with interest the review by Todd and Palmer\(^1\) published in a recent issue of CHEST (August 2011). It gives an excellent overview of the pathogenesis of bronchiolitis obliterans syndrome after lung transplantation, placing emphasis on future clinical translational treatment directions.

Montelukast for Bronchiolitis Obliterans Syndrome After Lung Transplantation

To the Editor:

We read with interest the review by Todd and Palmer\(^1\) published in a recent issue of CHEST (August 2011). It gives an excellent overview of the pathogenesis of bronchiolitis obliterans syndrome after lung transplantation, placing emphasis on future clinical translational treatment directions.
Although their review is thorough, we have missed one important translational development, namely, the leukotriene B4 pathway in the pathophysiology of bronchiolitis obliterans syndrome after lung transplantation. Leukotriene B4 is a lipid mediator that has been shown to have potent chemotactic activity for effector T lymphocytes mediated through its receptor, BLT1. In a murine model, Medoff and colleagues have demonstrated that BLT1 controls effector CD8+ T-cell trafficking into the lung and that BLT1-mediated CD8+ T-cell recruitment plays an important role in the development of airway fibroproliferation and obliteration. Indeed, in human lung transplant recipients, BLT1 is upregulated on T lymphocytes isolated from the airways of patients with bronchiolitis obliterans. Montelukast, a drug used for the treatment of asthma, inhibits leukotriene activity. In a pilot study of 11 patients with bronchiolitis obliterans syndrome after lung transplantation, the addition of montelukast to immunosuppressive drugs decreased FEV₁ decline after 6 months of treatment compared with a retrospective cohort of 11 patients. Intervening in the leukotriene B4 pathway through the addition of montelukast may thus be another promising research direction with clinically relevant treatment implications for bronchiolitis obliterans syndrome after lung transplantation.

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Response
To the Editor:

We thank Drs Simons and Reijers for their thoughtful correspondence regarding our recent review of bronchiolitis obliterans syndrome after lung transplantation. As they point out, emerging basic and clinical evidence suggests that manipulation of leukotriene B4 and its receptor BLT1 could be beneficial in bronchiolitis obliterans syndrome. We acknowledge that in order to maintain a concise submission, our review favored more mature lines of translational research and novel clinical therapeutics employed in larger cohorts of patients. As such, many preliminary yet promising areas of mechanistic and translational research were not discussed.

We, too, are intrigued by the basic observation that BLT1 receptor inhibition or deficiency results in attenuated effector T cell recruitment, airways epithelial damage, and obliterative airways disease in a variety of murine models. This work certainly provides a strong rationale for clinical trials of leukotriene receptor antagonists in patients with bronchiolitis obliterans syndrome who have received lung transplants. In our opinion, however, the clinical work to date in this area is still too preliminary to draw any definite conclusions. The clinical study noted by Simons and Reijers only included 11 patients, used historic controls, and had a relatively short follow-up time. We look forward to further prospective studies to confirm these promising initial results. Such well done prospective studies are critical to translate these and other exciting basic observations into meaningful clinical interventions and improved outcomes for lung transplant recipients.

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All That Glitters Is Not Gold in Pursuing the Diagnosis of Pulmonary Embolism

To the Editor:

We read with great interest the study by Moores and colleagues published in a recent issue of CHEST (August 2011). It gives an up-to-date, balanced overview of the approach to the diagnosis of acute nonmassive pulmonary embolism.

In their review, they elaborately discuss the benefits of clinical decision rules and D-dimer testing, making a compelling argument that the high sensitivity of D-dimer testing makes this test especially attractive for ruling out pulmonary embolism. They