and amphetamine) share the common ability to serve as potent substrates for SERT proteins. Stimulants that are not associated with IPAH, such as amphetamine and phentermine,5 are weak substrates for SERT.6 Chin et al1 correctly pointed out that our in vivo findings showed that d-amphetamine and METH are much more potent dopamine and norepinephrine transporter substrates when compared to their activities as SERTs. For example, the concentrations of d-amphetamine and METH as dopamine transporters having 50% of the effect compared to the control are approximately 25 nmol/L, whereas their corresponding values as SERTs are 1.785 and 736 nmol/L, respectively. Such data predict that pharmacologic doses of these drugs should not release 5-hydroxytryptamine (HT) in vivo. Consistent with this prediction, doses of d-amphetamine that elevate extracellular dopamine levels in the rat brain do not elevate extracellular 5-HT levels in nervous system tissue7 and do not increase 5-HT levels in plasma.8 In marked contrast, METH administration produces similar elevations in extracellular dopamine and 5-HT levels in the rat brain, and also increases plasma 5-HT levels.7,8 Our data indicate that, in the case of METH, the profile of transporter activity determined in vitro does not predict the profile of activity in vivo.

It should be noted that the stimulant medications currently being prescribed for the treatment of attention deficit disorder (eg, amphetamine, d-amphetamine, and methylphenidate) and appetite control (eg, phentermine, diethylpropion, and phendimetrazine) have minimal activity at SERTs and are taken at low oral doses, substantially reducing the possibility they will interact with SERT sites in human patients. Illicit METH, on the other hand, is often self-administered by smoked or IV routes where very high levels of drug would be expected to interact with SERTs in the brain and periphery. These considerations explain in part why METH, but not currently prescribed stimulants, might increase the risk of IPAH.

In summary, our data indicate that METH is a SERT substrate in vivo. In conjunction with the data reported by Chin et al,1 the evidence provides further support for the hypothesis that SERT substrate activity is an important factor contributing to an increased risk of IPAH.4 In light of these findings, it will be essential to determine whether the recreational use of other illicit SERT substrates like 3,4-methylenedioxymethamphetamine (ecstasy) increases the risk of IPAH.10

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Dexamethasone To Prevent Postbronchoscopy Fever in Children

To the Editor:

With interest we read the publication of Picard et al,1 who demonstrated that the prevalence of postbronchoscopic fever in children can be reduced by administration of dexamethasone. The study is well designed, and the results are convincing. We agree that postbronchoscopic fever is uncomfortable, occurs frequently, and sometimes causes distress with parents and physicians. However, one could argue that administration of 0.5 mg/kg of dexamethasone to avoid such distress is not necessary.

In our practice, we instruct parents that they should expect their child to be a little feverish in the 12 to 24 h following bronchoscopy, that this is normal, and that they only should contact a doctor when the fever persists for > 24 h or the child becomes ill. With that policy, parents contacted us in < 1% of cases (> 600 bronchoscopies in 10 years). Two of these patients were thought to have an infection for which antibiotic treatment was started.

Alternatively, one could prescribe oral antipyretics after the procedure if fever occurs, rather than immunosuppressing drugs. There is no proof that immunosuppression prevents any long-term sequelae, and there remains the small risk of bacteremia, especially in patients with chronic lung disease or immunologic disorders.2

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Promises and Limitations of the Bronchoscopic Microsampling Probe

To the Editor:

We read with interest the study by Sasabayashi et al in CHEST (February 2007) demonstrating the capacity of the bronchoscopic microsampling (BMS) probe to recover bacteria from suspensions. We agree that the BMS probe may be a promising tool for bacterial sampling compared to the actual standard BAL. Indeed, the variability of bacterial recovery by BAL is a major problem that has been underlined by a 2005 study showing that, after correction of the dilution of epithelial lining fluid by BAL, the bacterial count could vary up to 130-fold! However, although we ourselves believe in applications of the BMS probe such as the direct recovery of epithelial lining fluid for proteomic analysis, which we achieved with success, and although it has also been deemed promising in a recent review in CHEST, the BMS probe presents limitations that the authors fail to discuss.

While a bacterial recovery procedure using the BMS probe is less invasive than BAL, it still requires bronchoscopy and is not as uninvasive as blind techniques such as mini-BAL or protected-specimen brush. We have developed such a method in an animal study with the BMS probe inserted blindly through a catheter (Combicath; KOL Bio Medical Instruments; Chantilly, VA) that is routinely used for performing mini-BAL, although it has yet to be confirmed in a clinical setting. Furthermore, although the use of bronchoscopy may be an advantage in guiding the BMS probe toward an infected lung segment, it limits the widespread use of the probe compared to simple diagnostic methods such as quantitative cultures of endotracheal aspirates. Additionally, with the BMS probe it is impossible to explore great portions of the lung. Lung diseases such as pneumonia or ARDS are far from homogeneous, and the use of the BMS probe for diagnosis could lead to the failure of specimen recovery just as the BMS probe failed to recover detectable amounts of a lung inflammation marker (procollagen type III peptide) in ARDS patients. Further studies will define the clinical usefulness of the BMS probe.

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1414

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