regard, the recent article by Yellowlees and Kalucy (Chest 1990; 97:626-34) and the accompanying editorial by Gorman (Chest 1990; 97:514-15) were of great interest. With respect to the apparent efficacy of anti-depressant medications in asthmatic patients, however, it is important to consider the antihistaminic properties (which are not widely appreciated) that these agents possess.

Tricyclic antidepressant drugs are extraordinarily potent H1 and H2 antihistamines. Doxepin is approximately 775-fold more potent an H1 inhibitor than diphenhydramine on a molar basis.1 Anxiolytic properties of commonly used H1 antihistamines are well known. Antihistamines given to asthmatic patients can promote dose-related bronchodilatation and attenuate immediate bronchospastic responses to inhaled allergen and exercise.2,34 An increasing therapeutic role for H1 antihistamines in asthma has been suggested with recent availability of agents that are nonsedating and lack significant anticholinergic effects.3,4

It is important for both asthma and psychiatry/psychology subspecialists to be aware of the dual actions of antidepressant/antihistamine drugs—potentially effective in altering mood and also contributing to histamine-mediated responses. Ideally, the design of therapeutic trials using these agents for asthma should entail methods to identify these separate components of favorable response.

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To the Editor:

I basically support Dr Lang's comments regarding the antihistamine effects of tricyclic antidepressants and the need to separate these effects out from any possible central effects in clinical trials. It is my own view, however, that the primary effect of tricyclics is centrally mediated. At a clinical level, if in fact a major effect is due to the antihistamine action of tricyclcs, then one would expect respiratory function to improve very rapidly (within a matter of a few hours) after administration. This is not in fact what one finds, and it is generally necessary to persist with the antidepressants. The clinical improvement in asthma is often not seen for about a week or two. This, of course, is similar to the time scale for the central antidepressant action of the tricyclics. My views on the primarily central action of the antidepressant in improving asthma are supported by Dr Gorman's comments in his editorial. Dr Gorman cites the case of a patient with asthma who was put on a regimen of monoamine oxidase inhibitors (MAOIs), with which one would assume that any therapeutic effect on asthma was due to centrally mediated processes. I have also used MAOIs very cautiously in several asthmatic patients and have found the same sort of effects that Dr Gorman described. I do not, however, use MAOIs as a first-rank treatment in anxious or depressed asthmatic patients because of the potentially serious interactions. My final comment about Dr Lang's letter is that it is pleasing to see research interest and thinking being directed at this major clinical problem that besets all clinicians who work with asthmatic patients. There is a clear need for the sort of questions that Dr Lang asks to be investigated.

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Adenosine Deaminase and Lymphocytic Populations

To the Editor:

We read with interest the article by Fontes Baganha et al1 entitled "Serum and Pleural Adenosine Deaminase: Correlation with Lymphocytic Populations," which appeared in the March 1990 issue of Chest. Coincidentally, we reported in the May 1990 issue of the Journal of the Medical Association of Thailand the results of our study on the association of adenosine deaminase (ADA) activity with T-lymphocytes and subsets in pulmonary tuberculosis and bronchogenic carcinoma.2 To a major extent, both articles are based on similar working hypotheses. Therefore, in this short communication, we would like to draw attention to relevant and controversial data contained in both reports.

Results of analysis of serum ADA in both studies agree that serum ADA activity in the tuberculosis group (29.8 ± 10.0 U/L1 and 36.84 ± 10.9 U/L) was higher than that in the neoplasm group (14.5 ± 4.0 U/L1 and 27.85 ± 19.87 U/L). To our surprise, however, Fontes Baganha et al report that the serum ADA levels, when compared with normal values (21.3 ± 7.06 U/L), showed a significant elevation in the tuberculosis group and a decrease in the neoplasm group; whereas in our series, both patient groups showed higher values than the normal controls (19.45 ± 4.75 U/L), with slightly less prominence in the neoplasm group.

At this point, we would like to make note of information that is lacking on the types of diseases (especially neoplasms) and on the clinical condition of the subjects in the series of Fontes Baganha et al. Being aware of the variable degrees of depressed immunologic responses among patients with active tuberculosis2 and among patients with different neoplasms,3,4 we think that it is preferable to provide detailed descriptions of the subjects in order to avoid perplexing data derived from unspecified heterogeneous materials.

Local ADA activity at the sites of the pathologic affliction has been of more interest. That Fontes Baganha et al found higher ADA levels in tuberculous effusion (110.6 ± 35.2 U/L) than in neoplastic effusion (17.5 ± 8.4 U/L) is in agreement with the findings of others.5,6 Likewise, we have repeatedly shown increased ADA activity in bronchoalveolar lavage (BAL) fluids from lung with active tuberculosis compared with BAL fluids from cancerous lung and healthy lung.1,4,5

With regard to the study on lymphocytic populations, Fontes Baganha et al reported that the data comparing the two patient groups (data from normal controls were not shown) reveal higher percentages of CD4 T-cells in the group of patients with tuberculosis and higher percentages of CD8 T-cells in the group of patients with neoplasms, in both the blood and the pleural exudates. They were impressed by the conspicuously increased percentage of CD4 T-cells in tuberculous pleural effusions, which showed a positive correlation with pleural ADA levels; they proposed that ADA constitutes a marker of cell-mediated immune activity.

In contrast, the highlight of our report was the disclosure of an increase in ADA activity together with an increase in the percentage
of T-cells bearing interleukin-2 receptor (Tac cells) in the peripheral blood and BAL fluids only in patients with active pulmonary tuberculosis.

Since both ADA activity and T-cells bearing IL2 receptor are evidence of T-lymphocyte activation, both parameters may be used as indicators of a recent or currently active immunopathologic process in patients with pulmonary tuberculosis. In this regard, the conclusions of both studies adduce the same implications.

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Pleuroscopy—An Underestimated Diagnostic Procedure in Pleural Effusion

To the Editor:

We were pleased and interested to hear about the study by Bovornkitti et al. Both their work and ours are in the same area of investigation, ADA activity and lymphocyte populations in patients with tuberculosis and bronchogenic carcinoma.

However, the material used was considerably different in the two studies despite the overlapping etiologies—pleural tuberculosis and neoplastic effusions in our group and bronchial carcinoma and pulmonary tuberculosis in the work by Bovornkitti et al. The aim of our work was essentially the study of ADA and lymphocyte populations in pleural effusions and in peripheral blood, while Bovornkitti et al. studied those factors in the peripheral blood and BAL fluids.

Our group investigated the eventual correlations between variations in ADA activity and lymphocyte populations in pleural tuberculosis effusions (without parenchymatous pathologic changes) and in neoplasms (bronchogenic squamous cell carcinoma and adenocarcinoma), not specifically the function of the degree of pleuropulmonary involvement by the tuberculosis process or the histologic type of the tumors. Thus, the local activity of ADA and its correlation with the lymphocyte populations in the two studies was examined in different areas—the pleura and the bronchopulmonary structure. As a matter of fact, the differences observed in the local immunologic interventional capacity because of the existence or absence of immunologic structures, possibly only to local sequestration of immunocompetent cells with or without peripheral depletion, and major or minor cellular dynamic difficulties in these two areas may represent, among other factors, important conditioning factors of distinct immunologic responses in a given compartment. Therefore, these differences in the two studies may explain the variations detected in the findings. Our work was concerned with the lymphocyte populations CD4 and CD8, not only locally in immunologically different areas but also in the peripheral blood, on account of the eventual blood repercussions resulting from the local changes.

Finally, I was very interested in the correlation detected by Bovornkitti et al. between the increase in ADA activity and the rate of occurrence of T-cells bearing interleukin-2 (Tac cells) not only in BAL fluids but also in peripheral blood in patients with tuberculosis, which seems to point in a parallel way to the conclusions of our group concerning the correlation of ADA activity and the CD4 lymphocyte population.

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