LETTERS TO THE EDITOR

Effect of N-acetyl cysteine on thiol levels

We read with interest the article by Dr Bridge-
man and colleagues on the effect of oral N-acetyl cysteine (NAC) on thiol levels in
epithelial lining fluid (ELF) and lung tissue (July 1994;49:670–5). Their suggestion that NAC may
enhance the glutathione antioxidant potential of the
alveolar macrophage and reduce pulmonary
inflammation. sarcoidosis (Mycobacterium tuberculosis),
our patients were not markedly different from these patients. In addition, they did not perform functional measurements.

Several studies have investigated the anti-
oductive capacity of NAC, 600 mg daily. In
healthy smokers significant decreases in levels of
lactoferrin, ECP, and chemotactic activity
of neutrophils in BAL fluid, and of myelopero-
odase and elastase levels in serum, were
found. Treatment with NAC, 200 mg twice
or three times daily for more than one year,
was associated with a decrease in the number of bacteria, especially in patients with COPD. The design of some of these studies precludes firm conclusions, but they at least suggest that conventional doses of NAC may
influence the antioxidant capacity of the lung. This is of special relevance in those patients with a significantly disturbed pul-
omary oxidative/antioxidative balance, such as patients with COPD and smokers.Indeed, Lundbaeck et al recently showed that two years of treatment with NAC, 600 mg daily, reduced
the annual decline in FEV1, compared with a control group. This effect was most pronounced in smoking patients over 50 years of age with already considerably decreased FEV1.

Looking at the pharmacokinetic data, the authors showed in a group of patients con-
sisting of smokers, ex-smokers and non-
smokers that NAC, 600 mg daily for five days,
increased levels of glutathione in BAL fluid by 100% 1–3 hours after the last dose (p<0.05), and by 24% 16–20 hours after the last dose (NS). This "transient" increase in antioxidants in the lung apparently does not preclude a decrease in oxidative stress, or an improvement in antioxidant capacity of the lungs, or both.

PFR DEKHIJEN
CLA VAN HEERWAARDEN
Department of Pulmonology,
Academic Hospital Nijmegen,
PO Box 9121
6500 HB Nijmegen,
The Netherlands

3 Riise GC, Larsson S, Larsson P, Jeansson S, Anderson BA. The firm a microsomal micro-
4 Lundbaeck B, Lindstrom M, Anderson S, Ny-
strom L, Rosenhall L, Stjenberg N. Possible

AUTHORS' REPLY We thank Drs Dekhuijzen and van Herwaarden for their interest in our paper. We do not, however, agree with their conclusions. Although the patients in whom the thiol concentrations of NAC were measured,
BAL fluid and lung tissue were performed not specifically chosen as having COPD, these patients were all smokers or ex-smokers, as stated in the text, and table 1 clearly in-
dicates that some had airflow limitation as shown by the predicted values for FEV1. Thus, some of these patients had COPD. The results of measurements of thiol con-
centrations in BAL fluid precludes bacteria, especially
CF STANFORD
Department of Medicine,
KINGHD,
PO Box 1215,
Jeddah 21423,
Saudi Arabia

3 Riise GC, Larsson S, Larsson P, Jeansson S, Anderson BA. The firm a microsomal micro-
4 Lundbaeck B, Lindstrom M, Anderson S, Ny-
strom L, Rosenhall L, Stjenberg N. Possible

Asthma publications

The incidence of asthma is high in most communities and possibly increasing, and is associated with considerable morbidity.1

Physicians have been under-allergy throughout most of this century and, in broad outline, the concept of the interaction between IgE and antigens in the bronchi is similar to that held in the 1970s. The fact that historical changes similar to those found at post mortem examination are present even in mild asthma suggests that an immunological process is frequently going on in atopic subjects.1 It is well recognised that the respiratory infections and exposures to some antigens, in particular the house dust mite,1 associated with an increase in the incidence of asthma.

Since for all diseases it is logical to consider that prevention is better than cure, one would expect that the major push in research in asthma would be towards finding methods to reduce antigen exposure. In 1993 Thorax, the journal of the British Thoracic Society, published 280 papers, 30–7% of which were on the subject of asthma. In the same year the American Review of Respiratory Diseases, the journal of the American Thoracic Society, published 540 papers of which 20% were on the same subject. The distribution of the type of asthma research published in the two journals is shown in the table, although naturally there are some areas of overlap. It can be seen that neither journal has published so many papers on the household or external environment. The American Review of Respiratory Diseases has concentrated on allergy and general aspects of the disease, while Thorax has concentrated on clinical aspects.

It is natural that biological researchers should be interested in the details of pathophysiology and the exciting spectrum of lymphokines, adhesion molecules and mediators for their own sake, as well as the hope that in the future a cure for asthma might be found. Asthma patients and their physicians owe a great debt of gratitude to the pharmaceutic industry for the drugs they have produced, especially since the 1970s. We wonder, however, if, as physicians and researchers, we should be asking if the direction of asthma investigation has drifted too much away from the prevention of obvious excessive exposure to antigens.

1 Burney PGJ. Asthma mortality in England and Wales: evidence for a further increase, 1974–
2 Castle, Fuller R, Hall J, Palmer J. Serovet nationwide surveillance study: comparison of

Number (% of papers on each aspect of asthma research published in Thorax and the American Review of Respiratory Diseases (ARRD) in 1993

<table>
<thead>
<tr>
<th>Subject of paper</th>
<th>Thorax</th>
<th>ARRD</th>
<th>Significance of difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allergy</td>
<td>8 (35)</td>
<td>4 (20)</td>
<td>( \chi^2 = 4.7; df = 1; p &lt; 0.05 )</td>
</tr>
<tr>
<td>General</td>
<td>27 (71)</td>
<td>15 (51)</td>
<td>( \chi^2 = 4.3; df = 1; p &lt; 0.05 )</td>
</tr>
<tr>
<td>Therapy</td>
<td>46 (53)</td>
<td>15 (40)</td>
<td>( \chi^2 = 2.0; df = 1; p &gt; 0.05 )</td>
</tr>
<tr>
<td>Housing</td>
<td>3 (3)</td>
<td>1 (3)</td>
<td>( \chi^2 = 0.9; df = 1; p &gt; 0.05 )</td>
</tr>
<tr>
<td>Total</td>
<td>86 (100)</td>
<td>73 (100)</td>
<td></td>
</tr>
</tbody>
</table>
Effect of N-acetyl cysteine on thiol levels.

P N Dekhuijzen and C L Van Herwaarden

Thorax 1995 50: 215
doi: 10.1136/thx.50.2.215