episodes of otitis media, one episode of conjunctivitis, and oral thrush on three separate occasions (unrelated to antibi-
totic therapy). In May 1983 she was admitted to the hospital for management of herpetic stomatitis. In the course of the following year, she suf-
fiered numerous infections, consisting predominantly of rhinitis, pharyngitis, and otitis. In addition, she continued to have recurrent oral candidiasis. In April 1984, she developed pneumonia that was unresponsive to antibiotic therapy. Chest roentenogram re-
vealed interstitial pneumonia, and she was admitted for further evalua-
tion. At this time she was thin and irritable, but in no acute distress. Posi-
tive physical findings included acute otitis media, marked nasal congestion, injected pharyngeal mucosa, tonsilar hypertrophy, multiple dental caries, and oral candidiasis. There was gen-
eralized lymphadenopathy involving cervi-
cal, axillary, and inguinal regions. Examination of the lungs revealed scattered ronchi. The liver was palpable 3 cm below the right costal margin. The spleen was not enlarged. Examina-
tion of the extremities revealed moder-
ate clubbing. A chest roentenogram con-
formed diffuse interstitial pneumonia. Lung biopsy revealed lymphocytic interstitial pneumonitis (LIP), which is now considered an inclusion criterion (5) for pediatric AIDS. Activities of liver-origin enzymes in serum were above normal, and liver biopsy re-
valed chronic active hepatitis, also a feature common in pediatric AIDS (6). Among the other members of the family unit, the non-addict father, a five-year-old brother, and a "family friend" - a heroin-using man, were all in good health; the mother had been suffering from increased fatigability. Blood was sampled from each of these in April 1984 to provide further elabo-
ration as to the development of AIDS by the patient. Results (Table 1) indi-
cated that the mother and the patient had decreased T4/T8 T cell ratios. They also had a biochemically determinable immune dysfunction in the form of the presence of the α form of βm, as did the male "family friend," even though he was HTLV-III Ab negative on initial test. From these data, plus the subsequent development of ARC (AIDS-related complex) by the mother and a positive HTLV-III Ab test in the "family friend" in the fall of 1984, we concluded that the infant had devel-
oped AIDS because of intra-uterine transmission. The βm tests (4) were the most discriminant and earliest indi-
cators allowing the preclinical deter-
mination of AIDS in the mother and the "family friend." Whether intrave-
nous drug abuse and heterosexual transmis-
sion ("family friend" to mother), as has recently been documented (7, 8), or only the former was respon-
sible for the transmission of AIDS to the infant cannot be conclusively docu-
mented. However, the "family friend," who has subsequently been lost to fol-
low-up, most likely was an active carri-
er of AIDS, even though he was initially seronegative by HTLV-III Ab and only seroconverted later. This under-
scores the importance of performing a series of tests, including βm, to bio-
chemically define AIDS disease develop-
ment as early as possible.

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Correlation between Calcium and Zinc in Plasma

To the Editor:

Two recent Letters have discussed the relations between magnesium and calcium in serum and plasma (1, 2).

Subsequent to our systematic correlation studies, we were surprised to discover a significant positive simple correlation between plasma calcium (P-Ca) and plasma zinc (P-Zn) in 58 control men (3), 26 men with unstable angina pectoris (UA) (4), and 34 men with acute myocardial infarction (MI) on days 1 and 2 of hospitalization (Table 1).

Blood was sampled from the three groups during the same period. Plasma calcium (P-Ca) and plasma zinc (P-Zn) were measured by flame atomic ab-
sorption spectrometry in a Hitachi Model 180-80 with Zeeman effect, ac-
cording to a previously described proto-
col (3).

Simple (r) correlation coefficients were estimated after normality of dis-
tribution was checked by the chi-

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Table 1. Correlation between Ca and Zn in Plasma of Men

<table>
<thead>
<tr>
<th>Sample group</th>
<th>r</th>
<th>p</th>
<th>Ref. no.</th>
</tr>
</thead>
<tbody>
<tr>
<td>58 control men, ages 42.8 ± 15.9 years</td>
<td>0.54</td>
<td>&lt;0.001</td>
<td>3</td>
</tr>
<tr>
<td>26 men with unstable angina, ages 64.4 ± 10.5 years</td>
<td>0.59</td>
<td>&lt;0.02</td>
<td>4</td>
</tr>
<tr>
<td>34 men with myocardial infarction (day 1), ages 61.1 ± 10.9 years</td>
<td>0.44</td>
<td>&lt;0.01</td>
<td>unpublished</td>
</tr>
<tr>
<td>34 MI men (day 2)</td>
<td>0.37</td>
<td>&lt;0.05</td>
<td>unpublished</td>
</tr>
<tr>
<td>34 MI men (days 3 and 6)</td>
<td>&gt;0.05</td>
<td>unpublished</td>
<td></td>
</tr>
</tbody>
</table>

Square test. The significance of the simple correlation coefficient was determined by the t-test (3).

A study conducted at the same time in three groups of women selected by the same criteria did not show significant correlation between P-Ca and P-Zn (3, 4).

Serum Ca concentrations are affected by parathyryn through its renal reabsorption action, bone resorption, and formation of 1,25-dihydroxyvitamin D by the kidney (3). Serum Zn concentration, like that of Ca, is maintained within a narrow range in healthy humans (5). Many factors are known to affect serum Zn, such as neuroendocrine mechanisms and ecologic and genetic influences (3, 5). It has been noted that, even though the metabolism of Ca and Zn differ substantially, Ca-regulating hormones are known to affect Zn metabolism: parathyryn excess increases urinary and bone loss of Zn (5).

Markowitz et al. (5) found a high correlation (r = 0.923) between Zn and ionized Ca patterns in serum of healthy men, suggesting a common regulator, whereas, contrary to ours, their correlation between serum Zn and total serum Ca (r = 0.167; p > 0.05) was not significant.

Further studies are required to specify the regulatory factors and to account for the differences according to sex.

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