

Correspondence

Gold binding to red blood cells

Sir, We noted with interest the recent correspondence on the uptake of gold by red blood cells during treatment with sodium aurothiomalate and aurothioglucose.^{1,2} Two distinct groups have been identified, one with low concentrations of gold in red blood cells and the other with very much higher concentrations.¹⁻⁴ We have also studied the distribution of gold in blood of patients with rheumatoid arthritis during treatment with sodium aurothiomalate and confirmed these results on the intersubject differences in distribution of gold. We surveyed the patients to determine factors which might affect the distribution of gold. From this survey we have identified smoking as the major factor affecting the uptake of gold by red blood cells. The ratio of the gold concentrations in red blood cells to the plasma concentrations was 0.35 ± 0.07 , $n = 14$ (mean \pm standard error) in smokers and 0.028 ± 0.003 , $n = 23$ in nonsmokers.⁵ The difference was highly significant ($p < 0.0001$), and there was no overlap between the 2 groups.

The concentration of thiocyanate in plasma is markedly elevated by smoking.⁶ Preliminary studies in vitro indicate that thiocyanate significantly increases gold uptake by red blood cells. Whole blood from 5 healthy, nonsmoking volunteers was incubated with sodium aurothiomalate and varying concentrations of thiocyanate. The concentrations of gold in red blood cells after 24 hours' incubation with 0, 100 μ M, and 500 μ M concentrations of thiocyanate were 0.032 ± 0.002 μ g/ml (mean \pm SE), 0.084 ± 0.010 μ g/ml and 0.32 ± 0.05 μ g/ml respectively. This may be the explanation for the increased red blood cell gold uptake in smokers.

A manuscript outlining these findings will be presented shortly.

G. G. GRAHAM
Department of Physiology and Pharmacology,
University of NSW,
Kensington, NSW 2033

G. D. CHAMPION, T. M. HAAVISTO, P. J. MCNAUGHT
Departments of Rheumatology and Clinical
Pharmacology,
St Vincent's Hospital,
Darlinghurst, NSW 2010,
Australia

References

- Gottlieb N L. Gold binding to blood cells. *Ann Rheum Dis* 1980; **39**: 529-30.
- Goldberg I J L, Lawton K, Redding J H, Francois P E. Gold binding to blood cells. *Ann Rheum Dis* 1980; **39**: 530.
- Smith P M, Smith E M, Gottlieb N L. Gold distribution in whole blood during chrysotherapy. *J Lab Clin Med* 1973; **82**: 930-7.

⁴ Van de Stadt R J, Abbo-Tilstra B. Gold binding to blood cells and serum proteins during chrysotherapy. *Ann Rheum Dis* 1980; **39**: 31-6.

⁵ McNaught P J, Champion G D, Graham G G, Haavisto T M. The influence of smoking on the distribution of gold sodium thiomalate. Paper presented to Australian Rheumatism Association Annual Scientific Meeting, Adelaide, October, 1980. Abstract to be published in *Aust NZ J Med*.

⁶ Borgers D, Junge B. Thiocyanate as an indication of tobacco smoking. *Prevent Med* 1979; **8**: 351-7.

Gout in haemoglobinopathies

Sir,

We read with interest the paper of Rothschild *et al.*¹ reporting 2 patients with gout associated with sickle cell disease. Their review of the previously reported cases of uric acid deposition disease in association with haemoglobinopathies unfortunately failed to include the 2 patients previously reported by us in a comprehensive review.² Their paper makes a number of cogent points, but there are several that deserve additional comment.

(1) They suggest that the synovial fluid in patients with sickle cell anaemia and uric acid deposition disease is a transudate. There are insufficient reports of synovial fluid findings in such patients to document this. Our patient with haemoglobin SC disease² had an exudative fluid containing leucocytes $64\,800\text{ mm}^3$ ($64.8 \times 10^9/\text{l}$) (80% neutrophils), glucose 48 mg/dl (2.7 mmol/l), and protein 5.6 g/dl (56 g/l). Clearly, more data are required to resolve this question.

(2) Both of their patients had renal dysfunction, as have all previously reported cases.² It therefore seems likely that urate overproduction resulting from the increased red cell turnover which characterises chronic haemolytic haemoglobinopathies³ may of itself be insufficient to produce uric acid deposition disease. Relatively mild degrees of renal dysfunction, however, may predispose to its occurrence.

(3) Rothschild *et al.* note that the frequency of hyperuricaemia and impaired renal function in sickle cell disease is not accompanied by the expected incidence of gouty arthritis. They consider a number of possibilities for this discrepancy, including impaired leucocyte chemotaxis, reduced polymorphonuclear leucocyte activity due to anaerobic conditions, and decreased longevity of such patients. It should be emphasised, however, that the rarity of this association may well result from a failure to recognise clinical gout, the symptoms of which may be blunted by chronic analgesic use or may closely resemble those of the acute sickle crisis. Although hyperuricaemia may occur in up to 40% of adult patients,⁴ it is often relatively mild in degree. It is therefore essential that an