in a man with GPI who had been infected with the same strain and who died accidentally three months later. In both instances hundreds of sections of liver were examined. Moreover, very recently Krotosky *et al.* failed to find hypnozoites in this biopsy material. (ii) Similar negative results were found in the simian forms of quartan malaria—*P. inui* and *P. brasilianum*. (iii) Infections with these quartan parasites are easily cured with blood schizonticides such as chloroquine. It is therefore unnecessary to treat quartan malaria in man with primaquine (required for preventing the true relapse of *P. vivax* or *P. ovale* malaria). (iv) The persistent infections in the blood, whether blood- or sporozoite-induced, probably last for the life of the animal; thus quartan infections in man are the most common cause today of transfusion malaria.

But all is not myth. The quartan periodicity of *P. malariae* was recognized from Hippocratic times, while intermittent fevers were well known in Ancient Egypt from the Ptolemaic era; the priest, Neuchatis, in the Temple of Imhotep, besought the God to cure his mother of a quartan ague.

In the 13th century, Dante describes in the Inferno (Canto 17) the "shivering fit of quartan fever when the nails go dead and the victim trembles at the very sight of shade". The poet was evidently familiar with the clinical picture.

The death of two of our rulers in the 17th century was ascribed to quartan malaria. James I died in March, 1625, from a possible nephrotic sequela of the chronic disease, while the regicide, Cromwell, died in September, 1658 of an acute intermittent fever sometimes of a quartan nature. The finding at autopsy of the latter, of engorgement of cerebral vessels and the fatality of the condition suggest that *P. falciparum* was a more likely cause than the less severe *P. malariae*. However, was it warm enough in London in 1658 for the transmission of *P. falciparum* by mosquitoes?

Finally, Golgi in 1885 demonstrated the 72-hour cycle in the blood of the quartan parasite.

I am, etc.,

P. C. C. GARNHAM

Imperial College of Science and Technology, London SW7

References

Garnham, P. C. C. (1977). The continuing mystery of relapses in malaria. *Protozoological Abstracts,* **1,** 1-12.

Marchoux, E. (1926). *Paludisme.* Paris: Baillière et fils.

Accepted for publication 12th March, 1981.

Retinol, onchocerciasis and *Onchocerca volvulus* MADAM—In an attempt to investigate interactions of vitamin A (retinol) between the human host and *Onchocerca volvulus,* which were suggested by a recent clinical and epidemiological study (HALLER & LAUBER, 1980), we determined the retinol concentrations of the following specimens all obtained from Cameroonian adults: (a) plasma of onchocerciasis patients; (b) skin biopsies of the same individuals, presenting with onchocercomata; and (c) skin biopsies of clinically uninfected controls; and of (d) whole adult male and female *O. volvulus* isolated by collagenase from extirpated onchocercomata by SCHULZ-KEY *et al.* (1977). After saponification and extraction, the retinol concentrations were determined by high pressure liquid chromatography. Quantitation was carried out by planimetry of the peaks in relation to an external standard.

The median plasma retinol concentration in eight onchocerciasis patients was 1·4 IU/ml (range: 0·8-1·9). In all cases, concentrations were above the critical level of 0·3 IU/ml (=10 μg/100 ml), from below which clinical signs of retinol deficiency appear rapidly (WHO, 1976). The median skin retinol concentration in these individuals was 1·5 IU/g (range: 0·3-7·0). Four controls had a median skin retinol concentration of the same order as infected individuals: 1·2 IU/g (range: 0·6-2·8). The four batches of adult whole worms, derived from onchocerocomata of four different individuals, had a median tissue retinol concentration of 12·6 IU/g (range: 1·6-39·7).

Compared to the surrounding host tissue, the retinol concentration in tissues of *O. volvulus* is remarkably high, i.e., average eight times higher than in the skin. However the concentration does not reach those in fresh human liver, which are normally about 800 IU/g (KÖRNER & VÖLLM 1976). Our results suggest that further investigations should be made of possible relationships between *O. volvulus,* hypovitaminosis A and the clinical signs of onchocerciasis. They do not, however, solve the question of whether or not *O. volvulus* is dependent on host retinol for growth and reproduction, or if retinol depletion of host tissues might be a contributory factor in the pathogenesis of corneal opacities, localized dermatitis (sowda) and other symptoms occurring in both clinical onchocerciasis and hypovitaminosis A.

We are, etc.,

D. STÜRCHLER
FRANZISKA WYSS
A. HANCK

Swiss Tropical Institute, Medical Department, Socinstr. 57, CH-4051 Basel, Switzerland, and F. Hoffmann-La Roche, Pharmaceutical Division CH-4002 Basel, Switzerland.

References

Haller, L. & Lauber, E. (1980). Etude des interactions entre les taux sériques de vitamines et les parasitoses communément répandues en zone tropicale. *Acta tropica,* **37,** Suppl. 11, 110-119.

Körner, W. F. & Völlm, J. (1976). Vitamine, In: *Klinische Pharmakologie und Pharmakotherapie.* München: Urban & Schwarzenberg, pp. 361-402.

Schulz-Key, H., Albiez, E. J. & Büttner, D. W. (1977). Isolation of living adult *Onchocerca volvulus* from nodules. *Tropenmedizin und Parasitologie,* **28,** 428-430.

WHO Expert Committee (1976). Vitamin A deficiency and xerophthalmia. Technical report Series No. 590. Geneva: WHO.

Accepted for publication 2nd April, 1981.