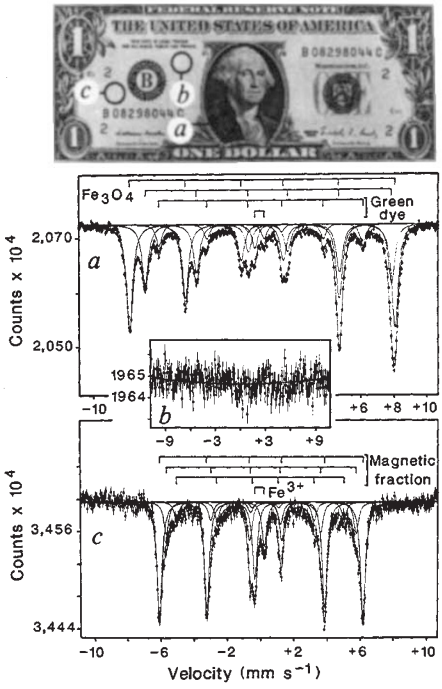
Mössbauer test for forgery

SIR—The relatively high concentration of iron-containing pigments in printers' ink and the specificity of their Mössbauer spectra indicate that Mössbauer spectroscopy could be used successfully for rapid checking of bank-note forgeries. The spectrum from the dollar note in the figure shows that Washington's lapel is printed in an ink whose main pigment is Fe_3O_4. The black iron oxide Fe_3O_4 is a specific, modern pigment which is not commonly used as a painting material[1]. Its strong magnetic properties make dollars 'magnetic'[2,3]. The ratio I_B/I_A of the two sextet intensities is specific and does not correspond to that of stoichiometric magnetite. X-ray fluorescence shows that parts of the iron atoms in B-sites are replaced by other atoms.

The figure shows a Mössbauer spectrum obtained from a region of green dye only. The Fe^{3+} irons are in several non-equivalent surroundings in the magnetic fraction and also in a paramagnetic compound. The spectrum in *b*, taken from a colourless region, shows that filaments used in the bank-note paper have no iron-containing compounds.

We have not yet studied the iron-containing dollar pigments in detail, but we can say that Mössbauer spectroscopy could become a powerful legal tool in the identification of fakes and forgeries. This conception is based on the pigment's Mössbauer-spectra specificity. Keisch has proposed[1] a similar technique for a similar problem, the investigation of iron-containing pigments used in paintings.

Development of the method as a tool for forgery detection requires first, Mössbauer spectroscopy of authentic bank-notes of different values printed in different years; and second, the same investigation on proven forgeries. We would like to appeal for collaborators in our attempts to develop the method. Such sponsors need not to worry about their money: the method is non-destructive.



Mössbauer spectra of a dollar bill. The three spectra *a*, *b* and *c* come from the marked regions of the bill.

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Still hope for malaria vaccine?

SIR—Malaria, a life-threatening disease caused by protozoan parasites of the genus *Plasmodium*, affects millions of people in the tropics annually. Resistance of the parasites to a wide range of drugs is now commonplace and spreading. In the search to improve the health of those at risk, vaccines are potentially significant components of an integrated worldwide strategy for malaria control. Gillett's suggestion in Scientific Correspondence (*Nature* **348**, 494; 1990) that this research effort is flawed, and has misunderstood or neglected the problems of genetic diversity generated by sexual reproduction of the parasite, is incorrect. Those involved in the search for effective vaccines have long been aware of the problems posed by polymorphism, not only of the parasite, but also of the mosquito vector and man. The problem of genetic diversity is not exclusive to antimalarial vaccine development: variability in parasite sensitivity to drugs and vector resistance to insecticides have been reported for many years, and significant effort is correctly being addressed to the problem in all three areas.

Certainly, malaria parasites undergo obligatory sexual reproduction to complete the life cycle, but Gillett may have failed to recognize that they are genetically haploid for all stages of the life cycle found in man, and are diploid only in the zygote and ookinete stages in the mosquito midgut. Therefore his implication that the "maintenance and increase in heterozygosity" confers advantage to the parasite is largely irrelevant. Clearly, sexual reproduction allows the generation of new phenotypes by both inter- and intragenic recombination. It must be recognized, however, that mutation can also readily generate polymorphism in the form of both vaccine-resistant (D. E. Hudson *et al. J. molec. Biol.* **203**, 707; 1988) and drug-resistant parasites.

Solutions to the problems raised by antigenic diversity could come from various well-established and widely published observations. (1) Within any one protein antigen there are both variable and conserved amino-acid sequences. Other constraints permitting, conserved epitopes that are the targets of protective immunity are clearly preferred. (2) For some antigens, functional constraints on the molecule may preclude variation in B- or T-cell epitopes. (3) Antigens in the sexual stages and the zygote not naturally expressed in the vertebrate host show very limited polymorphism. In the light of this discussion it is perhaps ironic to record that these antigens induce a transmission-blocking immunity which attacks, in the mosquito gut, the very stages of sexual reproduction that generate genetic diversity and which are diploid. (4) Attempts are now being made to control by immunization the disease state induced by secreted parasite antigens. The immunity generated need not be anti-parasitic, and the target antigens therefore not subject to the same selection pressures.

Significant progress has been made in our understanding of immunity to malarial parasites. Very successful early laboratory studies produced effective immunity with blood-stage parasites, sexual stages, sporozoites or zygotes, and thus provided an exciting basis for today's research. It has long been appreciated that these early studies used parasites of limited diversity in hosts of restricted genotype. To extend these investigations to the point where immunity can be induced in genetically diverse human populations against polymorphic populations of parasites will require extensive and careful study, but this study must be based upon a secure and informed understanding of the biology of the parasite and its hosts.

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SIR—Gillett[1] expresses an important and valid concern that sexual reproduction in parasites, with its potential for generating