Sir -- We do not agree with the way Watt et al (August 5, p 475) (1) interpret the results of the viral load measurements in patients dually infected with HIV-1 and scrub-typhus. We are not convinced that their study demonstrates that during acute scrub-typhus HIV-1 copy numbers decrease because HIV-1 suppressive factors are produced. Our interpretation of their results is that during scrub-typhus infection the viral load initially increases, but decreases during treatment, as has also been observed in malaria patients (2).

In the absence of viral load levels before the onset of scrub-typhus, we should take the viral load level on day 28 (after treatment) as the probable viral load level before the patient developed the disease. This would mean that the viral load levels during acute scrub-typhus infection had increased by 193% by day 3 of the infection.

What remains is to explain why viral load levels of scrub-typhus patients were lower than those of the non-typhus patients. We believe that the small number of patients enrolled in the study, the way they were selected and the way viral load levels were measured makes it impossible to interpret the differences in viral load levels between scrub-typhus and non-typhus patients.

The authors clearly describe the selection procedure of the 10 scrub-typhus patients, but not of the 5 control patients. Moreover, it is unclear why only 5 non-typhus patients were selected, since at the 2 study sites in Thailand there are most probably many more non-typhus than scrub-typhus patients.

The authors should specify which commercial viral load tests were used and for which patients. Ideally all viral load measurements should have been performed in scrub-typhus and non-typhus patients using the same tests. It is unclear whether the Amplicor HIV monitor test 1.0 or 1.5 has been used. The 1.0 version is known to be not sensitive enough to measure viral load levels of subtype A (3). Nine of the 10 HIV-1 infected
scrub-typhus patients were infected with the subtype E, but such strains are always recombinant A/E strains (4). The ultra-sensitive protocol of the 1.5 version is a very sensitive test for the detection of low viral load levels, but is unable to differentiate viral load levels > 50,000 copies/ml.

The in vitro data on suppression of HIV replication by serum from one scrub-typhus patient and on inhibition of syncytium-induction by sera from infected mice provide only weak evidence that cross-reacting antibodies might display some “HIV-neutralising” activity. Only one human serum was tested and we do not have information on whether this neutralisation persisted beyond day 4. Sera from HIV-negative subjects can temporarily display some “neutralising” activity at high concentrations (5).

For all these reasons we believe that the statement of Watt et al that “The characterisation of HIV-I suppressive factors produced during scrub-typhus may lead to novel strategies against AIDS” should be considered somewhat speculative.

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