the role of the host in this phenomenon has never been explored in such a way. Additionally, there is no question that any studies that allow a better understanding of the complex response of host to infection are very welcome.

Dr. Gosling [1] is correct in mentioning the role of immunity in the phenomenon we observed. However, it has now been well established by many immunological research teams that there is no pattern of immune response that is a correlate of protection. Therefore, Dr. Gosling’s statement that “the authors could demonstrate this if they examined serological samples,” [1, p. 147] is absolutely incorrect, because no demonstration of protection could possibly be performed [3]. Nevertheless, immunogenetic analyses are currently ongoing in the cohort of children that was studied.

We agree with Dr. Gosling, and wrote in the original article, that children harboring long-term parasitemia may constitute a major reservoir of the parasite during the nontransmission season. Our objective was not to criticize the interests of intermittent preventive treatment for infants. Of course, the treatment of infected individuals could be one interesting tool, among several others (such as vaccine development), to help the cause of malaria control. However, the application of such a strategy has to be well thought out with regard to all aspects, including the availability of treatment and socioeconomic factors. In our opinion, it is difficult to accept the idea that intermittent preventive treatment for infants alone would be able to eliminate or eradicate malaria. We all have to keep in mind the fact that the coverage of intermittent preventive treatment for pregnant women is now less than 50% (coverage was 29% in Malawi in 2000 [4]), which brings up the question of its pragmatic efficacy. What will the coverage of intermittent preventive treatment for infants be in several years? Nobody can answer this question, and unfortunately, nobody can contend that its coverage will be greater for infants than for pregnant women.

It is not time to forget any aspect of the complex infectious process leading to malaria disease, but on the contrary, it is time to pursue research efforts to better understand the physiopathology of malaria. Please, let the past be a lesson; stop bringing applied research and fundamental research into conflict [5], and we will all be winners.

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Recovery from Adult Measles Encephalitis Immediately after Early Immunomodulation

To the Editor—Acute measles encephalitis is an immune-mediated disorder that has similarities to acute disseminated encephalomyelitis [1, 2]. Acute measles encephalitis complicates ~1 of 1000 cases of acute measles [1] and has been reported to be associated with high mortality and morbidity rates [2]. In 2007 (as of 2 September), in Japan, 8 cases of encephalitis related to measles among 805 adult cases of measles were reported to the Infectious Disease Surveillance Center [3]. We describe 2 adult patients with acute measles encephalitis who presented with coma and who recovered within 24 h after receipt of high-dose intravenous immunoglobulin (IVIg) and dexamethasone.

Patient 1 was a 42-year-old woman who developed upper respiratory symptoms associated with a high-grade fever, followed by a typical measles exanthem and Koplik spots. Ten days after the onset of fever, she presented with drowsiness that had progressed over the past 2 days. She was mute and akinetic and had a stiff neck. Her Glasgow Coma Scale was 7. Analysis of a CSF specimen revealed mononuclear pleocytosis (cell count, 85 cells/mm³) and a protein level of 81 mg/dL. Magnetic resonance diffusion-weighted images demonstrated hyperintense lesions in the parietal cortices on both sides. She received a 5-day course of high-dose IVIg (0.4 g/kg per day) plus intravenous dexamethasone (0.4 mg/kg per day). Within 12 h, the patient was awake and able to speak a few words. Subsequently, she became fully conscious, and neurological functions became normal during the 5-day course of IVIg. Repeated analysis of CSF samples on day 6 revealed a normal WBC count of 4 cells/mm³.

Patient 2 was a 32-year-old woman who developed coma at the time of recovery from measles. The patient had a stiff neck and roving eye movements (Glasgow Coma Scale, 6). CSF analysis showed a WBC count of 600 cells/mm³ (500 neutrophils and 100 mononuclear cells), a protein level of 167 mg/dL, and a glucose level of 55 mg/dL. The findings of magnetic resonance diffusion-weighted imaging of the head were unremarkable. Electroencephalography revealed generalized slowing and occasional frontal intermittent rhythmic δ activities. She received a 5-day course of high-dose IVIg (0.4 g/kg...
per day) and intravenous dexamethasone (0.75 mg/kg per day). The next day, the patient was awake and comprehensive and complained of headache. Repeated analysis of CSF samples on day 7 revealed a normal WBC count (2 cells/mm³). In both patients, the encephalitis-phase CSF specimen demonstrated increased myelin basic protein level, negative oligoclonal band, and normal IgG index. The acute-phase serum sample revealed that the measles IgM and IgG antibodies had undergone seroconversion during the patient’s measles course before the development of encephalitis. Acute-phase CSF samples showed high IgM antibody titers for measles.

The 2 patients with adult acute measles encephalitis had a rapid recovery from coma within 24 h after receipt of high-dose IVIg and dexamethasone. Recent case reports of adult acute measles encephalitis from Japan showed that patients treated with corticosteroids and/or low-dose IVIg (2.5–5 g per day) varied with regard to neurological sequelae, but recovery within 24 h after commencing treatment, as was seen in our 2 patients, has (to our knowledge) never been reported [4]. In cases of acute disseminated encephalomyelitis, on the basis of empirical evidence, initial treatment with intravenous high-dose corticosteroids—and, for patients who do not respond adequately to corticosteroids, high-dose IVIg—is recommended [5]. The poorer outcome of acute measles encephalitis among patients with acute disseminated encephalomyelitis [2] may justify the simultaneous administration of corticosteroids and IVIg immediately after the patient demonstrates a reduced level of consciousness.

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