Trials have suggested cure rates of similar to that for incurable viral infections. This pattern is different from the pattern found more often in older women. Some of the women might have acquired infections during sexual contact that they did not report, and some might have had infections that were not detected at the baseline visit. However, many women were treated for infection, had negative test results, and then had positive test results again, which suggests that T. vaginalis was undetected by testing but still present for months after treatment. The possibility of long-term asymptomatic carriage is consistent with the age distribution of infected women. T. vaginalis is found more often in older women. This pattern is different from the pattern for bacterial sexually transmitted diseases but similar to that for incurable viral infections, such as herpes simplex virus type 2. Trials have suggested cure rates of >90%, but most have tested women once within a few weeks after treatment. When women were tested again a few months after treatment, some of the previously cured women had infection detected again, and none of the studies continued testing women beyond a few months. Cultures might not detect infections if the concentration of T. vaginalis is low, which would be expected in asymptomatic infections. Nucleic acid amplification tests may be better, but reports are inconsistent and the tests are not commercially available in the United States. Similarly, self-obtained vaginal swab specimens occasionally miss infections, but the sensitivity of tests performed with self-obtained specimens has compared favorably with that of tests performed with clinician-obtained specimens.

Treatment failure could explain many of our findings, because 13 women had a documented preceding infection. However, our results were not simply attributable to treatment failure. Most of the women (n = 11) had an intervening negative test result before having a positive result during an interval when they reported not having sex. This suggests that, after treatment, T. vaginalis infection can become nondetectable for months and then reappear. Because these findings were unexpected and obtained with a small number of participants, additional studies are needed to confirm or refute these observations.

Acknowledgments

Potential conflicts of interest. All authors: no conflicts.

Thomas A. Peterman,1 Lin H. Tian,1 Carol A. Metcalf,1 C. Kevin Malotte,1 Sindy M. Paul,7 John M. Douglas Jr,1 for the RESPECT-2 Study Group

1Division of Sexually Transmitted Diseases Prevention, Centers for Disease Control and Prevention, Atlanta, Georgia; 2California State University, Long Beach; 3New Jersey Department of Health and Senior Services, Trenton; and 1Human Sciences Research Council, Pretoria, South Africa

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Reprints or correspondence: Dr. Thomas A. Peterman, Mailstop E02, CDC, Atlanta, GA 30333 (peteman@dcdc.gov).

Clinical Infectious Diseases 2009;48:259–60 © 2009 by the Infectious Diseases Society of America. All rights reserved. 1058-4838/2009/4802-0014$15.00 DOI: 10.1086/595706

Detection of HIV Type 1 Load by the Roche Cobas TaqMan Assay in Patients with Viral Loads Previously Undetectable by the Roche Cobas Amplicor Monitor

To the Editor—In March 2008, the Roche Cobas TaqMan assay replaced the Roche Cobas Amplicor Monitor, version 1.5, for measuring plasma HIV type 1 (HIV-1) load in Japan. This has resulted
in the detection of an HIV-1 load >50 copies/mL in some of the patients whose HIV-1 load had been undetectable (<50 copies/mL) by the Amplicor Monitor over several years and for whom antiretroviral treatment regimens had not been changed [1, 2].

A total of 1387 HIV-1–infected patients visited our outpatient clinic from March through June 2008, and their HIV-1 load was measured by the TaqMan assay. Among these patients, 876 regularly visited the clinic (once every 1–3 months) and had an undetectable HIV-1 load by the Amplicor Monitor at the last visit. Surprisingly, the TaqMan assay detected an HIV-1 load >50 copies/mL in 253 (28.9%) of the 876 patients, although antiretroviral treatment had not been modified for these patients. Furthermore, another 22 patients (2.5%) were found to have an HIV-1 load ≥40 copies/mL with use of the TaqMan assay. The same assay also detected HIV-1 RNA at levels lower than the linear range of the assay (<40 copies/mL) in 128 (14.6%) of the 876 patients.

We analyzed the relationship between TaqMan detectability and time during which the HIV-1 load was undetectable by the Amplicor Monitor. This time was defined as the period from the first HIV-1 load undetectable by the Amplicor Monitor to the viral load first measured by the TaqMan assay, without any HIV-1 load rebound or blip detected during the period. Interestingly, among the patients who had a viral load undetectable by the Amplicor Monitor for <1 year, 45.7% had an HIV-1 load ≥50 copies/mL detected by the TaqMan assay; among the patients who had a viral load undetectable by the Amplicor Monitor for >4 years, 18.5% had an HIV-1 load ≥50 copies/mL detected by the TaqMan assay (figure 1). Conversely, 37.3% of patients who had a viral load undetectable by the Amplicor Monitor for <1 year had HIV-1 RNA undetectable by the TaqMan assay, and 70.2% of patients who had a viral load undetectable by Amplicor Monitor for >4 years had an HIV-1 load undetectable by the TaqMan assay. Thus, the proportion of patients who had an HIV-1 load ≥50 copies/mL was inversely correlated with the duration that the viral load was undetectable ($R^2 = 0.895$), and the proportion of patients with undetectable viral load was positively correlated with the duration that the viral load was undetectable ($R^2 = 0.979$). These findings indicate that the longer the effective treatment, the greater the number of patients with HIV-1 RNA undetectable by the TaqMan assay.

We observed significant discrepancy of HIV-1 detectability between the TaqMan assay and the Amplicor Monitor [3–5]. The TaqMan assay detected HIV-1 RNA in a significant percentage of treated patients with HIV-1 loads previously undetectable by the Amplicor Monitor; this is confusing to clinicians and patients and may be a critical problem in ongoing clinical trials of antiretroviral treatment. To determine the permissible range of detectable HIV-1 load during successful antiretroviral treatment, year-long clinical follow-up of treated patients is necessary. Our observation revealed that the detection rate of HIV-1 RNA with use of the TaqMan assay was inversely correlated with the previous duration of undetectable HIV-1 load, suggesting that long-term antiretroviral treatment can further suppress HIV-1 load even after it has decreased to below the detection limit of the Amplicor Monitor.

**Acknowledgments**

We thank Dr. Mahoko Kamimura, Kouji Watanabe, and Kunio Yanagisawa, for their helpful discussion and continuous support, and the nurses of AIDS Clinical Center Outpatient Clinic and the AIDS Clinical Center coordinator nurses, for their dedicated assistance.

**Financial support.** Ministry of Health, Labor, and Welfare of Japan grant-in-aid for AIDS research (H20-AIDS-002).

**Potential conflicts of interest.** All authors: no conflicts.

**Hiroyuki Gatanaga,** Kunihisa Tsukada, Haruhito Honda, Junko Tanuma, Hirohisa Yazaki, Tamayo Watanabe, Miwako Honda, Katsuji Teruya, Yoshimi Kikuchi, and Shinichi Oka

AIDS Clinical Center, International Medical Center of Japan, Tokyo, Japan

**References**


To the Editor—I appreciated the systematic review by McGinigle et al. [1] of active surveillance cultures (ASCs) for methicillin-resistant Staphylococcus aureus (MRSA) in the intensive care unit (ICU) but question their conclusions about the lack of enough robust evidence to provide definitive recommendations for the use of ASCs in the control of MRSA infection. The authors included 20 studies, but only 13 of these studies seem to be original intervention studies that assess the effect of ASCs on the rate of MRSA infection. In addition, as the authors indicate, the methodology and/or robustness of many of these studies are not optimal.

Because I have been interested in this subject for many years, I have collected the literature on another 7 published nonpediatric and nonneonatal ICU studies that merit inclusion in the systematic review by McGinigle et al. [1–8], as well as another 6 neonatal and/or pediatric ICU studies (not referenced). It would be interesting to understand why these adult ICU studies were not included in the systematic review by McGinigle et al. [1]. Three of these studies were interrupted-time series, and 1 was a controlled before-and-after study; both of these methodologies are fairly robust. It is true that not all of the studies included weekly ASCs, but this seems to be a questionable exclusion criteria if a reduction in the rate of MRSA infection was still reported. However, the consistency of positive findings in the adult ICU studies is worth emphasizing (i.e., ASCs can aid in the control of MRSA infection in the ICU, particularly when ASCs are combined with at least 1 of the following: patient and environmental decontamination and hand-hygiene initiatives).

It is noteworthy that, of the 20 studies (13 in the systematic review and the 7 aforementioned adult ICU studies), only 3 do not mention use of additional hygiene and/or decontamination procedures (4 of 26 studies, if the neonatal and/or pediatric ICU studies are also considered). Moreover, although all but 1 study reported a reduction in the rate of MRSA infection after introduction of ASCs, this 1 study was notable for its poor hand-hygiene compliance, late isolation of MRSA-positive patients, and absence of any decontamination or disinfection.

Finally, the rating of high-quality interrupted-time series as only “fair” evidence by McGinigle et al. [1] is debatable. The most important difference between my interpretation of the data and that of McGinigle and colleagues is my observation of the consistency, strength, temporal relationship, and plausibility of the evidence; this insight led me to conclude that ASCs should be recommended as standard practice, particularly in high-risk areas, such as ICUs, where there is a high rate of hospital-acquired MRSA infection and a great risk of MRSA infection.

Incidentally, my colleagues and I conducted a study [9] (which was incorrectly referenced in the systematic review) that demonstrated a two-thirds reduction in the rate of MRSA infection (a decrease from >15% to ~5% of ICU admissions, not the 11% reduction stated in the systematic review by McGinigle et al. [1]). Moreover, this reduction was entirely attributable to a reduction in the number of MRSA isolates from clinical specimens, not screening specimens. Although the number of MRSA isolates is only a surrogate for infection, it is more closely indicative of infection than colonization; that the number of MRSA isolates is a surrogate marker of colonization was wrongly implied by Milstone and Perl [10] in their accompanying editorial commentary. In support of the number of MRSA isolates being a surrogate marker of infection, there was a significant reduction in both length of stay and glycopeptide use associated with the introduction of ASCs.

Acknowledgments

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Ian M. Gould
Department of Medical Microbiology, Aberdeen Royal Infirmary, Foresthill, Aberdeen, Scotland, United Kingdom

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Use of Active Surveillance Cultures in Intensive Care Units

To the Editor—I appreciated the systematic review by McGinigle et al. [1] of active surveillance cultures (ASCs) for methicillin-resistant Staphylococcus aureus (MRSA) in the intensive care unit (ICU) but question their conclusions about the lack of enough robust evidence to provide definitive recommendations for the use of ASCs in the control of MRSA infection. The authors included 20 studies, but only 13 of these studies seem to be original intervention studies that assess the effect of ASCs on the rate of MRSA infection. In addition, as the authors indicate, the methodology and/or robustness of many of these studies are not optimal.

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Ian M. Gould
Department of Medical Microbiology, Aberdeen Royal Infirmary, Foresthill, Aberdeen, Scotland, United Kingdom

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