of *S aureus* bacteraemia, enabling timely, effective therapy that is associated with decreased length of hospital stay and health-care costs. However, they do require overnight incubation until a positive blood-culture signal is revealed. It would be desirable to identify staphylococcus species directly from whole-blood specimens.[1,14] Several PCR amplification-based methods are commercially available. An unanswered question for the systems reviewed is how accurate they are when more than one microbial species is present. Although mixed infections are infrequent, accurate detection of such mixed blood samples is important to monitor whenever a novel diagnostic method is selected for use. Rapid direct analysis of blood samples from patients by these new molecular techniques for bacterial pathogens allows targeted therapy at the point of care in real time and have improved the treatment of microbial sepsis.[15]

*Yi-Wei Tang, Lance R Peterson

Department of Laboratory Medicine and Department of Internal Medicine, Memorial Sloan-Kettering Cancer Center, New York, NY 10065, USA (Y-WT); and Department of Pathology and Laboratory Medicine, NorthShore University HealthSystem, Evanston, IL, USA (LRP)

tangy@mskcc.org

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A strategy to halt leprosy transmission

Leprosy is a chronic infectious disease caused by the bacillus *Mycobacterium leprae*. The organism multiplies very slowly and the incubation period is more than 2 years. Symptoms, including lesions of the skin, peripheral nerves, limbs, and eyes, can cause severe disability and take up to 20 years or more to develop after onset of infection. The mode of transmission of *M leprae* remains uncertain but is widely believed to occur from person to person via respiratory droplets. Close contacts of patients are at the highest risk of infection. The disease, however, is curable and early treatment can prevent disability. Standard treatment is multidrug therapy, consisting of the combined administration of three antibiotics (rifampicin, clofazimine, and dapsone), or two (rifampicin and dapsone), depending on the bacillary load.

Leprosy is no longer the scourge that had plagued humanity for countless centuries. However, as recently as 1985, its prevalence was 5·2 million and 122 countries, mostly in the developing world, were endemic for the disease. In 1991, the World Health Assembly passed

a resolution to "eliminate leprosy as a public health problem" by the year 2000. Elimination was defined as a reduction of global prevalence to less than one case per 10 000 population, equivalent to fewer than 600 000 cases worldwide. By 2005, global prevalence of the disease had fallen to about 300 000 and all but six countries had reached the elimination target.[1] This accomplishment has been attributed largely to the widespread availability of multidrug therapy since the early 1980s. Novartis, the Swiss pharmaceutical company that manufactures multidrug therapy, has been donating the drugs to all patients worldwide through WHO since 2000.

Nowadays, however, the problem facing antileprosy efforts is that since 2005, both the prevalence and the incidence of the disease have plateaued at about 200 000. Every year, for the past 8 years, more than 200 000 people with leprosy have been discovered in many foci in Africa, the Americas, and Asia.[2,3] Transmission of the infection is clearly continuing. A major obstacle to interrupting it is the paucity of information about where and to what extent transmission is occurring. Success in reducing global prevalence and incidence has led to complacency among health officials of many countries. Few countries now have a surveillance–response system that could provide the epidemiological data needed to map high-risk areas for leprosy, to monitor the changing epidemiological pattern of the disease, and to implement the required interventions. In the absence of such data, alternative approaches can be used. Potential sources of information include managers of leprosy programmes and historical data from past leprosy elimination campaigns. Patients with leprosy are likely to be found where patients with other poverty-associated diseases cluster. School surveys, too, might provide clues: the finding of school-age children with leprosy is a strong indicator of ongoing transmission.

Rigorous tracing of contacts of patients with leprosy is a prerequisite to stopping leprosy transmission. Every identified contact should receive information and counselling and be examined for clinical signs and symptoms. Symptomatic contacts should be given multidrug therapy and asymptomatic contacts should be given chemoprophylaxis with one dose of rifampicin. There is evidence that this postexposure prophylaxis can reduce detection rates of new patients—and thereby transmission of the infection—by about 50–60%. There

is also evidence, however, that its protective effect lasts for only about 2 years. This shortcoming underlines the need for research into alternative regimens. A serious obstacle, however, to gaining the full potential of contact tracing is the absence of a diagnostic test for early-stage or subclinical infection in contacts. Such a test would help with research into the mechanisms underlying the transmission of leprosy and make for better prevention and earlier treatment.[4–7]

We believe that leprosy transmission could be interrupted provided certain requirements were met. Governments of countries with leprosy must commit, politically and financially, to a transmission-lowering strategy. All leprosy control activities must, where possible, be integrated into national health systems or, where such systems are lacking or weak, share the facilities of control programmes for other diseases, such as poliomyelitis or guinea-worm disease. New instruments for early detection of leprosy infection, including subclinical infection, and for diagnosis of disease in contacts and other high-risk population groups, need to be developed and rapidly distributed.[8–10] School surveys and epidemiological mapping should be done regularly and active and passive contact tracing systematically implemented, followed by postexposure prophylaxis. Regimens for such chemoprophylaxis might need to be optimised. Innovative technologies need to be used for leprosy surveillance, which should include response mechanisms. The data generated by surveillance should be validated by an international commission of experts.

We propose that these conditions could form the basis of a leprosy endgame strategy. The mainstays of this strategy would be early diagnosis and prompt treatment of all patients. The time is ripe for implementing this strategy: at a meeting in Bangkok last July, health ministers of the 17 countries with ongoing leprosy transmission signed a declaration reaffirming their political commitment "towards a world free of leprosy".[11]

*Cairns S Smith, Shaik Kahder Noordeen,
Jan Hendrik Richardus, Hubert Sansarricq, Stewart T Cole,
Sumana Baruaf, Rosa Castãlia Soares, Lorenzo Savioli,
Ann Aertsh
Institute of Applied Health Sciences, School of Medicine and
Dentistry, University of Aberdeen, Foresterhill, Aberdeen AB25 2ZD,
UK (CSS); Chennai, India (SKN); Department of Public health,
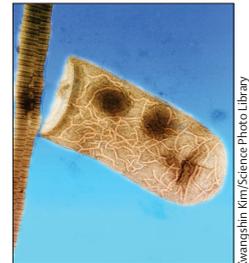
Kwangshin Kim/Science Photo Library

Erasmus Medical Center, Rotterdam, Netherlands (JHR); Saint Armou, France (HS); Global Health Institute, École Polytechnique Fédérale de Lausanne, Lausanne, Switzerland (STC); Ministry of Health, Brasília DF, Brazil (RCS); Department of Control of Neglected Tropical Diseases, WHO, Geneva, Switzerland (LS); and Novartis Foundation for Sustainable Development, Basel, Switzerland (AA)
cairns.smith@btinternet.com

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Corrections

Fry AM, Goswami D, Nahar K, et al. Efficacy of oseltamivir treatment started within 5 days of symptom onset to reduce influenza illness duration and virus shedding in an urban setting in Bangladesh: a randomised placebo-controlled trial. Lancet Infect Dis 2014; **14:** 109–18—In this Article, Mustafizur Rahman's surname was spelt incorrectly. This correction has been made to the online version as of Jan 20, 2014. The printed Article is correct.

Lake JE, Currier JS. Metabolic disease in HIV infection. Lancet Infect Dis 2013; **13:** 964–75—In paragraph five in Lipids section of this Review, stribild should be described as elvitegravir-cobicistat-emtricitabine-tenofovir DF and the comparator should be ritonavir plus atazanavir plus emtricitabine-tenofovir DF. Also, the comparator for the Striblid study (Rockstroh and colleagues[97]) should be ritonavir-atazanavir. Also, same paragraph, line 50, the comparator should be ritonavir-atazanavir. These corrections have been made to the online version as of Jan 20, 2014.