the blood units testing positive for hepatitis B surface (13%), HIV, and HTLV, which accounted for nearly one third of all donations in 2001. These findings argue in favor of maintaining a roster of regular, seronegative donors to save numbers of blood units.

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Prosthetic Valve Endocarditis due to *Kytococcus schroeteri*

To the Editor: Bacteria belonging to the former genus *Micrococcus*, the so-called micrococci, are usually regarded as contaminants from skin and mucous membranes. Nevertheless, micrococci have been reported as emerging pathogens in immunocompromised patients and have been described in severe infections (1–4). We describe what is, to our knowledge, the first case of prosthetic valve endocarditis caused by the newly described micrococcal species, *Kytococcus schroeteri*. Accurate identification of this species is of particular importance as kytococci—in contrast to other micrococcal species—are frequently resistant to penicillin and oxacillin (5).

A 34-year-old woman was admitted to the hospital with acute, severe aortic regurgitation, attributable to a dissection of both the ascending and descending aorta, which extended into the supraaortic and iliac arteries. Immediate surgical intervention was performed by implantation of an aortic arch (St. Jude Medical Inc., St. Paul, MN) conduit and reimplantation of the supraaortic arteries. Ten weeks later, the patient was admitted to the hospital because of fever of 39°C. Laboratory studies showed a leukocyte count of 15.3 x 10^9/L with 87% neutrophils and elevated C-reactive protein (180 mg/L). Transesophageal echocardiography and computed tomography suggested an abscess next to the prosthesis and showed vegetations on the prosthetic valve, which suggested endocarditis. Blood cultures yielded gram-positive cocci on four separate occasions during an 11-day period. Treatment, performed according to the antimicrobial susceptibilities of the isolates, consisted of vancomycin, gentamicin, and rifampin for 21 days. Within 1 week, the fever resolved and the leukocyte count returned to normal. Four days after antimicrobial therapy was initiated, right-sided hemiparesis and aphasia, thought to be due to an embolic cerebral stroke, developed. After those events, the aortic arch prosthesis was replaced without further complications.

Blood culture specimens were injected into BACTEC Plus culture vials for aerobic and anaerobic cultures and processed in BACTEC 9240 blood culture system (Becton Dickinson, Cockeysville, MD). Growth was detected in four different aerobic blood cultures after incubation of 3 to 5 days. Aerobic subcultures on Columbia agar supplemented with 5% sheep blood showed tiny, muddy-yellow colonies without hemolysis after 24 h of incubation. After 48 h, the size of colonies increased, a feature typical of *K. sedentariu*s, which is known to grow slightly more slowly than other members of the former *Micrococcus* genus. No or very weak reactions were found after 24 h incubation when the ID32 STAPH ATB gallery (bioMérieux Vitek, Hazelwood, MO)

was used. After 48 h, the reactions with this gallery resembled those of *M. luteus* or *M. lylae.* The probability of identification was indicated as 99.0% (*M. luteus*, T index of 0.77) for the profile 000003000 and 51.8% (*M. lylae*, T index of 0.98) and 47.2% (*M. luteus*, T index of 0.93), respectively, for the profile 000001000. When the ID-GPC card (VITEK 2, bioMérieux Vitek) was used, a poor selectivity was observed (*M. luteus*, T index 0.95; *Kocuria rosea*, T index 0.84). All isolates were resistant to oxacillin, penicillin, fosfomycin, ampicillin, and erythromycin and susceptible to vancomycin, teicoplanin, gentamicin, netilmicin, chloramphenicol, imipenem, rifampin, tetracycline, amoxicillin/clavulanate, and ciprofloxacin, as determined by disk diffusion method performed on Mueller-Hinton agar.

When arbitrarily primed-polymerase chain reaction with prolonged ramp times (6) was used, isolates were shown to be clonal, representing one strain (DSM 13884^T). Since colony formations, resistance pattern, and growth rate of this strain did not correspond with the species identification, as obtained by automated systems, further phenotypic and molecular studies were conducted, confirming the micrococcal nature of this unknown strain and justifying the classification as a distinct species, *Kytococcus schroeteri* sp. nov. (7).

In addition to the *Micrococcus* genus, bacteria belonging to the former genus *Micrococcus* were recently divided into the genera *Kocuria*, *Nesterenkonia*, *Kytococcus*, and *Dermacoccus,* followed by rearrangement into two families (*Micrococcaceae, Dermatophilaceae*) of the suborder *Micrococcineae* (5).

The traditional identification of the micrococci is based on their susceptibility to lysozyme and bacitracin and their resistance to lysostaphin and nitrofurantoin, in contrast to staphylococci, which display the opposite pattern. In automated identification systems, micrococci are included only in a limited manner. A prospective study showed an overall accuracy of results of 61.0% concerning *Micrococcus* species when the STAPH-IDENT strip (bioMérieux) was compared with conventional identification methods (8).

Micrococcal species are ubiquitous inhabitants of the human skin and mucous membranes and are usually disregarded as contaminants in clinical specimens. Yet, various severe infections such as arthritis, central nervous system infection, pneumonia, peritonitis, hepatic abscess, endocarditis, and nosocomial blood stream infections have been documented (1,3,4,9). Since early reports of endocarditis caused by gram-positive cocci that appear in tetrads and packets often did not reliably differentiate between micrococci and phenotypically similar microorganisms, such as coagulase-negative staphylococci, the frequency of micrococcal endocarditis is difficult to ascertain and might be underestimated. However, several cases of endocarditis attributable to *M. lylae*, *M. luteus*, *K. sedentarius,* and unspecified micrococci have been reported (1).

Regarding micrococci, data on antimicrobial susceptibilities are rare, and often the species affiliation remains unclear. In contrast to most micrococcal isolates, *K. sedentarius* isolates, as well as those reported here, are resistant to penicillin G and oxacillin. In the patient we describe, therapy was performed with vancomycin, gentamicin, and rifampin, resulting in bacteriologic eradication and clinical cure. However, a generally accepted therapeutic regime for severe infections with kytococcal species has not yet been defined. Concerning micrococci other than kytococci, a combination of rifampin with ampicillin has been effective (3). Successful treatment has also been achieved with vancomycin, clindamycin, penicillin, gentamicin, or a combination of these agents. Overall, rifampin shows the highest activity against all micrococcal species (10).

This report is the first case of *K. schroeteri* causing endocarditis on an artificial heart valve. The repeated recovery of this species from blood cultures strongly suggests a pathogenic role. We conclude that isolation of micrococci from blood specimens cannot always be disregarded as etiologically irrelevant. Results performed by automated identification systems should be interpreted with caution if micrococci are involved.

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When Is a Reservoir Not a Reservoir?

To the Editor: Some 80% of parasitic infections of humans are zoonoses (1). These infections are caused by multihost parasites for which the reservoir of infection depends on hosts other than *Homo sapiens*. But what is a reservoir of infection?

Haydon et al. (2) proposed a new series of definitions in connection with multihost pathogens, in which a target host is the host of interest in a particular context. The reservoir of infection included, for these authors, all hosts, whether incidental or not, that are epidemiologically connected to (i.e., contribute to transmission to) the target host.

The availability of three terms—reservoir, reservoir of infection, and reservoir host—frequently used interchangeably, leads to confusion. This confusion is, in part, what prompted me (3) to slightly redefine a reservoir (of infection) as an ecologic system in which an infectious agent survives indefinitely. Such a system includes all the component host populations, including that of any intermediate host or vector, in the context of any environmental component, and any quantitative requisite such as critical community size, which is required to maintain the agent indefinitely.

Vertebrate hosts that form an essential part of the system are reservoir hosts, though whether a whale or a fish is the reservoir host of *Anisakis* species can be a matter of debate. A host that becomes infected, but is not required for the maintenance of the population of a pathogen, can usefully be called an incidental host. (Accidental host is frequently used, but this is arguably a teleological term and therefore undesirable.) For incidental hosts that transmit pathogens from a reservoir to another incidental host, analogous to a pipe leading from a water reservoir, Garnham (4) coined the useful term "liaison host."

Haydon et al. dismiss my definition on two grounds. First, I exclude liaison hosts from the reservoir. This distinction is valid and could be argued either way, but I suggest that the pipes leading from a reservoir do not form part of the reservoir and that it is both conceptually and practically important to distinguish liaison hosts from reservoir hosts. The second objection is that many pathogens depend on the presence of several host species, at any given stage in the life history, for their maintenance. This concept is clearly considered in my article: together, such hosts collectively constitute part of the reservoir system, though no single one may be the reservoir host in its own right.

In good scientific English, each term should have a precise definition, and synonyms should be avoided. The Oxford English Dictionary (OED) (5) definitions of reservoir generally refer to a place or container used for the collection and storage of water, other fluids, or even solid material.

The OED definition of reservoir as a medical term is. "A population which is chronically infested with the causative agent of a disease and can infect other populations." While one might argue with the terms chronically, infested, and the infection of populations, this definition captures the usual sense in which reservoir host is used.

The quotations given in OED are more helpful. The earliest one given for reservoir in a medical context is from 1937, "For the continuous existence of a disease there must be some reservoir of infection… The most important reservoirs of infection are human or animal cases or carriers. Plants may be the reservoir of infection in some of the mycoses." However, according to OED, the compound term "reservoir host" was used earlier, in 1913, "The monkey is most probably the normal reservoir host [for *Physaloptera mordens*]."

The main conceptual difference between the proposal of Haydon et al. and my own is that mine is more generalized: for a given pathogen in a given place, there is a single reservoir. The proposal of Haydon et al. is more limited to practical considerations: the reservoir for one target host may not be the same as that for another target host in the same place.

The most important contribution of these two publications is that they raise an issue that has confused the literature for many years. Parasitologists, virologists, and bacteriologists should agree on a consensus set