This was the first case of high-level vancomycin-resistant enterococci with a class A phenotype isolated from a person in our hospital or in Ankara, Turkey. To prevent the organism’s spread, we implemented the recommendations of the Hospital Infection Control Practices Advisory Committee (5).

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care (13) and one of the lowest in hospitals (14). Similarly, Nordic European countries report both very low MRSA prevalence and antimicrobial-drug use (13,15). In Denmark, the prevalence of MRSA peaked at approximately 18% among all S. aureus isolates (and approximately 30% among blood isolates only) at the end of the 1960s, then regularly decreased during the 10 following years. This decrease has been attributed to various interventions, including increasing awareness of hospital hygiene and an intensive campaign to teach physicians the principles of prudent antimicrobial-drug use. Indeed, the decade witnessed a decrease in the use of streptomycin and tetracycline to which these MRSA strains were resistant. However, determining the relative contribution of these interventions to the disappearance of MRSA strains from Denmark has not been possible since all were implemented at approximately the same time. Since the beginning of the 1980s, the percentage of MRSA has remained extremely low, and below 1% among blood S. aureus isolates. Except for a very small number of localized hospital outbreaks, Danish MRSA isolates now represent imported cases from countries with high prevalence. To preserve this low level, patients admitted from foreign hospitals are isolated and screened for MRSA carriage. Health-care workers who have been working in foreign hospitals are screened before working in Danish hospitals. At the same time, both the overall level of antimicrobial-drug use and the fraction represented by broad-spectrum antimicrobial drugs, such as cephalosporins or fluoroquinolones, remain very low in Danish primary health care and hospitals, according to the 1999 report by the Danish Integrated Antimicrobial Resistance Monitoring and Research Programme (available from: URL: http://www.svs.dk/dk/Organisation/z/forsider/Danmap%20forsider.htm).

Additional research is certainly needed to fully understand the relationship between antimicrobial use and MRSA. However, the evidence supports implementation of programs to control or improve prescriptions when infection control alone does not control MRSA or the organization and resources for a “search-and-destroy” MRSA control strategy are not available.

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Lack of Evidence for Chloramphenicol Resistance in *Neisseria meningitidis*, Africa

To the Editor: High-level chloramphenicol resistance has been reported in 11 epidemiologically unrelated *Neisseria meningitidis* serogroup B strains in Vietnam and in a single strain in France, all isolated between 1987 and 1996 (1). Resistance was mediated by a chloramphenicol acetyltransferase (Cat) encoded by a *catP* gene homologous to *Clostridium perfringens* transposon Tn4451. While used infrequently in industrialized countries, chloramphenicol is often used to treat patients with meningococcal disease in Africa, especially during epidemics, when it frequently becomes the drug of choice because it can be administered intramuscularly (2).

To evaluate the presence of meningococcal chloramphenicol-resistant isolates in Africa, we assessed the frequency of the *catP* gene in 33 *N. meningitidis* serogroup A strains from the collection of the Centers for Disease Control and Prevention’s Epidemic Investigations Laboratory. The isolates, selected to give the maximum geographic and chronological representation, were collected during 1963 to 1998 from Chad, Egypt, Gambia, Ghana, Niger, Nigeria, South Africa, Tanzania, and Uganda, mostly during outbreaks. Thirteen (39.3%) of the strains were isolated during the 1990s, when chloramphenicol resistance was first described in Vietnam. All isolates were characterized by multilocus enzyme electrophoresis and represented four major electrophoretic subgroups (3,4). Chloramphenicol and penicillin MICs were determined for all isolates, according to the recommendations of the National Committee for Clinical Laboratory Standards, by the broth microdilution method using Mueller-Hinton broth with 5% lysed horse blood incubated in 5% CO₂ (5). All isolates were susceptible to both chloramphenicol (MIC <2 µg/mL) and penicillin (MIC <0.06 µg/mL). In addition, we tested all isolates for the presence of *catP* by polymerase chain reaction (PCR) using primers A, B, C, and D (1). Primers A and B, designed from the sequence of *catP*, amplify a 300-bp fragment only in chloramphenicol-resistant isolates. Primers C and D, designed on the basis of meningococcal sequences flanking the Tn4451-like insertion, amplify ~1200-bp fragment in resistant isolates and ~200-bp fragment in susceptible strains. Strain LNP13947 (kindly provided by Marc Galimand) was used as a positive control.

The *catP* gene was not detected in 32 of 33 *N. meningitidis* serogroup A strains. One isolate that was negative with primers C and D tested positive with primers A and B (M2786, Nigeria, 1963), which could suggest that *catP* was present but in a different location in the meningococcal genome. However, the chloramphenicol MIC of that strain was 2 µg/mL (susceptible). Repeated attempts to sequence the A/B amplicon were not successful with either primers A and B or another set of primers internal to primers A and B, implying that only a portion of the *catP* gene was present or (even more likely, given the conserved nature of this gene) that the PCR result was a false positive.

Chloramphenicol resistance was first described in meningococcal serogroup B isolates (1), but only serogroup A strains were included in this study since A is the most prevalent serogroup in Africa. (It accounts for most epidemics in Sub-Saharan regions.) Although our small sample size limited the chances of detecting a rare event, the data suggest that chloramphenicol resistance in Africa is relatively infrequent and that chloramphenicol is still an appropriate agent to treat meningococcal disease.

The acquisition of plasmids encoding Cat, which enzymatically inactivate chloramphenicol, is the most common mechanism of resistance in gram-positive and gram-negative organisms.