mechanisms contribute towards quinolone resistance.6 Mutations and MICs, suggesting that, in some populations, other have also found no correlation between increasing QRDR gyrA N. gonorrhoeae study enhances the existing knowledge of quinolone-resistant mutation. Although limited by the small number of isolates, this and number of mutations. We also describe a novel tinct from others and demonstrate no correlation between MICs paticular have mutation patterns similar to some countries yet dis-These, 9 had an additional silent mutation at codon 131 of parC! Asp-95! This pattern of mutation positions, involving gyrA positions 91 and 95 and parC position 86, was also predominant in Austria, Philippines, Thailand, Denmark and Hawaii, but was not seen in India and Japan.4 The geographic variations may be due to clonal or polyclonal spread of different local or imported strains.5,6 Typing methods such as PFGE may help elucidate the molecular epidemiology and transmission networks of quinolone-resistant N. gonorrhoeae. The commonest combination of mutations, seen in 11 (61%) strains, involved two mutations in gyrA (Ser-91 → Phe and Asp-95 → Gly) and one mutation in parC (Asp-86 → Asn); of these, 9 had an additional silent mutation at codon 131 of parC. Reported patterns of ORDR mutations vary between countries. This pattern of mutation positions, involving gyrA positions 91 and 95 and parC position 86, was also predominant in Austria, Philippines, Thailand, Denmark and Hawaii, but was not seen in India and Japan.4 The geographic variations may be due to clonal or polyclonal spread of different local or imported strains.5,6 Typing methods such as PFGE may help elucidate the molecular epidemiology and transmission networks of quinolone-resistant N. gonorrhoeae. In conclusion, quinolone-resistant N. gonorrhoeae at our hospital have mutation patterns similar to some countries yet distinct from others and demonstrate no correlation between MICs and number of mutations. We also describe a novel gyrA mutation. Although limited by the small number of isolates, this study enhances the existing knowledge of quinolone-resistant N. gonorrhoeae.

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Transparency declarations

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First detection of plasmid-mediated quinolone resistance (qnrA and qnrS) in Escherichia coli strains isolated from humans in Scandinavia

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Sir,

Plasmid-mediated quinolone resistance linked to qnr genes (A, B and S) has recently been discovered.1–4 These encode Qnr proteins that are members of the pentapeptide family and are able to protect topoisomerases and thus reduce their susceptibility to fluoroquinolones and increase the mutant selection window, therefore increasing the likelihood of selection of mutations.4,5 Plasmid-mediated fluoroquinolone resistance associated with qnr genes was first detected in the USA in 1994 in an isolate of Klebsiella pneumoniae; later it has been found also in Asia and in several countries in Europe.4,5 These resistance determinants
might be a threat, allowing fast spread of resistance. Besides, *qnr* genes are often linked to resistance to cephalosporins.\(^4,6\)

During a study that was performed to screen an *Escherichia coli* strain collection including 83 nalidixic-acid-resistant and 5 susceptible isolates from humans and 39 nalidixic-acid-resistant and 3 susceptible isolates from pigs in Denmark for characterization of quinolone resistance mechanisms, we found two isolates that showed reduced susceptibility to ciprofloxacin (MICs = 0.5 mg/L), but were susceptible to nalidixic acid (MICs = 4 and 8 mg/L). Both isolates were from patients at Hvidovre Hospital, Denmark: one from a urine sample and one from a blood sample.

In the first case, the patient was an 88-year-old woman hospitalized in mid-December 2005 with recurrent pneumonia, sepsis and bilateral crural ulcers. She was successfully treated with first mecillinam orally, then later cefuroxime and gentamicin iv and finally dicloxacillin orally due to a positive culture of *Staphylococcus aureus* from her crural ulcers. From a urine specimen, an *E. coli* resistant to ampicillin, cefuroxime, gentamicin, trimethoprim, sulfamethoxazole, ciprofloxacin and nitrofurantoin was cultured. This isolate, which showed no difference detected by antibiogram to the isolate later found, was, however, not further investigated or kept. In January 2006, the patient was hospitalized with pneumonia and dysuria and from a urine culture, furantoin was cultured. This isolate, which showed no difference for characterization of quinolone resistance mechanisms, we found, was, however, not further investigated or kept. In January 2006, the patient was hospitalized with pneumonia and dysuria and from a urine culture, furantoin was cultured. This isolate, which showed no difference

In the second case, the patient was a 72-year-old male with disseminated colon cancer. His recent medical history included three abdominal operations and during the last month, he had received several antibiotics: ampicillin, gentamicin, metronidazole and meropenem. In April 2002, he showed clinical sepsis and an ESBL-producing *E. coli* was cultured from the blood (H93). This isolate was initially found to be resistant towards ampicillin, cefuroxime, ceftazidime and cefpodoxime, but intermediate to ceftriaxone and susceptible towards cefoxitin, cefotaxime, piperacillin + tazobactam and ciprofloxacin. The patient was treated successfully (clinically) with mecillinam 400 mg three times a day for 5 days.

In the case of strain H93, no transformants were obtained. Transformation was performed with plasmid DNA from both strains by electroporation into TG1 competent cells (Stratagene). In the case of strain H93, no transformants were obtained.

Hybridization assays on Southern blots showed hybridization of a *qnrS* probe to a plasmid in strain H93, at an ~20 kb EcoRI fragment or an ~10 kb *SmaI* fragment of the donor strain and its transformants. In the case of strain H93, hybridization with the *qnrS* probe was observed to the chromosomal DNA. Although *qnrS* is generally referred to as plasmid-mediated, this gene is normally found on class I integrons that might jump to the chromosome, which is a possible explanation for the findings in this strain.

This is the first description of plasmid-mediated fluoroquinolone resistance in Scandinavia and in both isolates, the quinolone resistance was found in ESBL-producing multiresistant strains.

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**Transparency declarations**

None to declare.

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