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BACKGROUND

a) For the SCIs, fluoroquinolones are considered first-line treatment of MDR pneumococci. However, there was concern that short-term breakthrough success might be followed by emerging Resistant pneumococci strains by accumulation of additional resistance mutations.

b) In recent years, the role of efflux in non-fermenting FG-R has become more and more appreciated. Apart from a few isolates resistant in MBC increased effect is associated with rising resistance frequencies in the GNRs (4). When clinical resistance in attributed to the efflux resistance mechanism, the ABE effect might play a role (5).

c) Although FG-R pneumococci often show resistance to other antibiotics, recent work indicated that for every subject pneumococci case there are three additional infections (6). Here, we present data on FG-R in 422 non-invasive pneumococci, collected during seven seasons between 1995 and 2014 across 15 Belgian clinical laboratories.

MAJOR OBSERVATIONS

(i) Rising resistance to older fluoroquinolones since 2011

All isolates were assessed on FG-R using broth microdilution (Table 1).

(iii) Topoisomerase mutations explain a lot in high-level resistance, but not all

• Clinical topoisomerase mutations in gyrA (parC) and gyrB (parE) were found in varying combinations, arguing against clonal expansion of FG-R.

• Geographical difference.

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Table 2. Overview of the various FG-R genotypes encountered in 422 clinical pneumococcal strains. Staphylococcus pneumoniae strains are shaded in grey.

References