Seirbhís Sláinte Níos Fearr á Forbairt | Building a Better Health Service

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Annual Epidemiological Report

December 2018

Antimicrobial Resistance in Ireland, 2017

Key Facts

- There were 3,180* invasive *Escherichia coli* infections, with 3rd-generation cephalosporin resistance increasing to 13.2%* and extended-spectrum beta-lactamase (ESBL) production at 11.4%*. Five (0.1%) infections were due to carbapenemase-producing *E. coli* (CPE), all of which were OXA-48
- The proportion of *Staphylococcus aureus* bloodstream infections that were meticillin resistant (MRSA) (n=1,175) increased slightly to 16.3%
- There were 493* invasive *Klebsiella pneumoniae* infections, of which four (0.8%) were carbapenem resistant. Four infections were due to carbapenemase-producing *K. pneumoniae* (CPE), all of which were OXA-48
- There were 454* *Enterococcus faecium* bloodstream infections, with vancomycin resistance (VRE or VREfm) decreasing to 39.4%
- There were 415 invasive *Streptococcus pneumoniae* infections, with penicillin non-susceptible *S. pneumoniae* decreasing to 15.7%^
- Additional data on carbapenemase-producing *Enterobacterales*† (CPE):
    - There were 14 cases of invasive CPE, of which 13 were OXA-48
    - CPE was detected in 447 patients: 79% were associated with screening (i.e. from patients colonised with CPE) and 21% with clinical samples (i.e. from infection). Almost three-quarters of CPE were OXA-48

* highest / ^lowest number or proportion over the period 2008-2017; † includes all *Enterobacterales* species not just *E. coli* and *K. pneumoniae*

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Table of Contents

Background to Antimicrobial Resistance Surveillance

Antimicrobial resistance (AMR) presents a major challenge to healthcare in Ireland, as infections caused by AMR pathogens result in higher morbidity and mortality, extended hospital stays and increased healthcare costs.

The European Antimicrobial Resistance Surveillance Network (EARS-Net), previously the European Antimicrobial Resistance Surveillance System (EARSS), collects routinely-generated antimicrobial susceptibility testing (AST) data on eight important bacterial pathogens. Participating clinical microbiology laboratories in Ireland submit data on the "primary" or first isolate from blood or cerebrospinal fluid (CSF) per patient per quarter. EARS-Net does not distinguish clinically significant isolates from contaminants, nor does it distinguish between hospital-acquired, healthcare-associated and community-acquired infections. EARS-Net primarily serves as a surveillance system to measure national levels of AMR.

In 2017, all laboratories participated in EARS-Net resulting in 100% coverage of the Irish population. Population coverage has remained at over 95% since 2004.

EARS-Net encourages the use of EUCAST guidelines and clinical breakpoints for AST in line with the EU case definitions. By the end of 2017, 36 of the 39 clinical microbiology laboratories in Ireland had switched to EUCAST, with just three laboratories still using CLSI guidelines. The figures presented in this report (Table 1) are based on data extracted from the EARS-Net database on **1st November 2018**.

Additional data on pneumococcal serotyping were provided by the Irish Meningitis and Sepsis Reference Laboratory (IMSRL) at the Children's University Hospital, Temple Street.

The voluntary EARS-Net enhanced surveillance programme was established in 2004. Participating laboratories are invited to provide additional demographic and clinical data on invasive pathogens causing BSI. In 2017, enhanced surveillance data were submitted on 2,546 individual records (cases or isolates under the EARS-Net definition) by 19 participating laboratories, representing 40% of all reports to EARS-Net. Table 2 displays demographic and other basic data for the major resistance profiles of pathogens reported to the EARS-Net enhanced surveillance.

Table 1. Summary of antimicrobial resistance to key antibiotics by pathogen in Ireland, 2013-2017 (data correct as of 09/11/2018)

Pathogen	Year 2013	2014	2015	2016	2017
Number laboratories by year-end	**41**	**39**	**38**	**37**	**39**
%Coverage of population	*100*	*100*	*97*	*99*	*100*
E. coli					
Number of isolates	**2530**	**2770**	**2697**	**3059**	**3180**
%Ampicillin-R*	*70.9*	*69.9*	*66.7*	*68.4*	*69.9*
%3GC-R*	*12.3*	*12.0*	*12.5*	*12.2*	*13.2*
%ESBL-producers*	*10.5*	*10.2*	*10.6*	*11.1*	*11.4*
%Ciprofloxacin-R*	*25.3*	*26.2*	*24.4*	*24.1*	*26.0*
%Gentamicin-R*	*9.8*	*11.2*	*11.0*	*10.2*	*11.4*
%Gentamicin/Amikacin/Tobramycin-R*	*12.9*	*14.5*	*13.4*	*13.2*	*14.2*
%Carbapenem†-R*	*0.1*	*0.1*	*0.2*	*0.2*	*0.3*
%MDR*	*6.1*	*6.1*	*6.5*	*6.6*	*7.0*
S. aureus					
Number of isolates	**1094**	**1116**	**1082**	**1168**	**1175**
Number Meticillin-R (or MRSA)	**222**	**217**	**199**	**172**	**192**
%Meticillin-R (or MRSA)	*20.3*	*19.4*	*18.4*	*14.7*	*16.3*
K. pneumoniae					
Number of isolates	**326**	**358**	**401**	**469**	**494**
%Ampicillin-R*	*99.1*	*100.0*	*99.3*	*99.4*	*99.6*
%3GC-R*	*21.2*	*13.1*	*17.5*	*16.9*	*16.9*
%ESBL-producers*	*18.4*	*11.0*	*13.3*	*12.9*	*12.6*
%Ciprofloxacin-R*	*20.9*	*17.3*	*21.6*	*16.6*	*20.7*
%Gentamicin-R*	*16.9*	*12.6*	*17.0*	*11.5*	*12.0*
%Gentamicin/Amikacin/Tobramycin-R*	*17.8*	*13.2*	*18.0*	*12.6*	*13.6*
%Carbapenem†-R*	*1.2*	*1.1*	*2.2*	*1.1*	*0.8*
%MDR*	*12.9*	*8.7*	*9.5*	*8.3*	*7.3*
E. faecium					
Number of isolates	**409**	**404**	**421**	**431**	**454**
%Ampicillin-R*	*93.2*	*95.3*	*94.3*	*94.6*	*93.5*
%Vancomycin-R (VREfm)	*43.1*	*45.8*	*45.6*	*44.4*	*39.4*
%HLG-R*	*41.4*	*44.4*	*49.5*	*58.3*	*67.7*
%Linezolid-R*	*1.2*	*2.0*	*0.7*	*0.2*	*0.2*
%MDR*	*19.6*	*22.2*	*21.3*	*28.2*	*30.5*
E. faecalis					
Number of isolates	**336**	**315**	**294**	**297**	**345**
%Ampicillin-R*	*2.7*	*1.6*	*0.7*	*0.7*	*0.6*
%Vancomycin-R (VREfa)	*2.1*	*2.9*	*1.4*	*1.0*	*0.6*
%HLG-R*	*33.6*	*32.8*	*28.0*	*29.4*	*31.0*
%Linezolid-R*	*0.6*	*1.0*	*0.4*	*0.0*	*0.9*

* Not all isolates tested; † Carbapenems include imipenem, meropenem and ertapenem
Number of isolates presented in **bold**; proportions (%) presented in *italics*
R, Resistant; NS, Non-Susceptible [includes isolates with intermediate (Int) and high-level resistance (HLR)]
MRSA, Meticillin-Resistant *S. aureus* ; VREfm, Vancomycin-Resistant *E. faecium*
HLG, High-Level Gentamicin; 3GC, 3rd-Generation Cephalosporin (includes cefotaxime, ceftriaxone, ceftazidime)
ESBL, Extended-Spectrum Beta-Lactamase; MDR, Multi-Drug Resistant (see definition in text for each pathogen)

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Table 1 (continued). Summary of key antimicrobial resistance to key antibiotics by pathogen in Ireland, 2013-2017 (data correct as of 09/11/2018)

Pathogen	Year				
	2013	2014	2015	2016	2017
Number laboratories by year-end	**41**	**39**	**38**	**37**	**39**
%Coverage of population	*100*	*100*	*97*	*99*	*100*
S. pneumoniae					
Number of isolates	**311**	**331**	**304**	**364**	**415**
%Penicillin-NS*	*20.7*	*17.1*	*17.5*	*16.5*	*15.7*
of which: %HLR	*2.6*	*2.4*	*0.3*	*0.0*	*1.2*
%Int	*18.0*	*14.5*	*17.2*	*16.5*	*14.5*
%Erythromycin-R*	*17.9*	*13.8*	*15.2*	*13.2*	*13.1*
%Penicillin-NS/Erythromycin-R*	*13.0*	*11.0*	*10.8*	*9.9*	*9.1*
P. aeruginosa					
Number of isolates	**207**	**182**	**201**	**251**	**296**
%Piperacillin/tazobactam-R*	*15.7*	*16.5*	*14.0*	*17.1*	*16.0*
%Ceftazidime-R*	*10.7*	*8.9*	*8.5*	*13.1*	*11.8*
%Imipenem/meropenem-R*	*13.1*	*11.6*	*16.4*	*13.1*	*13.5*
%Ciprofloxacin-R*	*15.0*	*13.7*	*13.5*	*16.7*	*16.6*
%Gentamicin-R*	*11.6*	*4.9*	*3.5*	*11.2*	*8.9*
%Gentamicin/Amikacin/Tobramycin-R*	*11.6*	*5.5*	*7.0*	*12.8*	*9.1*
%MDR*	*9.4*	*6.7*	*7.5*	*13.1*	*10.1*
***Acinetobacter* spp.**					
Number of isolates	**89**	**91**	**86**	**68**	**73**
%Ciprofloxacin-R*	*3*	*8*	*7*	*1*	*8*
%Gentamicin-R*	*0*	*3*	*4*	*2*	*2*
%Gentamicin/Amikacin/Tobramycin-R*	*1*	*3*	*5*	*3*	*3*
%Imipenem/meropenem-R*	*4*	*4*	*6*	*0*	*8*
%MDR*	*0*	*2*	*3*	*0*	*2*

* Not all isolates tested

Number of isolates presented in **bold**; proportions (%) presented in *italics*

R, Resistant; NS, Non-Susceptible [includes isolates with intermediate (Int) and high-level resistance (HLR)]

Multi-Drug Resistant (see definition in text for each pathogen)

Table 2. Age and gender breakdown of patients by pathogen with major resistance profiles in Ireland (data from laboratories participating in enhanced surveillance 2017 only). The proportion of isolates detected <48 hours and >5 days post-admission is also shown

Pathogen	Total for 2017	Percent female	Median age in years	Detected <48 hours after admission	Detected >5 days after admission
Escherichia coli	**1221**	39%	75.0	78%	18%
Fluoroquinolone Resistant	**330 (27%)**	43%	78.0	77%	20%
Fluoroquinolone Susceptible	**891**	56%	74.0	79%	17%
Staphylococcus aureus	**612**	35%	68.0	65%	24%
Meticillin Resistant (MRSA)	**100 (16%)**	34%	74.0	50%	41%
Meticillin Susceptible (MSSA)	**512**	35%	66.0	67%	21%
Klebsiella pneumoniae	**170**	39%	68.0	61%	35%
Enterococci	**270**	39%	70.5	41%	51%
Vancomycin Resistant (VRE)	**51 (19%)**	39%	63.0	22%	73%
Vancomycin Sensitive (VSE)	**219**	39%	72.0	46%	47%
Streptococcus pneumoniae	**161**	48%	68.0	97%	3%
Penicillin non-Susceptible	**19 (12%)**	53%	81.0	79%	21%
Penicillin Susceptible	**142**	47%	67.0	99%	1%
Pseudomonas aeruginosa	**112**	34%	71.5	55%	41%

Epidemiology

Escherichia coli

Escherichia coli is found in the normal gut flora of humans and animals. Most strains of *E. coli* are harmless, but some can be pathogenic and can cause disease in vulnerable patients. *E. coli* is the most frequent cause of urinary tract infections (UTI) and bloodstream infections (BSI) in humans, but is also associated with many other infections, including gastroenteritis, intra-abdominal infections and neonatal meningitis.

E. coli can develop resistance to the key antimicrobials used for treatment, potentially resulting in strains that are multi-drug resistant.

Resistance to third-generation cephalosporins (3GC) in *E. coli* and other *Enterobacterales* (previously known as *Enterobacteriaceae*) is most often due to the production of extended-spectrum beta-lactamases (ESBL), enzymes that confer resistance to most beta-lactams, but not to carbapenems. ESBL-producers are also often resistant to other antimicrobial classes.

As the incidence of ESBLs increases, this results in reliance on last-resort antimicrobials, such as carbapenems for treatment of severe or invasive infections. This in turn facilitates the emergence and spread of carbapenem resistance, in particular due to carbapenemases, enzymes that confer resistance to almost all beta-lactams, including carbapenems. The genes that encode carbapenemase production are found on plasmids, mobile genetic elements, facilitating spread between different species of *Enterobacterales*.

Antimicrobial resistance in *Escherichia coli*

In 2017, there were 3,180 reports of invasive *E. coli* infections (3,171 from blood and nine from CSF) from 3,126 patients, an increase of 4% compared with 2016 (n=3,059).

Table 1 displays the annual data since 2013 on the proportion of *E. coli* isolates resistant to the five indicator antimicrobials/antimicrobial classes: aminopenicillins (ampicillin or amoxicillin), third-generation cephalosporins (3GCs; cefotaxime, ceftriaxone or ceftazidime), fluoroquinolones (ciprofloxacin), aminoglycosides (gentamicin, tobramycin or amikacin) and carbapenems (meropenem or ertapenem):

- Resistance to third-generation cephalosporins, fluoroquinolones and aminoglycosides increased slightly in 2017 compared with 2016. While there was no significant increase over the period 2013-2017, there has been an overall upward trend since 2008 (Figure 3)
- ESBL-producers accounted for 11.4% of isolates, the highest proportion since surveillance began (Figure 1)
- Aminopenicillin resistance was observed in 69.9% of isolates although this has remained relatively stable. Ireland has the 2nd highest proportion of aminopenicillin resistance in Europe. When resistance profiles to all indicator antimicrobials are

examined, Ireland had the third lowest proportion of fully susceptible isolates in Europe (Figure 4)

- Nine isolates (0.3%) were resistant to carbapenems:
  - Four were carbapenemase-producers, or CPE (all OXA-48-type)
  - In addition, another isolate that was carbapenem-sensitive on susceptibility testing was found to be CPE (Table 3)
  - In the period 2012-2017, 26 *E. coli* invasive isolates were reported as carbapenem-resistant, with eight confirmed to be CPE, of which three were resistant to all five indicator antimicrobials
  - Carbapenem resistance among invasive *E. coli* isolates remains at low levels to date in Ireland and across Europe
- Multi-drug resistance (MDR) in *E. coli* is of increasing concern:
  - Multi-drug resistant (MDR) *E. coli* is defined as combined resistance to 3GCs, fluoroquinolones and aminoglycosides
    Please note: the definition of MDR has been updated to match that used by ECDC: previously our definition for MDR was three or more of the indicator antimicrobials OR any isolate with resistance to carbapenems
  - MDR *E. coli* accounted for 7.0% of isolates, an increase from 6.6% in 2016. While MDR increased over the period 2013-2017, the trend was not significant. In 2017, Ireland had one of the highest proportions of combined resistance in North and Western Europe (Figure 5)
  - One isolate was resistant to all five indicator antimicrobials, compared with four in 2016. Across Europe, only 24 such isolates displayed this resistance profile, an increase from 11 in 2016
- The majority of *E. coli* (80%) and MDR-*E. coli* (74%) were detected within two days of admission to hospital
- The majority of *E. coli* (78%) occurred in patients over 60 years (median age=74 years)

Enhanced surveillance findings:

- 39% of fluoroquinolone-resistant *E. coli* (FQREC) BSIs were reported as healthcare-associated infection, which contrasts with 31% for fluoroquinolone-susceptible *E. coli* (FQSEC)
- The most common source of *E. coli* BSI was urinary tract infection, with 47% FQREC BSI and 42% FQSEC reported in association with the presence of a urinary catheter
- Recent antimicrobial exposure was noted in 13% of cases of *E. coli* BSI

Figure 1. **Trends for invasive *E. coli* infections in Ireland, 2008-2017: total numbers of *E. coli* with percentage third-generation cephalosporin resistant (3GC-R) and ESBL producer (ESBL+ve)**

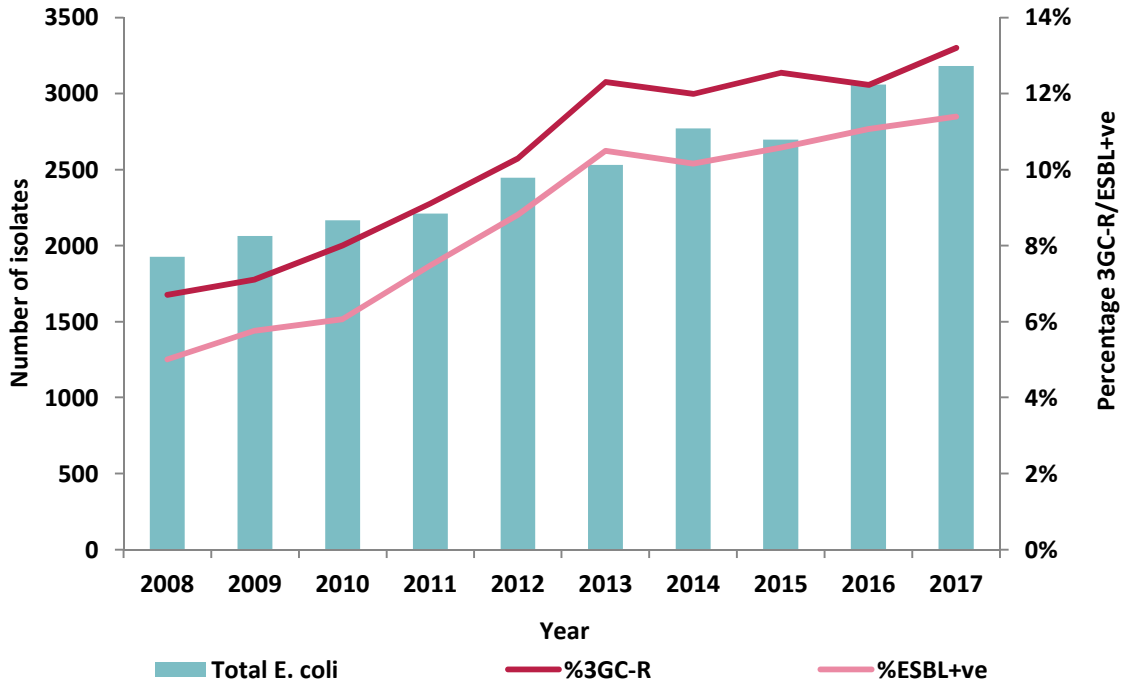
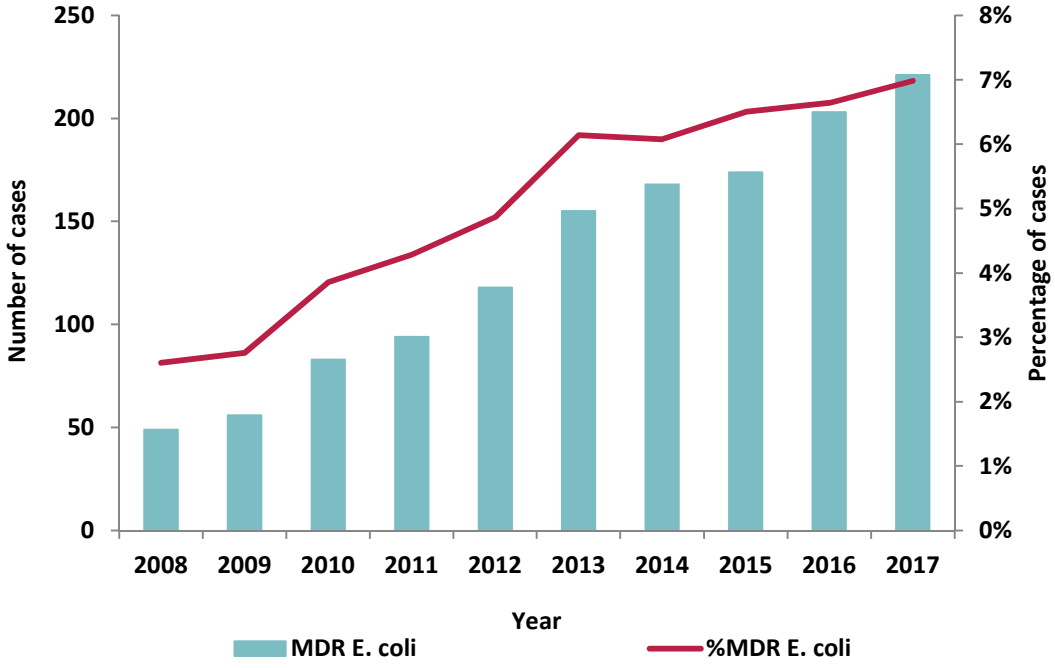


Figure 2. **Trends for invasive *E. coli* infections in Ireland: total numbers of *E. coli* tested for multi-drug resistance (MDR)* with number and percentage that are MDR**



*Multi-drug resistance (MDR) defined as resistance to combined resistance to 3GCs, fluoroquinolones and aminoglycosides

Table 3. Data on carbapenem resistant compared with carbapenemase-producing *E. coli* in Ireland, 2011-2017

Year	Number CRE (*E. coli*)	Number CPE (*E. coli*)	Enzymes detected (numbers)
2011	0	0	
2012	2	0	
2013	2	0	
2014	2	1	NDM (1)
2015	6	2	NDM (1), OXA-48 (1)
2016	5	1	NDM (1)
2017	9	4	OXA-48 (4)*
Total	26	8	5 OXA-48 (5), NDM (3)

CRE, carbapenem-resistant *Enterobacterales* ; CPE, carbapenemase-producing *Enterobacterales*

*Plus an additional CPE where carbapenem-sensitive (OXA-48)

Figure 3. Distribution of *E. coli* isolates from fully susceptible (blue) to resistance to 1-5 of the indicator antimicrobials in EU/EEA countries, 2017

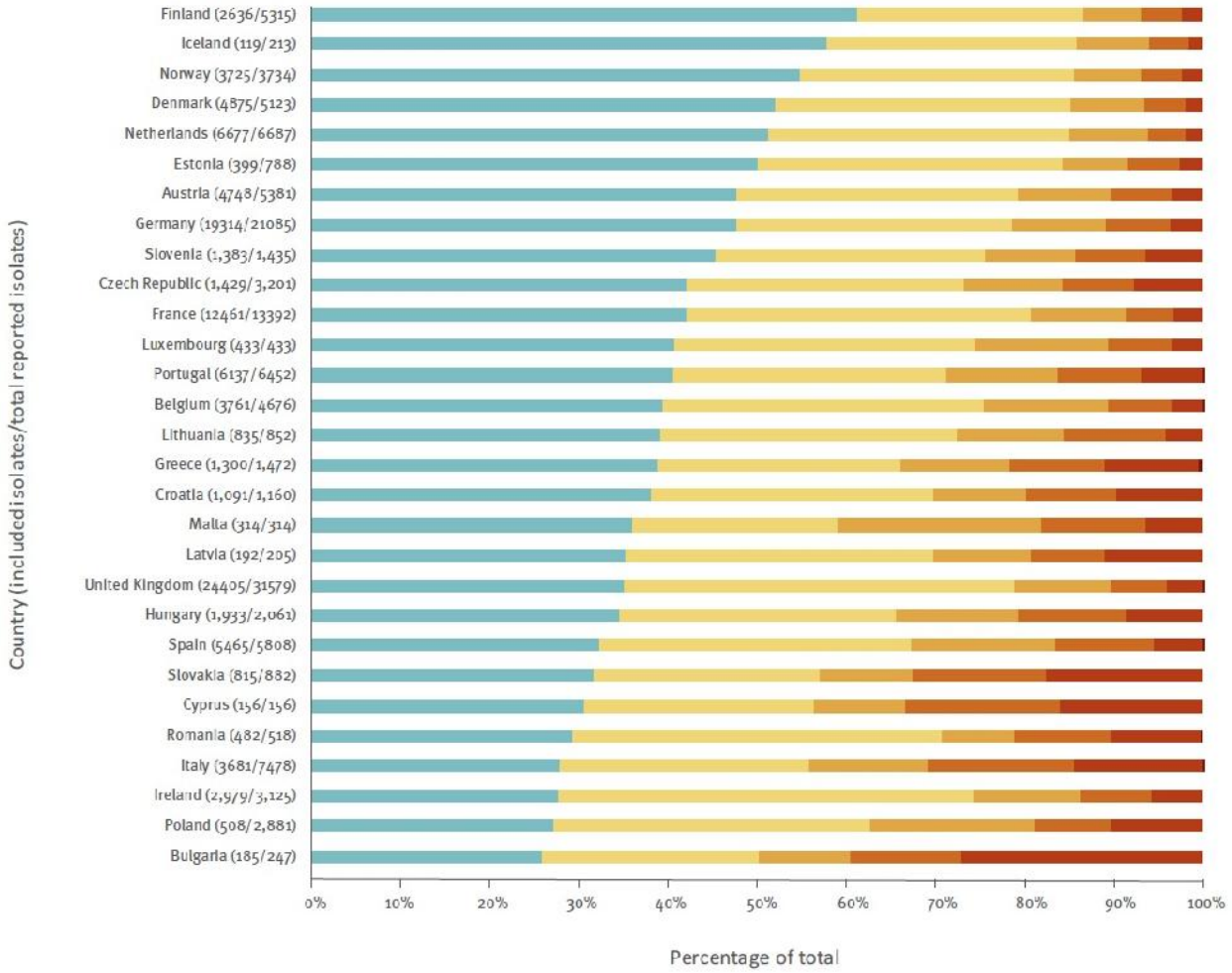
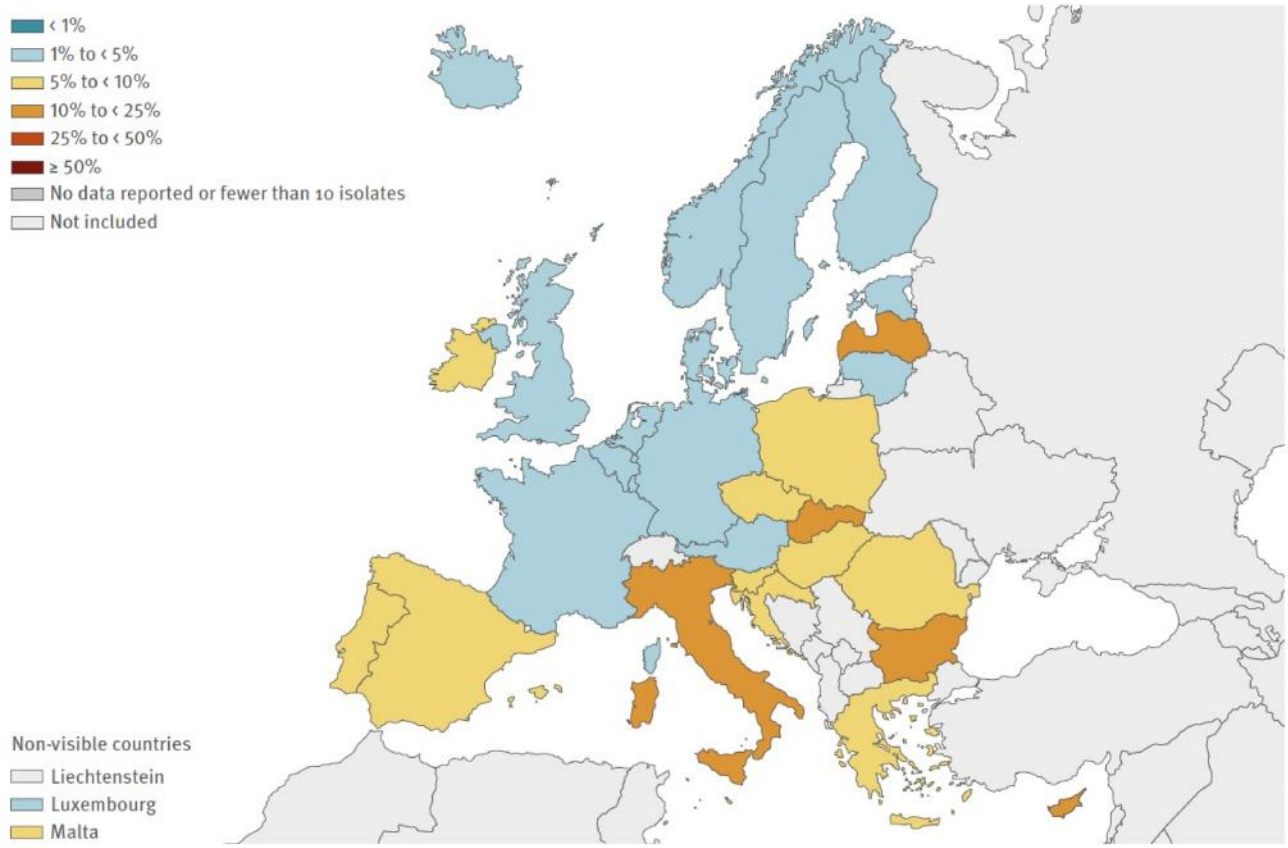


Figure kindly provided by Liselotte Diaz Högberg, European Centre for Disease Prevention and Control (ECDC)

Figure 4. Distribution of *E. coli* with multi-drug resistance (defined as combined resistance to 3GCs, fluoroquinolones and aminoglycosides) in EU/EEA countries reported to EARS-Net in 2017



Map kindly provided by Liselotte Diaz Högberg, European Centre for Disease Prevention and Control (ECDC)

Staphylococcus aureus

Staphylococcus aureus is commonly found on the skin and mucous membranes of healthy people. In addition to being one of the commonest causes of BSI, *S. aureus* is a frequent cause of skin, soft tissue and bone infections.

Flucloxacillin is the treatment of choice for *S. aureus* infections. Resistance to flucloxacillin, better known as meticillin resistance, is mediated by *mec*A, a gene that confers resistance to beta-lactams.

Antimicrobial resistance in *S. aureus*

There were 1,175 reports of *S. aureus* BSI from 1,146 patients, which is similar to 2016 (n=1,169).

- In 2017, 16.3% of cases were MRSA, which represents a slight increase from 14.7% in 2016. (Table 1 shows data for the period 2013-2017). The trend for the past five years remains downwards (Figure 5)
- Overall, there was an 11.6% increase in the number of reported MRSA BSI compared with 2016 (192 versus 172 cases). In contrast, the total number of MSSA BSI decreased by 1.4% (983 versus 987 cases)
- Calculating separate rates (numbers of cases per 1,000 patient days) for MRSA and MSSA, which are generally independent of each other, gives a better indication of the burden of these infections on acute care hospitals in Ireland. In 2017, the MRSA rate was 0.046 cases per 1,000 patient days, a small increase from 0.043 in 2016, while the MSSA rate decreased from 0.245 to 0.238 (Figure 6)
- Despite decreasing trends in recent years, MRSA continues to be a problem in Irish hospitals. In 2017, Ireland had one of the highest proportions of MRSA in North and Western Europe (Figure 7)
- A higher proportion of MRSA (44%) were temporally associated with an admission episode at the reporting hospital (i.e. were isolated 3 days or more after admission) than MSSA (35%)
- The majority of *S. aureus* (55%) and MRSA (56%) isolates were reported by the nine tertiary hospitals, which comprised 45% of all patient bed days in 2017
- MRSA is more likely to occur in older patients than MSSA: 78% of MRSA occurred in patients over 60 years (median age=78 years), compared with 58% of MSSA (median age=58 years); with almost two-thirds of *S. aureus* infections (regardless of whether MRSA or MSSA) occuring in males

Figure 5. Trends for *S. aureus* bloodstream infections in Ireland, 2008-2017: total numbers of *S. aureus*/MRSA and percentage MRSA

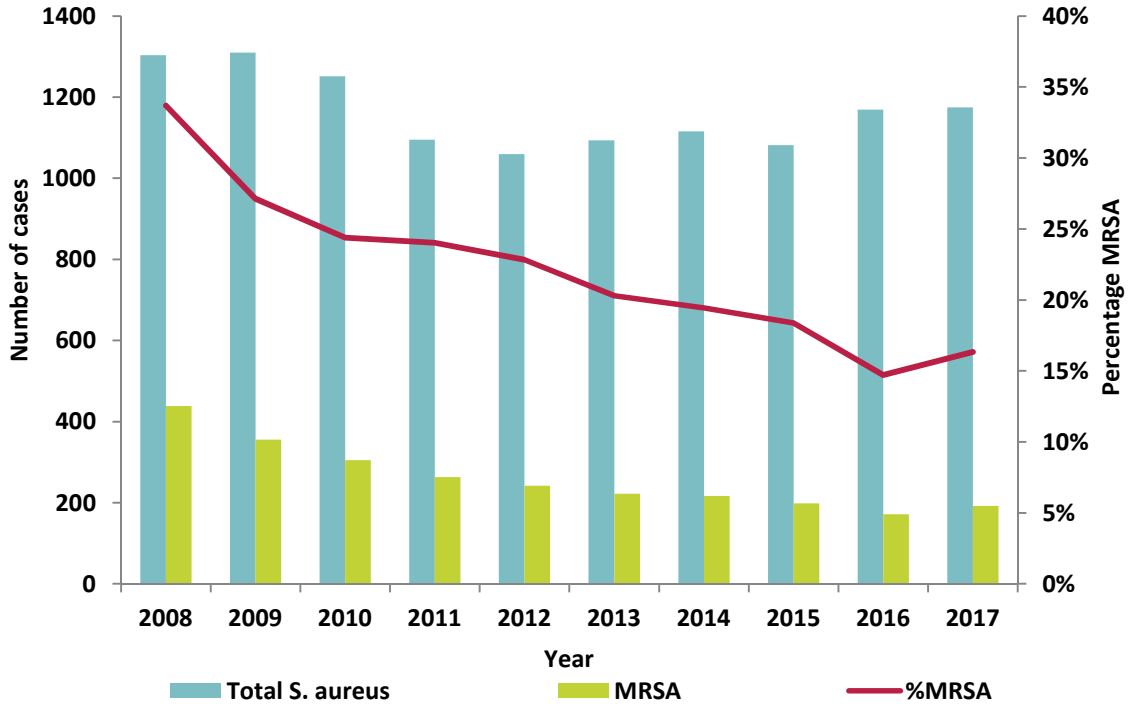
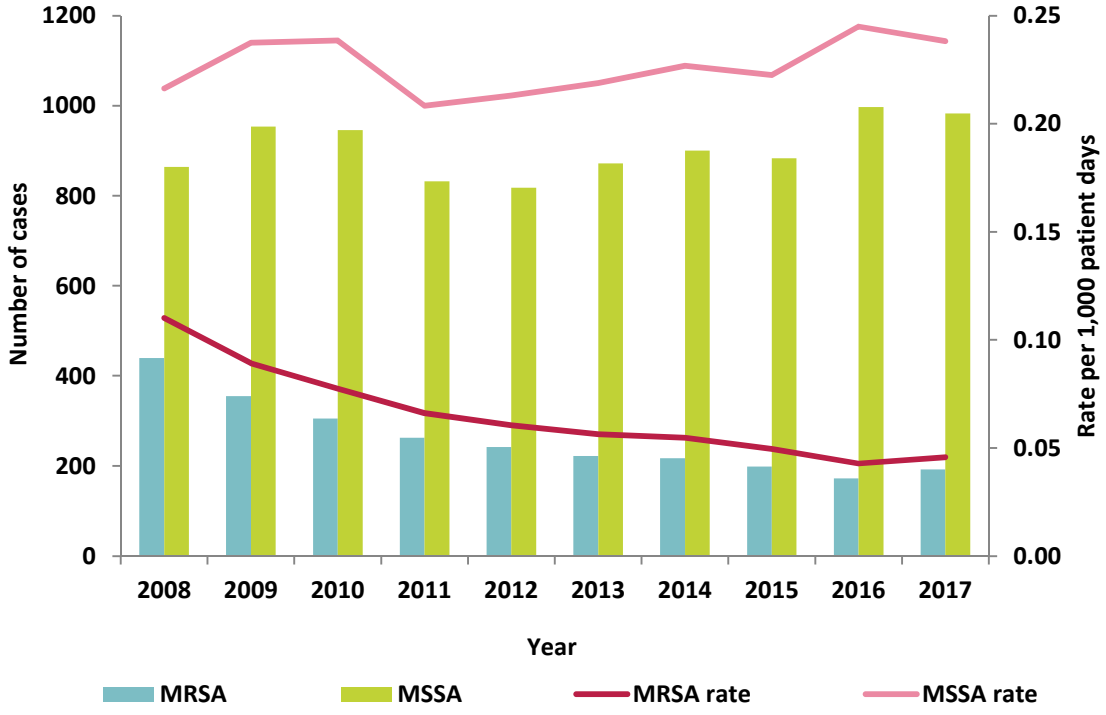


Figure 6. Trends for *S. aureus* bloodstream infections in Ireland, 2008-2017: total numbers of *S. aureus*/MRSA and percentage MRSA



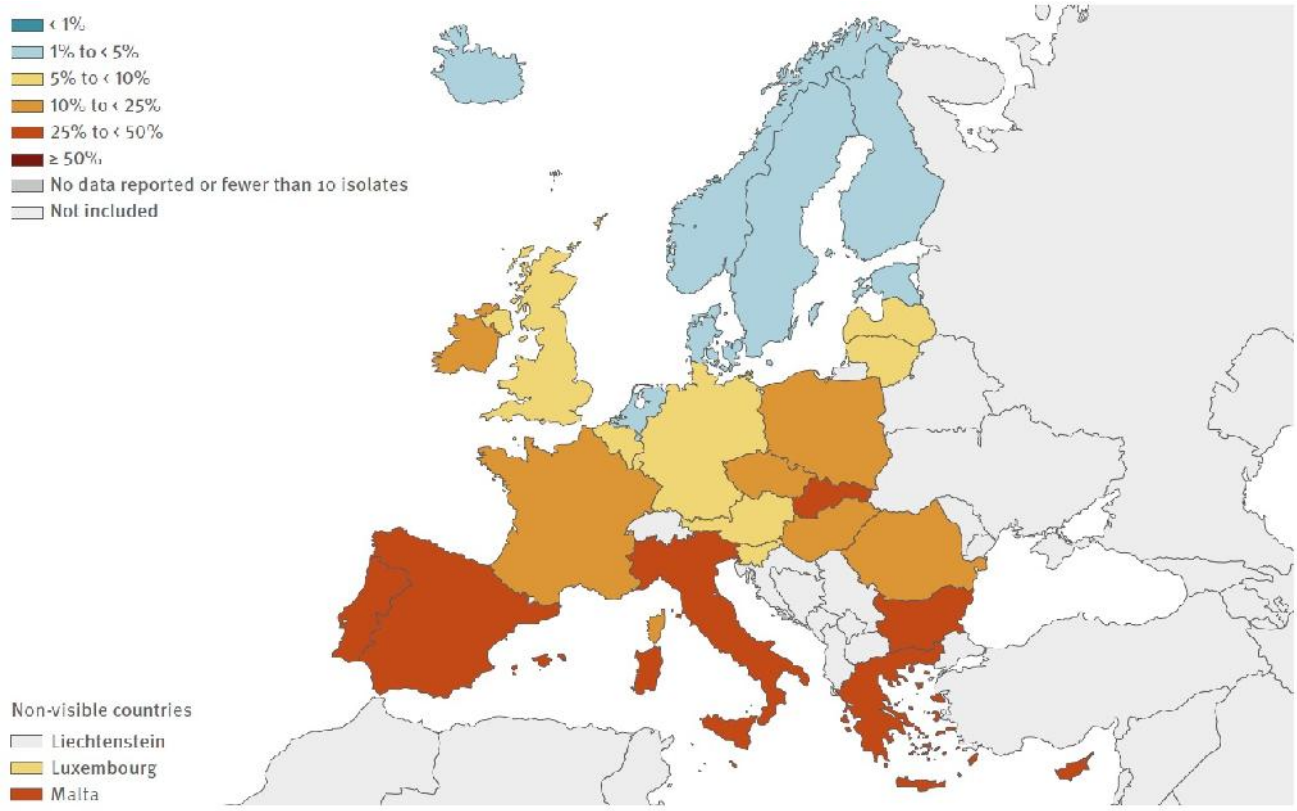
Enhanced surveillance findings:

- 55% of MRSA and 50% of MSSA BSIs were reported as healthcare-associated
- 11% of MRSA and 16% of MSSA BSIs were reported as device-associated
- 24% of patients with MRSA and 23% of patients with MSSA BSIs were noted to have had recent antimicrobial exposure
- The most common source for *S. aureus* BSI was non-surgical wound (skin and soft tissue infection): MRSA; 21%, MSSA; 20%

Further data on *S. aureus* BSI in Ireland for the period 2008-2017, including breakdown by MRSA and MSSA, by acute hospital (both public and private) is available at the following link: http://www.hpsc.ie/a-z/microbiologyantimicrobialresistance/europeanantimicrobialresistancesurveillancesystemearss/referenceandeducationalresourcematerial/saureusmrsa/latestsaureusmrsadata/

For more detailed European data on *S. aureus* and all other pathogens described in this report, including tables, charts and maps, see https://ecdc.europa.eu/en/antimicrobial-resistance/surveillance-and-disease-data/data-ecdc

Figure 7. Distribution of MRSA in EU/EEA countries reported to EARS-Net in 2017



Map kindly provided by Liselotte Diaz Högberg, European Centre for Disease Prevention and Control (ECDC)

Klebsiella pneumoniae

Klebsiella pneumoniae resides in the normal human gut flora. Most *K. pneumoniae* infections are healthcare-associated, including pneumonia, wound, UTI and BSI.

Similar to *E. coli*, *K. pneumoniae* can develop resistance to key antimicrobials. Indeed, many ESBLs and carbapenemases were initially identified in *K. pneumoniae,* before spreading to *E. coli* and other *Enterobacterales* species.

Antimicrobial resistance in *K. pneumoniae*

There were 493 reports of invasive *K. pneumoniae* infection (492 from blood and one from CSF) from 481 patients, an increase of 5% from 2016 (n=469).

Table 1 displays annual data since 2013 on the proportion of *K. pneumoniae* isolates resistant to the five indicator antimicrobials (as for *E. coli* above; note: *K. pneumoniae* are naturally resistant to aminopenicillins and any susceptible isolates that are reported represent either an error in identification or susceptibility testing) :

- Resistance to 3GC was reported for 16.9% of isolates, while 12.7% were ESBL producers, similar to 2016 (16.8% and 12.9%, respectively) (Figure 8)
- Resistance to fluoroquinolones and aminoglycosides increased compared with 2016
- Four (1.1%) isolates were resistant to carbapenems (Figure 8):
    - Three isolates were carbapenemase-producers, or CPE (all OXA-48-type)
    - An additional isolate was sensitive to carbapenems, but confirmed to be a CPE (also an OXA-48-type)
    - In the period 2011-2017, 33 invasive *K. pneumoniae* isolates were reported as carbapenem-resistant, with 22 confirmed as CPE, of which 18 were resistant to all five indicator antimicrobials. In addition, another isolate that was carbapenem-sensitive was found to be a CPE (Table 4)
    - As shown in Figure 9, carbapenem resistance among invasive isolates of *K. pneumoniae* in Ireland remains at a low level, unlike southern and eastern European countries, including Greece (64.7%) and Italy (29.7%)
- MDR in *K. pneumoniae* is a major concern:
    - MDR-*K. pneumoniae* is defined as combined resistance to 3GCs, fluoroquinolones and aminoglycosides
      Please note: the definition of MDR has been updated to match that used by ECDC: previously our definition for MDR was three or more of the indicator antimicrobials OR any isolate with resistance to carbapenems
    - MDR accounted for 7.3% of isolates, a decrease from 2016 (8.3%). A significant decrease in MDR *K. pneumoniae* was observed from 2013-2017 (Figure 10)

  - No isolates were resistant to all five indicator antimicrobials, compared with four in 2016. Across Europe, 4.5% of isolates displayed this resistance profile, an increase from 4.4% in 2016

- AMR among invasive *K. pneumoniae* isolates, including MDR, in Ireland is amongst the lowest in Europe (Figure 11)
- The majority of *K. pneumoniae* (53%) and MDR *K. pneumoniae* (59%) isolates were reported by the nine tertiary hospitals, which comprised 45% of all patient days in 2017
- The majority of MDR *K. pneumoniae* (57%) were associated with the reporting hospital (i.e. were isolated three days or more after admission)
- The majority of *K. pneumoniae* infections (71%) occurred in patients over 60 years (median age=69 years), with almost two-thirds of *K. pneumoniae* infections occuring in males

Figure 8. Trends for invasive *K. pneumoniae* infections in Ireland, 2008-2017: total numbers of *K. pneumoniae* with percentage 3GC resistant (3GC-R), ESBL producer (ESBL+ve) and carbapenem resistant (CBP-R)

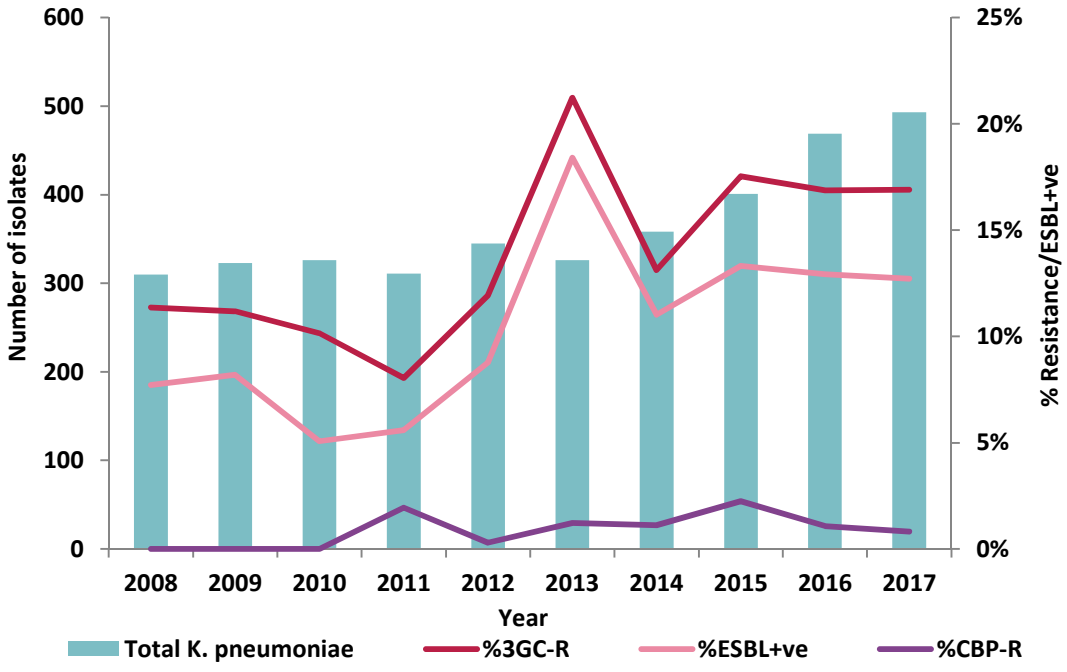


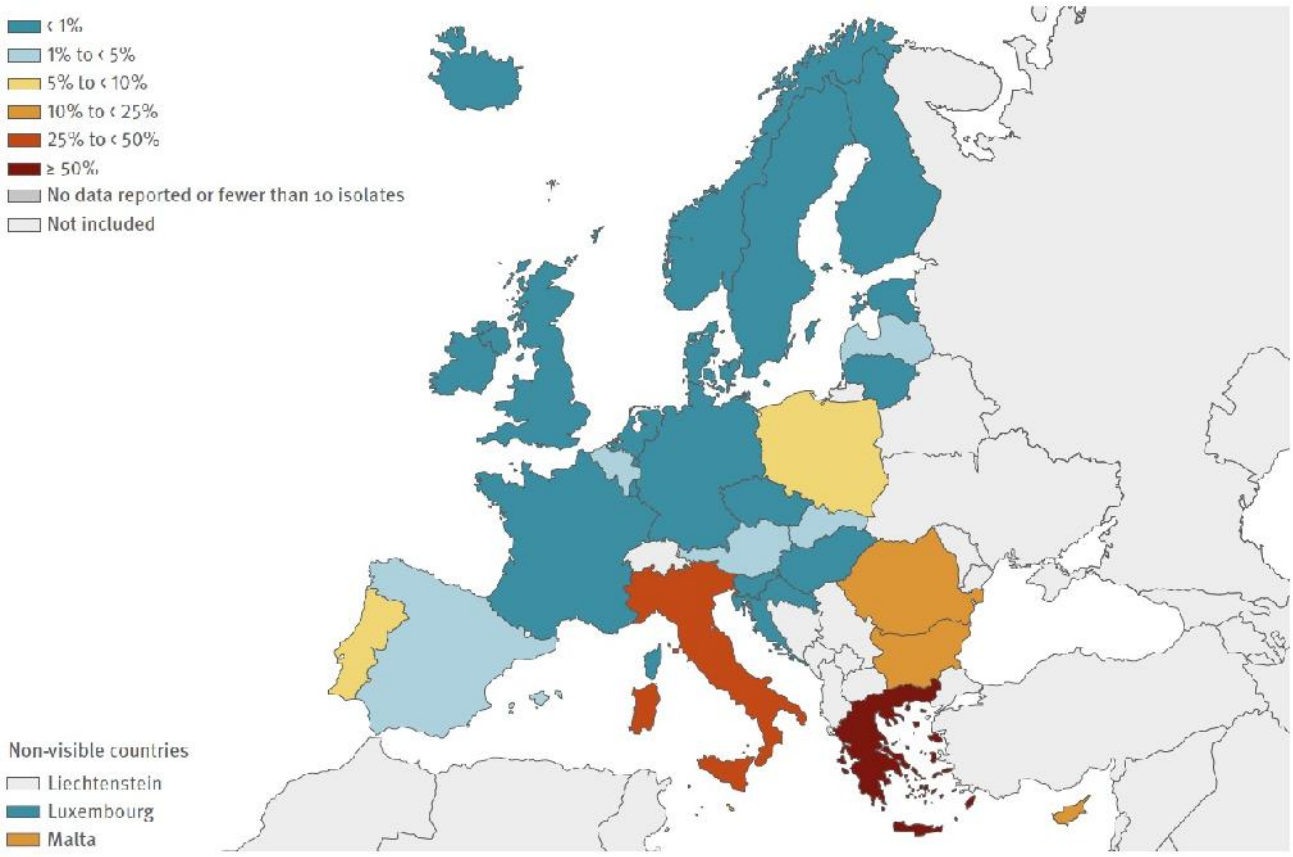
Table 4. Data on carbapenem resistant *K. pneumoniae* compared with carbapenemase-producing *K. pneumoniae* in Ireland, 2011-2017

Year	Number CRE (*K. pneumoniae*)	Number CPE (*K. pneumoniae*)	Enzymes detected (numbers)
2011	6	4	OXA-48 (3), KPC(1)
2012	1	0	
2013	4	2	OXA-48 (2)
2014	4	2	OXA-48 (1), KPC(1)
2015	9	7	OXA-48 (6), KPC(1)
2016	5	4	KPC(3), OXA-48 (1)
2017	4	3	OXA-48 (3)*
Total	33	22	16 OXA-48 (16), KPC (6)

CRE, carbapenem-resistant *Enterobacterales* ; CPE, carbapenemase-producing *Enterobacterales*

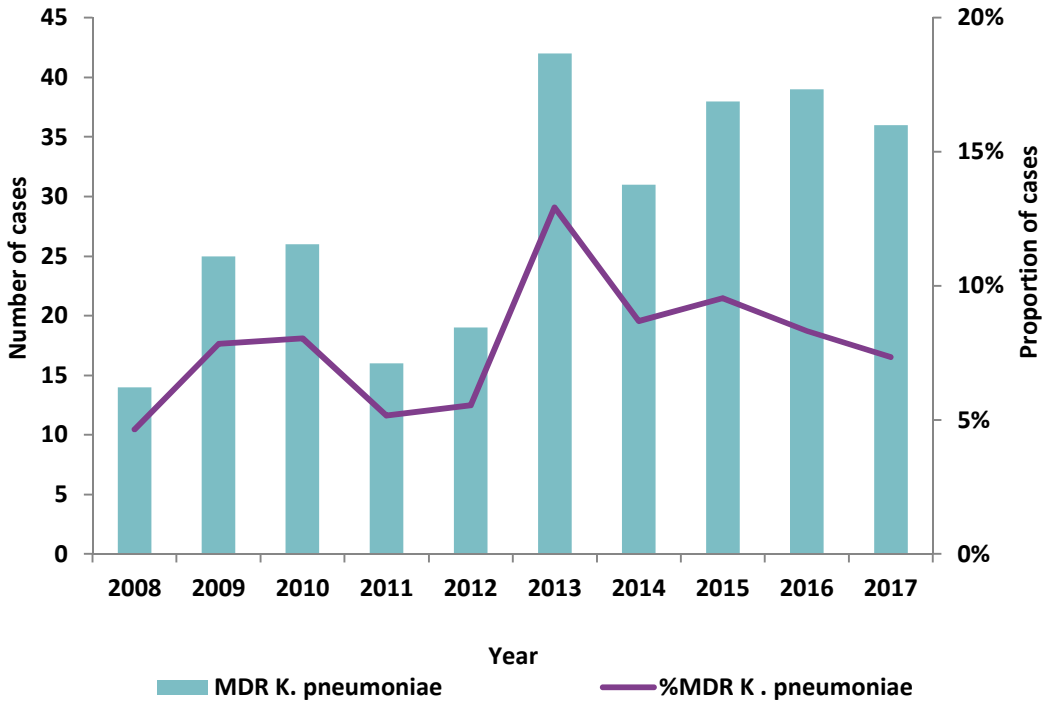
*Plus an additional CPE where carbapenem-sensitive (OXA-48)

Figure 9. Distribution of *K. pneumoniae* with carbapenem resistance in EU/EEA countries reported to EARS-Net in 2017



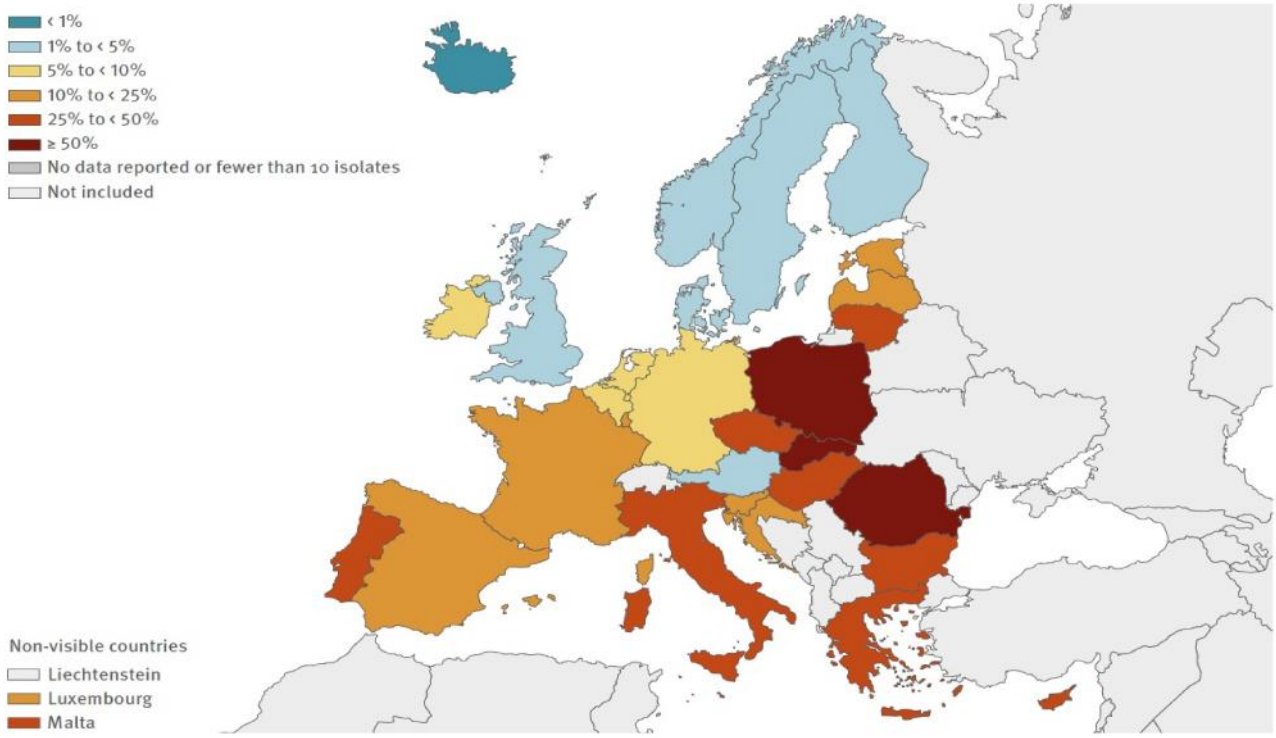
Map kindly provided by Liselotte Diaz Högberg, European Centre for Disease Prevention and Control (ECDC)

Figure 10. Trends for invasive *K. pneumoniae* infections in Ireland, 2008-2017: total numbers of *K. pneumoniae* tested for MDR*, with number and percentage MDR



*Multi-drug resistance defined as resistance to combined resistance to 3GCs, fluoroquinolones and aminoglycosides

Figure 11. Distribution of MDR *K. pneumoniae* (defined as combined resistance to 3GCs, fluoroquinolones and aminoglycosides) in EU/EEA countries reported to EARS-Net in 2017



Map kindly provided by Liselotte Diaz Högberg, European Centre for Disease Prevention and Control (ECDC)

Enterococci

Enterococci reside in the normal human gut flora. They can cause infections, including BSI, in severely ill and immunocompromised patients. The majority of infections are due to *Enterococcus faecium* and *Enterococcus faecalis*.

The enterococci are naturally resistant to many antimicrobials, including to low levels of aminoglycosides (e.g. gentamicin). Resistance to vancomycin and high-level gentamicin can emerge by the bacteria acquiring specific resistance genes.

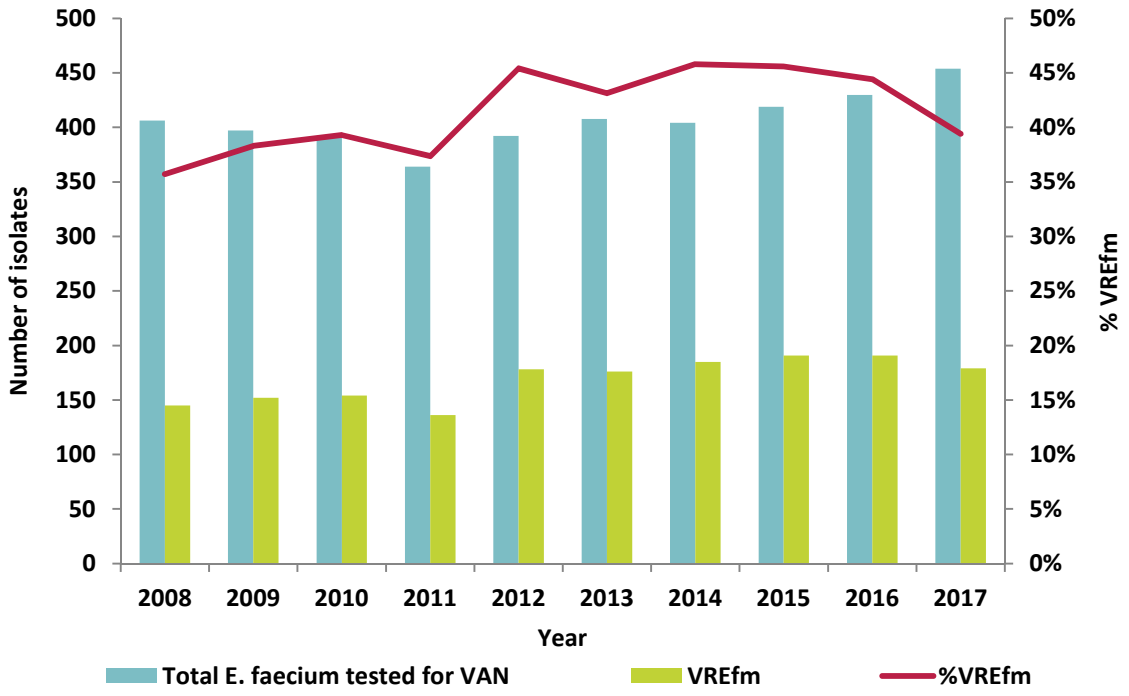
Antimicrobial resistance in *E. faecium*

In 2017, there were 454 reports of *E. faecium* BSI from 442 patients, an increase of 5% from 2016 (n=431).

Table 1 displays the annual trends since 2013 in the proportion of *E. faecium* isolates resistant to the three indicator antimicrobials: ampicillin, vancomycin and high-level gentamicin:

- Vancomycin resistance was observed in 39.4% of isolates (VREfm), which represents a decrease from 44.4% in 2016 (Figure 12)

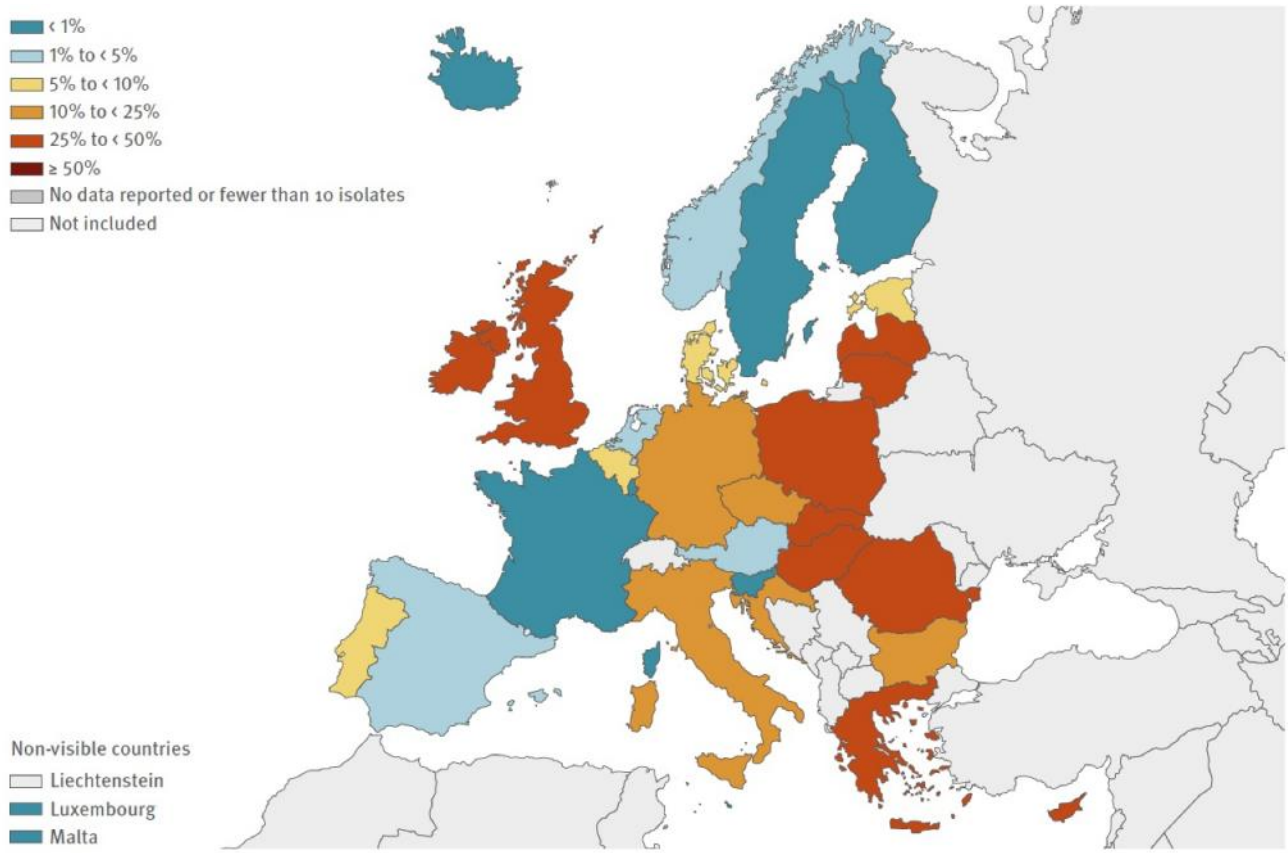
Figure 12. Trends for *E. faecium* BSI in Ireland, 2008-2017: total numbers of *E. faecium* and percentage vancomycin-resistant *E. faecium* (VREfm)



- Resistance to all three indicator antimicrobials (MDR-*E. faecium*) was reported for 30.5% of isolates, an increase from 28.2% in 2016

- In 2017, Ireland ranked second after Cyprus (46.3%) and was among ten countries in Europe reporting proportions of VREfm over 25% (Figure 13). Nine countries have reported increasing trends, compared with two reporting decreasing trends, including Ireland
- One isolate (0.2%) was reported as resistant to linezolid
- The majority of *E. faecium* (63%) and VREfm (74%) BSI were reported by the nine tertiary hospitals (which comprised 45% of all patient days in 2017)
- The majority of *E. faecium* (70%) and VREfm (69%) BSI were associated with the hospital of admission (i.e. were isolated three days or more after admission)
- The majority of *E. faecium* (73%) BSI occurred in patients over 60 years (median age=70 years), with nearly two-thirds occuring in males

Figure 13. Distribution of VREfm in EU/EEA countries reported to EARS-Net in 2017



Map kindly provided by Liselotte Diaz Högberg, European Centre for Disease Prevention and Control (ECDC)

Antimicrobial resistance in *E. faecalis*

There were 345 reports of *E. faecalis* BSI from 341 patients, an increase of 16% from 2016 (n=297).

Table 1 displays annual trends since 2013 in the proportions of *E. faecalis* isolates with resistance to the three indicator antimicrobials (as for *E. faecium* above):

- Two isolates (0.6%) were resistant to vancomycin (VREfa). VREfa remains at low levels in Ireland
- Three isolates (0.9%) were resistant to linezolid, of which one was confirmed to be positive for optrA, an emerging transferrable gene that confers linezolid resistance on enterococci
- The majority of *E. faecalis* (53%) isolates were reported by the nine tertiary hospitals (which comprised 45% of all patient days in 2017)
- The majority of *E. faecalis* (59%) were detected within two days of admission to hospital. The majority of *E. faecalis* (71%) occurred in patients over 60 years (median age=72 years) with approximately two-thirds of *E. faecium* infections occuring in males

Enhanced surveillance findings (data for *E. faecium* and *E. faecalis* combined):

- Healthcare-association was reported for 94% of the vancomycin-resistant enterococcal (VRE) and 69% of the vancomycin-susceptible enterococcal (VSE) BSIs
- Device-association was reported for 6% of VRE and 5% of VSE BSI
- Recent antimicrobial exposure was noted for 8% of patients with VRE and 9% with VSE BSI

Streptococcus pneumoniae

Streptococcus pneumoniae resides in the normal upper respiratory tract flora. It is the main cause of community-acquired pneumonia and otitis media and is also associated with invasive infections, including BSI and meningitis.

The treatment of choice for pneumococcal infections is penicillin. Resistance to penicillin can arise due to alterations in the penicillin-binding proteins (PBPs), the target sites for penicillin in the bacterial cell wall, resulting in lowered affinity of these PBPs, for penicillin. The level of resistance to penicillin can be intermediate or high-level depending on the exact alteration to the PBP. It is important to determine the exact level of resistance (or non-susceptibility), as this will influence the treatment options that are available, depending on the site of infection.

Antimicrobial resistance in *S. pneumoniae*

There were 415 reports of invasive *S. pneumoniae* infection (413 from blood and two from CSF) from 412 patients, an increase of 14% from 2016 (n=364).

Table 1 displays annual trends since 2013 in the proportions of *S. pneumoniae* isolates non-susceptible/resistant to penicillin and erythromycin:

- Penicillin non-susceptible *S. pneumoniae* (PNSP) accounted for 15.7% (14.5% intermediately-resistant and 1.2% high-level resistant, or HLR) of all isolates tested against penicillin in 2017, which represents a decrease from 16.5% in 2016
- Five isolates displayed HLR to penicillin, an increase from zero in 2016. However, there has been an overall decreasing trend from 2011 onwards (Figure 14)
- Erythromycin resistance was reported for 13.1% of isolates, similar to 2016 (13.2%)
- PNSP and erythromycin co-resistance was reported for 9.6% of isolates, similar to 2016 (9.9%)
- The proportion of PNSP in Ireland has decreased over the period 2013-2017 (Figure 14). Ireland has moderately-high proportions of PNSP compared to other EU/EEA countries. However, data may not be comparable across all countries due to the potential for different interpretive criteria to be applied depending on the guidelines used and the site of infection
- Serotyping results were available for 92% of invasive pneumococcal isolates:
    - Of all invasive *S. pneumoniae* cases, eight serotypes comprised over two-thirds of all isolates: 8 (17%), 12F (14%), 19A† (9%), 9N (7%), 3† (6%), 15A and 22F (5% each), and 33F (4%); 26 serotypes comprised the remaining third
    - Among PNSP isolates, three serotypes predominated: 19A (31%), 15A (21%) and 23B (14%), with nine serotypes making up the remainder. Of the five PNSP isolates with HLR to penicillin, four belonged to serotype 19A

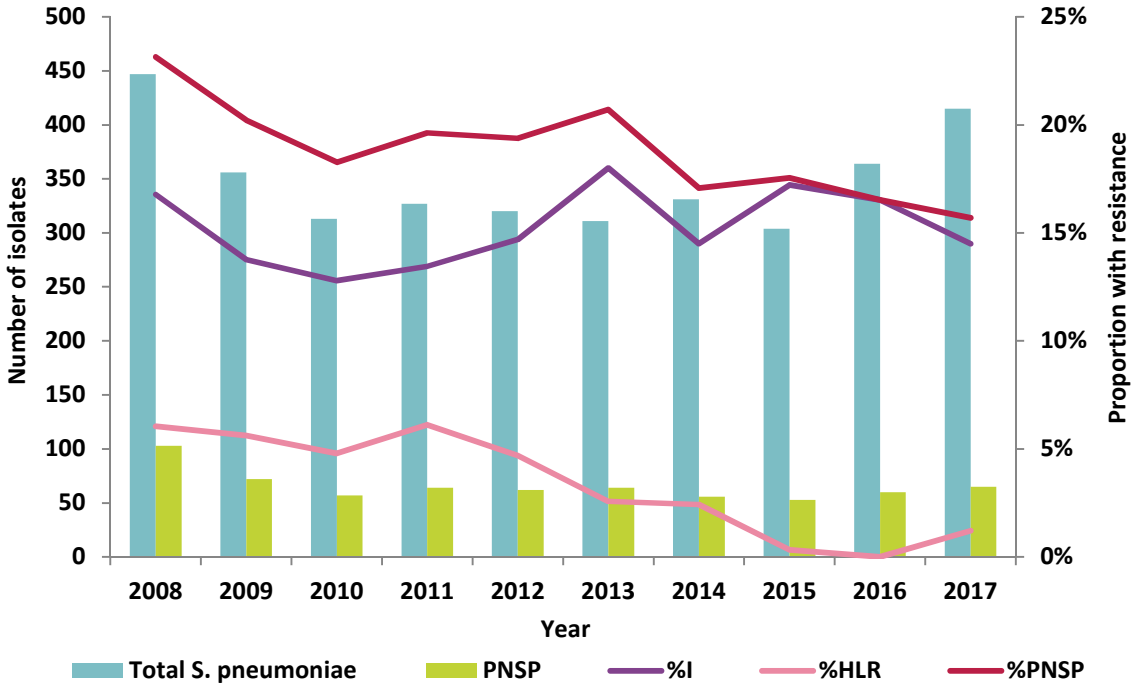
- Following the introduction of pneumococcal conjugate vaccines (PCV) into the childhood vaccination schedule (PCV7 in 2008 replaced by PCV13 in 2010), there has been a shift in the predominant serotypes causing invasive pneumococcal disease (IPD):
  - Of all invasive *S. pneumoniae* cases, non-PCV13 serotypes comprised almost 30% of all serotypes in 2008 compared with almost 80% in 2017
  - Among PNSP cases, non-PCV13 serotypes comprised just over 10% of all serotypes in 2008 compared with almost 60% in 2017. Of approximately 40% of PCV13 serotypes, the majority (78%) were 19A

†Serotypes 3 and 19A are included in PCV13. However, the vaccine may not always result in a strong immune response against these serotypes

For more details on the effects of vaccination on IPD, see the separate annual epidemiological report on IPD:

http://www.hpsc.ie/abouthpsc/annualreports/

Figure 14. Trends for invasive *S. pneumoniae* infections in Ireland, 2008-2017: total numbers of *S. pneumoniae* and penicillin non-susceptible *S. pneumoniae* (PNSP) and percentage PNSP, including penicillin intermediately-resistant (%I) and high-level resistant (%HLR)



- The majority of *S. pneumoniae* (96%) and PNSP (87%) were isolated within two days of hospital admission. The majority of *S. pneumoniae* (61%) occurred in patients over 60 years (median age=66 years) with males and females equally likely to get a *S. pneumoniae* infection

Enhanced surveillance findings:

- The majority of both PNSP and PSSP BSIs were community-acquired
- Respiratory tract infection remained the most common source of pneumococcal BSI

Pseudomonas aeruginosa and Acinetobacter spp.

Pseudomonas aeruginosa and *Acinetobacter* spp. are widely found in the environment and thrive in moist conditions, including on medical equipment such as catheters and ventilators. They are opportunistic pathogens in immunocompromised patients and cause infections, such as pneumonia, UTI and BSI.

P. aeruginosa and *Acinetobacter* spp. are inherently resistant to many antimicrobials. Hence, if resistance develops to those agents to which they remain susceptible, this can seriously compromise treatment options.

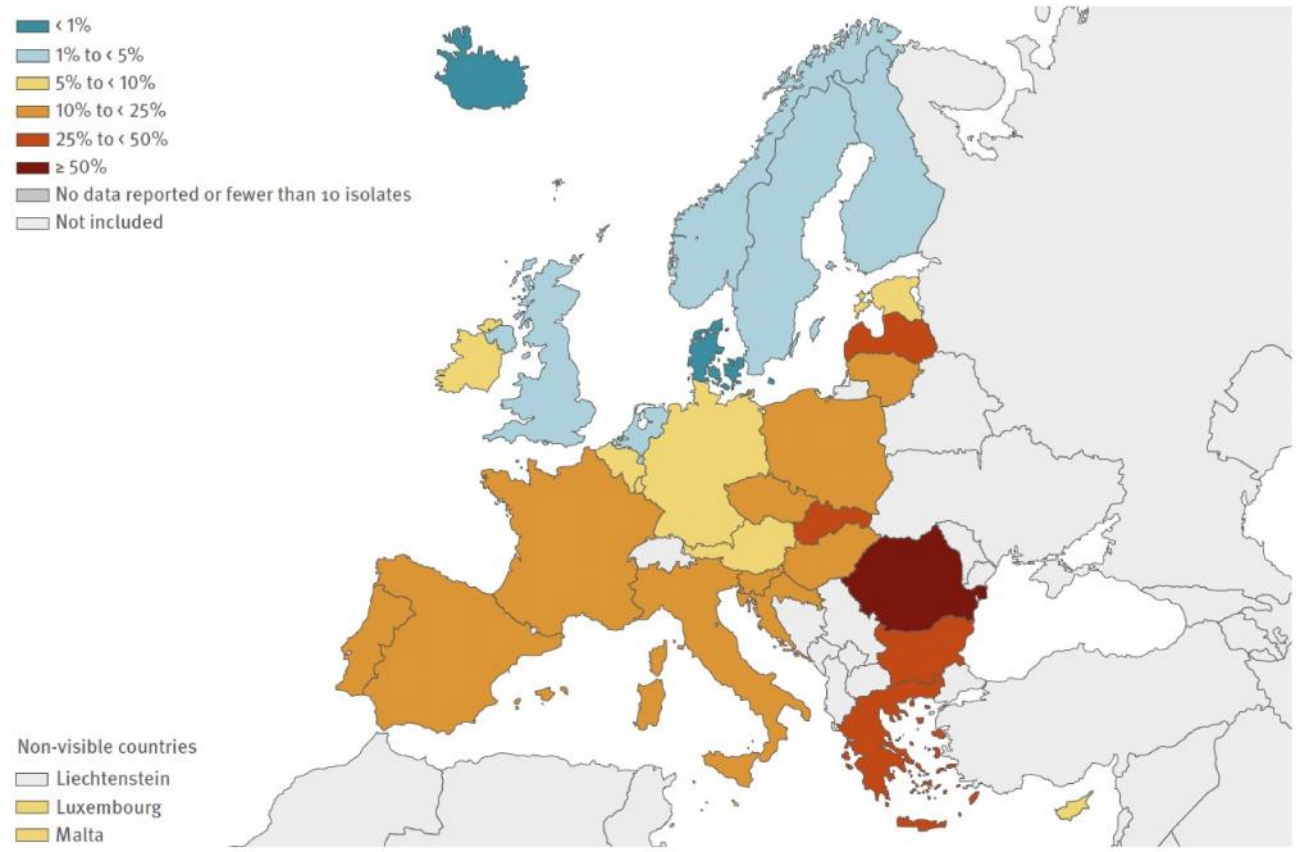
Antimicrobial resistance in *Pseudomonas aeruginosa*

In 2017, there were 296 reports of invasive *P. aeruginosa* infection (294 from blood and two from CSF) from 288 patients, an increase of 18% from 2016 (n=251).

Table 1 displays annual trends since 2013 in the proportion of *P. aeruginosa* isolates resistant to the five indicator antimicrobials/antimicrobial classes: piperacillin-tazobactam, ceftazidime, carbapenems (meropenem or imipenem), fluoroquinolones (ciprofloxacin) and aminoglycosides (gentamicin, amikacin or tobramycin):

- Of isolates tested against all five indicator antimicrobials, 10.1% were identified as MDR *P. aeruginosa*, defined as resistance to three or more of the indicator antimicrobials. This represents a decrease from 2016 (13.1%)
- AMR of *P. aeruginosa* isolates in Ireland for each of the five indicator antimicrobials, as well as for MDR (Figure 15), are at moderately low levels in comparison with other European countries
- The majority of *P. aeruginosa* (57%) and MDR *P. aeruginosa* (68%) isolates were reported by the nine tertiary hospitals (which comprised 45% of all patient days in 2017)
- The majority of *P. aeruginosa* (73%) occurred in patients over 60 years (median age=70 years) with approximately two-thirds of *P. aeruginosa* infections occuring in males

Figure 15. Distribution of MDR *Pseudomonas aeruginosa* in EU/EEA countries reported to EARS-Net in 2017



Map kindly provided by Liselotte Diaz Högberg, European Centre for Disease Prevention and Control (ECDC)

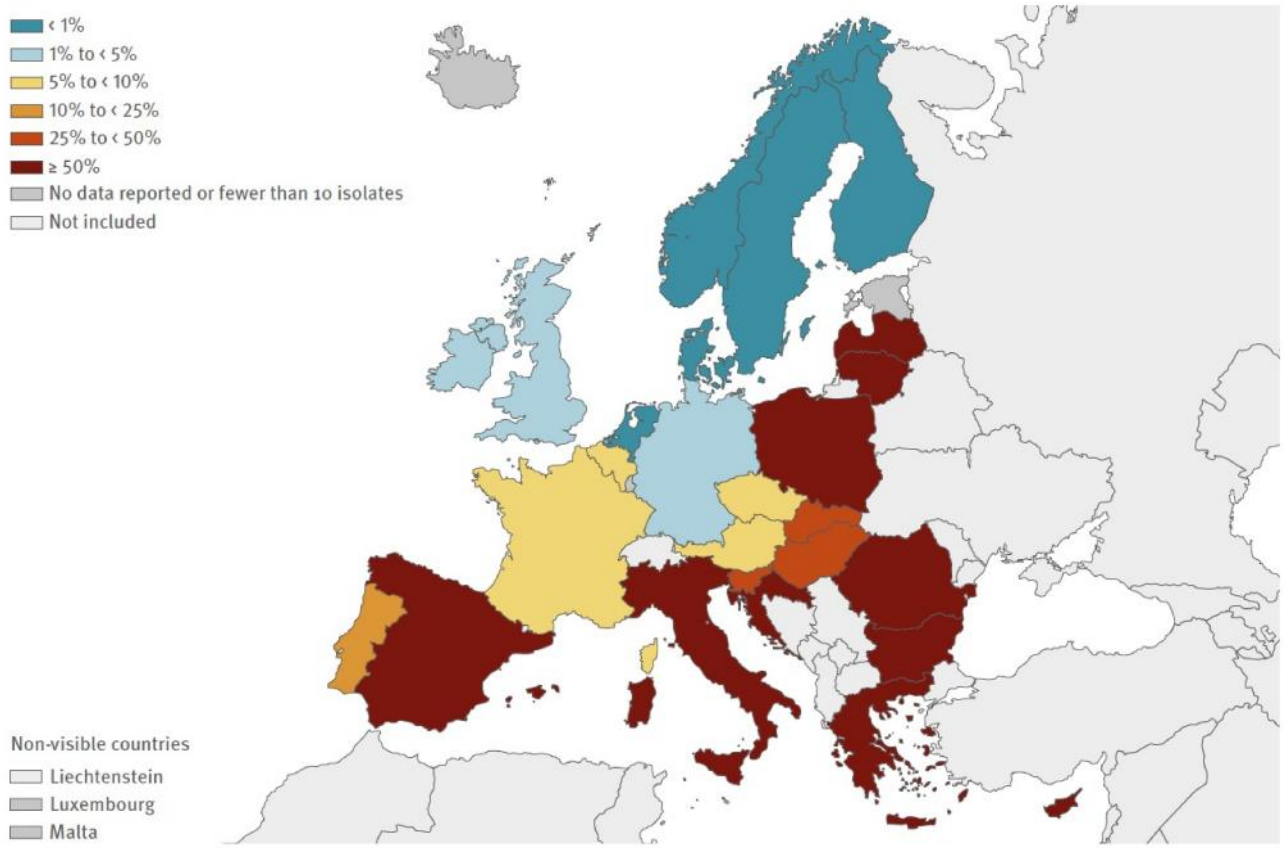
Antimicrobial resistance in *Acinetobacter* spp.

There were 73 reports of invasive infection caused by *Acinetobacter* spp. (all from blood) from 73 patients, compared with 68 reports in 2016.

Table 1 displays annual data since 2013 in the proportion of *Acinetobacter* spp. isolates with resistance to the three indicator antimicrobials/antimicrobial classes: carbapenems (meropenem or imipenem), fluoroquinolones (ciprofloxacin) and gentamicin:

- One isolate was identified as MDR *Acinetobacter* spp., i.e. resistant to all three indicator antimicrobials. In addition, this isolate was found to be a carbapenemase producer (OXA-23/51)
- AMR among *Acinetobacter* spp. in Ireland remains at low levels (Figure 16)
- The majority of Acinetobacter spp. (67%) were isolated within two days of hospital admission. The median age of patients with invasive infection due to *Acinetobacter* spp. was 53 years

Figure 16. Distribution of MDR-*Acinetobacter* spp. in EU/EEA countries reported to EARS-Net in 2017



Map kindly provided by Liselotte Diaz Högberg, European Centre for Disease Prevention and Control (ECDC)

Further information available on HPSC website

Further information on EARS-Net and antimicrobial resistance in Ireland can be found at:

http://www.hpsc.ie/a-z/microbiologyantimicrobialresistance/antimicrobialresistance/

European data can be found at:

https://ecdc.europa.eu/en/about-us/partnerships-and-networks/disease-and-laboratory-networks/ears-net

Proposed changes to EARS-Net AMR surveillance in 2019

In 2019, a number of changes to the surveillance of AMR in Ireland are proposed:

- Amendment of the case definition to reflect that used by EARS-Net at ECDC (moving from collecting data on the first isolate per patient **per quarter** to the first isolate per patient **per year**)
- Addition of group B streptococci (from patients of all ages) to the list of pathogens under surveillance
- Consolidation of the surveillance of *Candida* spp. from BSI, which commenced in 2017 (data not presented here)

Invasive Carbapenemase-producing Enterobacterales (CPE) infection

Background

Invasive carbapenem-producing *Enterobacterales* (CPE) infection, previously known as carbapenem-resistant *Enterobacteriaceae* (CRE), became notifiable in Ireland from January 2012.

For surveillance purposes, the case definition is based on laboratory criteria only: any person with reference laboratory confirmation of CPE from a normally sterile site should be reported to public health as an invasive CPE case.

Data were extracted from the Computerised Infectious Diseases Reporting (CIDR) system on 1st December 2018.

Epidemiology

In 2017, there were 14 notifications of invasive CPE:

- 13 of the cases were associated with five tertiary hospitals, all of which reported outbreaks in 2017, with one case coming from a general hospital
- The majority of cases were from BSI (n=11) with three isolates from normally sterile sites: prostate fluid, aneurysm plaque and tissue
- The predominant carbapenemase was OXA-48 (n=13); one IMP was also identified
- Four *Enterobacterales* species accounted for all invasive CPE infections: *E. coli* (n=5*), *K. pneumoniae* (n=5*), *Enterobacter cloacae* (n=3) and *Klebsiella oxytoca* (n=2)
- The median patient age was 56 years (range, 33-89), with a male predominance (n=9; 64%)

*CPE was identified in both *E. coli* and *K. pneumoniae* for one case

Figure 17. Distribution of carbapenemases causing invasive CPE infection in Ireland, 2011-2017

Carbapenemase type	2011	2012	2013	2014	2015	2016	2017	2011-2017
IMP	0	0	0	0	0	1	1	2
KPC	0	0	0	2	2	3	0	7
NDM	0	0	0	0	0	3	0	3
OXA	0	0	0	2	6	6	13	27
VIM	0	0	0	1	0	1	0	2
Total	**0**	**0**	**0**	**5**	**8**	**14**	**14**	**41**

There were no invasive CPE infections notified from 2011-2013. Since 2014, 41 invasive cases of CPE were notified, with OXA-48 accounting for almost two-thirds of these (Figure 17). Although the total number of CPE reported in 2017 was the same as in 2016, the numbers of CPEs that were OXA-48 more than doubled over the same period.

Enhanced CPE Surveillance

Background to Enhanced CPE Surveillance

Carbapenemase producing *Enterobacterales* (CPE), also known as carbapenem resistant *Enterobacterales* (CRE), are a growing threat to public health due to very limited options for treatment of infection.

A voluntary, paper-based enhanced surveillance system for all CRE isolates was launched in 2011 and invasive CRE infection was made legally notifiable.

In response to the increasing national incidence of CPE, quarterly mandatory enhanced CPE surveillance was established in January 2017.

The National Carbapenemase Producing *Enterobacterales* Reference Laboratory Service (NCPERLS), based at Galway University Hospital, has provided reference services since October 2012, with the annual total number of patients with newly-confirmed CPE increasing from 50 in 2013 to 433 in 2017.

In 2017, the proportion of invasive carbapenem resistant *K. pneumoniae* isolates causing BSI reported to the EARS-Net varied from 0% in Norway and Estonia to 32% in Italy and Romania and 66% in Greece. However, BSI represent the tip of the iceberg, as other more common infection types (e.g. urinary tract or wound infections) and asymptomatic, and often unrecognised, enteric/gut colonisation also contribute to the successful dissemination of CPE, particularly in healthcare settings.

Case definition

- The first isolate per patient per year of any *Enterobacterales* species that is a confirmed carbapenemase-producer from any specimen type, either infection or carriage (e.g., if the first isolate is a screening specimen, a subsequent BSI due to the same isolate won't be counted in surveillance)
- If the same carbapenemase is found in isolates of two or more species from the same patient, then only the first species is included (e.g. OXA-48 *E. coli* followed by OXA-48 *Enterobacter cloacae*; only the OXA-48 *E. coli* will be counted in surveillance)
- If a different carbapenemase is found in an isolate of any species in a subsequent specimen from the same patient, then the first isolate with this other carbapenemase is included (e.g., OXA-48 *E. coli*, followed by NDM-1 *K. pneumoniae*; both will be counted in surveillance)
- If an organism is not isolated or fails to grow, but a carbapenemase is detected by direct PCR on the specimen, such CPEs should not be reported
- The case definition for enhanced surveillance does not distinguish between isolates from the same patient identified in different hospitals

Results

Thirty-two microbiology laboratories reported 449 CPE isolates from 447 patients, with seven laboratories reporting zero isolates. In 2017, two laboratories did not submit data for the complete year, due to ongoing resource issues.

- If probable duplicate patients between hospitals are excluded (based on date of birth, gender or specific information supplied), the total number of patients reported to enhanced CPE surveillance in 2017 was 427, which is very similar to the NCPERLS total of 433 patients with newly-confirmed CPE. It is noteworthy that there are differences in what is counted: NCPERLS reports are based on the date an isolate is received, while CPE enhanced surveillance is based on the specimen collection date and reports on the first isolate per patient per year, which may include more than newly-confirmed patients (i.e. when a patient was previously known with CPE in the preceding year)
- As the case definition only requires reporting of the first isolate of any *Enterobacterales* species with the same enzyme for the year, patients are only counted once in this surveillance programme, unless a subsequent isolate from the same patient is reported with a different enzyme:
    - Two patients had two different carbapenemases reported: both with OXA-48 and NDM isolated from the same screening specimens
    - Two patients had two different carbapenemases reported from two separate specimens:
        - OXA-48 *Enterobacter cloacae* from a clinical specimen and VIM *E. cloacae* from a screening specimen where both specimens were taken on the same date
        - OXA-48 *E. coli* from a screening specimen and OXA-181/232 *E. coli* from a subsequent clinical specimen
- Three species accounted for 75% of all CPE isolates: *K. pneumoniae* (31%), *E. coli* (23%) and *Enterobacter cloacae* (21%) (Table 5, Figure 18)
- Nationally, the majority of CPE were OXA-48 (73%), with KPC predominant in the mid-west (n=45; 79% of KPC isolates) (Figure 19)
- Males (57%) and patients aged ≥57 years (75%) accounted for the majority of cases
- The majority of isolates were detected from screening specimens (n=353; 79%), with the remainder from clinical specimens (n=96; 21%) (Figure 20), of which seven were from BSI
- Inpatients in 33 hospitals accounted for the majority of carbapenemases (n=378; 78%):
    - Admission and specimen dates were reported for 321 (85%), with a median interval between admission and first positive result of eight days (range, 0-458 days)

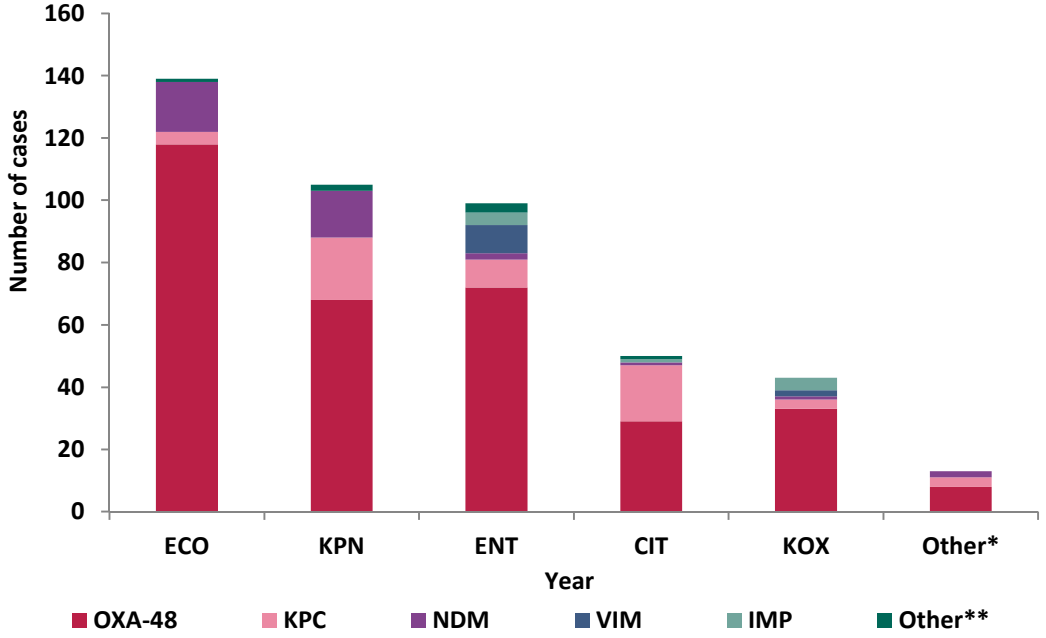
- Of clinical specimens, the majority were detected from inpatients (n=72; 75%). Of those, information on antimicrobial therapy was provided for 45 (63%), with 25 of those (60%) having required antimicrobial therapy active against a carbapenemase for suspected infection prior to case notification. However, for over one-third of clinical isolates from inpatients (37%), information on antimicrobial therapy by the time of case notification was not reported
- Information on inpatient isolation status within 24 hours of the laboratory reporting a suspected carbapenemase was provided for 277 isolates (73%), with the majority of patients isolated (n=254; 92%) and 16 (8%) who were discharged prior to the result. In three cases, the patient was not isolated within 24 hours. However, for 28% of inpatient isolates (n=105), isolation status was not reported
- In 2017, the majority of inpatient CPEs were reported from screening specimens (rectal swab or faeces) (Figure 18). The following hospitals accounted for the majority of reported CPE and all also reported and managed CPE outbreaks in 2017:
  - Tallaght University Hospital (n=76; 97% on screening)
  - Galway University Hospital (n=45; 88% on screening)
  - University Hospital Limerick (n=39; 87% on screening)
  - Beaumont Hospital (n=33; 74% on screening)
  - St James's Hospital (n=33; 76% on screening)
  - University Hospital Waterford (n=26; 69% on screening)
  - St Luke's Hospital, Kilkenny (n=17; 76% on screening)
  - Naas General Hospital (n=16; 100% on screening)
  - St Vincent's University Hospital (n=15; 78% on screening)
  - In 2017, additional CPE outbreaks were notified to Departments of Public Health by the Mater Misericordiae University Hospital, Our Lady's Hospital, Navan, Sligo University Hospital and the Beacon Hospital, as well as by two long-term care facilities (LTCF) in different regions

- The remaining isolates were detected from outpatients attending 12 hospitals (n=30), LTCF residents (n=29) and patients attending general practitioners (GP) (n=12)
- Outcome data at the time of reporting was not provided for 27% of inpatient isolates. Of 276 inpatients, 28 (10%) were reported to have died at the time of reporting. However, cause of death was not ascertained

Table 5. Summary of *Enterobacterales* and carbapenemase type in Ireland, 2017

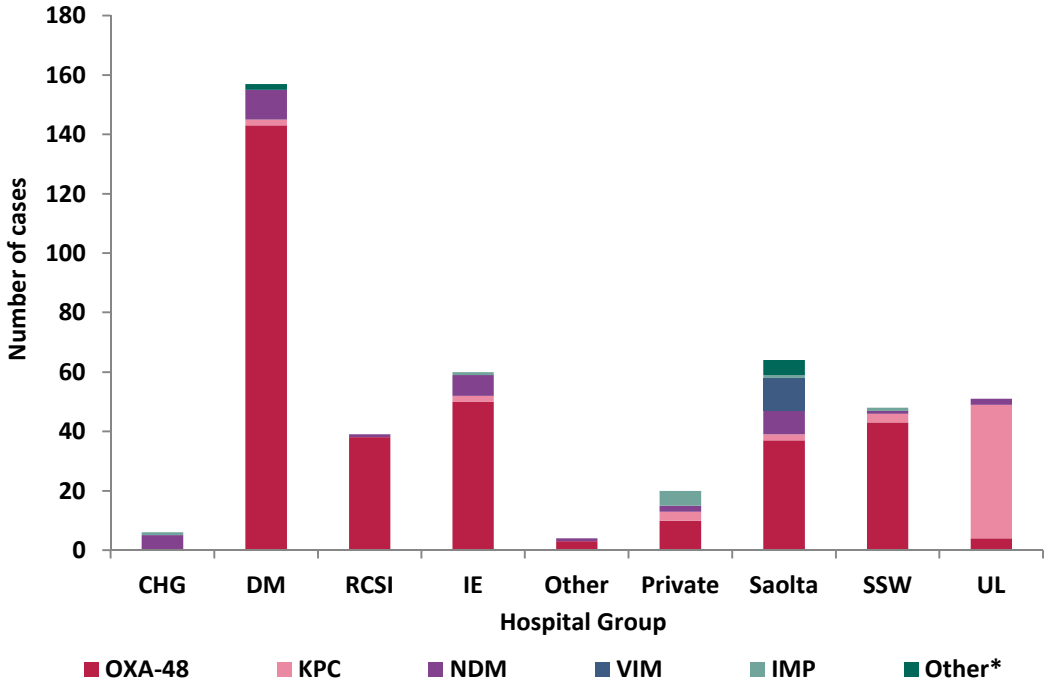
Enterobacterales species	Enzyme						
	OXA-48	KPC	NDM	VIM	IMP	Other**	Total
E. coli	118	4	16			1	**139**
K. pneumoniae	68	20	15			2	**105**
Enterobacter spp.	72	9	2	9	4	3	**99**
Citrobacter spp.	29	18	1		1	1	**50**
K. oxytoca	33	3	1	2	4		**43**
Other *Enterobacterales* *	8	3	2				**13**
TOTAL	**328**	**57**	**37**	**11**	**9**	**7**	**449**

Figure 18. *Enterobacterales* and carbapenemase type in Ireland, 2017



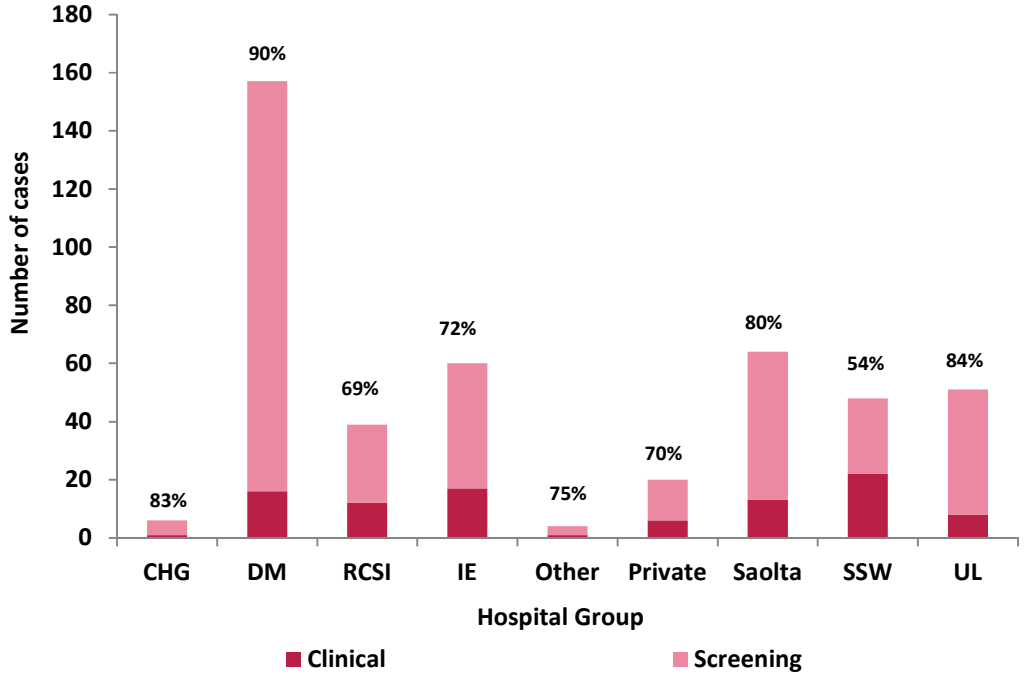
ECO, *E. coli*; KPN, *K. pneumoniae*; ENT, *Enterobacter* spp; CIT, *Citrobacter* spp; KOX, *K. oxytoca*; Other*, Other *Enterobacterales*; Other**, Other enzymes: IMI, OXA-181/232, OXA-48/NDM

Figure 19. Distribution of carbapenemase type by hospital group in Ireland, 2017



CHG, Children's Hospital Group; DM, Dublin Midlands Group; DNE, RCSI, RCSI Group; IE, Ireland East Group; Other, Other non-acute; Saolta Group, West North-West Group; SSW, South South-West Group; UL, University of Limerick Group; *Other, Other enzymes: IMI, OXA-181/232, OXA-48/NDM

Figure 20. Distribution of cases from clinical and screening specimens (and proportion from screening) by hospital group in Ireland, 2017



Further information available on HPSC website

Further information on Enhanced CPE Surveillance in Ireland, including data by acute hospital and hospital group, can be found at:

http://www.hpsc.ie/a-z/microbiologyantimicrobialresistance/strategyforthecontrolofantimicrobialresistanceinirelandsari/carbapenemresistantenterobacteriaceaecre/surveillanceofcpeinireland/cpequarterlysurveillancereports/

Public health implications

AMR remains an important threat in Ireland. In October 2017, Ireland's national action plan on AMR (iNAP) was published outlining the strategy to prevent AMR from 2017 to 2020.

Over the past five years, the numbers of invasive infections have steadily increased: *E. coli* increased by 26%, *K. pneumoniae* by 52%, *E. faecium* by 11%, *S. pneumoniae* by 33% and *P. aeruginosa* by 43%.

Infections due to ESBL-producing-*E. coli* and *K. pneumoniae*, as well as with resistance to many of the other commonly used antimicrobials (combined resistance or MDR) have become more common. Recent increases in resistance to carbapenems, regarded as reserve antimicrobials for treatment of AMR infections and infections in seriously ill patients have also been observed. Increasing incidence of carbapenemase-producing *Enterobacterales* (CPE) in Ireland prompted declaration of a national public health emergency in October 2017. Across Europe, resistance levels are generally higher in *K. pneumoniae* than in *E. coli* but this is not the case for Ireland, where resistance levels have been lower in *K. pneumoniae*.

Meticillin resistance in *S. aureus* has decreased significantly over the past 10 years from 34% in 2008 to 16% in 2017.

Ireland has long had one of the highest proportions of vancomycin resistance in *E. faecium* (VREfm) in Europe and was one of only two countries in 2017 where the numbers of *E. faecium* exceeded those of *E. faecalis*. The reasons for these are poorly understood. However, there are encouraging signs that the proportion of vancomycin resistant *E. faecium* is decreasing.

With the recent increase in invasive *S. pneumoniae* infections, serotyping data from IMSRL has provided invaluable data on the different serotypes in circulation. Ongoing surveillance of the predominant serotypes is required, as strains with non-vaccine serotypes are known to increase in prevalence following the introduction of conjugate vaccines and this has been demonstrated in Ireland. Such surveillance data will inform subsequent vaccine development in order to target emerging serotypes not covered by current vaccines. The separate chapter on invasive pneumococcal disease (IPD) in Ireland in 2017 contains additional information on pneumococcal serotyping.

Despite the overall increase in the number of invasive *S. pneumoniae* infections, the steady decrease in the proportion of isolates with reduced susceptibility, or high-level resistance, to penicillin is encouraging. Some of this may be related to conjugate pneumococcal vaccines selectively targeting serotypes that are typically associated with resistance, but is also likely to be driven by decreasing community antimicrobial use (for which there is a temporal association).

It is important that AMR surveillance in Ireland continues to evolve, to detect other new and emerging pathogens and resistance mechanisms. For example, *Candida auris* is a relatively new pathogen, first identified in 2009, that is often MDR. To date, it has not been reported in Ireland. In recent years, it has emerged in a number of countries as a cause of

hospital outbreaks. Strains of *E. coli* with resistance to colistin were reported from China in 2015. Colistin is a critically important antimicrobial, used to treat MDR infections, such as those due to CPE. The gene responsible for this resistance is called *mcr*1 and is carried on a plasmid that can be transferred easily to other strains of *E. coli* and to other bacterial species. Linezolid resistance in *E. faecalis* due to the presence of another gene (*optr*A) with the potential to spread to other strains is a further example of an emerging resistance mechanism and has already been detected in Ireland.

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