Ongoing measles outbreak in Elche, Spain, 29 January to 9 March 2012

J A Delgado de los Reyes (delgado_josdel@gva.es)[1], M Arencibia Jiménez[1], J F Navarro Gracia[1], E Alonso Echabe[2], P García Puente[2], E M Banqueri Guerrero[1], G Pérez Torregrosa[1], J D Calle Barreto[1], R Zurriaga Carda[1]
1. Preventive Medicine Service, Hospital General Univesitario of Elche, Elche, Spain
2. Public Health Center, Elche, Spain

Citation style for this article:
Delgado de los Reyes JA, Arencibia Jiménez M, Navarro Gracia JF, Alonso Echabe E, García Puente P, Banqueri Guerrero EM, Pérez Torregrosa G, Calle Barreto JD, Zurriaga Carda R. Ongoing measles outbreak in Elche, Spain, 29 January to 9 March 2012.
Euro Surveill. 2012;17(11):pii=20119. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20119

Article published on 15 March 2012

On 29 January 2012, the first case of measles in Elche, Spain, since 2001 was notified through the epidemiological surveillance system of the Valencian Community. As of 9 March, 109 cases have been notified. The outbreak started in a neighbourhood where the vaccination coverage of the population is inadequate. This report highlights the need to vaccinate the susceptible population and also points to the importance of developing coordinated measures between public health centres and hospital preventive services.

Outbreak description

On 29 January 2012, a 13-year-old child was diagnosed with suspected measles at the emergency service of the Hospital General Universitario of Elche (HGUE), Spain. The infection was confirmed by serology (IgM positive) at the hospital.

By 9 March, 109 cases in Elche have been confirmed through serology or an epidemiological link to confirmed cases (Figure 1). Of the 109 cases, 44 were notified from the same neighbourhood, Los Palmerales, where the index case lived (Figure 2). The mean age of the cases was 15 years (range: 25 days to 50 years). A total of 42 cases were adults aged from 20 to 50 years; 37 of the cases were children aged from 25 days to four years. Eight cases were aged under one year. Among the young adult cases, three were pregnant.

Of the 109 cases, 80 were diagnosed in the emergency services of the HGUE (66 cases) and the Hospital of Vinalopo in Elche, far from the HGUE, on the other side of the city (14 cases). Eleven of the 80 cases examined in the emergency services were admitted to inpatient care because of the following: impaired general condition (n=6), pneumonia (n=2), bronchitis (n=2) or otitis (n=1). Their ages ranged from 25 days to 17 years; the mean duration of hospitalisation was four days (range: two to seven days).

Cases not diagnosed at hospital (n=29) were diagnosed at primary healthcare centres.

Of the 109 cases, 66 had not been vaccinated with measles-mumps-rubella (MMR) vaccine (Table 1). A total of 28 cases had received the first dose of MMR vaccine; two cases had received both doses.

Four of the cases were healthcare workers in the HGUE: two were working in the emergency service; they were not vaccinated against measles, The MMR vaccination status of the other two was unknown: these cases had no direct link with the measles patients treated in the hospital.

Background

Measles is a highly contagious viral disease that can be prevented by a safe and effective vaccine, such as the measles-mumps-rubella (MMR) vaccine [1]. It is recommended that the MMR vaccine be given in two doses. The vaccine was introduced into the vaccination schedule in Spain in 1981. In the Valencian Community – one of the autonomous communities of Spain – the first dose is given at 15 months of age and the second at the age of five years [2].

As this infectious disease is preventable, a plan of action for measles elimination was implemented in Spain in 2001 [3]; the objective was expected to be achieved by 2005. In spite of the efforts made, however, there have been some measles outbreaks among the autonomous communities of Spain. The deadline for the elimination goal has therefore been postponed, in line with the plan of the World Health Organization Regional Office for Europe to eliminate measles and rubella by 2015 [4].

One of the strategies of the plan of action has been to enhance the epidemiological surveillance system at the national level, in order to facilitate early detection of measles cases and transmission control.

Consequently, in the Valencian Community, measles notification is mandatory [5]. Another important strategy has been to increase the measles vaccination coverage: the target vaccination coverage is above 95% for the first and second MMR doses).

In 2010, four cases of measles were notified in the Valencian Community: three of them were laboratory confirmed, imported from France. The fourth case was confirmed by epidemiological link to the laboratory-confirmed cases [6]. In 2011, the number of notified cases increased to 236. Most were notified in the Valencia health department (120 cases); 30 cases were notified in Alicante.

The city of Elche, with a population of 230,354, is located 25 km from Alicante. Until the outbreak described here, the last case of measles in Elche was

Figure 1

Confirmed measles cases in Elche, Spain, 29 January–9 March 2012 (n=109)

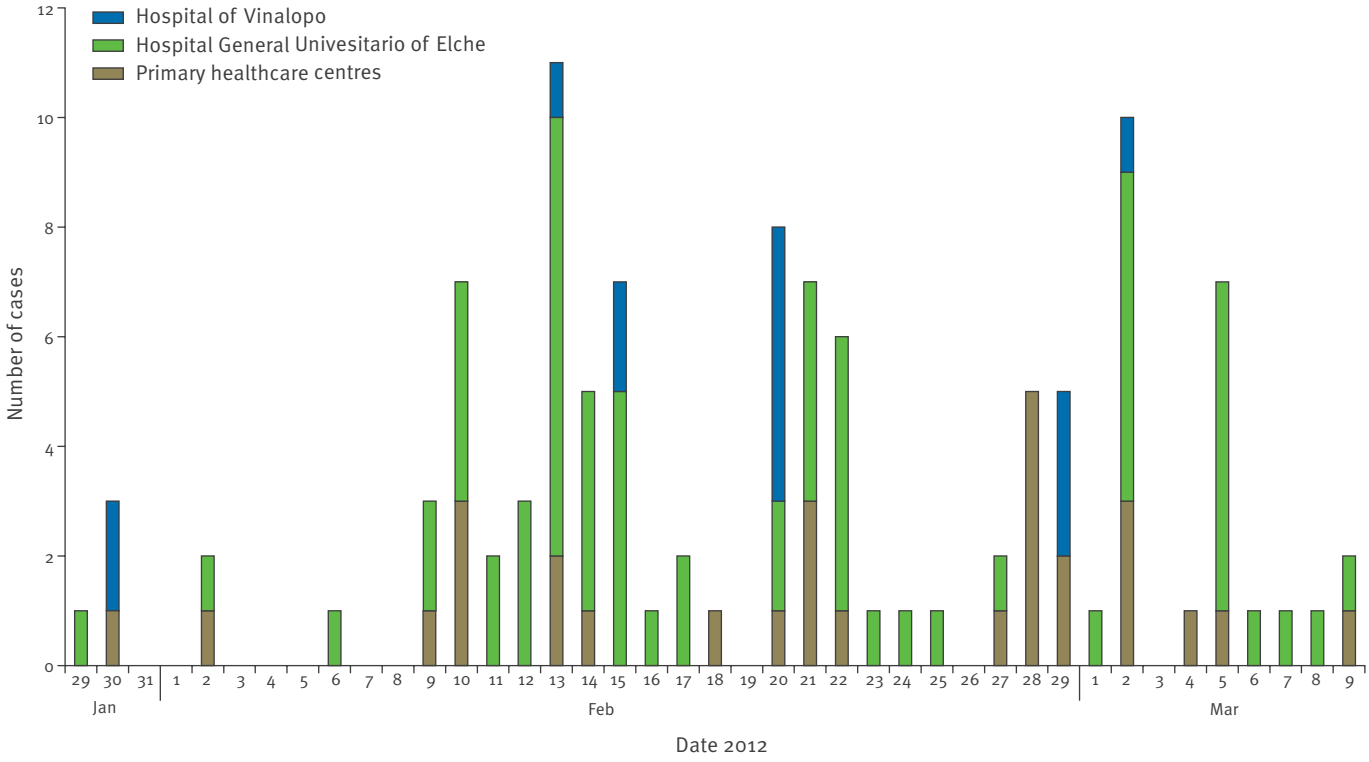
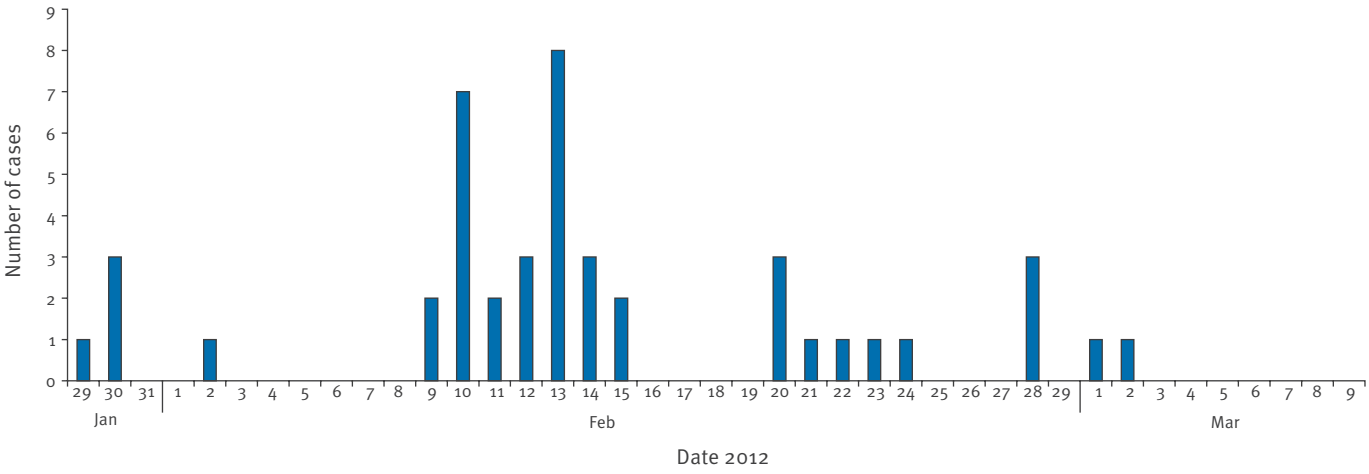


Figure 2

Confirmed measles cases in Los Palmerales, Elche, Spain, 29 January–9 March 2012 (n=44)



notified in 2001. In 2011, The MMR vaccination coverage was 96.4% for the first dose (measured between 12 and 24 months of age) and 90.4% for the second dose (measured between the ages of three and seven years) [7].

There are a number of groups with low vaccination coverage in Elche. These include people living in Los Palmerales, a neighbourhood near the HGUE. In this neighbourhood, people of Roma ethnicity are the main ethnic group, many of whom refuse vaccination due to cultural objections [8,9]. Other groups are also at risk: children younger than 15 months who have not received the first dose of MMR vaccine, because vaccination is not indicated; some children who have received only one dose and who have not developed a full immune response; and adults aged between 20 and 45 years who did not receive the MMR vaccine, because it was not included in the vaccination schedule when they were young, and who have not been affected by measles. Non-immunised young healthcare professionals in the hospitals are also a susceptible group [10].

Control measures

As there were reports of eight measles outbreaks, with a total of 236 cases, elsewhere in the Valencian Community in 2011 [11], preventive measures have been taken in Elche since 11 January 2012. They consisted of information sessions, mainly given to the staff in the HGUE emergency services as well as to the coordinators of the primary care centres. Once the outbreak was declared in Elche (on 29 January), these measures were extended to other clinical services. In addition, a catch-up vaccination campaign against measles was implemented, between 6 and 17 February, among the healthcare professionals aged from 20 to 45 years, particularly those working in the services of paediatrics, emergencies, obstetrics and oncology: 56 doses of MMR vaccine have been given and 98 blood tests have been carried out to check immunity against measles.

In order to prevent intrahospital transmission, it was recommended that suspected measles cases be attended only by staff who have a documented serological measles immunity or documented vaccination. In addition, new staff are being tested and we are also working to increase the percentage of vaccinated staff among the healthcare workers younger than 45 years of age.

In addition, a practical guide was distributed among the healthcare professionals who work at the primary health care centres. It encourages staff to treat suspected cases as outpatients and to send just the severe cases to hospital emergency services, where triage of patients with fever and rash is carried out in order to assess them promptly.

At the community level, the Public Health Center in Elche has been responsible for monitoring and controlling the outbreak through enhancing the MMR vaccination coverage. The actions taken in the city have been as follows: to move forward the first dose of MMR vaccine from 15 months to 12 months of age; to keep suspected cases isolated at home; to move forward the second dose of MMR vaccine to children younger than four years of age who have been in contact with a suspected case (ensuring that there is an interval of four weeks between the first and second dose); and carrying out catch-up vaccination for children older than five years of age and adults younger than 45 years of age in Elche.

On 14 February, 323 doses of MMR vaccine were administered in Los Palmerales (Table 2).

Table 1

Confirmed measles cases by age group and MMR vaccination status, Elche, Spain, 29 January–9 March 2012 (n=109)

Age group	MMR vaccination status				Total
	1 dose	2 doses	Unknown	Unvaccinated	
	n	n	n	n	
≤15 months	3	0	0	15	**18**
16 months–4 years	9	0	1	9	**19**
5–9 years	3	1	1	7	**12**
10–14 years	2	0	0	6	**8**
15–19 years	6	1	2	1	**10**
20–24 years	3	0	3	11	**17**
25–29 years	1	0	3	2	**6**
30–34 years	1	0	1	8	**10**
35–39 years	0	0	0	3	**3**
≥40 years	0	0	2	4	**6**
Total	**28**	**2**	**13**	**66**	**109**

MMR: measles-mumps-rubella.

Discussion

Elche is experiencing an ongoing measles outbreak. Even though the MMR vaccination coverage is high in the city, measles has been easily spread in pockets of unvaccinated people. The virus has been transmitted to children under 15 months of age and to people with incomplete vaccination.

The high number of patients diagnosed with measles at the emergency service of the HGUE has been due to the proximity of the most affected areas, including Los Palmerales, to the hospital.

The high number of doses MMR vaccine given in Los Palmerales in mid-February, in addition to other actions taken in the city – lowering the ages of the first and second doses of MMR vaccine administration, as well as vaccinating children older than five years of age and young adults who had not received both doses – may have a positive effect in controlling the outbreak.

Coordinated work among the hospital and public health services has been an important factor in ensuring that the appropriate measures have been undertaken and that the status of the outbreak is reported, giving accurate and timely information.

References

1. European Centre for Disease Prevention and Control (ECDC). European monthly measles monitoring (EMMO). Issue 8: 21 February 2012. Stockholm: ECDC; 2012. Available from: http://ecdc.europa.eu/en/publications/Publications/SUR_EMMO_European-monthly-measles-monitoring-February-2012.pdf
2. Conselleria de Sanitat. Orden de 16 de junio de 2008, de la Conselleria de Sanidad, por la que se modifica el calendario de vacunación sistemática infantil en la Comunitat Valenciana. [Order 16 June 2008, Regional Ministry of Health, by amending the vaccination schedule of children in the Valencian Community]. Official Diary of the Valencian Community. Num. 5812 / 23.07.2008. Valencian/Spanish. Available from: http://www.docv.gva.es/datos/2008/07/23/pdf/2008_8694.pdf
3. López Hernández B, Laguna Sorinas J, Marín Rodríguez I, Gallardo García V, Pérez Morilla E, Mayoral Cortés JM. Spotlight on measles 2010: an ongoing outbreak of measles in an unvaccinated population in Granada, Spain, October to November 2010. Euro Surveill. 2010;15(50):pii=19746. Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19746
4. World Health Organization (WHO) Regional Office for Europe. Resolution. Renewed commitment to elimination of measles and rubella and prevention of congenital rubella syndrome by 2015 and sustained support for polio-free status in the WHO European Region. Regional Committee for Europe. Sixtieth session. Moscow, 13–16 September 2010. Copenhagen: WHO Regional Office for Europe; 2010. EUR/RC60/R12. Available from: http://www.euro.who.int/__data/assets/pdf_file/0016/122236/RC60_eRes12.pdf
5. Guaita Calatrava R, Giner Ferrando E, Bayo Jimeno J, Yuste Muñoz L, Saiz Sánchez C, Ortí Lucas R, et al. Brote nosocomial de sarampión.[Nosocomial measles outbreak], Madrid: National Centre of Epidemiology, Institute of Health Carlos III. Boletín epidemiológico semanal. Weeks 46-47. 2011;19(16); 220-7. Spanish. Available from: http://revistas.isciii.es/bes/public/journals/1/pdf_79.pdf
6. Sub-Directorate of Epidemiology, General Directorate of Investigation and Public Health Valencian Community. Plan Sarampión de la Comunitat Valenciana. Vigilancia epidemiologica. Año 2010. [Measles Plan in the Valencian Community. Year 2010. Valencia: Sub-Directorate of Epidemiology, General Directorate of Investigation and Public Health Valencian Community; 2011. Spanish. Available from: http://dgsp.san.gva.es/DgspPortal/docs/sarampion_informe2010.pdf
7. General Directorate of Investigation and Public Health Valencian Community. Cobertura vacunal de triple vírica en Elche, España en 2011. [MMR vaccination coverage in Elche, Spain in 2011. Valencia: General Directorate of Investigation and Public Health Valencian Community. [Accessed 18 Feb 2012]. Spanish. Available from: http://www.sp.san.gva.es/rvn/403.htm (restricted-access website).
8. Peña-Rey I, Martínez de Aragón V, Mosquera M, de Ory F, Echevarría JE; Measles Elimination Plan Working Group in Spain. Measles risk groups in Spain: implications for the European measles-elimination target. Vaccine. 2009:27(30)3927-34.
9. Health Minister of Spain. Guía para la actuación con la Comunidad Gitana en los Servicios Sanitarios. [Guide for action with the Roma community in health services]. Madrid: Health Minister of Spain; 2006. Spanish. Available from: http://www.msc.es/profesionales/saludPublica/prevPromocion/promocion/desigualdadSalud/docs/guiaSaludFSG.pdf
10. Botelho-Nevers E, Chevereau L, Brouqui P. Letter to the editor. Spotlight on measles 2010: Measles in healthcare workers – vaccination should be revisited. EuroSurveill. 2010;15(41):pii=19687. Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19687
11. Sub-Directorate of Epidemiology, General Directorate of Investigation and Public Health Valencian Community. Informe Sarampión de la Comunitat Valenciana. Año 2011. [Measles report, Valencian Community. Year 2011]. Valencia: Sub-Directorate of Epidemiology, General Directorate of Investigation and Public Health Valencian Community; 2012. Available from: http://www.sp.san.gva.es/DgspPortal/docs/sarampion_informe2011.pdf

Table 2

MMR vaccinations administered by age group in Los Palmerales, Elche, Spain, 14 February 2012 (n=323)

Age group	Number of MMR vaccinations
≤15 months	1
16 months–4 years	35
5–14 years	87
≥15 years	194
Unknown age	6

MMR: measles-mumps-rubella.

Identification of OXA-23-producing *Acinetobacter baumannii* in Greece, 2010 to 2011

A Liakopoulos[1], V Miriagou[2], E A Katsifas[3], A D Karagouni[3], G L Daikos[4], L S Tzouvelekis[5], E Petinaki (petinaki@med.uth.gr)[1]
1. Department of Microbiology, University Hospital of Larissa, Larissa, Greece
2. Laboratory of Bacteriology, Hellenic Pasteur Institute, Athens, Greece
3. Faculty of Biology, Department of Botany, Microbiology Group, University of Athens, Athens, Greece
4. 1st Department of Propaedeutic Medicine, Medical School, University of Athens, Athens, Greece
5. Department of Microbiology, Medical School, University of Athens, Athens, Greece

Citation style for this article:
Liakopoulos A, Miriagou V, Katsifas EA, Karagouni AD, Daikos GL, Tzouvelekis LS, Petinaki E. Identification of OXA-23-producing Acinetobacter baumannii in Greece, 2010 to 2011.
Euro Surveill. 2012;17(11):pii=20117. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20117

Article published on 15 March 2012

We report on the sequence type and beta-lactamase content of 174 carbapenem-resistant *Acinetobacter baumannii* isolates recovered from clinical specimens during 2010 and 2011 in a tertiary care hospital in central Greece. Carbapenem resistance was associated mainly with carriage of the *bla*$_{OXA-23}$ gene (in 72.4% of the isolates). To our knowledge, this is the first description of *A. baumannii* strains producing OXA-23 in Greece. During 2011, in our hospital they rapidly 'replaced' the previously predominant OXA-58-positive *A. baumannii* strains.

Previous studies have documented the predominance of oxacillinase (OXA)-58-producers among carbapenem-resistant *Acinetobacter baumannii* in Greek hospitals [1]. Here, we report the isolation of OXA-23-producing *A. baumannii* from clinical specimens taken in 2010 and 2011 at the University Hospital of Larissa in central Greece. To the best of our knowledge, this is the first description of OXA-23-producing *A. baumannii* in this country.

Background

A. baumannii has emerged as a leading cause of nosocomial infections, particularly among critically ill patients in intensive care units [2]. *A. baumannii* clinical isolates are commonly resistant to multiple antimicrobial drug classes and have the ability to survive in the environment for prolonged periods of time, which facilitates their persistence in hospitals [3]. Carbapenems have been widely used to treat infections caused by the microorganism but a trend of increasing resistance to these antibiotics associated with the production of acquired carbapenem-hydrolysing OXA-type class D beta-lactamases has been reported worldwide [4]. Three distinct groups of such acquired beta-lactamases, OXA-23-like, OXA-24/40-like and OXA-58-like, have been described [4].

Collection and analysis of isolates

The University Hospital of Larissa has 600 beds, with medical, surgical and paediatric subspecialties and one intensive care unit. It serves as one of the main tertiary care hospitals in the district of Thessaly, which has a population of 1,000,000 inhabitants.

Examination of records kept by the infection control committee of the University Hospital of Larissa showed that carbapenem-resistant *A. baumannii* was isolated more frequently in 2011 than in 2010 (2.1 vs 1.5 cases, respectively, per 1,000 patient-days), while the overall isolation rates of *A. baumannii* remained essentially similar (2.1 and 2.0 cases per 1,000 patient-days, respectively). Furthermore, it was noticed that the carbapenem-resistant *A. baumannii* isolates recovered during 2011 exhibited higher meropenem minimal inhibitory concentrations (MICs), ranging from 128 to 256 mg/L, with both MIC_{50} and MIC_{90} values at 256 mg/L. For the isolates from 2010, the range was 16–256 mg/L, with MIC_{50} and MIC_{90} values at 24 and 32, respectively). These findings prompted us to explore any changes in the *A. baumannii* population.

We therefore in December 2011, after the annual evaluation of the infection control committee, studied retrospectively single carbapenem-resistant *A. baumannii* clinical isolates (i.e. one per patient) obtained during July to December 2010 (n=47) and those from January to December 2011 (n=127). The isolates were recovered from various clinical specimens including, in descending frequency, bronchial secretions, blood, pus and urine. When necessary, species identity was confirmed with VITEK 2 (bioMérieux, France) and/or detection by PCR of *bla*$_{OXA-51}$ the intrinsic carbapenemase gene of *A. baumannii* [5]. MICs of various antimicrobials, including imipenem and meropenem, were re-determined by the Etest (bioMérieux, Sweden). Susceptibility status was defined according to the latest European Committee on Antimicrobial Susceptibility Testing breakpoints [6].

All 174 isolates were typed by three loci-sequencing typing [5,7]; allele numbers and sequence types were assigned based on the information in the respective databases. Identification of CHDL-encoding genes and mapping of their genetic environment were performed by PCR assays and sequencing of the respective amplicons, as described elsewhere [8,9].

Characteristics of the carbapenem-resistant *A. baumannii* population

Of the 174 carbapenem-resistant *A. baumannii* isolates, 125 (71.8%) carried bla_{OXA-23}, 48 (27.6%) carried bla_{OXA-58} and one (0.6%) was positive for both genes. The majority of bla_{OXA-58} carriers (n=42) were isolated during 2010. Five of the bla_{OXA-23} carriers were isolated in late 2010 from patients in the intensive care unit. The first two were isolated from patients in October 2010. The first patient was in their mid20s and the second in their early 70s. Both patients had no previous medical history of underlying disease such as diabetes, cancer or immunosuppression before being hospitalised. These two patients had been treated for at least two weeks before the isolates were cultured and their infections (both bacteraemia) were considered as hospital acquired, as was the case for the remaining patients infected with OXA-23-producing *A. baumannii*. Medical records of the first two patients did not indicate prior hospitalisation in the past six months or any travel abroad. It is therefore possible that OXA-23-positive *A. baumannii* had already circulated in the hospital.

Environmental screening was not carried out. We cannot provide reliable hypotheses regarding potential routes of introduction of OXA-23 producers to the hospital. Among the carbapenem-resistant *A. baumannii* isolates detected in 2011, the bla_{OXA-23} carriers predominated (120/127; 95%): 43 were isolated from patients in the intensive care unit and the rest from various medical (n=60) and surgical wards (n=17). Of the other seven isolates, recovered in a sporadic fashion throughout 2011, six were positive for bla_{OXA-58} and one carried both bla_{OXA-58} and bla_{OXA-23}. According to the three loci-sequencing typing, 24 bla_{OXA-58}-positive isolates belonged to sequence type (ST) 106, 23 to ST201 and one exhibited a novel allelic profile (2-1-2). ST101 was prevalent among bla_{OXA-23} carriers (n=101), the remaining 24 isolates being classified as ST201. The isolate harbouring both carbapenemase genes belonged to ST106. The isolation frequency of the isolates is shown in the Figure.

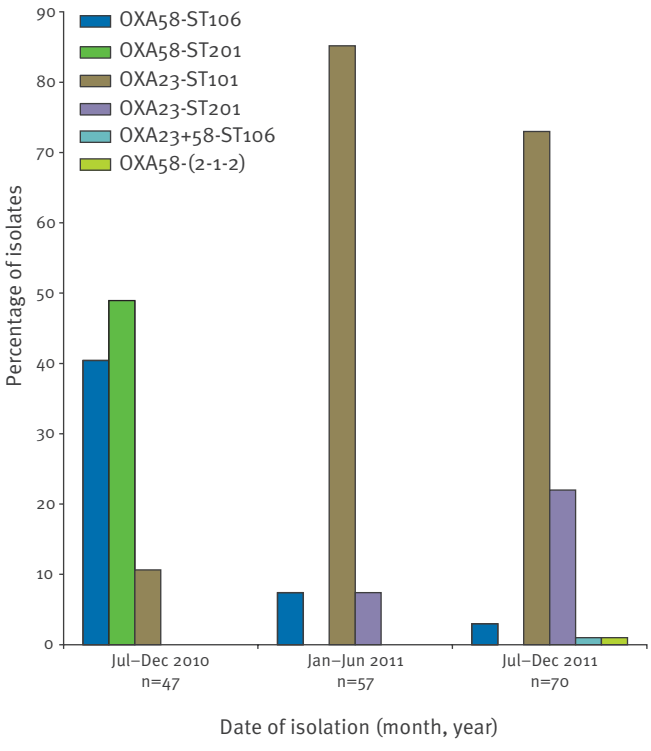
Apart from resistance to at least one carbapenem (imipenem and/or meropenem), all 174 isolates exhibited resistance to multiple drugs including penicillin-inhibitor combinations, oxyimino-cephalosporins, aminoglycosides and fluoroquinolones. All were susceptible to colistin. The bla_{OXA-23}-positive isolates were generally inhibited by higher concentrations of meropenem as compared with bla_{OXA-58} carriers. This is consistent with previous findings indicating that OXA-23 confers to *A. baumannii* higher levels of carbapenem resistance, as compared with OXA-58, although both enzymes exhibit relatively weak carbapenemase activities [10]. The genetic environment of bla_{OXA-23} gene was investigated in 20 isolates representing all sequence types and beta-lactamase combinations found. In all cases, bla_{OXA-23} occurred as part of the previously characterised Tn*2006* transposon bracketed by two IS*Aba1* (insertion sequences) in opposite orientation [9].

Discussion

During 1999 to 2009, *A. baumannii* strains carrying the bla_{OXA-58} carbapenemase gene predominated among carbapenem-resistant isolates of this species in the hospital flora in various Mediterranean countries including Italy, Greece, Lebanon and Turkey [1,11]. Since 2009, isolation of *A. baumannii* producing the OXA-23 carbapenemase has been increasingly reported in European countries [12-14]. Of note, a massive 'replacement' of OXA-58 *A. baumannii* by OXA-23 producers, similar to that observed here, has recently been described in Italian hospitals [15]. It was hypothesised that the higher carbapenem MICs of the bla_{OXA-23}-positive isolates may provide a selective advantage in the hospital setting. Yet, the change of the *A. baumannii* population seen in our study was too abrupt to be explained by the operation of carbapenem selective pressure alone. Whatever the reasons for the shift in

Figure

Isolation frequencies of carbapenem-resistant *Acinetobacter baumannii* strains, University Hospital of Larissa, Thessaly, Greece, July 2010–December 2011 (n=174)



ST: sequence type.

the *A. baumannii* population, the spread of strains producing OXA-23 seems to reflect a global trend towards a predominance of bla_{OXA-23} carriers belonging to the international clonal lineages I and II [16,17]. Spread of multidrug-resistant strains producing OXA-23 may aggravate the therapeutic problems caused by this species by further limiting treatment options.

It is highly likely that similar changes have also occurred in the *A. baumannii* population in tertiary care hospitals in the major urban centres of Greece, Athens and Thessaloniki. The available resources however, have been almost exclusively allocated to detecting and containing carbapenemase-producing *Enterobacteriaceae*. Yet, we believe that a low-cost study involving a limited number of sentinel hospitals would provide a comprehensive picture regarding the *A. baumannii* strains circulating in this country.

Hospital personnel have been informed about our findings and infection control measures have been reinforced.

Acknowledgments

This work was partially supported by the Medical School of the University of Thessalia (Grant No. 3917) and the Hellenic Pasteur Institute, Athens, Greece. We thank A. Papaioannou for technical assistance.

References

1. Gogou V, Pournaras S, Giannouli M, Voulgari E, Piperaki ET, Zarrilli R, et al. Evolution of multidrug-resistant Acinetobacter baumannii clonal lineages: a 10 year study in Greece (2000-09). J Antimicrob Chemother. 2011;66(12):2767-72.
2. Chastre J. Infections due to Acinetobacter baumannii in the ICU. Semin Respir Crit Care Med. 2003;24(1):69-78.
3. Neonakis IK, Spandidos DA, Petinaki E. Confronting multidrug-resistant Acinetobacter baumannii: a review. Int J Antimicrob Agents. 2011;37(2):102-9.
4. Poirel L, Nordmann P. Carbapenem resistance in Acinetobacter baumannii: mechanisms and epidemiology. Clin Microbiol Infect. 2006;12(9):826-36.
5. Turton JF, Gabriel SN, Valderrey C, Kaufmann ME, Pitt TL. Use of sequence-based typing and multiplex PCR to identify clonal lineages of outbreak strains of Acinetobacter baumannii. Clin Microbiol Infect. 2007;13(8):807-15.
6. European Committee on Antimicrobial Susceptibility Testing (EUCAST). Breakpoint tables for interpretation of MICs and zone diameters. 1 Jan 2012. Available from: http://www.eucast.org/fileadmin/src/media/PDFs/EUCAST_files/Breakpoint_tables/Breakpoint_table_v_2.0_120221.pdf
7. Health Protection Agency (HPA). Acinetobacter baumannii sequence typing home page. London: HPA. [Accessed 1 Jan 2012]. Available from: http://www.hpa-bioinformatics.org.uk/AB/home.php
8. Woodford N, Ellington MJ, Coelho JM, Turton JF, Ward ME, Brown S, et al. Multiplex PCR for genes encoding prevalent OXA carbapenemases in Acinetobacter spp. Int J Antimicrob Agents. 2006;27(4):351-3.
9. Corvec S, Poirel L, Naas T, Drugeon H, Nordmann P. Genetics and expression of the carbapenem-hydrolyzing oxacillinase gene blaOXA-23 in Acinetobacter baumannii. Antimicrob Agents Chemother. 2007;51(4):1530-3.
10. Héritier C, Poirel L, Lambert T, Nordmann P. Contribution of acquired carbapenem-hydrolyzing oxacillinases to carbapenem resistance in Acinetobacter baumannii. Antimicrob Agents Chemother. 2005;49(8):3198-202.
11. Di Popolo A, Giannouli M, Triassi M, Brisse S, Zarrilli R. Molecular epidemiological investigation of multidrug-resistant Acinetobacter baumannii strains in four Mediterranean countries with a multilocus sequence typing scheme. Clin Microbiol Infect. 2011;17(2):197-201.
12. Brigante G, Migliavacca R, Bramati S, Motta E, Nucleo E, Manenti M, et al. Emergence and spread of a multidrug-resistant Acinetobacter baumannii clone producing both the OXA-23 carbapenemase and the 16S rRNA methylase ArmA. J Med Microbiol. 2012 Jan 26. [Epub ahead of print].
13. Karah N, Giske CG, Sundsfjord A, Samuelsen Ø. A diversity of OXA-carbapenemases and class 1 integrons among carbapenem-resistant Acinetobacter baumannii clinical isolates from Sweden belonging to different international clonal lineages. Microb Drug Resist. 2011;17(4):545-9.
14. Bonnin RA, Poirel L, Licker M, Nordmann P. Genetic diversity of carbapenem-hydrolysing β-lactamases in Acinetobacter baumannii from Romanian hospitals. Clin Microbiol Infect. 2011;17(10):1524-8.
15. D'Arezzo S, Principe L, Capone A, Petrosillo N, Petrucca A, Visca P. Changing carbapenemase gene pattern in an epidemic multidrug-resistant Acinetobacter baumannii lineage causing multiple outbreaks in central Italy. J Antimicrob Chemother. 2011;66(1):54-61.
16. Higgins PG, Dammhayn C, Hackel M, Seifert H. Global spread of carbapenem-resistant Acinetobacter baumannii. J Antimicrob Chemother. 2010;65(2):233-8.
17. Mugnier PD, Poirel L, Naas T, Nordmann P. Worldwide dissemination of the blaOXA-23 carbapenemase gene of Acinetobacter baumannii. Emerg Infect Dis. 2010;16(1):35-40.

Moderate influenza vaccine effectiveness in Victoria, Australia, 2011

J E Fielding (james.fielding@mh.org.au)[1,2], K A Grant[1], T Tran[1], H A Kelly[1,2]
1. Victorian Infectious Diseases Reference Laboratory, Melbourne, Australia
2. National Centre for Epidemiology and Population Health, The Australian National University, Canberra, Australia

Citation style for this article:
Fielding JE, Grant KA, Tran T, Kelly HA. Moderate influenza vaccine effectiveness in Victoria, Australia, 2011.
Euro Surveill. 2012;17(11):pii=20115. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20115

Article published on 15 March 2012

We used a sentinel general practitioner (GP) network to conduct surveillance for laboratory-confirmed influenza amongst patients presenting with influenza-like illness (ILI) in Victoria, Australia in 2011. The test-negative variation of the case control study design was used to estimate effectiveness for seasonal trivalent influenza vaccine. Cases and controls were ILI patients that tested positive and negative for influenza, respectively. Vaccination status was recorded by GPs and vaccine effectiveness (VE) was calculated as (1-adjusted odds ratio)x100%. There were 529 patients included in the study, of which 29% were influenza-positive. Twelve percent of study participants were reported as vaccinated, 6% of cases and 15% of controls. Adjusted VE against all influenza was 56%, but not statistically significant. There was generally little variation in VE estimates when stratified by virus type and subtype, which is consistent with good matches between circulating strains and the vaccine strains. The VE was higher among adults of working age than among children.

Introduction

Victoria accounts for approximately 25% of Australia's population of 23 million people. It has a temperate climate, and the influenza season usually occurs between June and October. Each season, the Victorian Infectious Diseases Reference Laboratory uses a network of sentinel general practitioners (GPs) to conduct surveillance for influenza-like illness (ILI) and laboratory-confirmed influenza. The system has been operational since 1998, with an average of 60 GPs participating each year. This surveillance system is used to estimate vaccine effectiveness (VE) of the seasonal influenza vaccine.

Seasonal influenza vaccination in Australia is a publicly funded programme. The Australian government provides free influenza vaccination to all Australians aged 65 years and older, Aboriginal and Torres Strait Islander people over 15 years of age, pregnant women and individuals aged six months and older with medical conditions predisposing to severe influenza [1]. Individuals may also be vaccinated outside the funded programme, such as through workplaces. The influenza virus composition of the seasonal trivalent influenza vaccine (TIV) in Australia in 2011 was A/California/7/2009 (H1N1)-like virus, A/Perth/16/2009 (H3N2)-like virus, and B/Brisbane/60/2008-like virus (of the B/Victoria/2/87 lineage) [2].

Here we use the results from laboratory-confirmed influenza surveillance in Victoria to estimate TIV effectiveness in 2011 using the prospective test-negative variation of the case control study. This design has been used in Europe, North America and Australia [3-6]. We aimed to calculate type- and subtype-specific VE estimates and used them in combination with surveillance data to make inferences how well the 2011 seasonal TIV matched circulating strains. The strain composition recommended for use in the 2011 southern hemisphere influenza vaccine was the same as the one subsequently used in the 2011/12 northern hemisphere seasonal vaccine [7].

Methods

In 2011, 97 GPs participated in the surveillance system which operated from 2 May to 30 October inclusive. Advertising in GP circulars was used to encourage GPs to participate in the programme and targeted recruitment was undertaken in geographical areas considered to be poorly represented. A relatively even and widespread distribution suggested adequate representation of the 97 GPs throughout the metropolitan and most rural areas of the state. GPs reported the total number of consultations per week from which proportions were calculated as the number of ILI patients per 1,000 consultations. ILI was defined as fever (or history of fever), cough, and either fatigue or malaise [8]. GPs were asked to collect a nose and/or throat swab from patients with an ILI within four days of the onset of the patient's symptoms and provide data on the patient's age, sex, date of symptoms onset, influenza vaccination status in 2011 and 2010, date of vaccination and presence of comorbid conditions for which influenza

vaccination is indicated. Patients were chosen for swabbing at the discretion of the GP.

To test for influenza viruses, RNA was extracted from clinical specimens using a Corbett extraction robot followed by reverse transcription using random hexamers. cDNA was amplified using an ABI-7500 Fast Real-Time PCR System incorporating primers and probes specific for the detection of type A, B and C influenza viruses. Samples that tested positive for influenza type A in this assay were subtyped in a second real-time PCR assay incorporating primers and probe specific for influenza A(H1N1)pdm09, A(H1) (non-pandemic) and A(H3) haemagglutinin genes.

VE was defined as (1-adjusted odds ratio)x100%, where the odds ratio is the ratio of odds of laboratory-confirmed influenza cases being vaccinated to the odds of controls (those that tested negative for influenza) being vaccinated. Logistic regression was used to calculate odds ratios and 95% confidence intervals that were adjusted for the variables of age group, month of specimen collection and comorbidity. There was not sufficient statistical power to generate age-specific VE estimates for the age group ≥65 years or to further stratify the age group of 0–19 year-olds. Patients were excluded from the VE analysis if vaccination status was unknown, if the date of symptom onset was unknown or if there was an interval greater than four days between symptom onset and specimen collection, based on the decreased likelihood of a positive result after this time [9,10]. Patients were considered not vaccinated if there was less than 14 days between the date of vaccination and symptom onset. All analyses were conducted using Stata (version 10.0; StataCorp LP). The chi-squared test was used to compare proportions, with $p<0.05$ considered statistically significant.

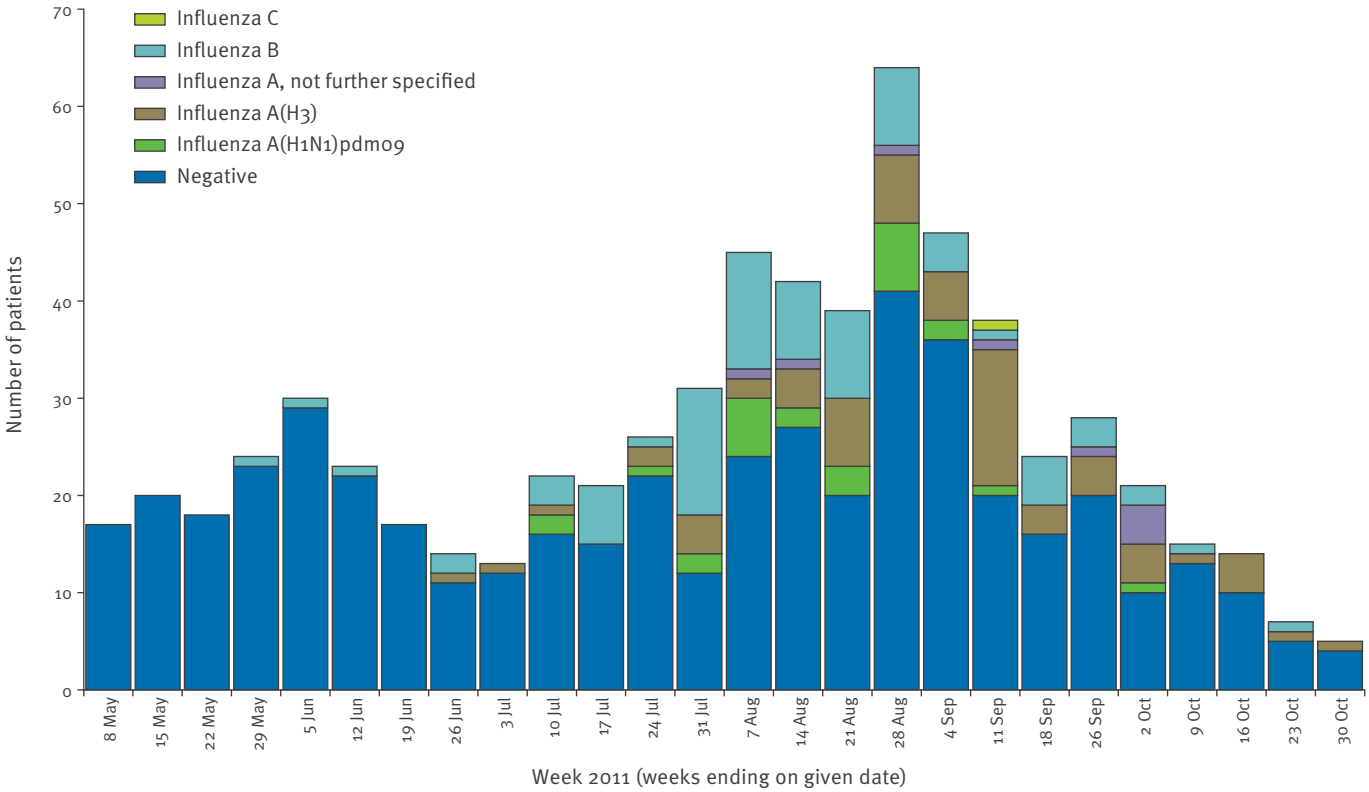
Results

Participating GPs reported seeing a total of 194,295 patients during the reporting period, of whom 945 (0.5%) met the ILI case definition, a proportion that was consistent with previous years. As the reporting of ILI cases is not identifiable and separate to those who are swabbed (for whom data are recorded on a laboratory test request form), we are unable to assess any demographic or vaccination status differences between those who were swabbed and those who were not. Of the 945 ILI cases, 665 (70%) were swabbed and 185 (28%) tested positive for influenza. In general, influenza A(H1N1)pdm09 predominated during the first half of the season, A(H3) during the middle to latter part, whilst cases of influenza B were detected throughout (Figure). One case of influenza type C infection was also detected.

We excluded 136 swabbed patients (20%) from the VE analysis due to unknown vaccination status (n=25), unknown date of symptom onset (n=44) or more than four days between symptom onset and specimen

Figure

Influenza-positive and -negative patients at sentinel general practices by week, Victoria, 2 May to 30 October (n=665)



collection (n=80); some were excluded for more than one reason. The case of influenza type C infection was also excluded. There was no statistically significant difference between the swabbed patients that were included and those that were excluded from the study by vaccination status (p=0.11), influenza positivity (p=0.07), age group (p=0.72), presence of a comorbid condition (p=0.21) or vaccination in 2010 (p=0.10).

Of the 529 patients included in the study, 155 (29%) were cases and 374 (71%) were controls. Cases were significantly younger than controls (p=0.004) and more common in August and September (p<0.001), but there was no statistically significant difference between cases and controls by sex (p=0.31) (Table 1). There was no statistically significant difference between cases and controls with respect to presence of a comorbidity recommended for influenza vaccination (p=0.15), although those with a comorbid condition were more likely to be older (p<0.001) and to be vaccinated (p<0.001). Being vaccinated in 2010 was not associated with testing positive for influenza (p=0.21), but was associated with older age (p<0.001) and with vaccination in 2011 (p<0.001).

Of the 529 patients eligible for the VE analysis, 65 (12%) were reported as vaccinated, with a statistically significant difference between cases (6%) and controls (15%) (p=0.008) (Table 2). No cases of influenza A(H1N1)pdm09 were reported as vaccinated. The proportion vaccinated was significantly higher in older age groups (p<0.001), but there was no statistically significant difference between those vaccinated and not vaccinated by month of testing (p=0.63).

There was little difference in the overall crude (60%) and adjusted (56%) point estimates for VE against all influenza, although only the crude estimate was statistically significant (Table 3). Although slightly higher against influenza A(H1N1)pdm09, age-adjusted VE estimates were generally consistent when stratified by type and subtype, however, 95% confidence intervals for estimates in the age group of 0–19 year-olds were very wide. Crude VE against influenza A(H1N1)pdm09 was 100% because none of 24 cases with confirmed influenza A(H1N1)pdm09 were vaccinated, but the VE was reduced after adjustment.

A sensitivity analysis conducted by restricting inclusion of cases and controls to the influenza season in 2011 when cases are more likely to be detected (the period from 20 June to 30 October when at least one influenza case was detected in consecutive weeks) resulted in changes to the point estimates from 0% to 1%. Not censoring patients for whom there were more than four days between symptom onset and specimen collection reduced the crude and overall adjusted VE estimates from 0% to 25% and from 2% to 14%, respectively.

Discussion

Using a population of patients with ILI who consulted sentinel GPs in Victoria, Australia, we have estimated a moderate effectiveness of 56% for the 2011 seasonal TIV against all influenza, although this was not statistically significant. VE estimates for the age group of 0-19 year-olds (childhood) were lower and considerably less precise than those for the age group of 20-64 year-olds. This is consistent with our observations in previous years which have highlighted the utility of this GP surveillance programme for estimating VE among working age adults who comprise most of the surveillance population [11,12].

Table 1

Characteristics of cases and controls, vaccine effectiveness study, Victoria, 2 May to 30 October (n=529)

	Number of controls (%)	Number of cases (%)	p value
Sex			
Female[a]	189 (51)	86 (55)	0.31
Age			
0–19 years	108 (29)	67 (43)	0.004
20–64 years	249 (67)	85 (55)	
≥65 years	17 (5)	3 (2)	
Month of swab collection			
May	71 (19)	2 (1)	<0.001
June	64 (17)	1 (<1)	
July	59 (16)	30 (19)	
August	107 (29)	74 (48)	
September	49 (13)	39 (25)	
October	24 (6)	9 (6)	
Comorbid condition[b]	43 (13)	12 (9)	0.15
Previously vaccinated[c]	76 (22)	25 (17)	0.21
Total	**374**	**155**	

[a] No data for one control.
[b] No data for 50 controls and 15 cases.
[c] No data for 27 controls and seven cases.

Table 2

Number and vaccination status of cases and controls by age group, vaccine effectiveness study, Victoria, 2 May to 30 October (n=529)

		Age group (years)			
		0–19	20–64	≥65	**Total**
Controls	n	108	249	17	**374**
	Vaccinated (%)	2 (2)	43 (17)	10 (59)	**55 (15)**
All influenza cases	n	67	85	3	**155**
	Vaccinated (%)	1 (1)	6 (7)	3 (100)	**10 (6)**
Influenza A(H1N1)pdm09 cases	n	4	20	0	**24**
	Vaccinated (%)	0 (0)	0 (0)	0 (0)	**0 (0)**
Influenza A(H3) cases	n	24	29	1	**54**
	Vaccinated (%)	0 (0)	3 (10)	1 (100)	**4 (7)**
Influenza B cases	n	37	30	2	**69**
	Vaccinated (%)	1 (3)	1 (3)	2 (100)	**4 (6)**

Strain typing surveillance data suggested good matches to the vaccine strains: 89% of 87 influenza A(H1N1) isolates were A/California/7/2009-like with the remainder A/California/7/2009-like (low reactor); 96% of 122 type A(H3N2) isolates were A/Perth/16/2009-like with the remainder A/Perth/16/2009-like (low reactor); 96% of 136 type B isolates were B/Brisbane/60/2008-like, 4% were B/Brisbane/60/2008-like (low reactor) and fewer than 1% were B/Florida/4/2006-like (low reactor) of the B/Yamagata/16/88 lineage (personal communication: K O'Bryan, World Health Organization Collaborating Centre for Reference and Research on Influenza, December 2011). Thus, the type- and subtype-stratified VE point estimates are broadly consistent with a good match to the circulating strains. However, none of the adjusted VE estimates was statistically significant suggesting insufficient study power. This is particularly evident in the childhood age group of the 0–19 year-olds.

To our knowledge there are no other published data for 2011 southern hemisphere seasonal influenza vaccine effectiveness. However, a point of comparison to other studies exists given the strain composition has not changed for the 2010/11 northern hemisphere and 2010 and 2011 southern hemisphere seasonal TIVs. In general the estimates obtained from our study were higher than those from other comparable studies. Using the same method we were able to demonstrate an effectiveness of 89% for the 2010 TIV against influenza A(H1N1)pdm09 among working age adults [12], compared with the 78% effectiveness observed this year. A study conducted amongst inpatients in 15 Australian hospitals in the same period in 2010 estimated a statistically significant effectiveness of 49% for TIV against hospitalisation with influenza A(H1N1) pdm09 [13]. Similarly in Europe, preliminary estimates for seasonal influenza vaccine effectiveness against all influenza using the test-negative variation of the case control study design among ILI patients seen in primary care were lower than our study, ranging from 5% to 50% [14-17]. The pooled end-of-season analysis of the European data resulted in lower adjusted estimates of VE against both influenza A(H1N1)pdm09 (27%) and type B influenza (64%) in working age adults compared to our study, although neither was statistically significant [18].

In our analysis we attempted to control for variables generally considered to be confounders [19], that is, those assumed to be associated with both exposure (vaccination) and outcome (influenza) but not on the causal pathway. These include age, month of swab collection and presence of a comorbid condition for which influenza vaccine is indicated. We observed generally little variation between crude VE estimates and those adjusted for these confounding variables. Only age was significantly associated with both vaccination and influenza. Month of swab collection and comorbidity were significantly associated with outcome and exposure respectively, but neither was significantly associated with both. Other studies using the same variation of the test-negative case control study as this one have also adjusted for receipt of influenza vaccine within a year before the study [16,18]. Whilst we collected this data field in 2011, its inclusion as a covariate in the adjusted model resulted in considerable variation from the crude and the age-, month- and comorbidity-adjusted VE estimates. However, further statistical analysis did not support inclusion of previous vaccination in the model because it assumes that previous vaccination has the same effect regardless of vaccination in the current season, and because of its high degree of correlation with current vaccination status which skews and reduces the precision of the VE estimate.

While variables may be considered to be theoretical confounders they may result in biases that could under- or over-estimate the VE. Results from influenza VE studies in Europe for the 2010/11 season included comments about the need for a cautious approach to dealing with such variables [17,20] and highlight the need for further clarification of the optimal analysis for the test-negative design when used to estimate influenza VE. Whilst relatively new, the method is administratively practical and theoretically acceptable, and

Table 3

Crude and adjusted vaccine effectiveness of seasonal vaccine against influenza by age group and type/subtype, Victoria, 2 May to 30 October (n=529)

	Influenza vaccine effectiveness (95% confidence interval)			
	Crude	Adjusted[a]		
		0–19 years	20–64 years	All ages
All	60 (19 to 80)	33 (-676 to 94)	61 (-3 to 85)	56 (-2 to 81)
Influenza A(H1N1)pdm09	100 (6 to 100)[b]	Not defined	77 (-44 to 100)[b,c]	78 (-38 to 100)[b,c]
Influenza A(H3)	54 (-34 to 84)	-44 (-1,757 to 100)[b,c]	48 (-99 to 86)	58 (-53 to 89)
Influenza B	64 (-2 to 88)	-16 (-1,298 to 90)	78 (-77 to 97)	53 (-68 to 87)

[a] Adjusted for month of swab collection and comorbidities.

[b] Calculated using exact method.

[c] Median unbiased estimates.

we will continue to refine it in collaboration with other investigators that have adopted it.

As previously discussed, other limitations of the study must also be taken into account when considering the results [6,12,21]. Briefly, the study was conducted in a general practice setting and the results are thus representative of the mid-range of the influenza clinical spectrum. Those not sick enough to attend a medical practitioner and more severe cases requiring hospitalisation were not part of the sampling frame. We were unable to quantify immunity from previous infection or healthy vaccinee bias, both of which overestimate VE. Conversely though, when conducted retrospectively, the test-negative case control design generally underestimates true VE under most conditions of test sensitivity, specificity and the ratio of influenza to non-influenza attack rates [22].

Overall, the seasonal TIV was moderately effective against medically attended influenza in Victoria, Australia during the 2011 southern hemisphere season. These VE estimates were generally consistent among working age adults when stratified by type and influenza A subtype, and consistent with an apparent good match between TIV and circulating strains during a season which saw the re-emergence of the influenza A(H3N2) subtype [23].

Acknowledgments

We thank Sheena Sullivan from the World Health Organization Collaborating Centre for Reference and Research on Influenza, Melbourne, Australia and Nevil Pierse from the Department of Public Health, University of Otago, Wellington, New Zealand for statistical advice. We especially thank the general practitioners that participated in the surveillance system during 2011 and staff of the Viral Identification Laboratory at the Victorian Infectious Diseases Reference Laboratory who conducted the influenza testing. The sentinel general practice surveillance programme is funded by the Victorian Government Department of Health.

References

1. Australian Government Department of Health and Ageing. Influenza (Flu). Canberra: Department of Health and Ageing. [Accessed Dec 15 2011]. Available from: http://health.gov.au/internet/immunise/publishing.nsf/Content/immunise-influenza
2. Recommended viruses of influenza vaccines for use in the 2011 influenza season (southern hemisphere). Wkly Epidemiol Rec. 2010;85(41):402-12.
3. Kissling E, Valenciano M, Falcao J, Larrauri A, Widgren K, Pitigoi D, et al. "I-MOVE" towards monitoring seasonal and pandemic influenza vaccine effectiveness: lessons learnt from a pilot multi-centric case-control study in Europe, 2008-9. Euro Surveill. 2009;14(44):pii=19388. Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19388
4. Belongia EA, Kieke BA, Donahue JG, Coleman LA, Irving SA, Meece JK, et al. Influenza vaccine effectiveness in Wisconsin during the 2007-08 season: comparison of interim and final results. Vaccine. 2011;29(38):6558-63.
5. Skowronski DM, De Serres G, Crowcroft NS, Janjua NZ, Boulianne N, Hottes TS, et al. Association between the 2008-09 seasonal influenza vaccine and pandemic H1N1 illness during Spring-Summer 2009: four observational studies from Canada. PLoS Med. 2010;7(4):e1000258.
6. Fielding JE, Grant KA, Papadakis G, Kelly HA. Estimation of type- and subtype-specific influenza vaccine effectiveness in Victoria, Australia using a test negative case control method, 2007-2008. BMC Infect Dis. 2011;11:170.
7. Recommended composition of influenza virus vaccines for use in the 2011-2012 northern hemisphere influenza season. Wkly Epidemiol Rec. 2011;86(10):86-90.
8. Thursky K, Cordova SP, Smith D, Kelly H. Working towards a simple case definition for influenza surveillance. J Clin Virol. 2003;27(2):170-9.
9. Carrat F, Vergu E, Ferguson NM, Lemaitre M, Cauchemez S, Leach S, et al. Time lines of infection and disease in human influenza: a review of volunteer challenge studies. Am J Epidemiol. 2008;167(7):775-85.
10. Cowling BJ, Chan KH, Fang VJ, Lau LL, So HC, Fung RO, et al. Comparative epidemiology of pandemic and seasonal influenza A in households. N Engl J Med. 2010;362(23):2175-84.
11. Kelly H, Carville K, Grant K, Jacoby P, Tran T, Barr I. Estimation of influenza vaccine effectiveness from routine surveillance data. PLoS One. 2009;4(3):e5079.
12. Fielding JE, Grant KA, Garcia K, Kelly HA. Effectiveness of seasonal influenza vaccine against pandemic (H1N1) 2009 virus, Australia, 2010. Emerg Infect Dis. 2011;17(7):1181-7.
13. Cheng AC, Kotsimbos T, Kelly HA, Irving LB, Bowler SD, Brown SG, et al. Effectiveness of H1N1/09 monovalent and trivalent influenza vaccines against hospitalization with laboratory-confirmed H1N1/09 influenza in Australia: a test-negative case control study. Vaccine. 2011;29(43):7320-5.
14. Kissling E, Valenciano M. Early estimates of seasonal influenza vaccine effectiveness in Europe, 2010/11: I-MOVE, a multicentre case-control study. Euro Surveill. 2011;16(11):p=19818. Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19818
15. Pebody R, Hardelid P, Fleming DM, McMenamin J, Andrews N, Robertson C, et al. Effectiveness of seasonal 2010/11 and pandemic influenza A(H1N1)2009 vaccines in preventing influenza infection in the United Kingdom: mid-season analysis 2010/11. Euro Surveill. 2011;16(6):pii=19791. Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19791
16. Savulescu C, Jimenez-Jorge S, de Mateo S, Ledesma J, Pozo F, Casas I, et al. Effectiveness of the 2010/11 seasonal trivalent influenza vaccine in Spain: preliminary results of a case-control study. Euro Surveill. 2011;16(11):p=19820. Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19820
17. Steens A, van der Hoek W, Dijkstra F, van der Sande M. Influenza vaccine effectiveness, 2010/11. Euro Surveill. 2011;16(15):p=19843. Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19843
18. Kissling E, Valenciano M, Cohen JM, Oroszi B, Barret AS, Rizzo C, et al. I-MOVE Multi-Centre Case Control Study 2010-11: Overall and Stratified Estimates of Influenza Vaccine Effectiveness in Europe. PLoS One. 2011;6(11):e27622.
19. Valenciano M, Kissling E, Ciancio BC, Moren A. Study designs for timely estimation of influenza vaccine effectiveness using European sentinel practitioner networks. Vaccine. 2010;28(46):7381-8.
20. Puig-Barbera J. 2010-2011 influenza seasonal vaccine, preliminary mid-season effectiveness estimates: reason for concern, confounding or are we following the right track? Euro Surveill. 2011;16(11):p=19821. Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=19821
21. Kelly HA, Grant KA, Fielding JE, Carville KS, Looker CO, Tran T, et al. Pandemic influenza H1N1 2009 infection in Victoria, Australia: no evidence for harm or benefit following receipt of seasonal influenza vaccine in 2009. Vaccine. 2011;29(37):6419-26.
22. Orenstein EW, De Serres G, Haber MJ, Shay DK, Bridges CB, Gargiullo P, et al. Methodologic issues regarding the use of three observational study designs to assess influenza vaccine effectiveness. Int J Epidemiol. 2007;36(3):623-31.
23. Lopez Chavarrias V, Broberg E, Nicoll A. Preliminary implications for Europe of the 2011 influenza season in five temperate southern hemisphere countries. Euro Surveill. 2011;16(50):p=20044. Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20044

Outbreak investigation of brucellosis in Thassos, Greece, 2008

I Karagiannis[1], K Mellou[1], K Gkolfinopoulou (kassy1golf@yahoo.com)[1], G Dougas[1], G Theocharopoulos[1], D Vourvidis[2], D Ellinas[3], M Sotolidou[3], T Papadimitriou[1], R Vorou[1]
1. Hellenic Center for Disease Control and Prevention, Athens, Greece
2. Ministry of Rural Development and Food, Athens, Greece
3. Primary Health Care Center of Prinou, Thassos, Greece

Citation style for this article:
Karagiannis I, Mellou K, Gkolfinopoulou K, Dougas G, Theocharopoulos G, Vourvidis D, Ellinas D, Sotolidou M, Papadimitriou T, Vorou R. Outbreak investigation of brucellosis in Thassos, Greece, 2008.
Euro Surveill. 2012;17(11):pii=20116. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20116

Article published on 15 March 2012

In spring 2008, the Hellenic Center for Disease Control and Prevention was notified about human brucellosis cases in Thassos, a Greek island that had been up to that point under a brucellosis eradication programme. Following the verification of the outbreak a 1:1 case–control study was conducted in the island. The study revealed that consumption of locally produced raw cheese was a risk factor for *Brucella melitensis* infection (odds ratio (OR): 15.1, 95% confidence interval (CI): 6.56–34.7). *Brucella melitensis* biotype 3 was identified in two clinical samples. As a result of the outbreak, the island is no longer officially considered as an area with farms free of brucellosis and is currently under a brucellosis control programme. The investigation of this outbreak demonstrated that control and eradication of brucellosis is not only a question of designing a strategy, but rather of ensuring its continuous, strict implementation. Furthermore, it revealed the lack of appropriate education of the public regarding the risks associated with raw, non heat-treated cheese consumption.

Introduction

Brucellosis is a common disease worldwide, representing a serious public health problem in many countries, especially those around the Mediterranean Sea [1-6]. In Greece, since 1998, the strategy of confronting brucellosis in small ruminants consists of two main components: (i) the implementation of a control programme (mass vaccination policy) in the mainland of the country and (ii) the implementation of an eradication programme in the islands, based on a test and slaughter policy [7,8].

On 16 May 2008, the Hellenic Centre for Diseases Control and Prevention (HCDCP) was notified about seven human cases of brucellosis in Thassos, the northernmost Greek island, located in the region of East Macedonia and Thrace. Thassos is only 10 km away from the mainland and has 13,720 inhabitants (census 2011) and an estimated population of goats and sheep of around 60,000. There are no cattle herds on the island. At the time of the notification, Thassos was under a brucellosis eradication programme for livestock, which had been in place since 1998 [9]. Routine serological testing of animals conducted by the competent veterinary services had not resulted in any reports of animal cases since 2005. According to information from the mandatory notification system, no laboratory-confirmed human cases among the residents of Thassos or human cases epidemiologically linked to the island had been reported since 2005.

During the following days, more cases were notified from the island and an outbreak was verified. An outbreak investigation was initiated in order to identify the source of infection, investigate possible ways of the pathogen introduction into the island, control the outbreak and prevent the occurrence of future outbreaks in the area.

Methods

Epidemiological investigation

A 1:1 case–control study was conducted. Ninety-eight cases and 98 controls were invited to participate. A case was defined as any resident of Thassos aged ≥ 18 years who, from January until the end of August 2008, presented at least two of the following symptoms: fever, night sweats, arthralgia, malaise, headache, and who also had a positive Standard Agglutination Test (SAT) in a Wright test titre of at least 1:160. Controls were selected via simple random sampling of the general adult population using the municipality registries and a random number table. Person-to-person interviews were taken locally via a structured questionnaire, by healthcare professionals of the regional primary healthcare centre. The questionnaire included demographic information, clinical symptoms and exposure to possible risk factors for up to two months before symptom onset for cases, and in weeks 14 to 23 of 2008 for controls, such as participation in common meals, possession of or contact with farm animals,

manure handling, visits to areas with farm animals/breeding environments, consumption of dairy products and site of purchase thereof. The main hypothesis based on the available descriptive epidemiological data was that the consumption of a traditional cheese variety that is produced locally around Orthodox Easter had been the source of the outbreak.

Epidata (v3.1, Denmark) was used for data entry and Stata 11.0 software (STATA, College Station, Texas, USA) for data analysis. Each binomial exposure was analysed individually (univariate analysis). Odds ratios (OR) and the corresponding 95% confidence intervals (CIs) were calculated in Stata v11. When zero counts were reported for the control group, Cornfield's approximation was used. Variables that were statistically significant at the α=0.2 level in the univariate analysis and to which at least 20% of the cases had been exposed were included in the multivariable analysis. The latter was conducted through the use of multiple logistic regression using backwards elimination techniques.

Laboratory and environmental investigation

Blood samples were obtained from eight patients in order to confirm the aetiological agent of the outbreak and identify the *Brucella* species involved. Further laboratory investigation including agglutination with specific antisera was performed for identifying the biotype of the bacterium. The Prefectural Public Health Directory of Kavala carried out hygienic inspections in shops and local cheese producers of the area. The Prefectural Veterinary Directorate tested herds in the area for *Brucella* spp. during the second half of May 2008.

Results

Epidemiological investigation

Ninety-eight cases and 63 controls responded (response rates: 100% and 64% respectively). Age did not statistically significantly differ between the two groups. Fifty-five cases (56%) and 19 controls (30%) were male. The most common clinical manifestations reported by cases are presented in Table 1. Malaise and fever were the two predominant symptoms, followed by night sweats, arthralgia and headache. Back pain was less frequent, being present in approximately one quarter of the cases.

Table 1

Clinical manifestations among brucellosis cases in Thassos, Greece, February–August 2008 (n=98)

Symptom	N (%)
Fever	62 (63)
Night sweats	60 (61)
Arthralgia	52 (53)
Back pain	28 (29)
Malaise	74 (76)
Headache	50 (51)

The distribution of week of symptom onset for the cases is depicted (Figure). The geographical distribution of cases and controls was similar. Most cases were residents of villages A (60%) and B (24%), while the retrospective percentages among controls were 62% and 27%.

Exposures that were ultimately included in the multivariable model are presented in Table 2 (univariable analysis results). After adjusting for age and sex and controlling for covariation of independent variables in the multivariable analysis, only consumption of fresh cheese from a local producer was a risk factor (OR:15.09, 95% CI: 6.56–34.7).

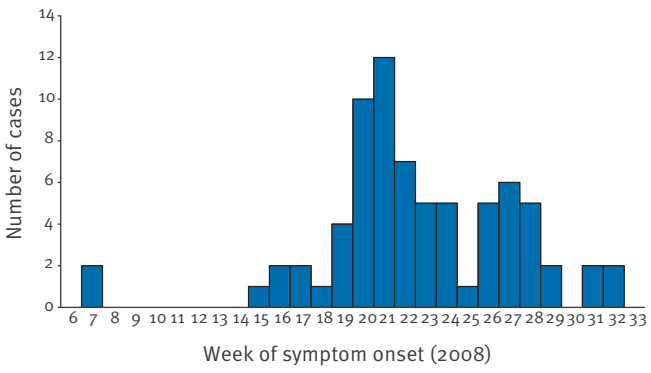
Laboratory and environmental investigation

Brucella melitensis was isolated in five of eight blood samples that were acquired from patients. In two of the positive samples, agglutination with specific antisera, revealed *B. melitensis* biotype 3, while no biotype testing was carried out in the other three positive samples. The environmental investigation in July 2008 did not pinpoint any specific establishment or market unit that could be linked to the outbreak. During the second half of May 2008, before the implementation of a mass vaccination control programme on the island, 30 herds were randomly tested for *Brucella* spp. including 4,585 sheep and goats. A total of 488 animals belonging to 18 (60%) of the 30 herds were found positive, resulting in a seroprevalence of brucellosis of 11% among sheep and goats. Further investigations for the identification of a specific biotype were not conducted.

In August 2008, epidemiological evidence showed that cheese consumption from a specific breeder of the island was linked to the occurrence of brucellosis; 79 of the 85 (93%) cases who had consumed fresh non heat-treated cheese, made with goat and sheep milk, could remember that the cheese had been purchased from that breeder. Cheese products were actually distributed by the specific breeder to consumers personally

Figure

Notified cases of brucellosis, by week of symptom onset, Thassos, Greece, 4 February–17 August 2008 (n=98)



The week of symptom onset is not known for 24 cases. These cases are not shown on the Figure.

and not through market stores. During the environmental investigation in July, raw unpasteurised cheese had been found in his premises but he claimed it was intended for personal consumption after maturation.

Measures implemented

In May 2008, the Prefectural Public Health Directorate of Kavala orally informed the residents about the basic preventive measures for the disease such as consuming only properly processed milk and dairy products and disseminated written information regarding the nature, the transmission route, and the main clinical manifestations of brucellosis. Written information on preventive measures was also given to local market stores and to taverns, to the Health Care Center of Prinos and to the General Hospital of Kavala. Local authorities also focused on informing local breeders and producers on correct practices and the risks associated with raw cheese consumption.

Following the results of the environmental investigation, the veterinary services of the Prefecture of Kavala moved on to a massive vaccination programme among young and adult female goats and sheep and to the slaughter of male animals which tested positive for *Brucella* spp..

In August, when the epidemiological link was made, all raw cheese in the premises of the implicated breeder had been consumed or discarded and his herds had already been included in the implemented massive vaccination programme of animals.

Discussion

In spring and summer 2008, there was an outbreak of brucellosis in the Greek island of Thassos that had been under a veterinary eradication programme since 1998. According to the available data from HCDCP, a similar outbreak had never been notified in the past. HCDCP worked closely with the Prefectoral Public Health Directory of Kavala to identify the aetiological agent of the outbreak, timely control the outbreak and take specific measures for the prevention of similar future incidents.

The results of the analytical epidemiological study showed that the consumption of raw non heat-treated cheese was the main risk factor for brucellosis infection, a finding that is consistent with previous reports in the literature [10-15]. Most cases, according to the epidemic curve, were exposed to the pathogen between week 15 and week 32 of 2008. Based on the incubation period of brucellosis (1–9 weeks), the time of exposure was, most probably, between week 14 and week 23 of 2008. The consumption of raw cheese is a local custom among the island inhabitants around the celebration of Orthodox Easter as part of a local tradition; in 2008, Easter was in the end of week 17. According to veterinary authorities, the most probable cause of re-emergence of brucellosis on the island was the illegal importation of animals from the mainland. However, we cannot be sure that this was actually the way that brucellosis was imported in Thassos.

A possible limitation of the study is that no breeders were included in the control group. This may have been a random result or due to the fact that breeders did not agree to participate. However, we believe that not having breeders in the control group could have actually led to an underestimation of the association between consumption of raw cheese from the specific breeder and the disease's occurrence. There are numerous breeders in the island that have small flocks and produce their own milk and cheese. One could thus assume that breeders mainly consume their own milk products and less so products of other producers. Thus, breeders would be less likely to consume raw cheese from the specific breeder involved in the outbreak compared to the general population. Even though some of the breeders in the cases' group had consumed raw cheese from the specific breeder during visits to friends or relatives, almost a third of them had become sick via other routes; this is why the consumption of cheese from the specific breeder does not

Table 2

Independent risk factors for acquisition of brucellosis in Thassos, Greece, February–August 2008

Risk factor	Cases (N=98) n (%)	Controls (N=63) n (%)	Odds ratio	95% Confidence interval
Men	55 (56)	19 (30)	3.03	1.48–6.31
Contact with farm animals (sheep, goats, cattle or pigs)	27 (28)	3 (5)	11.61	3.35–61.3
Visit to area with farm animals	29 (30)	17 (27)	1.50	0.72–3.22
Visit to breeding flocks	27 (28)	2 (3)	16.2	3.82–143.2
Manure handling	36 (37)	14 (22)	2.50	1.18–5.45
Goats' manure	27 (28)	10 (16)	2.23	1.02–5.08
Occupation relative to animals	26 (27)	0 (0)	1.78	0.8–4.09
Animal breeding	22 (22)	0 (0)	1.54	0.69–3.56
Fresh cheese consumption from a local producer	85 (87)	3 (5)	105.7	30.0–447.3

Variables were inserted in the multivariable analysis model before backwards elimination.

explain 100% of the cases. Based on this assumption, if the control group had also included breeders, the total number of controls who were not exposed to the cheese of the specific breeder could have actually been greater, leading to an even higher odds ratio.

Limited ability of performing bacteria cultures or more specific serological tests at the local level (such as Enzyme-linked immunosorbent assay (ELISA) or complement fixation test) and the unavailability of samples from the raw cheese leftovers, hampered the microbiological confirmation of the apparent epidemiological association.

Data from the mandatory notification system show that the last two cases of human brucellosis among residents of the island were notified in the beginning of 2009. There have not been any human cases identified ever since (until the end of October 2011) and the routine serological testing of local herds by the competent veterinary authorities also demonstrates the effectiveness of the implemented measures. The vaccination programme on the island is still ongoing. However, it is still soon to say that the island is free of brucellosis again, based on the data provided by the local veterinary authorities.

This outbreak has clearly demonstrated that control and eradication of brucellosis is not only a question of designing a strategy, but rather of ensuring its continuous, strict implementation through well organised policies and programmes. Political will, commitment for inter-sectoral collaboration between all involved parties and close monitoring and evaluation of the measures implemented are unquestionable prerequisites for disease control and eradication [16].

Acknowledgments

V Batziliotis, I Angeli, A Panteliadou, General Veterinary Directorate, Ministry of Rural Development and Food, Athens, Greece; A Tataridis, Thassos, Greece; A Kansouzidou, T Mantoudi, F Kamaria, A Bakas, Thessaloniki Hospital for Infectious Diseases, Thessaloniki, Greece; S Koutlas, T Ainalidou, T Eleftheriadis, Veterinary Directorate of Kavala Prefecture, Kavala, Greece; C Papadopoulos, D Hizaris, K Vamvakis, Directorate of Public Health of Kavala Prefecture, Kavala, Greece.

References

1. Seimenis AM. Zoonotic diseases in the Mediterranean region: a brief introduction. Vet Ital. 2008;44(4):573-6
2. Mantovani A. General epidemiological aspects of major zoonoses in the Mediterranean Region. In report of: Mediterranean Zoonoses Control Programme (MZCP)/Workshop on Zoonoses Surveillance and Control in the Mediterranean Region. Athens: World Health Organization/ Mediterranean Zoonoses Control Centre. 1998. Available from: http://www.mzcp-zoonoses.gr/pdfen/Report.pdf
3. Taleski V, Zerva L, Kantardjiev T, Cvetnic Z, Erski-Biljic M, Nikolovski B, et al. An overview of the epidemiology and epizootology of brucellosis in selected countries of Central and Southeast Europe. Vet Microbiol. 2002; 20;90(1-4):147-55.
4. Avdikou I, Maipa V, Alamanos Y. Epidemiology of human brucellosis in a defined area of Northwestern Greece. Epidemiol Infect. 2005;133(5):905-10.
5. Bosilkovski M, Krteva L, Dimzova M, Vidinic I, Sopova Z, Spasovska K. Human brucellosis in Macedonia - 10 years of clinical experience in endemic region. Croat Med J. 2010;51(4):327-36.
6. Pappas G, Papadimitriou P, Akritidis N, Christou L, Tsianos EV. The new global map of human brucellosis. Lancet Infect Dis. 2006;6(2):91-9.
7. European Commission. Final report of a mission carried out in Greece from 23 to 27 September 2002. In order to evaluate the progress of the bovine Brucellosis eradication programme. DG(SANCO)/8629/2002. MR Final. 2002. Available from: http://ec.europa.eu/food/fs/inspections/vi/reports/greece/vi_rep_gree_8629-2002_en.pdf
8. Jelastopulu E, Bikas C, Petropoulos C, Leotsinidis M. Incidence of human brucellosis in a rural area in Western Greece after the implementation of a vaccination programme against animal brucellosis. BMC Public Health. 2008;8:241.
9. Vorou R, Gkolfinopoulou K, Dougas G, Mellou K, Pierroutsakos IN, Papadimitriou T. Local brucellosis outbreak on Thassos, Greece: a preliminary report. Euro Surveill. 2008; 13(25):pii=18910. Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=18910
10. Alterkruse SF, Timbo BB, Mowbray JC, Bean NH, Potter ME. Cheese-associated outbreaks of human illness in the United States, 1973 to 1992: sanitary manufacturing practices protect consumers. J Food Prot. 1998;61(10):1405-7
11. Farina F, Fuser R, Rossi M, Scotton PG. Outbreak di brucellosi nella provincia di Treviso da formaggio pecorino importato [Brucellosis outbreak in Treviso province caused by infected cheese from an endemic area]. Infez Med. 2008;16(3):154-7. Italian.
12. Minas M, Minas A, Gourgulianis K, Stournara A. Epidemiological and clinical aspects of human brucellosis in Central Greece. Jpn J Infect Dis. 2007;60(6):362-6.
13. Mendez Martinez C, Paez Jimenez A, Cortés-Blanco M, Salmoral Chamizo E, Mohedano Mohedano E, Plata C, et al. Brucellosis outbreak due to unpasteurized raw goat cheese in Andalucia (Spain), January-March 2002. Euro Surveill. 2003; 8(7):pii=421. Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=421
14. Ramos JM, Bernal E, Esguevillas T, Lopez-Garcia P, Gaztamibide MS, Gutierrez F. Non-imported brucellosis outbreak form unpasteurized raw milk in Moroccan immigrants in Spain. Epidemiol Infect 2008; 136:1552-5
15. Hadjichristodoulou C, Papatheodorou C, Soteriades E, Panagakos G, Kastritis I, Goutziana G, et al. Epidemiological study of brucellosis in eight Greek villages using a computerised mapping programme. Eur J Epidemiol. 1999;15(7):671-80.
16. Robinson A. Guidelines for coordinated human and animal brucellosis surveillance. Food and Agriculture Organization of the United Nations (FAO) animal production and health paper 156. Rome: FAO. 2003. Available from: ftp://ftp.fao.org/docrep/fao/005/y4723e/y4723e00.pdf

Carbapenem-resistant *Acinetobacter baumannii* carrying the NDM-1 gene, Czech Republic, 2011

A Nemec (anemec@szu.cz)[1], L Krizova[1]
1. Laboratory of Bacterial Genetics, Centre for Epidemiology and Microbiology, National Institute of Public Health, Prague, Czech Republic

Citation style for this article:
Nemec A, Krizova L. Carbapenem-resistant Acinetobacter baumannii carrying the NDM-1 gene, Czech Republic, 2011.
Euro Surveill. 2012;17(11):pii=20121. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20121

Article published on 15 March 2012

To the editor: We read with interest the paper of Hrabák et al. [1] on the isolation of a bla_{NDM-1}-carrying *Acinetobacter baumannii* strain. However, although the authors believed to be the first in the Czech Republic to report on a New Delhi metallo-beta-lactamase-1 (NDM-1)-producing bacterium, there have been other reports on the same *A. baumannii* strain in the country [2,3]. Here, we would like to comment on some points of these studies.

In our report published in a Czech national bulletin in September 2011 [2], we presented epidemiological and microbiological data on a high-level carbapenem-resistant strain (designated ANC 4097) isolated from a patient hospitalised in an intensive care unit in the Czech Republic. Based on the available information, we concluded that the strain had been imported to the Czech Republic from Egypt in mid-2011 [2]. The strain was shown to be resistant in vitro to all beta-lactams and to most other clinically relevant antimicrobial agents, and to carry the genes encoding the NDM-1 and OXA-23 carbapenemases together with a number of other resistance determinants [2]. In a later paper, we provided additional genetic characterisation of ANC 4097 focused on the genetic structures associated with the bla_{NDM-1} and bla_{OXA-23} genes [3]. We have only recently learned that the laboratory where the original clinical specimens were processed had provided the same bacterial strain to two research groups. Thus, two independent investigations on the same strain were conducted leading to our papers [2,3] and that of Hrabák et al. [1].

Even though the data on the NDM-1 carrying strain presented in the independent studies are mostly congruent, some findings and conclusions by Hrabák et al. [1] deserve a commentary. Firstly, Hrabák et al. claimed that the strain was a producer of the NDM-1 carbapenemase based on the presence of the bla_{NDM-1} gene, carbapenemase activity, and the inhibitory effect of ethylenediaminetetraacetic acid (EDTA) on the carbapenem resistance phenotype. However, ANC 4097 was also shown by us to harbour the bla_{OXA-23} and bla_{OXA51}-like genes, both carrying IS*Aba1* in their promoter regions [2,3]. Therefore, the strain may produce at least three different carbapenemases, each of which can be responsible for the carbapenem resistance. As EDTA inhibition of carbapenemase activity is not a specific marker to detect the metallo-beta-lactamase production in *A. baumannii* [4,5], unambiguous evidence that ANC 4097 is a genuine producer of NDM-1 (and not only a carrier of a silent bla_{NDM-1} gene) is still missing.

Secondly, the minimum inhibitory concentration (MIC) for chloramphenicol (8 mg/L) reported by Hrabák et al. was surprisingly low seeing as *A. baumannii* is typically resistant to this antibiotic. In contrast, we found a chloramphenicol MIC of ≥256 µg/mL in ANC 4097 using Etest (bioMérieux), and the strain yielded a positive PCR signal for the *catA1* gene encoding chloramphenicol acetyltransferase (unpublished data). It is of note that the catA1 gene is part of the AbaR3 resistance island which, or variants of which, are commonly present in *A. baumannii* European clone I to which ANC 4097 belongs [3].

Finally, although Hrabák et al. [1] have reported their isolates to be susceptible only to colistin, we found that ANC 4097 was also susceptible to at least tobramycin and doxycycline [2,3]. Even though these antimicrobials may have limited value in the treatment of systemic *Acinetobacter* infections they have been recommended for consideration when defining the level of multidrug resistance in *A. baumannii* for epidemiological purposes [6].

Despite these points, the epidemiological part of the report of Hrabák et al. [1] has valuably contributed to the comprehensiveness of the information on the first bacterial strain with the bla_{NDM-1} gene isolated in the Czech Republic.

References

1. Hrabák J, Štolbová M, Študentová V, Fridrichová M, Chudáčková E, Zemlickova H. NDM-1 producing Acinetobacter baumannii isolated from a patient repatriated to the

Czech Republic from Egypt, July 2011. Euro Surveill. 2012;17(7):pii=20085. Available from: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20085

2. Nemec A, Křížová L, Maixnerová M, Šmejcová A, PSA. Multirezistentní Acinetobacter baumannii nesoucí geny pro karbapenemázy NDM-1 a OXA-23 importovaný do České republiky. [Multidrug-resistant Acinetobacter baumannii strain carrying genes encoding carbapenemases NDM-1 and OXA-23 imported to the Czech Republic]. Zprávy CEM. 2011;20(8):295–8. Czech. Available from: http://www.szu.cz/uploads/documents/CeM/Zpravy_EM/20_2011/08_srpen/295_298.pdf
3. Krizova L, Bonnin RA, Nordmann P, Nemec A, Poirel L. Characterization of a multidrug-resistant Acinetobacter baumannii strain carrying the blaNDM-1 and blaOXA-23 carbapenemase genes from the Czech Republic. J Antimicrob Chemother. 2012 Feb 24. [Epub ahead of print].
4. Segal H, Elisha BG. Use of Etest MBL strips for the detection of carbapenemases in Acinetobacter baumannii. J Antimicrob Chemother. 2005;56(3):598.
5. Bonnin RA, Naas T, Poirel L, Nordmann P. Phenotypical-, biochemical- and molecular-based techniques for detection of metallo-β-lactamase NDM in Acinetobacter baumannii. J Clin Microbiol. 2012 Jan 18. [Epub ahead of print].
6. Magiorakos AP, Srinivasan A, Carey RB, Carmeli Y, Falagas ME, Giske CG, et al. Multidrug-resistant, extensively drug-resistant and pandrug-resistant bacteria: an international expert proposal for interim standard definitions for acquired resistance. Clin Microbiol Infect. 2012;18(3):268-81.

The European Union Summary Report on antimicrobial resistance in zoonotic and indicator bacteria from humans, animals and food in 2010

Eurosurveillance editorial team (eurosurveillance@ecdc.europa.eu)[1]
1. European Centre for Disease Prevention and Control (ECDC), Stockholm, Sweden

Citation style for this article:
Eurosurveillance editorial team. The European Union Summary Report on antimicrobial resistance in zoonotic and indicator bacteria from humans, animals and food in 2010.
Euro Surveill. 2012;17(11):pii=20118. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20118

Article published on 15 March 2012

On 14 March, the European Food Safety Authority (EFSA) and the European Centre for Disease Prevention and Control (ECDC) published the second joint report on antimicrobial resistance in zoonotic bacteria affecting humans, animals and food [1]. The report presents antimicrobial resistance data for 2010. Data from 26 European Union (EU) Member States were analysed by ECDC and EFSA to give an overall picture of antimicrobial resistance among *Salmonella* and *Campylobacter* isolates and indicator *Escherichia coli* and *Enterococcus* isolates. Limited data on meticillin-resistant *Staphylococcus aureus* in animals and food were also included

This joint scientific report contributes to the work currently being carried out at EU level to fight antimicrobial resistance in zoonotic bacteria.

References

1. European Food Safety Authority and European Centre for Disease Prevention and Control. The European Union Summary Report on antimicrobial resistance in zoonotic and indicator bacteria from humans, animals and food in the European Union in 2010. EFSA Journal 2012;10(3):2598. doi:10.2903/j.efsa.2012.2598. Available from: www.efsa.europa.eu/efsajournal

Call for expression of interest for membership in EFSA's Stakeholder Platform

Eurosurveillance editorial team (eurosurveillance@ecdc.europa.eu)[1]
1. European Centre for Disease Prevention and Control (ECDC), Stockholm, Sweden

Citation style for this article:
Eurosurveillance editorial team. Call for expression of interest for membership in EFSA's Stakeholder Platform.
Euro Surveill. 2012;17(11):pii=20120. Available online: http://www.eurosurveillance.org/ViewArticle.aspx?ArticleId=20120

Article published on 15 March 2012

The European Food Safety Authority (EFSA) has launched a public call for expression of interest for organisations who wish to be considered for membership in the EFSA Stakeholder Platform.

All interested European Union-wide organisations involved in the food chain are invited to submit their expressions of interest in joining the Platform by 6 April 2012.

Read more at: http://www.efsa.europa.eu/en/stakeholders/cp.htm.