REVIEW 10.1111/1469-0691.12000

Leading infectious diseases problems in Turkey

H. Erdem[1] **and M. Akova**[2]

1) *Department of Clinical Microbiology and Infectious Diseases, GATA Haydarpasa Training and Research Hospital, Istanbul and* 2) *Section of Infectious Diseases, Hacettepe University School of Medicine, Ankara, Turkey*

Abstract

Turkey has significant geographical and socio-economic differences throughout a vast area of the country. These characteristics affect the epidemiology of infectious diseases, some of which are rarely seen in western Europe. However, effectively implemented control measures have resulted in decreased rates of many community-acquired infections, including tuberculosis and malaria, that were major health problems only a few decades ago. There are high rates of antimicrobial resistance in various nosocomial isolates of Gram-positive and Gram-negative bacteria. A recently implemented, nationwide, electronic resistance surveillance system in hospitals is expected to produce reliable data, and possibly will help to develop an effective strategy to decrease antimicrobial resistance in bacteria that currently plague many tertiary-care hospitals in the country. This article summarizes the most frequently encountered community-acquired infections, and gives an overview of current antimicrobial resistance in both outpatient and hospital settings in Turkey.

Keywords: Antimicrobial resistance, infectious diseases, Turkey
Article published online: 11 September 2012
Clin Microbiol Infect 2012; **18:** 1056–1067

Corresponding author: M. Akova, Section of Infectious Diseases, School of Medicine, Hacettepe University, 06100 Ankara, Turkey.
E-mail: makova@hacettepe.edu.tr

Introduction

Turkey is at the crossroads between Europe, Asia, and Africa. This geographical situation has not only had significant economic and cultural impacts over the centuries, but has also been responsible for the epidemiology of various infectious diseases occurring throughout the country. As of 2012, Turkey has 75 million inhabitants, and the population growth rate is 13.5 per thousand. Three-quarters of the country's population resides in cities and large towns, and one-quarter resides in villages and rural areas (http://www.tuik.gov.tr/IcerikGetir.do?istab_id=139).

According to the data released by the Statistical Institute of Turkey, 4226 patients lost their lives because of infectious diseases in Turkey in 2008, and 102 528 and 33 188 died because of circulatory system disorders and neoplasms, respectively. Thus, communicable diseases contributed to mortality in <2% of all deaths in the country (http://www.tuik.gov.tr/IcerikGetir.do?istab_id=1). The current review focuses on the epidemiology of the most common infectious diseases in Turkey, including data on regional antibiotic resistance patterns.

Bacterial Infections

Brucellosis

Brucellosis is a zoonotic infection transmitted to humans from infected animals and animal food products. In 2002, 17 765 cases of human brucellosis were reported in Turkey, with a prevalence rate 25 per 100 000 population [1]. However, this figure almost halved to 9324 cases with a prevalence rate of 13 per 100 000 in 2009, owing to extensive control efforts [2]. The disease is mostly prevalent in the central and south-eastern parts of the country, where animal husbandry is an important source of living [3]. *Brucella melitensis*

accounts for the majority of cases of brucellosis in humans in Turkey, followed by *Brucella abortus*, the second most frequent agent [4].

Brucellosis has traditionally been included in the differential diagnosis of persistent fever in both adults and in children [5], including those with neutropenia and cancer [6–9], and causes a significant threat to laboratory workers [4,10]. The various complications and rare forms of clinical presentation have been extensively reported [11–14]. Several papers have been published on various modalities of treatment, including early reports on the ineffectiveness of quinolone monotherapy and the comparative efficacy between quinolone plus rifampin combinations and the classical WHO-suggested regimen of doxycycline plus rifampin [15–17].

Tularaemia

Tularaemia had been historically known to cause sporadic outbreaks, particularly in the north-western parts of the country [18]. However, tularaemia has now become an emerging zoonotic disease disseminating over a wider geographical area in Turkey [19–22]. Genetic analyses of *Francisella tularensis* recovered from various sites in the country showed close linkages with neighbouring countries such as Bulgaria [23]. Most of the cases described were of the pharyngeal type of disease, and the classical case description included a patient with fever, sore throat and cervical lymphadenopathy who failed to respond to β-lactam antibiotic therapy [21]. Furthermore, seroepidemiological evidence of the infection was detected in villagers dealing with farming and animal husbandry in the eastern part of the country [22].

The disease is prominent during winter and autumn; unchlorinated and contaminated water is the major cause of dissemination for oropharyngeal infection [24]. Occasionally, the oculoglandular form has been reported, owing to washing of the face with contaminated water. Although the microorganism could not be cultured from the suspected water sources, molecular evidence of tularaemia was found by PCR on the same samples [25]. *F. tularensis* ssp. *holarctica* (type B) was found to be the predominant subtype in Turkey [23]. Fishing, animal farming and hunting were proved to be additional risk factors in a study performed in an endemic area with an overall attack rate of 2.3 per 1000 population [26].

In an *in vitro* study, 39 isolates of *F. tularensis* were susceptible to aminoglycosides, tetracyclines, chloramphenicol, rifampicin, and fluoroquinolones, but were resistant to macrolides, clindamycin, and β-lactams. Furthermore, fluoroquinolones, levofloxacin in particular, were found to have the lowest MIC_{50} and MIC_{90} values [27].

Tuberculosis (TB)

Currently, the WHO estimates that 12 million people are living with tuberculosis (TB) worldwide (http://www.globalhealthfacts.org/data/topic/map.aspx?ind=16). According to a recent WHO report based on the geographical profile in 2010, the prevalence of the disease per 100 000 population was 5.9 in Germany, 15 in the UK, 18 in Spain, 29 in Poland, and 24 in Turkey (http://apps.who.int/ghodata/?vid=500). Overall, during the last 15 years, there has been a steady decline in the prevalence of TB cases in Turkey [28]; the rate of <25 cases per 100 000 means that the country is classified as a low-endemicity region according to the WHO definition. However, TB still continues to be included in the differential diagnosis of fever of unknown origin in clinical practice in Turkey [29–31].

The prevalence of multidrug-resistant (MDR) *Mycobacterium tuberculosis* (defined as isolates that are non-susceptible to isoniazid and rifampicin) is relatively low [32]; the reported figures vary between 2% and 21% [33–36]. According to the 'Fight against Tuberculosis 2009 Report' of the Turkish Ministry of Health (MoH), the frequency of MDR strains was 3% in new cases, whereas it was reported to be 18% in previously treated patients. Overall, this report indicated that the rate of MDR TB was 5% [37]. On the other hand, extensively drug-resistant TB (defined as MDR isolates resistant to any fluoroquinolone and one or more of the three injectable anti-TB drugs: capreomycin, kanamycin, and amikacin) was reported to be approximately 0–2.5% in Turkey [38,39].

Lyme disease

There have been many case reports of Lyme disease from Turkey [40–43]. The seropositivity rate was reported to be 17% in patients with a history of tick bite in central Anatolia [44], and *Borrelia burgdorferi*, the agent of Lyme disease, was found to be active in *Ixodes ricinus* ticks in Turkey [45]. However, solid data seem to be substantially lacking for the epidemiology of Lyme disease in Turkey.

Rickettsioses

In the past history, rickettsioses had subtantial impacts on the Turkish population [46–48]. The use of molecular genetic tools and cell culture assays have significantly improved the discovery of new agents, and consequently three new ehrlichioses and 12 rickettsioses have been described worldwide since 1980 [49]. On the other hand, old rickettsioses, such as epidemic typhus or scrub typhus, have re-emerged because of poor living standards [50].

A few small clusters of patients with disease related to *Rickettsia conorii* infection were observed in different parts of Turkey in the last decade [51–53]. Also, there were sporadic case reports of rickettsioses throughout the country [54,55].

Viral Infections

Human immunodeficiency virus (HIV) infection

In 2010, 32.4 million people were living with HIV worldwide, and 1.8 million died of AIDS-related illnesses (http://www.who.int/gho/HIV/en/index.html). The first case in Turkey was reported in 1985 [56], and, by the end of December 2011, there were 5224 confirmed cases (http://pozitifyasam.org/tr/turkiyede-HIV-AIDS.html). Although it is very probable that there has been under-reporting, the current prevalence of the disease is accepted to be <0.1% [57–59]. Turkey seems to have the lowest HIV prevalence rate after Bosnia and Herzegovina in the Balkan region, and the most frequent route of transmission is the heterosexual one [60]. Inadequate knowledge on sexually transmitted diseases, a relatively younger population, ease of travel, poor socio-economic conditions, an increase in the number of unregistered sex workers and intravenous drug use have all been reported to contribute to the prevalence of HIV infection in Turkey [61–64]. On the other hand, several nationwide awareness and counselling programmes for HIV and related diseases are available from different non-governmental organizations. Currently, all HIV-infected patients are covered by the national insurance system, which provides free access to all diagnostic procedures and antiretroviral treatment, including the up-to-date theraputic regimens.

Crimean–Congo haemorrhagic fever (CCHF)

Crimean–Congo haemorrhagic fever is a tick-borne infection caused by the genus *Nairovirus* from the family *Bunyaviridae* [65]. The disease was initially recognized by Russian scientists among Soviet military recruits in 1940s [66], and Crimean haemorrhagic fever was subsequently found to be indistinguishable from Belgian Congo fever [67]. Thus, the two names were combined.

The virus has been known to affect human populations in neighbouring countries such as Bulgaria [68], Greece [69], Iraq [70], and Iran [71]. The first symptomatic human CCHF case was noted in 2002 in Turkey, with serious impacts on healthcare workers [72–74]. Consequently, awareness of the disease has been raised among medical personnel [75]. In 2009, *c.* 1300 cases were reported, with a mortality rate of 4.8% [76]. The large number of cases admitted to hospitals during the epidemic season of the disease in certain areas has significantly increased the workload of blood banks and transfusion centres, owing to excessive consumption of blood and blood products for the management of the disease [77].

The potential vectors for CCHF transmission in Turkey are *Rhipicephalus bursa*, *Hyalomma marginatum marginatum* and *Haemaphysalis parva* ticks [78]. Although CCHF is usually diagnosed during spring and summer, when ticks are active, the disease has also been reported during the winter season [79].

The clinical presentation may range from a mild clinical course to severe and fatal progression. Recent outbreaks in Turkey have provided opportunities to detail the characteristics of the disease [80–86]. Patients frequently present with fever, fatigue, generalized pain, myalgia, nausea and vomiting, and diarrhoea, and in severe cases epistaxis, haematemesis, melena, haematuria, gingival bleeding, vaginal bleeding, petechiae or ecchymosis are also seen. In the latter cases, death occurs as a result of multi-organ failure, disseminated intravascular coagulation, and circulatory shock [87,88]. A recent seroepidemiological survey estimated that up to 88% of infections were subclinical [89].

The use of ribavirin remains controversial; however, the WHO recommends the use of both oral and intravenous formulations of ribavirin in the management of CCHF (http://www.Who.Int/mediacentre/factsheets/fs208/en/). Recently, many studies have been published on unsettled issues regarding the use of ribavirin for CCHF management [88,90–94].

Hantavirus infections

At least five hantavirus subtypes—Puumala, Dobrava, Saaremaa, Tula and Seoul subtypes—are found across Europe [95]. The virus is transmitted to humans through infected rodents and their excreta, in contrast to other genera of the family *Bunyaviridae*, which are usually transmitted through arthropods [96]. Hantaviruses cause two different types of disease in humans: haemorrhagic fever with renal syndrome (HFRS) and hantavirus pulmonary syndrome. HFRS is seen mainly in the Eurasian region, and hantavirus pulmonary syndrome is usually detected in the Americas [97].

Although there have been recent reports on hantavirus infections, there are no nationwide data on this disease. In 2009, 12 confirmed HFRS cases were reported from Zonguldak province, located in north-western Turkey. Although the results showed serological positivity for the Puumala subtype, the specificity of this test remains unconfirmed, owing to cross-reactivity among hantavirus subtypes. Another limitation of this study was the negative RT-PCR result; consequently, sequence analysis could not be performed [98]. Five cases of Dobrava subtype infection were reported from Giresun (two cases), a city located in north-eastern Turkey [99], Istanbul (two cases) [100,101], and Ankara [102]. The only death occurred in one of the Istanbul cases. In an animal study, the Puumala subtype was found in 6% of *Microtus* wild rodents in Trabzon and Izmir provinces, located in the north-eastern and western parts of the country, respectively [103].

West Nile virus (WNV) infection

West Nile virus (WNV), a member of the Japanese encephalitis virus antigenic complex, causes a wide range of clinical pictures, from asymptomatic disease to severe meningitis and encephalitis. Most of the people infected with WNV are asymptomatic, and symptoms are seen in only 20–40% of infected patients. Although fever is one of the characteristic symptoms caused by WNV, some patients report low-grade fever or no fever at all [104].

The initial evidence of WNV infection in Turkey obtained from a seroprevalence survey was obtained in 1977 [105]. There had been several case reports of WNV infection by the end of summer 2010 [106–109]. Thirty-five probable and 12 confirmed cases, with 21% mortality, were reported from 15 provinces, mainly located in the western part of the country [110]. WNV infections were included in the list of national notifiable diseases as of April 2011.

Seroprevalence studies indicated that serological evidence of WNV infection was present in approximately 1% of healthy blood donors [111,112]. Although WNV was not detected in the usual vectors, such as *Culex pipiens*, *Ochlerotatus caspius*, and *Aedes* spp., in the south-eastern provinces of Turkey, 16% of 181 human subjects screened in the same region were found to be seropositive [113].

Currently, there is no nationwide donor screening programme for WNV for organ transplantation.

Hepatitis A–D viruses

In 2011, the annual incidence rates of acute hepatitis A virus (HAV) and acute hepatitis B virus (HBV) infections, respectively, were 5.21 and 3.79 per 100 000 population in Turkey. Although mortality was not recorded for HAV infection, six patients lost their lives because of acute HBV infection and five because of hepatitis C virus (HCV) infection in 2011 (data from Turkish MoH, personal communication with S. Tosun). In 2008, 79% of acute HAV infections were detected among the younger age group (<15 years), and 93% and 97%, respectively, of acute HBV and HCV infections were seen among those who were >15 years of age. Currently, HAV infection still appears to be a childhood disease in Turkey, and HAV vaccine has been recently included in the National Immunization Programme. Thus, a consequent decrease in the incidence of the disease should be expected.

The main transmission route of HBV is through heterosexual contact [114]. The prevalence of HBV infection is higher (3–10%) in the eastern and south-eastern parts of Turkey [115,116], where the general socio-economic level of the population is lower than in the rest of the country. On the other hand, the prevalence rates vary between 1.7% and 3% in the western provinces [117–121]. This figure makes the central and western parts of the country low-endemicity and moderate-endemicity regions, and the eastern and south-eastern provinces high-endemicity regions, according to the WHO classification, in which a <2% threshold determines low endemicity and >8% high endemicity. Genotype D is known to be the predominant HBV genotype in Turkey [122,123]. HBV vaccination has been included in the National Immunization Programme for newborns and young children since 1998, and the cost of the vaccine is also reimbursed for those who are at high risk of acquiring the infection [124].

Although hepatitis D virus (HDV) infection prevalence seems to have declined recently, it is still a significant public health problem in Turkey [125]. The disease is frequent in the eastern and south-eastern provinces [126], and HDV seroprevalence is 3–5% among HBV carriers [127–129], 16–45% in chronic hepatitis cases [128,130], and 45% in patients with cirrhosis [128]. HDV-1 seems to be the predominant genotype in patients with HDV infections in Turkey [131,132].

The prevalence of HCV infections was reported to be 0.3–0.7% [116–120,133]. The mode of transmission was by transfusion of blood and blood products in most of the cases who received these products before the nationwide blood screening process for HCV had been implemented [134]. Most HCV infections in Turkey are caused by genotype 1b [134–136]. Currently, the country's stockpile of blood is safe, and all donations are serologically screened for HBV, HCV, and HIV, among other blood-borne pathogens. Moreover, couples are encouraged to be screened for these viruses before marriage, and those who are found to be positive are counselled with regard to preventive measures [121].

Parasitic Infections: Malaria

Malaria has been known since early in Turkish history [46], and today chloroquine-sensitive *Plasmodium vivax* is the only common aetiological agent of the disease throughout the country [137]. Sporadically, imported cases of *Plasmodium falciparum* have been reported [137]. In 1997, *c.* 35 000 cases of *P. vivax* malaria were notified. The number of infected cases fell to *c.* 10 000 in 2002, owing to intensive preventive efforts, including active and pasive surveillance and vector control measures (http://www.Saglik.Gov.Tr/tr/belge/1-3416/sitma-savas-daire-baskanliginin-sitma-ile-ilgili-istati-.Html).

Most of the patients were from Cukurova province, which is a predominantly irrigated-farming region of south-eastern

Turkey. During 2009, 84 cases were reported, and in 2010 not a single case was detected [138].

Antibacterial Resistance

High antibiotic resistance rates in Turkey have been linked to extensive antibiotic consumption [139,140]. In February 2003, the MoH issued a 'Budget Enforcement Document' that delineated the antibiotic prescription policy in the country. According to this regulation, extended-spectrum parenteral antibiotics can only be prescribed by the infectious diseases specialists. If an infectious disease specialist is not available, only an internist or a paediatrician can make the prescription. Any antibiotic usage that is not compliant with this regulation will not be reimbursed by the state [141]. Currently, there are few initial reports on the financial gains, along with decreasing nosocomial infection and resistance rates, related to the 2003 legislation [142,143].

Antibiotic resistance in community-acquired pathogens

Gram-positive bacteria. . Renal and cardiac sequelae of *Streptococcus pyogenes* infections continue to be significant health problems in Turkey, although their incidence rates have declined during the last decade [144–147]. The high frequency of these complications indicates that a lack of early diagnosis and treatment could be the main problem, rather than antimicrobial resistance, as low-level antibiotic resistance has been reported only against macrolides and clindamycin in this bacterium [148,149]. On the other hand, the rate of resistance to tetracycline was reported to be 18% in both of these studies.

Epidemiological data obtained before the introduction of the pneumococcal conjugate vaccine PCV7 indicated that 19F and 6B were the major serotypes recovered from invasive pneumococcal infections [150]. The data obtained before the new CLSI breakpoints were established showed that the rate of non-susceptibility to penicillin was as high as 35%, including a high-level resistance rate of 7%[139]. The reported resistance rates for other antibiotics included the following: ceftriaxone, 1%; levofloxacin, 2%; erythromycin, 18%; tetracycline, 19%; trimethoprim–sulphamethoxazole, 39%; chloramphenicol, 5%; and rifampicin, 2% [151]. According to the new criteria for penicillin susceptibility, intermediate resistance is 0–3% and high-level resistance is 0–0.5% for parenteral penicillin in non-meningeal strains, whereas up to one-third of meningitis isolates have been reported to be penicillin-resistant in large series [152–154].

Community-acquired *Staphylococcus aureus* infections are rarely reported, and do not seem to be a significant problem [155,156].

Gram-negative bacteria. . Antibiotic resistance does not seem be a problem in the management of invasive diseases caused by *Haemophilus influenzae*. The most problematic drug is trimethoprim–sulphamethoxazole, with one-third of all isolates being non-susceptible. Other antimicrobials, including β-lactams, quinolones, macrolides, and tetracyclines, seem to be effective in >90% of *H. influenzae* isolates in Turkey (Table 1) [157–164]. Although ampicillin resistance appears to mainly correlate with β-lactamase production, 0–2.8% (median, 1%) of all *H. influenzae* isolates were identified as β-lactamase-negative ampicillin-resistant strains [158,159,161,163].

High-level antimicrobial resistance was reported in *Escherichia coli* and *Klebsiella* spp. isolated from community-acquired urinary tract infections (CA-UTIs) (Table 2) [165–170]. High-rate of quinolone resistance seems to compromise the empirical usage of these antibiotics in CA-UTIs in Turkey [166–170]. Extended-spectrum β-lactamase (ESBL) production by community-acquired *E. coli* isolates was reported in 5–6% of uncomplicated CA-UTIs [169,171] and in 12–17% of complicated CA-UTIs [169,171]. The most frequent type of ESBL detected was CTX-M-15 [171–173].

ESBL production was also reported in community-acquired isolates of non-typhoidal *Salmonella* strains [174–176]. In one study, 29% of *Salmonella typhimurium* isolates were found to produce ESBLs, and those strains commonly co-produced resistance mechanisms for trimethoprim–sulphamethoxazole and aminoglycosides [177]. Multiresistance patterns were frequently detected in *Shigella flexneri* and *S. typhimurium* [176]. In general, the rate of quinolone resistance was low. In a recent analysis, among 620 clinical *Salmonella* isolates (18 *Salmonella typhi* and 602 various non-typhoidal *Salmonella*), only one *Salmonella enteritidis* isolate

TABLE 1. Rates of antibiotic resistance in *Haemophilus influenzae* isolates from invasive infections in Turkey[a] [157–164]

AMP	A-CL	Amp-S	CEC	CTX	CIP	TET	SXT	CLA	CLR	AZT	BLa
3–13 (6)	0–0 (0)	0.5–1 (0.7)	0–2 (0.7)	0–2 (0)	0–3.7 (0)	1–9 (3)	23–32 (26)	2–6 (2)	2–4 (3)	0–3 (0.5)	6–6 (6)

AMP, ampicillin; A-CL, amoxycillin–clavulanate; Amp-S, ampicillin–sulbactam; CEC, cefaclor; CTX, ceftriaxone; CIP, ciprofloxacin; TET, tetracycline; SXT, trimethoprim–sulphamethoxazole; CLA, chloramphenicol; CLR, clarithromycin; AZT, azithromycin. BLa, β-lactamase activity.
[a]Figures indicate % range of resistance (median) from published studies.

TABLE 2. Rates of antibiotic resistance in *Escherichia coli* and *Klebsiella* spp. isolates from community-acquired urinary tract infections in Turkey (adapted from [165])

	E. coli		*Klebsiella* spp.	
Antibiotics	**Range (%)**	**Median (%)**	**Range (%)**	**Median (%)**
Ampicillin	37–82	55	79–100	91
Ampicillin–sulbactam	15–57	45	42–60	51
Amoxycillin–clavulanate	10–40	26	6–64	41
Cefazolin	7–49	29	100	100
Cefuroxime	5–34	22	20–54	42
Ceftriaxone	2–30	7	5–36	27
Ceftazidime	2–10	6	5–25	25
Cefepime	2–13	10	0–13	8
Trimethoprim–sulphamethoxazole	12–63	40	16–48	35
Gentamicin	3–47	11	12–30	18
Amikacin	1–32	4	4–33	19
Fosfomycin	0–3	1	NK	NK
Nitrofurantoin	0–18	5	10–76	12
Ciprofloxacin	6–39	18	6–30	18
Piperacillin–tazobactam	3–17	10	15–35	23
Imipenem	0–3	1	0–5	0

NK, not known.

was found to be fully resistant to ciprofloxacin. However, 75 isolates (12.1%) had MICs between 0.125 and 0.5 mg/L, indicating decreased susceptibility [178].

Antibiotic resistance in the hospital setting

A nationwide nosocomial infection surveillance system (Turkish National Nosocomial Infections Surveillance Network (UHESA)) was initiated in 2006 by the MoH, and data from >1200 participating hospitals were collected by the use of standardized, hand-filled forms during 2006 and 2007, and through a web-based notification system from 2008 (http://rshm.gov.tr/enfeksiyon/dosya/rehber.pdf). The 2010 data from this network are detailed below. It should be noted that not all participating hospitals are large tertiary-care centres; many are small-scale regional hospitals. As the UHESA contains pooled data, these may differ from those reported in surveillance studies, which are usually performed in tertiary-care, teaching university hospitals.

High antimicrobial resistance rates in nosocomial isolates have contributed significantly to the increasing cost of nosocomial infections nationwide [179–181]. In Istanbul, the daily cost of antibiotic use for all hospital-acquired infections in a tertiary-care, training and research hospital, which pooled 553 patients on the cross-sectional study day, was 2137 USD [182].

Nosocomial Gram-positive bacteria. . *Staphylococcus aureus* remains a significant pathogen in Turkish hospitals. According to the 2010 UHESA data, the pooled mean methicillin resistance rate was 53% for this bacterium (http://www.rshm.gov.tr/enfeksiyon/dosya/analiz_2010.pdf). In a single-centre report, the rate of heterogeneously vancomycin-intermediate *Staphylococcus aureus* was 18% among 256 isolates [183]. However, a recent study reported a much lower rate [184]. On the other hand, vancomycin-intermediate *Staphylococcus aureus* was not detected in a study with 390 methicillin-resistant *Staphylococcus aureus* isolates [185].

The first vancomycin-resistant *Enterococcus faecium* (VRE) isolate in Turkey was obtained in 1997 [186]. At the turn of the new millennium, only sporadic cases of VRE had been reported [187]. Currently, VRE comprises 11% of all enterococcal nosocomial isolates, according to a UHESA report (http://rshm.gov.tr/enfeksiyon/dosya/rehber.pdf). So far, no linezolid and daptomycin resistance has been reported for *Staphylococcus aureus* and enterococcal isolates [188–191].

Nosocomial Gram-negative bacteria. . Common resistance problems in tertiary-care hospitals in Turkey include high rates of ESBL-producing enteric Gram-negative bacteria, and

TABLE 3. Rates (%) of antibiotic resistance among hospital-acquired Gram-negative bacteria in two large surveillance studies inTurkey [194,196]

	Escherichia coli		*Klebsiella pneumoniae*		*Pseudomonas aeruginosa*		*Acinetobacter* spp.	
	HITIT-2[a]	**MYSTIC[b]**	**HITIT-2**	**MYSTIC**	**HITIT-2**	**MYSTIC**	**HITIT-2**	**MYSTIC**
Ceftriaxone	ND	21	ND	41	ND	83	ND	90
Ceftazidime	31	15	36	55	26	54	87	83
Cefepime	27	14	31	22	24	56	76	71
Imipenem	0	1	3	15	30	52	55	33
Cefoperazone–sulbactam	10	ND	25	ND	30	ND	52	ND
Piperacillin–tazobactam	18	16	25	27	18	31	85	84
Ciprofloxacin	58	38	18	29	27	54	87	78
Amikacin	5.5	ND	12	ND	23	ND	63	ND
Tobramycin	ND	21	ND	55	ND	59	ND	44
ESBL-positive	42	15	41	40	ND	ND	ND	ND

ESBL, extended-spectrum β-lactamase; ND, not determined.
[a]The HITIT-2 study was performed in 13 tertiary-care centres in 2007.
[b]The MYSTIC study was performed in nine tertiary-care centres in 2000.

carbapenem resistance in *Acinetobacter* spp. and *Pseudomonas aeruginosa* [192–195]. The data from the 2010 UHESA report indicated the following overall resistance rates in participating hospitals: ESBL production in *Klebsiella pneumoniae* and *E. coli* was 45.8% and 40%, respectively; and carbapenem resistance in *Pseudomonas aeruginosa* and *Acinetobacter baumannii* was 31% and 69%, respectively. Two recent multicentre surveillance studies in large tertiary-care university centres reported resistance data for various Gram-negative nosocomial pathogens that are generally in accordance with the resistance patterns described above [194,196] (Table 3).

PER-1, a class A ESBL, has been found to be widespread in MDR *Pseudomonas aeruginosa* and *A. baumannii* [197–199]. Among the carbapenemase-producing bacteria, OXA-48 was first detected in a *K. pneumoniae* strain in Istanbul in 2001 [200]. Subsequently, the same enzyme was found in other *Enterobacteriaceae* [201]. Outbreaks have been described [202], and apparently these strains have disseminated to other European countries from Turkey [203]. There is preliminary evidence that OXA-48-producing isolates may be circulating in the community [201]. OXA-23, OXA-51 and OXA-58 were the most common carbapenemases in MDR *A. baumannii* isolates from several centres [204,205]. Metallo-β-lactamases, mainly of the VIM and IMP types, were also reported in *Enterobacteriaceae* and *Pseudomonas aeruginosa* [201]. The first NDM-1 in Turkey was recently reported in a *K. pneumoniae* strain isolated from a patient transferred from Iraq [206].

Conclusions

Although the implementation of intensive vaccination policies and preventive public health measures has caused a significant decline in the prevalence of many important infectious diseases, several community-acquired infections, including brucellosis, tularaemia, and CCHF, remain important health challenges in Turkey. The country has recently adopted a universal health insurance policy for all citizens. High rates of antimicrobial resistance in both outpatient and inpatients settings that compromise effective healthcare are prevalent, and contribute substantially to increased health expenditure by the state. In response to this, a nationwide resistance surveillance system has recently been initiated, and a restricted antimicrobial prescription policy has been implemented in all secondary-care and tertiary-care hospital settings. It is expected that these actions will have a favourable effect, leading to a decline in antibiotic resistance in the near future, but, currently, solid data supporting these expectations are lacking.

Transparency Declaration

The authors declare no conflict of interest related to this work.

References

1. Bruselloz (brucellosis), monthly epidemiology report. RHSM publications. 2004, 89–96.
2. Irmak H. Brusellozun kontrolü amaciyla saglik bakanliginca yapilan calişmalar [The activities of the ministry of health for the control of brucellosis]. *3. Türkiye Zoonotik Hastaliklar Simpozyumu Kitabi (3rd Turkey zoonotic diseases symposium book)*. Ankara: , 2010; 254–258.
3. Yuce A, Alp-Cavus S. Türkiye'de bruselloz: Genel bakis [Brucellosis in Turkey: an overview]. *Klimik Derg* 2006; 19: 87–97.
4. Yumuk Z, O'Callaghan D. Brucellosis in Turkey—an overview. *Int J Infect Dis* 2012; 16: e228–e235.
5. Ciftdogan DY, Bayram N, Vardar F. Brucellosis as a cause of fever of unknown origin in children admitted to a tertiary hospital in the Aegean region of Turkey. *Vector Borne Zoonotic Dis* 2011; 11: 1037–1040.
6. Citak EC, Arman D. *Brucella melitensis*: a rare cause of febrile neutropenia. *Pediatr Hematol Oncol* 2011; 28: 83–85.
7. Ozbalci D, Ergene U, Cetin CB. Brucellosis: a rare cause of febrile neutropenia in acute myeloblastic leukemia. *Med Oncol* 2011; 28: 255–257.
8. Sari I, Altuntas F, Hacioglu S *et al.* A multicenter retrospective study defining the clinical and hematological manifestations of brucellosis and pancytopenia in a large series: hematological malignancies, the unusual cause of pancytopenia in patients with brucellosis. *Am J Hematol* 2008; 83: 334–339.
9. Metan G, Sardan YC, Hascelik G. Brucellosis in all patients with febrile neutropenia. *Leuk Lymphoma* 2006; 47: 954–956.
10. Sayin-Kutlu S, Kutlu M, Ergonul O *et al.* Laboratory-acquired brucellosis in Turkey. *J Hosp Infect* 2012; 80: 326–330.
11. Erdem H, Ulu-Kilic A, Kilic S *et al.* Efficacy and tolerability of antibiotic combinations in neurobrucellosis: results of the Istanbul study. *Antimicrob Agents Chemother* 2012; 56: 1523–1528.
12. Buzgan T, Karahocagil MK, Irmak H *et al.* Clinical manifestations and complications in 1028 cases of brucellosis: a retrospective evaluation and review of the literature. *Int J Infect Dis* 2010; 14: e469–e478.
13. Calik S, Gokengin D. Human brucellosis in Turkey: a review of the literature between 1990 and 2009. *Turk J Med Sci* 2011; 41: 549–555.
14. Koruk ST, Erdem H, Koruk I *et al.* Management of brucella endocarditis: results of the Gulhane study. *Int J Antimicrob Agents* 2012; 40: 145–150.
15. Akalin HE, Unal S, Gur D, Baykal M. Ofloxacin in the treatment of brucellosis. *Eur J Clin Microbiol Infect Dis* 1990; 326–328.
16. Akova M, Uzun O, Akalin HE, Hayran M, Unal S, Gur D. Quinolones in treatment of human brucellosis: comparative trial of ofloxacin–rifampin versus doxycycline–rifampin. *Antimicrob Agents Chemother* 1993; 37: 1831–1834.
17. Kocagoz S, Akova M, Altun B, Gur D, Hascelik G. In vitro activities of new quinolones against *Brucella melitensis* isolated in a tertiary-care hospital in Turkey. *Clin Microbiol Infect* 2002; 8: 240–242.
18. Golem SB. Luleburgaz'da yeni bir tularemi epidemisi [A recent tularemia outbreak in Luleburgaz]. *Turk Hyg Exp Biol Bull* 1945; 5: 28–40.
19. Kilic S. A general overview of *Francisella tularensis* and the epidemiology of tularemia in Turkey. *Flora Derg* 2010; 15: 37–58.

20. Celebi G, Baruonu F, Ayoglu F *et al.* Tularemia, a reemerging disease in northwest Turkey: epidemiological investigation and evaluation of treatment responses. *Jpn J Infect Dis* 2006; 59: 229–234.
21. Gurcan S, Otkun MT, Otkun M, Arikan OK, Ozer B. An outbreak of tularemia in western Black Sea region of Turkey. *Yonsei Med J* 2004; 45: 17–22.
22. Yazgi H, Uyanik MH, Ertek M *et al.* Tularemia seroprevalence in the risky population living in both rural and urban areas of Erzurum. *Mikrobiyol Bul* 2011; 45: 67–74.
23. Gurcan S, Karabay O, Karadenizli A, Karagol C, Kantardjiev T, Ivanov IN. Characteristics of the Turkish isolates of *Francisella tularensis*. *Jpn J Infect Dis* 2008; 61: 223–225.
24. Ulu Kilic A, Kilic S, Sencan I *et al.* A water-borne tularemia outbreak caused by *Francisella tularensis* subspecies *holarctica* in central Anatolia region. *Mikrobiyol Bul* 2011; 45: 234–247.
25. Ozdemir D, Sencan I, Annakkaya AN *et al.* Comparison of the 2000 and 2005 outbreaks of tularemia in the Duzce region of Turkey. *Jpn J Infect Dis* 2007; 60: 51–52.
26. Leblebicioglu H, Esen S, Turan D *et al.* Outbreak of tularemia: a case-control study and environmental investigation in Turkey. *Int J Infect Dis* 2008; 12: 265–269.
27. Yesilyurt M, Kilic S, Celebi B *et al.* Antimicrobial susceptibilities of *Francisella tularensis* subsp. *holarctica* strains isolated from humans in the central Anatolia region of Turkey. *J Antimicrob Chemother* 2011; 66: 2588–2592.
28. Ciftci F, Tozkoparan E, Deniz O, Bozkanat E, Kibaroglu E, Demirci N. The incidence of tuberculosis in the armed forces: a good reflection of the whole population. *Int J Tuberc Lung Dis* 2004; 8: 965–968.
29. Tabak F, Mert A, Celik AD *et al.* Fever of unknown origin in Turkey. *Infection* 2003; 31: 417–420.
30. Kucukardali Y, Oncul O, Cavuslu S *et al.* The spectrum of diseases causing fever of unknown origin in Turkey: a multicenter study. *Int J Infect Dis* 2008; 12: 71–79.
31. Saltoglu N, Tasova Y, Midikli D, Aksu HS, Sanli A, Dundar IH. Fever of unknown origin in Turkey: evaluation of 87 cases during a nine-year-period of study. *J Infect* 2004; 48: 81–85.
32. Baylan O. Extensively drug resistant and extremely drug resistant tuberculosis forms after multi-drug resistant tuberculosis: new faces of the old disease. *Mikrobiyol Bul* 2011; 45: 181–195.
33. Tahaoglu K, Kizkin O, Karagoz T, Tor M, Partal M, Sadoglu T. High initial and acquired drug resistance in pulmonary tuberculosis in Turkey. *Tuber Lung Dis* 1994; 75: 324–328.
34. Kartaloglu Z, Bozkanat E, Ozturkeri H, Okutan O, Ilvan A. Primary antituberculosis drug resistance at Turkish military chest diseases hospital in Istanbul. *Med Princ Pract* 2002; 11: 202–205.
35. Kilicaslan Z, Albal H, Kiyan E, Aydemir N, Seber E. Drug resistance in pulmonary tuberculosis in Istanbul. *Eur J Clin Microbiol Infect Dis* 2002; 21: 763–764.
36. Durmaz R, Ozerol IH, Durmaz B, Gunal S, Senoglu A, Evliyaoglu E. Primary drug resistance and molecular epidemiology of *Mycobacterium tuberculosis* isolates from patients in a population with high tuberculosis incidence in Turkey. *Microb Drug Resist* 2003; 9: 361–366.
37. Anonymous. Report on fight against tuberculosis in Turkey. 24 March 2009. Ankara: Department of Fight Against Tuberculosis, Turkish Republic of Ministry of Health.
38. Ozkutuk N, Surucuoglu S, Gazi H, Coskun M, Ozkutuk A, Ozbakkaloglu B. Second-line drug susceptibilities of multidrug-resistant *Mycobacterium tuberculosis* isolates in Aegean region—Turkey. *Turk J Med Sci* 2008; 38: 245–250.
39. Kayali R, Coplu N, Ceyhan I, Ocak F, Citil BE, Esen B. The rates of resistance to second-line drugs in multidrug resistant *Mycobacterium tuberculosis* strains. *Mikrobiyol Bul* 2006; 40: 1–7.
40. Eroglu C, Esen S, Hokelek M *et al.* Menenjit ve ensefalit bulgulari ile karakterize bir lyme menenjiti olgusu [A case of lyme meningitis characterized with meningitis and encephalitis findings]. *Infeks Derg* 2002; 16: 225–228.
41. Polat E, Turhan V, Aslan M, Musellim B, Onem Y, Ertugrul B. First report of three culture confirmed human lyme cases in Turkey. *Mikrobiyol Bul* 2010; 44: 133–139.
42. Koc F, Bozdemir H, Pekoz T, Aksu HS, Ozcan S, Kurdak H. Lyme disease presenting as subacute transverse myelitis. *Acta Neurol Belg* 2009; 109: 326–329.
43. Bulut C, Tufan ZK, Altun S, Altinel E, Kinikli S, Demiroz AP. An overlooked disease of tick bites: Lyme disease. *Mikrobiyol Bul* 2009; 43: 487–492.
44. Demirci M, Yorgancigil B, Tahan V, Arda M. Isparta yöresinde kene isirigi oykusu olanlarda lyme hastaligi seropozitifligi [The lyme disease seropositivity in Isparta province in those with a history of tick-bite]. *Infeks Derg* 2001; 15: 17–20.
45. Sen E, Uchishima Y, Okamoto Y *et al.* Molecular detection of *Anaplasma phagocytophilum* and *Borrelia burgdorferi* in *Ixodes ricinus* ticks from Istanbul metropolitan area and rural Trakya (Thrace) region of north-western Turkey. *Ticks Tick Borne Dis* 2011; 2: 94–98.
46. Erdem H, Tetik A, Arun O, Besirbellioglu BA, Coskun O, Eyigun CP. War and infection in the pre-antibiotic era: the third Ottoman army in 1915. *Scand J Infect Dis* 2011; 43: 690–695.
47. Karatepe M. The role of Turkish physicians in the vaccination against typhus during the years of World War I. *Mikrobiyol Bul* 2008; 42: 301–313.
48. Payzin S, Akan E. Residual agglutinins against rickettsial agents in human sera from central and eastern Turkey and their relation to cardiovascular diseases. *Bull World Health Organ* 1966; 35: 111–117.
49. Raoult D. Introduction to rickettsioses, ehrlichioses, and anaplasmosis. In: Mandell GL, Bennett JE, Dolin R, eds. *Mandell, Douglas, and Bennett's principles and practice of infectious diseases*. Philadelphia, PA: Churchill Livingstone, 2010; 2495–2499.
50. Badiaga S, Brouqui P. Human louse-transmitted infectious diseases. *Clin Microbiol Infect* 2012; 18: 332–337.
51. Kuloglu F, Rolain JM, Fournier PE, Akata F, Tugrul M, Raoult D. First isolation of *Rickettsia conorii* from humans in the Trakya (European) region of Turkey. *Eur J Clin Microbiol Infect Dis* 2004; 23: 609–614.
52. Mert A, Ozaras R, Tabak F, Bilir M, Ozturk R. Mediterranean spotted fever: a review of fifteen cases. *J Dermatol* 2006; 33: 103–107.
53. Kuloglu F, Rolain JM, Celik AD, Akata F, Tugrul M, Raoult D. Prospective evaluation of rickettsioses in the Trakya (European) region of Turkey in 2005. *Clin Microbiol Infect* 2009; 15 (suppl 2): 220–221.
54. Oztoprak N, Celebi G, Aydemir H *et al.* Mediterranean spotted fever due to contact with dog-tick. *Mikrobiyol Bul* 2008; 42: 701–706.
55. Ozkan A, Ozkalemkas F, Ali R *et al.* Mediterranean spotted fever: presentation with pancytopenia. *Am J Hematol* 2006; 81: 646–647.
56. Erbay A, Kayaaslan B, Akinci E *et al.* HIv/AIDS olgularinin epidemiyolojik, klinik ve laboratuvar özelliklerinin degerlendirilmesi [Evaluation of epidemiological, clinical and laboratory features of HIV/AIDS cases]. *Flora Derg* 2009; 14: 36–42.
57. Afsar I, Gungor S, Sener AG, Yurtsever SG. The prevalence of HBV, HCV and HIV infections among blood donors in Izmir, Turkey. *Indian J Med Microbiol* 2008; 26: 288–289.
58. Coskun O, Gul C, Erdem H, Bedir O, Eyigun CP. Prevalence of HIV and syphilis among Turkish blood donors. *Ann Saudi Med* 2008; 28: 470.
59. Guzelant A, Kurtoglu MG, Kaya M, Kesli R, Baysal B. Kan vericilerinde ve bir ağız-diş sağliği merkezi çalişanlarinda Hepatit B, Hepatit C ve HIV seroprevalansi ile vericilerde risk faktörlerinin arastirilmasi [The seroprevalence of Hepatitis B, Hepatitis C and HIV in blood donors and workers in a dentistry center and risk factors for infection in blood donors]. *Infeks Derg* 2008; 22: 189–195.

60. Stanojevic M, Alexiev I, Beshkov D *et al.* HIV-1 molecular epidemiology in the Balkans—a melting pot for high genetic diversity. *AIDS Rev* 2012; 14: 28–36.
61. Tumer A. Dünyada ve Türkiye'de güncel verilerle HIV/AIDS [HIV/AIDS in Turkey and in the world according to current data]. *Turk HIV/AIDS Derg* 2006; 9: 99–103.
62. Hariri AG, Karadag F, Gokalp P, Essizoglu A. Risky sexual behavior among patients in Turkey with bipolar disorder, schizophrenia, and heroin addiction. *J Sex Med* 2011; 8: 2284–2291.
63. Avcikurt C, Koroglu O, Koroglu A, Avcikurt AS. HIV/AIDS awareness and attitudes of tour guides in Turkey. *Cult Health Sex* 2011; 13: 233–243.
64. Celikbas A, Ergonul O, Baykam N *et al.* Epidemiologic and clinical characteristics of HIV/AIDS patients in Turkey, where the prevalence is the lowest in the region. *J Int Assoc Physicians AIDS Care (Chic)* 2008; 7: 42–45.
65. Ergonul O. Crimean–Congo hemorrhagic fever virus: new outbreaks, new discoveries. *Curr Opin Virol* 2012; 2: 215–220.
66. Chumakov MP. A new tick-borne virus disease—Crimean hemorrhagic fever. In: Sokolov AA, Chumakov MP, Kolachev AA, eds. *Crimean hemorrhagic fever (acute infectious capillary toxicosis)*. Moscow: Izd Otd Primorskoi Armii, 1945; 13–45.
67. Simpson DI, Knight EM, Courtois G, Williams MC, Weinbren MP, Kibukamusoke JW. Congo virus: a hitherto undescribed virus occurring in Africa. I. Human isolations—clinical notes. *East Afr Med J* 1967; 44: 86–92.
68. Vorou RM. Crimean–Congo hemorrhagic fever in southeastern europe. *Int J Infect Dis* 2009; 13: 659–662.
69. Papa A, Dalla V, Papadimitriou E, Kartalis GN, Antoniadis A. Emergence of Crimean–Congo haemorrhagic fever in Greece. *Clin Microbiol Infect* 2010; 16: 843–847.
70. Tantawi HH, Al-Moslih MI, Al-Janabi NY *et al.* Crimean–Congo haemorrhagic fever virus in Iraq: isolation, identification and electron microscopy. *Acta Virol* 1980; 24: 464–467.
71. Chinikar S, Ghiasi SM, Moradi M *et al.* Geographical distribution and surveillance of Crimean–Congo hemorrhagic fever in Iran. *Vector Borne Zoonotic Dis* 2010; 10: 705–708.
72. Gozalan A, Akin L, Rolain JM *et al.* Epidemiological evaluation of a possible outbreak in and nearby Tokat province. *Mikrobiyol Bul* 2004; 38: 33–44.
73. Karti SS, Odabasi Z, Korten V *et al.* Crimean–Congo hemorrhagic fever in Turkey. *Emerg Infect Dis* 2004; 10: 1379–1384.
74. Bakir M, Ugurlu M, Dokuzoguz B, Bodur H, Tasyaran MA, Vahaboglu H. Crimean–Congo haemorrhagic fever outbreak in middle Anatolia: a multicentre study of clinical features and outcome measures. *J Med Microbiol* 2005; 54: 385–389.
75. Kader C, Erbay A, Aker S, Alper S. Kastamonu ili aile hekimlerinin Kırım-Kongo kanamalı atesi konusunda bilgi düzeylerinin degerlendirilmesi [Evaluation of the knowledge of family physicians regarding Crimean–Congo haemorrhagic fever in Kastamonu]. *Flora Derg* 2011; 16: 61–66.
76. Maltezou HC, Andonova L, Andraghetti R *et al.* Crimean–Congo hemorrhagic fever in Europe: current situation calls for preparedness. *Euro Surveill* 2010; 15: 19504.
77. Elaldi N. Kırım-Kongo kanamalı atesi'nin kan merkezi is yüküne etkisi [The effect of Crimean–Congo hemorrhagic fever on blood transfusion center workload]. *Cumhuriyet Tıp Derg* 2010; 32: 292–297.
78. Hekimoglu O, Ozer N, Ergunay K, Ozkul A. Species distribution and detection of Crimean Congo hemorrhagic fever virus (CCHFV) in field-collected ticks in Ankara province, central Anatolia, Turkey. *Exp Appl Acarol* 2012; 56: 75–84.
79. Koksal I, Yilmaz G, Iskender S *et al.* The first Crimean–Congo hemorrhagic fever case in the winter season from Turkey. *Intervirology* 2011; 54: 144–145.
80. Aksoy HZ, Yilmaz G, Aksoy F, Koksal I. Crimean–Congo haemorrhagic fever presenting as epididymo-orchitis. *J Clin Virol* 2010; 48: 282–284.
81. Yilmaz G, Koksal I, Topbas M, Yilmaz H, Aksoy F. The effectiveness of routine laboratory findings in determining disease severity in patients with Crimean–Congo hemorrhagic fever: severity prediction criteria. *J Clin Virol* 2010; 47: 361–365.
82. Kaya S, Yilmaz G, Ertunc B, Koksal I. Parotitis associated with Crimean Congo hemorrhagic fever virus. *J Clin Virol* 2012; 53: 159–161.
83. Engin A, Erdogan H, Ozec AV *et al.* Ocular findings in patients with Crimean–Congo hemorrhagic fever. *Am J Ophthalmol* 2009; 147: 634–638.
84. Engin A, Yilmaz MB, Elaldi N *et al.* Crimean–Congo hemorrhagic fever: does it involve the heart? *Int J Infect Dis* 2009; 13: 369–373.
85. Celikbas A, Ergonul O, Dokuzoguz B, Eren S, Baykam N, Polat-Duzgun A. Crimean Congo hemorrhagic fever infection simulating acute appendicitis. *J Infect* 2005; 50: 363–365.
86. Ergonul O, Celikbas A, Yildirim U *et al.* Pregnancy and Crimean–Congo haemorrhagic fever. *Clin Microbiol Infect* 2010; 16: 647–650.
87. Appannanavar SB, Mishra B. An update on Crimean Congo hemorrhagic fever. *J Glob Infect Dis* 2011; 3: 285–292.
88. Ergonul O, Celikbas A, Dokuzoguz B, Eren S, Baykam N, Esener H. Characteristics of patients with Crimean–Congo hemorrhagic fever in a recent outbreak in Turkey and impact of oral ribavirin therapy. *Clin Infect Dis* 2004; 39: 284–287.
89. Bodur H, Akinci E, Ascioglu S, Onguru P, Uyar Y. Subclinical infections with Crimean–Congo hemorrhagic fever virus, Turkey. *Emerg Infect Dis* 2012; 18: 640–642.
90. Ergonul O. Crimean–Congo haemorrhagic fever. *Lancet Infect Dis* 2006; 6: 203–214.
91. Ergonul O. Debate (see Elaldi N *et al.*, efficacy of oral ribavirin treatment in Crimean–Congo haemorrhagic fever: a quasi-experimental study from Turkey. *Journal of Infection* 2009; 58: 238–244). Biases and misinterpretation in the assessment of the efficacy of oral ribavirin in the treatment of Crimean–Congo hemorrhagic fever. *J Infect* 2009; 59: 284–286. Author reply 286–289.
92. Elaldi N, Bodur H, Ascioglu S *et al.* Efficacy of oral ribavirin treatment in Crimean–Congo haemorrhagic fever: a quasi-experimental study from Turkey. *J Infect* 2009; 58: 238–244.
93. Ozkurt Z, Kiki I, Erol S *et al.* Crimean–Congo hemorrhagic fever in eastern Turkey: clinical features, risk factors and efficacy of ribavirin therapy. *J Infect* 2006; 52: 207–215.
94. Cevik MA, Elaldi N, Akinci E *et al.* A preliminary study to evaluate the effect of intravenous ribavirin treatment on survival rates in Crimean–Congo hemorrhagic fever. *J Infect* 2008; 57: 350–351.
95. Heyman P, Ceianu CS, Christova I *et al.* A five-year perspective on the situation of haemorrhagic fever with renal syndrome and status of the hantavirus reservoirs in Europe, 2005–2010. *Euro Surveill* 2011; 16: 19961.
96. Lednicky JA. Hantaviruses. A short review. *Arch Pathol Lab Med* 2003; 127: 30–35.
97. Bi Z, Formenty PB, Roth CE. Hantavirus infection: a review and global update. *J Infect Dev Ctries* 2008; 2: 3–23.
98. Ertek M, Buzgan T. An outbreak caused by hantavirus in the Black Sea region of Turkey, January–May 2009. *Euro Surveill* 2009; 14: 19214.
99. Kaya S, Yilmaz G, Erensoy S, Yagci Caglayik D, Uyar Y, Koksal I. Hantavirus infection: two case reports from a province in the eastern Black Sea region, Turkey. *Mikrobiyol Bul* 2010; 44: 479–487.
100. Sariguzel N, Hofmann J, Canpolat AT *et al.* Dobrava hantavirus infection complicated by panhypopituitarism, Istanbul, Turkey, 2010. *Emerg Infect Dis* 2012; 18: 1180–1183.
101. Oncul O, Atalay Y, Onem Y *et al.* Hantavirus infection in Istanbul, Turkey. *Emerg Infect Dis* 2011; 17: 303–304.

102. Sarı T, Temuçin F, Kaçar M, Oral B, Tülek N. Renal sendromla seyreden hantavirus enfeksiyonu; olgu sunumu [A hantavirus infection with renal syndrome: case presentation]. In: *Klimik Congress Book*. Antalya, 322, 2011; 18–13. Please reformat this reference in journal style, and query author for editors, publisher, the meaning of '322', and the meaning of '18-13'.In: *Klimik Congress Book*. Antalya, 322, 2011; 18–13..
103. Laakkonen J, Kallio-Kokko H, Oktem MA *et al.* Serological survey for viral pathogens in Turkish rodents. *J Wildl Dis* 2006; 42: 672–676.
104. Gubler DJ. The continuing spread of West Nile virus in the western hemisphere. *Clin Infect Dis* 2007; 45: 1039–1046.
105. Meco O. West Nile arbovirus antibodies with hemagglutination inhibition (HI) in residents of southeast Anatolia. *Mikrobiyol Bul* 1977; 11: 3–17.
106. Ergunay K, Ozkul A. Confirmation of West Nile virus seroreactivity in central nervous system infections of unknown etiology from Ankara province, central anatolia, Turkey. *Mikrobiyol Bul* 2011; 45: 381–383.
107. Tapisiz A, Emiralioglu N, Vural O *et al.* The first report of West Nile virus infection in a child from Turkey. *Turk J Pediatr* 2011; 53: 317–319.
108. Arpaci F, Cetin T, Kubar A. West Nile virus infection in a patient with acute graft-versus-host disease. *Haematologica* 2009; 94: 687.
109. Kocak-Tufan Z, Bulut C. Batı Nil virüsü Infeksiyonları [West Nile virus infections]. *Flora Derg* 2011; 16: 1–9.
110. Kalaycioglu H, Korukluoglu G, Ozkul A *et al.* Emergence of West Nile virus infections in humans in Turkey, 2010 to 2011. *Euro Surveill* 2012; 17: 20182.
111. Hizel K, Yenicesu I, Erdal B *et al.* Investigation of West Nile virus seroprevalence in healthy blood donors. *Mikrobiyol Bul* 2010; 44: 425–430.
112. Ayturan S, Aydogan S, Ergunay K, Ozcebe OI, Us D. Investigation of West Nile virus seroprevalence in Hacettepe University hospital blood donors and confirmation of the positive results by plaque reduction neutralization test. *Mikrobiyol Bul* 2011; 45: 113–124.
113. Ozer N, Ergunay K, Simsek F *et al.* West Nile virus studies in the Sanliurfa province of Turkey. *J Vector Ecol* 2007; 32: 202–206.
114. Toy M, Onder FO, Wormann T *et al.* Age- and region-specific hepatitis B prevalence in Turkey estimated using generalized linear mixed models: a systematic review. *BMC Infect Dis* 2011; 11: 337.
115. Tunc N, Eraydin H, Cetinkaya E, Oduncu MK, Toy S. Siirt Devlet Hastanesi'ne başvuran hastalarda HbsAg, anti-Hbs, anti-HCV ve anti-HIV seroprevalansı [HbsAg, anti-Hbs, anti-HCV and anti-HIV seroprevalence of the patients admitted to Siirt Public Hospital]. *Viral Hepatit Derg* 2011; 17: 7–11.
116. Demiraslan H, Aksoz S. Adıyaman ili kan vericilerindeki HbsAg ve anti-HCV sıklığının değerlendirilmesi [The evaluation of HbsAg and anti-HCV seroprevalences of blood donors in Adiyaman]. *Viral Hepatit Derg* 2008; 13: 23–26.
117. Kose S, Ece G, Gozaydin A, Ergin O. İzmir Sosyal Hizmetler ve Cocuk Esirgeme Kurumuna bağlı yetiştirme yurtlarında yaşayan çocuklarda Hepatit B ve Hepatit C seroprevalansı [Seroprevalence of hepatitis B and hepatitis C in children living in Izmir Social Services and Child Protection Institution]. *Viral Hepatit Derg* 2010; 16: 64–68.
118. Altuntas-Aydin O, Kumbasar-Karaosmanoglu H, Kokrek A, Isik ME, Nazlican Ö. İstanbul bölgesi kan donörlerinde HbsAg, anti-HCV ve anti-HIV seroprevalansı [Seroprevalences of HbsAg, anti-HCV and anti-HIV among blood donors in Istanbul]. *Viral Hepatit Derg* 2009; 14: 69–73.
119. Oksuz S, Yildirim M, Ozaydin C, Sahin I, Sencan I. Düzce bölgesi kan vericilerinde HbsAg, anti-HCV ve anti-HIV seroprevalansı [Seropositivity rates of HbsAg, anti-HCV and anti-HIV in blood donors in Düzce area]. *Viral Hepatit Derg* 2008; 13: 27–30.
120. Sahin D, Sahin I, Sozer F, Onder F. Kırklareli Devlet Hastanesi kan merkezine başvuran donörlerde HBV, HCV ve HIV seroprevalansı: Retrospektif bir calisma [HBV, HCV and HIV seroprevalance in applicant donors of Kirklareli State Hospital blood center: a retrospective study]. *Viral Hepatit Derg* 2008; 13: 31–35.
121. Kumbasar Karaosmanoglu H, Altuntas Aydin O, Sandikci S, Yamanlar ER, Nazlican O. Seroprevalence of hepatitis B: do blood donors represent the general population? *J Infect Dev Ctries* 2012; 6: 181–183.
122. Atalay MA, Gokahmetoglu S, Aygen B. Genotypes of hepatitis B virus in central Anatolia, Kayseri, Turkey. *Saudi Med J* 2011; 32: 360–363.
123. Kulah C, Cirak MY. Determination of hepatitis B virus genotypes by DNA sequence analysis in patients from Ankara, Turkey. *Mikrobiyol Bul* 2010; 44: 245–253.
124. Tosun S, Deveci S, Kaplan Y, Kasirga E. Should a booster dose be administered in children after mass immunization for hepatitis B? *Hepat Mon* 2011; 11: 440–444.
125. Degertekin H, Yalcin K, Yakut M, Yurdaydin C. Seropositivity for Delta hepatitis in patients with chronic hepatitis B and liver cirrhosis in Turkey: a meta-analysis. *Liver Int* 2008; 28: 494–498.
126. Abbas Z, Jafri W, Raza S. Hepatitis D: scenario in the Asia-Pacific region. *World J Gastroenterol* 2010; 16: 554–562.
127. Demirdal T, Demirturk N, Asci Z. Afyonkarahisar ilinde Hepatit Delta virusu seroprevalansı [Seroprevalence of hepatitis delta virus in Afyonkarahisar]. *Viral Hepatit Derg* 2009; 14: 104–107.
128. Turkdogan MK, Bozkurt H, Uygan I *et al.* Chronic hepatitis delta virus infection in Van region of eastern Turkey. *Turk J Gastroenterol* 2005; 16: 17–20.
129. Izmirli S, Gozde-Celik D, Gungordu Z *et al.* Hepatit Delta virüsü infeksiyonu seroprevalansı:Retrospektif temelli seroepidemiyolojik bir değerlendirme [Seroprevalence of hepatitis delta virus infection: retrospective-based seroepidemiologic assessment]. *Flora Derg* 2011; 16: 120–126.
130. Bahcecioglu IH, Aygun C, Gozel N, Poyrazoglu OK, Bulut Y, Yalniz M. Prevalence of hepatitis delta virus (HDV) infection in chronic hepatitis B patients in eastern Turkey: still a serious problem to consider. *J Viral Hepat* 2011; 18: 518–524.
131. Celik I, Karatayli E, Cevik E *et al.* Complete genome sequences and phylogenetic analysis of hepatitis delta viruses isolated from nine Turkish patients. *Arch Virol* 2011; 156: 2215–2220.
132. Le Gal F, Badur S, Hawajri NA *et al.* Current hepatitis delta virus type 1 (HDV1) infections in central and eastern Turkey indicate a wide genetic diversity that is probably linked to different HDV-1 origins. *Arch Virol* 2012; 157: 647–659.
133. Coskun O, Erdem H, Besirbellioglu BA, Eyigun CP. Distribution of hepatitis C virus infection in the male Turkish population. *Int J Infect Dis* 2006; 10: 481.
134. Keskin F, Ciftci S, Turkoglu S, Badur S. Transmission routes of chronic hepatitis C and their relation to HCV genotypes. *Turk J Gastroenterol* 2010; 21: 396–400.
135. Aktas E, Ogedey ED, Kulah C, Begendik Comert F. Hepatitis C virus genotypes in a province of western Black-Sea region, Turkey. *Mikrobiyol Bul* 2010; 44: 647–650.
136. Kuckoztas MF, Ozgunes N, Yazici S. Investigation of the relationship between hepatitis C virus (HCV) genotypes with HCV-RNA and alanine aminotransferase levels in chronic hepatitis C patients. *Mikrobiyol Bul* 2010; 44: 111–115.
137. Karahocagil MK, Baran AI, Yaman G *et al.* Case report: two *Plasmodium vivax* malaria cases in the Van province. *Turkiye Parazitol Derg* 2009; 33: 172–173.
138. Ozbilgin A, Topluoglu S, Es S, Islek E, Mollahaliloglu S, Erkoc Y. Malaria in Turkey: successful control and strategies for achieving elimination. *Acta Trop* 2011; 120: 15–23.
139. Erdem H, Pahsa A. Antibiotic resistance in pathogenic *Streptococcus pneumoniae* isolates in Turkey. *J Chemother* 2005; 17: 25–30.

140. Ozgenc O, Genc VE, Ari AA *et al.* Evaluation of the therapeutic use of antibiotics in Aegean region hospitals of Turkey: a multicentric study. *Indian J Med Microbiol* 2011; 29: 124–129.
141. Erdem H, Kurtaran B, Arun O *et al.* The place and the efficacy of infectious disease consultations in the hospitals. *Infect Dis Clin Pract* 2012; 20: 131–136.
142. Arda B, Sipahi OR, Yamazhan T *et al.* Short-term effect of antibiotic control policy on the usage patterns and cost of antimicrobials, mortality, nosocomial infection rates and antibacterial resistance. *J Infect* 2007; 55: 41–48.
143. Kurt H, Karabay O, Birengel S, Memikoglu O, Yilmaz Bozkurt G, Yalci A. Effects of legal antibiotic restrictions on consumption of broad-spectrum beta-lactam antibiotics, glycopeptides and amphotericin B. *Chemotherapy* 2010; 56: 359–363.
144. Oner A, Demircin G, Bulbul M. Post-streptococcal acute glomerulonephritis in Turkey. *Acta Paediatr* 1995; 84: 817–819.
145. Orun UA, Ceylan O, Bilici M *et al.* Acute rheumatic fever in the central Anatolia region of Turkey: a 30-year experience in a single center. *Eur J Pediatr* 2012; 171: 361–368.
146. Karademir S, Demirceken F, Atalay S, Demircin G, Sipahi T, Tezic T. Acute rheumatic fever in children in the Ankara area in 1990–1992 and comparison with a previous study in 1980–1989. *Acta Paediatr* 1994; 83: 862–865.
147. Ozer S, Hallioglu O, Ozkutlu S, Celiker A, Alehan D, Karagoz T. Childhood acute rheumatic fever in Ankara, Turkey. *Turk J Pediatr* 2005; 47: 120–124.
148. Dundar D, Sayan M, Tamer GS. Macrolide and tetracycline resistance and emm type distribution of *Streptococcus pyogenes* isolates recovered from Turkish patients. *Microb Drug Resist* 2010; 16: 279–284.
149. Bayramoglu G, Topkaya AE, Balikci A, Aydin F. Serotypes and antimicrobial susceptibilities of invasive group A streptococci identified in eastern Black Sea region of Turkey. *Mikrobiyol Bul* 2011; 45: 446–453.
150. Erdem H, Sener B. Pneumococcal seroepidemiology in Turkey: implications for vaccine coverage. *Vaccine* 2008; 26: 1271–1273.
151. Erdem H. An update on invasive pneumococcal antibiotic resistance in Turkey, 2008. *J Chemother* 2008; 20: 697–701.
152. Telli M, Eyigör M, Gültekin B, Aydin N. Streptococcus pneumoniae'nin menenjit disi klinik izolatlarinda penisilin direnci ile serotip iliskisi ve bazı antibiyotiklere direnc [Penicillin resistance and serotype relationship in *Streptococcus pneumoniae* clinical isolates and resistance to some antibiotics]. *Ankem Derg* 2010; 24: 55–60.
153. Dogan O, Gulmez D, Hascelik G. Effect of new breakpoints proposed by clinical and laboratory standards institute in 2008 for evaluating penicillin resistance of *Streptococcus pneumoniae* in a Turkish university hospital. *Microb Drug Resist* 2010; 16: 39–41.
154. Pehlivanoğlu F, Kart-Yaşar K, Şengöz G. Beyin omurilik sıvısından izole edilen mikroorganizmalar ve antibiyotik duyarlılıkları [Microorganisms isolated from cerebrospinal fluid and their antibiotic resistance]. *Ankem Derg* 2011; 25: 1–5.
155. Ulug M, Ayaz C, Celen MK. A case report and literature review: osteomyelitis caused by community-associated methicillin resistant *Staphylococcus aureus*. *J Infect Dev Ctries* 2011; 5: 896–900.
156. Kara A, Tezer H, Devrim I *et al.* Primary sternal osteomyelitis in a healthy child due to community-acquired methicillin-resistant *Staphylococcus aureus* and literature review. *Scand J Infect Dis* 2007; 39: 469–472.
157. Ilki A, Sagiroglu P, Elgormus N, Soyletir G. Trends in antibiotic susceptibility patterns of *Streptococcus pneumoniae* and *Haemophilus influenzae* isolates: four years follow up. *Mikrobiyol Bul* 2010; 44: 169–175.
158. Gonullu N, Catal F, Kucukbasmaci O, Ozdemir S, Torun MM, Berkiten R. Comparison of in vitro activities of tigecycline with other antimicrobial agents against *Streptococcus pneumoniae*, *Haemophilus influenzae* and *Moraxella catarrhalis* in two university hospitals in Istanbul, Turkey. *Chemotherapy* 2009; 55: 161–167.
159. Torun MM, Namal N, Demirci M, Bahar H. Nasopharyngeal carriage and antibiotic resistance of *Haemophilus influenzae*, *Streptococcus pneumoniae* and *Moraxella catarrhalis* in healthy school children in Turkey. *Indian J Med Microbiol* 2009; 27: 86–88.
160. Uncu H, Colakoglu S, Turunc T, Demiroglu YZ, Arslan H. In vitro resistance rates of *Streptococcus pneumoniae* and *Haemophilus influenzae* clinical isolates to the antibiotics used in therapy. *Mikrobiyol Bul* 2007; 41: 441–446.
161. Sener B, Tunckanat F, Ulusoy S *et al.* A survey of antibiotic resistance in *Streptococcus pneumoniae* and *Haemophilus influenzae* in Turkey, 2004–2005. *J Antimicrob Chemother* 2007; 60: 587–593.
162. Torun MM, Namal N, Demirci M, Bahar H, Kocazeybek B. Pharyngeal carriage and antimicrobial resistance of *Haemophilus influenzae* in non-type-B-vaccinated healthy children attending day care centers in Turkey. *Chemotherapy* 2007; 53: 114–117.
163. Altun B, Gur D. Hacettepe Üniversitesi Çocuk Hastanesi'nde klinik örneklerden izole edilen Haemophilus influenzae suşlarının antibiyotiklere direnç durumu (2002–2007) [Antimicrobial resistance in *Haemophilus influenzae* strains isolated from various clinical samples in Hacettepe University Children's Hospital (2002–2007)]. *Cocuk Enf Derg* 2008; 2: 50–54.
164. Özkul H, Alpay-Özbek Ö, Çoban H, Gülay Z. Dokuz Eylül Üniversitesi Hastanesinde 2003–2006 yillarinda uretilen Haemophilus influenzae suslarinin antibiyotik duyarliliklari. Ankem Derg 2007; 21: 86–90.
165. Coskun O, Erdem H, Avci A. Management of community-acquired acute bacterial cystitis in Turkey. *Turk J Med Sci* 2011; 41: 149–157.
166. Ceran N, Mert D, Kocdogan FY *et al.* A randomized comparative study of single-dose fosfomycin and 5-day ciprofloxacin in female patients with uncomplicated lower urinary tract infections. *J Infect Chemother* 2010; 16: 424–430.
167. Aypak C, Altunsoy A, Duzgun N. Empiric antibiotic therapy in acute uncomplicated urinary tract infections and fluoroquinolone resistance: a prospective observational study. *Ann Clin Microbiol Antimicrob* 2009; 8: 27.
168. Guneysel O, Onur O, Erdede M, Denizbasi A. Trimethoprim/sulfamethoxazole resistance in urinary tract infections. *J Emerg Med* 2009; 36: 338–341.
169. Arslan H, Azap ÖK, Ergönül Ö, Timurkaynak F. Risk factors for ciprofloxacin resistance among *Escherichia coli* strains isolated from community-acquired urinary tract infections in Turkey. *J Antimicrob Chemother* 2005; 56: 914–918.
170. Karaca Y, Coplu N, Gozalan A, Oncul O, Citil BE, Esen B. Co-trimoxazole and quinolone resistance in *Escherichia coli* isolated from urinary tract infections over the last 10 years. *Int J Antimicrob Agents* 2005; 26: 75–77.
171. Azap OK, Arslan H, Serefhanoglu K *et al.* Risk factors for extended-spectrum β-lactamase positivity in uropathogenic *Escherichia coli* isolated from community-acquired urinary tract infections. *Clin Microbiol Infect* 2010; 16: 147–151.
172. Celik AD, Yulugkural Z, Kuloglu F *et al.* CTX-M type extended spectrum beta-lactamases in *Escherichia coli* isolates from community acquired upper urinary tract infections at a university in the European part of Turkey. *J Microbiol Immunol Infect* 2010; 43: 163–167.
173. Yumuk Z, Afacan G, Nicolas-Chanoine MH, Sotto A, Lavigne JP. Turkey: a further country concerned by community-acquired *Escherichia coli* clone o25-st131 producing CTX-M-15. *J Antimicrob Chemother* 2008; 62: 284–288.
174. Bahar G, Mert A, Catania MR, Koncan R, Benvenuti C, Mazzariol A. A strain of *Salmonella enterica* serovar *virchow* isolated in Turkey and carrying a CTX-M-3 extended-spectrum beta-lactamase. *J Chemother* 2006; 18: 307–310.

175. Budak F, Nordmann P, Girlich D, Gur D. Characterization of extended-spectrum beta-lactamase-producing *Salmonella* isolates in a children's hospital in Ankara—first report of SHV-2a and SHV-9 in *Salmonella* spp. from Turkey. *Turk J Pediatr* 2009; 51: 28–34.
176. Kilic D, Tulek N, Tuncer G, Doganci L, Willke A. Antimicrobial susceptibilities and ESBL production rates of *Salmonella* and *Shigella* strains in Turkey. *Clin Microbiol Infect* 2001; 7: 341–342.
177. Otkun M, Erdem B, Akata F *et al.* Antibiotic resistance patterns and plasmid profiles of *Salmonella typhimurium* isolates in Turkey. *Eur J Clin Microbiol Infect Dis* 2001; 20: 206–209.
178. Ercis S, Erdem B, Hascelik G, Gur D. Nalidixic acid resistance in *Salmonella* strains with decreased susceptibility to ciprofloxacin isolated from humans in Turkey. *Jpn J Infect Dis* 2006; 59: 117–119.
179. Yalcin AN, Hayran M, Unal S. Economic analysis of nosocomial infections in a Turkish university hospital. *J Chemother* 1997; 9: 411–414.
180. Khan MM, Celik Y. Cost of nosocomial infection in Turkey: an estimate based on the university hospital data. *Health Serv Manage Res* 2001; 14: 49–54.
181. Inan D, Saba R, Gunseren F *et al.* Daily antibiotic cost of nosocomial infections in a Turkish university hospital. *BMC Infect Dis* 2005; 5: 5.
182. Inan A, Dagli O, Şenbayrak-Akçay S, Öztürk-Engin D, Karagül E, Çelik-Özyürek S. Antibiotic use and cost in a teaching hospital in Istanbul. *J Microbiol Infect Dis* 2011; 1: 128–133.
183. Sancak B, Ercis S, Menemenlioglu D, Colakoglu S, Hascelik G. Methicillin-resistant *Staphylococcus aureus* heterogeneously resistant to vancomycin in a Turkish university hospital. *J Antimicrob Chemother* 2005; 56: 519–523.
184. Kuscu F, Ozturk DB, Gurbuz Y, Tutuncu EE, Sencan I, Gul S. Investigation of reduced vancomycin susceptibility in methicillin-resistant staphylococci. *Mikrobiyol Bul* 2011; 45: 248–257.
185. Aktas E, Mengeloglu FZ, Kulah C, Comert FB. Evaluation of reduced susceptibility to vancomycin among MRSA strains isolated from clinical specimens. *Mikrobiyol Bul* 2010; 44: 339–341.
186. Vural T, Sekercioglu AO, Ogunc D *et al.* Vankomisine dirençli *Enterococcus faecium* suşu [Vancomycin-resistant *Enterococcus faecium* strain]. *Ankem Derg* 1999; 13: 1–4.
187. Erdem H, Oncul O. A review of the current place of glycopeptides in Turkish medical practice. *Curr Ther Res Clin Exp* 2007; 68: 49–66.
188. Yucel N, Citak S, Bayhun S. Antimicrobial resistance profile of *Staphylococcus aureus* isolated from clinical samples and foods of animal origin. *Foodborne Pathog Dis* 2011; 8: 427–431.
189. Efe S, Sinirtas M, Ozakin C. In vitro susceptibility to linezolid in methicillin-resistant *Staphylococcus aureus* and vancomycin-resistant enterococcus strains. *Mikrobiyol Bul* 2009; 43: 639–643.
190. Tunger A, Aydemir S, Uluer S, Cilli F. In vitro activity of linezolid & quinupristin/dalfopristin against gram-positive cocci. *Indian J Med Res* 2004; 120: 546–552.
191. Pelitli TS, Cesur S, Kinikli S, Irmak H, Demiroz AP, Karakoc E. Evaluation of vancomycin, teicoplanin, linezolide and tigecycline susceptibilities of nosocomial methicillin-resistant *Staphylococcus* strains by e-test. *Mikrobiyol Bul* 2011; 45: 758–761.
192. Eser OK, Ergin A, Tunckanat F, Hascelik G. In vitro activity of tigecycline as a therapeutic option against multidrug-resistant *Acinetobacter* spp. *New Microbiol* 2008; 31: 535–542.
193. Bayramoglu G, Kaya S, Besli Y *et al.* Molecular epidemiology and the clinical significance of *Acinetobacter baumannii* complex isolated from cerebrospinal fluid in neurosurgical intensive care unit patients. *Infection* 2011; 40: 163–172.
194. Eraksoy H, Basustaoglu A, Korten V *et al.* Susceptibility of bacterial isolates from Turkey—a report from the meropenem yearly susceptibility test information collection (MYSTIC) program. *J Chemother* 2007; 19: 650–657.
195. Souli M, Galani I, Giamarellou H. Emergence of extensively drug-resistant and pandrug-resistant gram-negative bacilli in Europe. *Euro Surveill* 2008; 13: 19045.
196. Gur D, Hascelik G, Aydin N *et al.* Antimicrobial resistance in gram-negative hospital isolates: results of the Turkish HITIT-2 surveillance study of 2007. *J Chemother* 2009; 21: 383–389.
197. Vahaboglu H, Coskunkan F, Tansel O *et al.* Clinical importance of extended-spectrum beta-lactamase (PER-1-type)-producing *Acinetobacter* spp. and *Pseudomonas aeruginosa* strains. *J Med Microbiol* 2001; 50: 642–645.
198. Kolayli F, Gacar G, Karadenizli A, Sanic A, Vahaboglu H. PER-1 is still widespread in Turkish hospitals among *Pseudomonas aeruginosa* and *Acinetobacter* spp. *FEMS Microbiol Lett* 2005; 249: 241–245.
199. Vahaboglu H, Ozturk R, Aygun G *et al.* Widespread detection of PER-1-type extended-spectrum beta-lactamases among nosocomial *Acinetobacter* and *Pseudomonas aeruginosa* isolates in Turkey: a nationwide multicenter study. *Antimicrob Agents Chemother* 1997; 41: 2265–2269.
200. Poirel L, Heritier C, Tolun V, Nordmann P. Emergence of oxacillinase-mediated resistance to imipenem in *Klebsiella pneumoniae*. *Antimicrob Agents Chemother* 2004; 48: 15–22.
201. Canton R, Akova M, Carmeli Y *et al.* Rapid evolution and spread of carbapenemases among *Enterobacteriaceae* in Europe. *Clin Microbiol Infect* 2012; 18: 413–431.
202. Carrer A, Poirel L, Yilmaz M *et al.* Spread of OXA-48-encoding plasmid in Turkey and beyond. *Antimicrob Agents Chemother* 2010; 54: 1369–1373.
203. Levast M, Poirel L, Carrer A *et al.* Transfer of OXA-48-positive carbapenem-resistant *Klebsiella pneumoniae* from Turkey to France. *J Antimicrob Chemother* 2011; 66: 944–945.
204. Meric M, Kasap M, Gacar G *et al.* Emergence and spread of carbapenem-resistant *Acinetobacter baumannii* in a tertiary care hospital in Turkey. *FEMS Microbiol Lett* 2008; 282: 214–218.
205. Vahaboglu H, Budak F, Kasap M *et al.* High prevalence of OXA-51-type class D beta-lactamases among ceftazidime-resistant clinical isolates of *Acinetobacter* spp.: co-existence with OXA-58 in multiple centres. *J Antimicrob Chemother* 2006; 58: 537–542.
206. Poirel L, Ozdamar M, Ocampo-Sosa AA, Turkoglu S, Ozer UG, Nordmann P. NDM-1-producing *Klebsiella pneumoniae* now in Turkey. *Antimicrob Agents Chemother* 2012; 56: 2784–2785.