Hospital-acquired pneumonia (HAP)

Pneumonia is one of the most common types of hospital-acquired infection[1–3]

Hospital-acquired pneumonia (HAP)

- Occurs ≥48 hours after hospital admission[1]
- Contributes to 26% of all hospital-acquired infections in Europe[2]
- Is a leading cause of death in hospitals and ICUs[2]

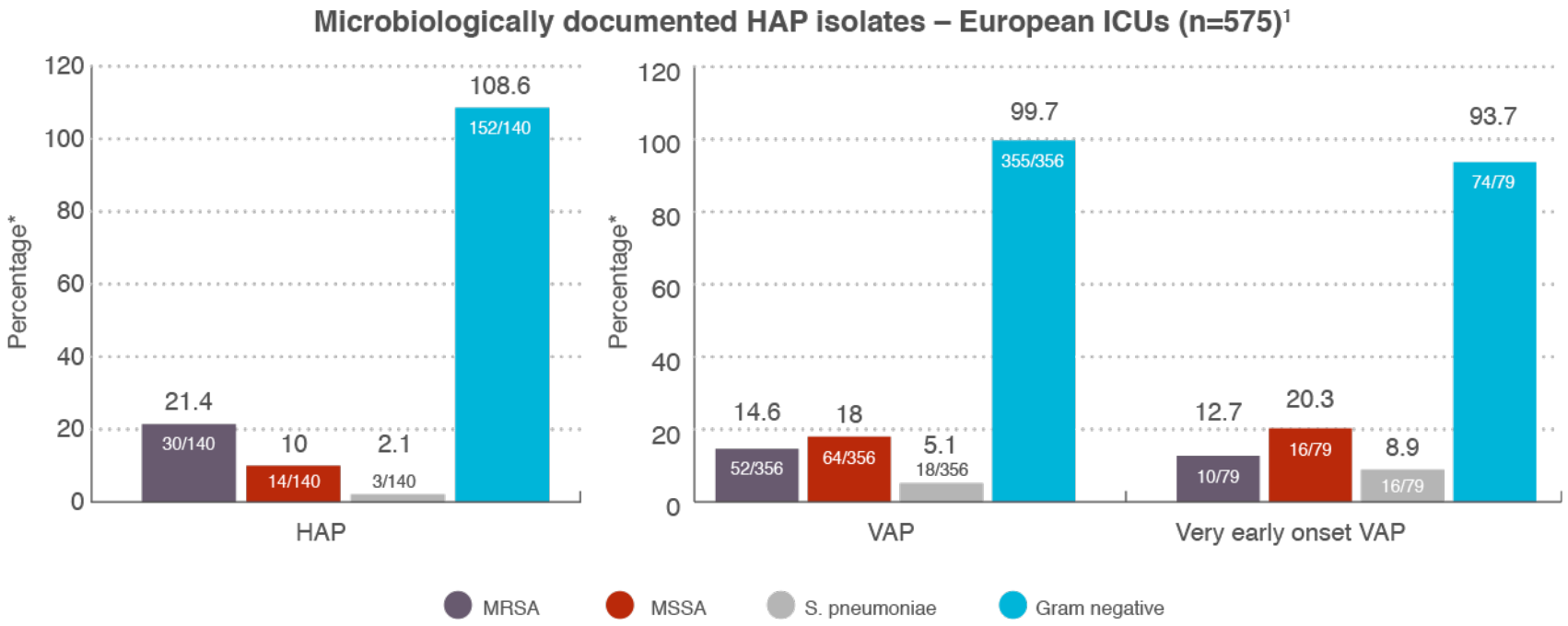
Ventilator-associated pneumonia (VAP)

- A subset of HAP[1]
- Occurs ≥48 hours after endotracheal intubation[1]
- Reported in 29% of ventilated patients with ARDs[4]
  - Median time from diagnosis to death, 8.5 days[4]

ICU, intensive care unit; ARDS, acute respiratory distress syndrome

1. American Thoracic Society, Infectious Diseases Society of America. *Am J Respir Crit Care Med* 2005; **171**: 388–416.
2. Zarb P *et al. Euro Surveill* 2012; **17**(46): 20316. **3.** Rubinstein E *et al. Clin Infect Dis* 2011; **52**(1): 31–40. **4.** Forel J-M *et al. Critical Care* 2012, 16: R65.

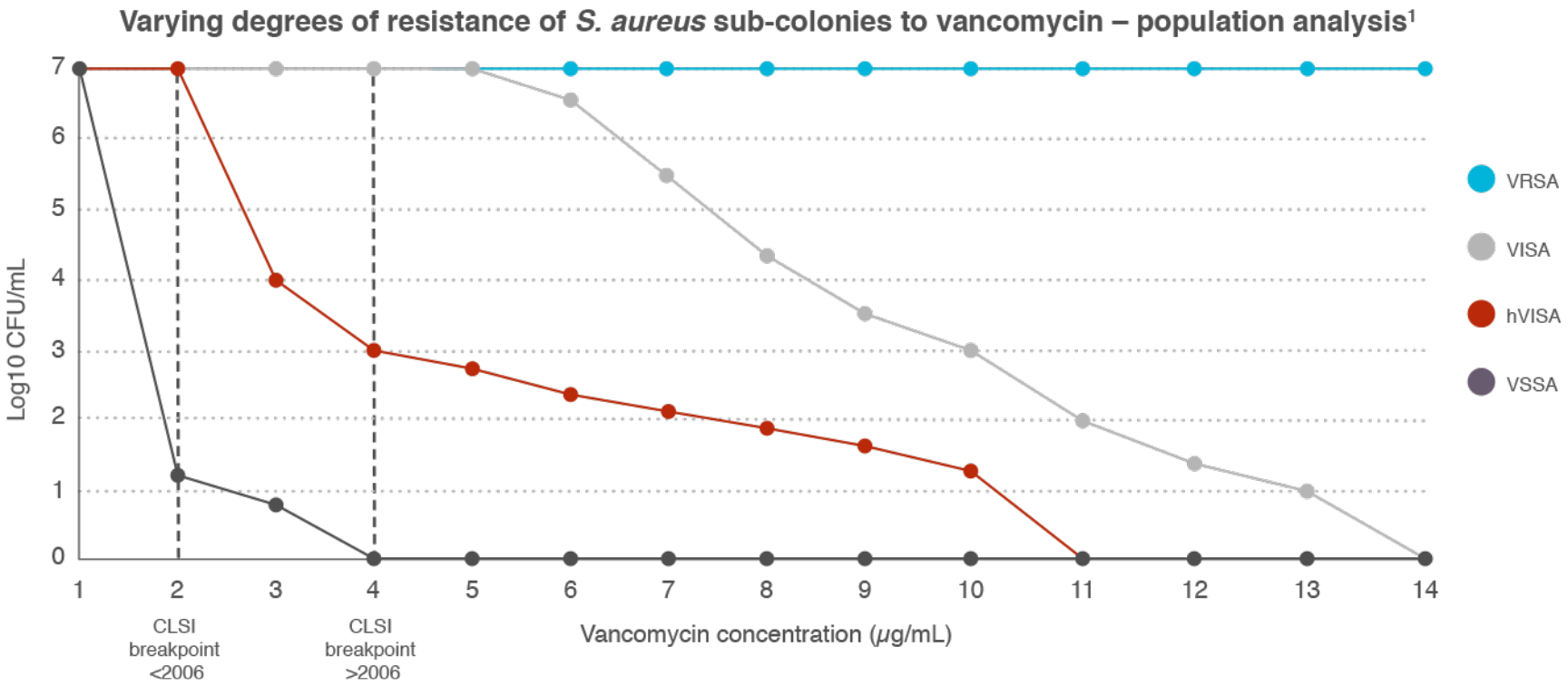
S. aureus is a major pathogen for hospital-acquired pneumonia (HAP)[1,2]



*Percentages are higher than 100 due to polymicrobial infection.
ICU, intensive care unit; MRSA, methicillin-resistant *S. aureus*; MSSA, methicillin-sensitive *S. aureus*; VAP, ventilator-associated pneumonia.
1. Koulenti D *et al. Crit Care Med* 2009; **37**: 2360–8. **2.** Rubinstein E *et al. Clin Infect Dis* 2011; **52**(1): 31–40.

Treatment of HAP is complicated by *S. aureus* strains with decreasing vancomycin susceptibility[1]

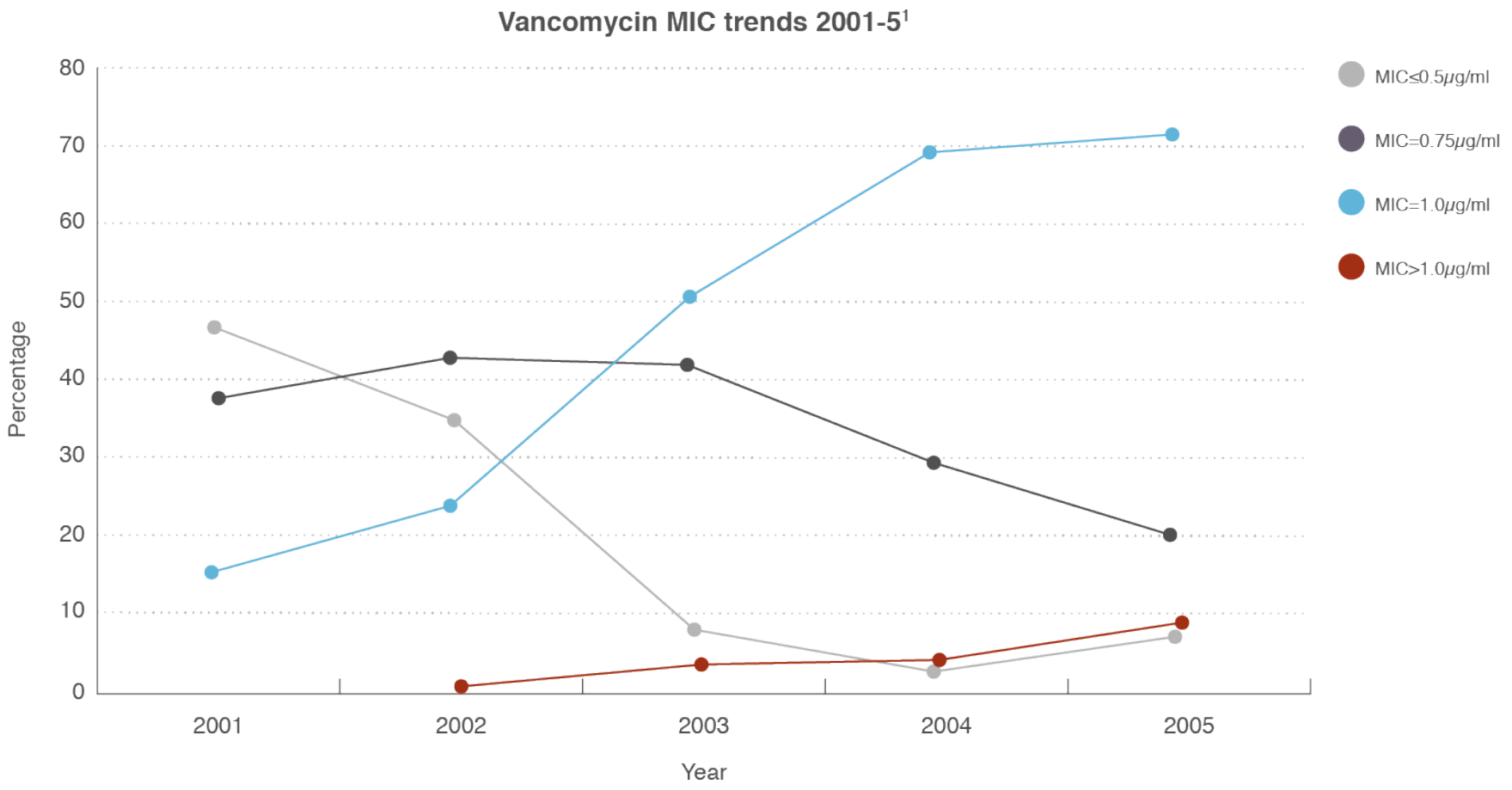
- While >99% of *S. aureus* isolates remain susceptible to vancomycin, strains of MRSA with reduced susceptibility are beginning to appear[1]



HAP, hospital-acquired pneumonia; MRSA, methicillin-resistant *S. aureus*; CFU, colony-forming units; VRSA , vancomycin-resistant *S. aureus*; VISA, vancomycin-intermediate *S. aureus*; hVISA, heterogenous VISA; VSSA, vancomycin-sensitive *S. aureus*; CLSI, Clinical and Laboratory Standards Institute

1. Dhand A & Sakoulas G. *F1000 Medicine Reports* 2012; **4**: 4.

Vancomycin MIC “creep”



MIC, minimum inhibitory concentration

1. Steinkraus G *et al. J Antimicrob Chemother* 2007;**60**:788–794.

Staphylococcus aureus Vancomycin MIC "creep" into resistance[1]

- TEST Programme = International surveillance with standardised broth microdilution methodology
- The proportion of S. Aureus, MRSA and MSSA isolates with reduced susceptibility to vancomycin has increased in recent years.

Year	Isolates	Phenotype		
		S. aureus	MRSA	MSSA
2004-2009	All (*n*)	20004	8249	11755
	VAN MIC≥2µg/ml [*n* (%)]	797 (4.0)	439 (5.3)	358 (3.0)
2004	All (*n*)	2525	1158	1367
	VAN MIC≥2µg/ml [*n* (%)]	101 (4.0)	65 (5.6)	36 (2.6)
2005	All (*n*)	2930	1411	1519
	VAN MIC≥2µg/ml [*n* (%)]	62 (2.1)	39 (2.8)	23 (1.5)
2006	All (*n*)	3612	1531	2081
	VAN MIC≥2µg/ml [*n* (%)]	94 (2.6)	50 (3.3)	44 (2.1)
2007	All (*n*)	4944	2028	2916
	VAN MIC≥2µg/ml [*n* (%)]	160 (3.2)	78 (3.8)	82 (2.8)
2008	All (*n*)	4348	1481	2867
	VAN MIC≥2µg/ml [*n* (%)]	253 (5.8)	136 (9.2)	117 (4.1)
2009	All (*n*)	1645	640	1005
	VAN MIC≥2µg/ml [*n* (%)]	127 (7.7)	71 (11.1)	56 (5.6)

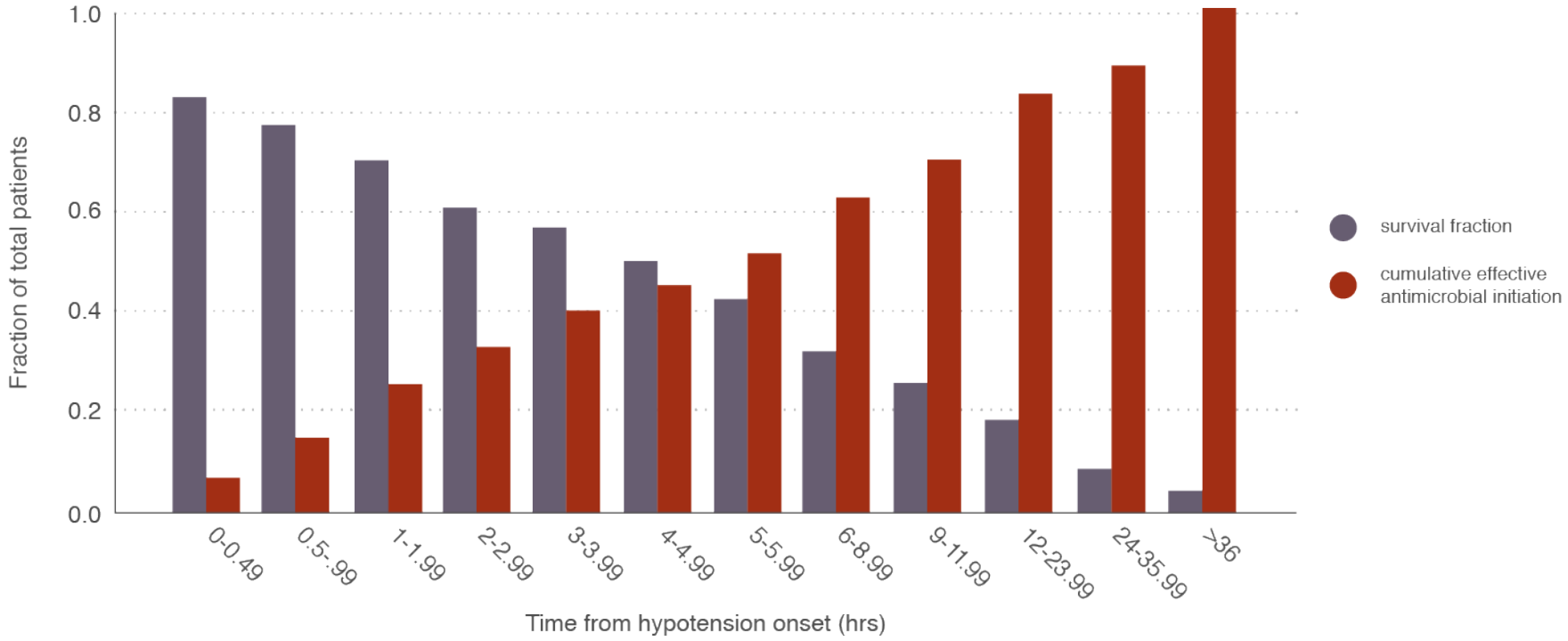
= $p < 0.001$ Difference between number of isolates with VAN MIC ≥2µg/ml in 2004 vs 2009

1. Hawser SP *et al. IJAA* 2011; **37**: 219–224

Each hour of delay in initiation of effective antimicrobial therapy reduces survival[1]

- Effective therapy for septic shock initiated within the first 'golden hour' after onset of hypotension is associated with 80% survival, with a mean decrease in survival of 7.6% for each hour of delay[1]

Cumulative effective antimicrobial initiation following onset of septic shock-related hypotension and associated survival[1]



1. Kumar A *et al. Crit Care Med* 2006. **34**:1589–96.