



What is Community-Associated MRSA?

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Plan for Today

- *Staphylococcus aureus*
- Antimicrobial resistance history
- CA-MRSA epidemiology, 1995-present
- The genomic revolution & MRSA
- MRSA & the Environment

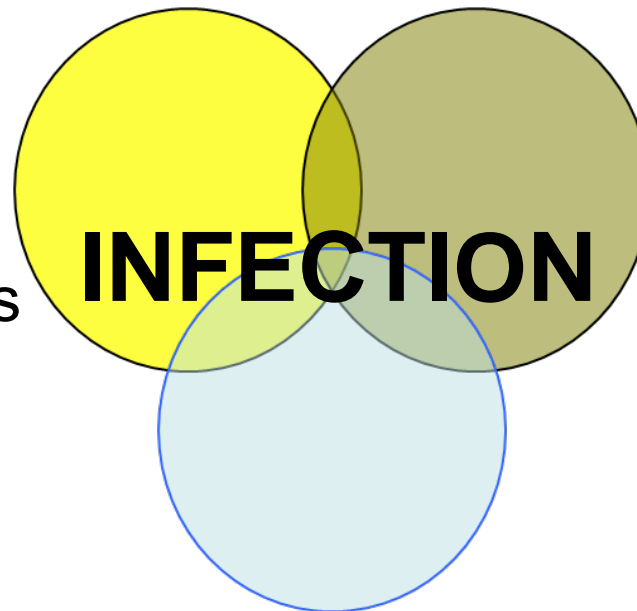
Our Planet Belongs to Microbes

- There are more viruses in 1 drop of seawater than there are people on the Earth
- Bacteria always live on us & live on objects around us
- We are rarely infected by them
- Even the bacteria in our mouths are capable of infecting us
- Why are we not all infected right now?

What is an infection?

HOST

- Immunity
- Habits
- Exposures
- Other diseases predispose



PATHOGEN

- Bacteria
- Viruses
- Parasites

ENVIRONMENT

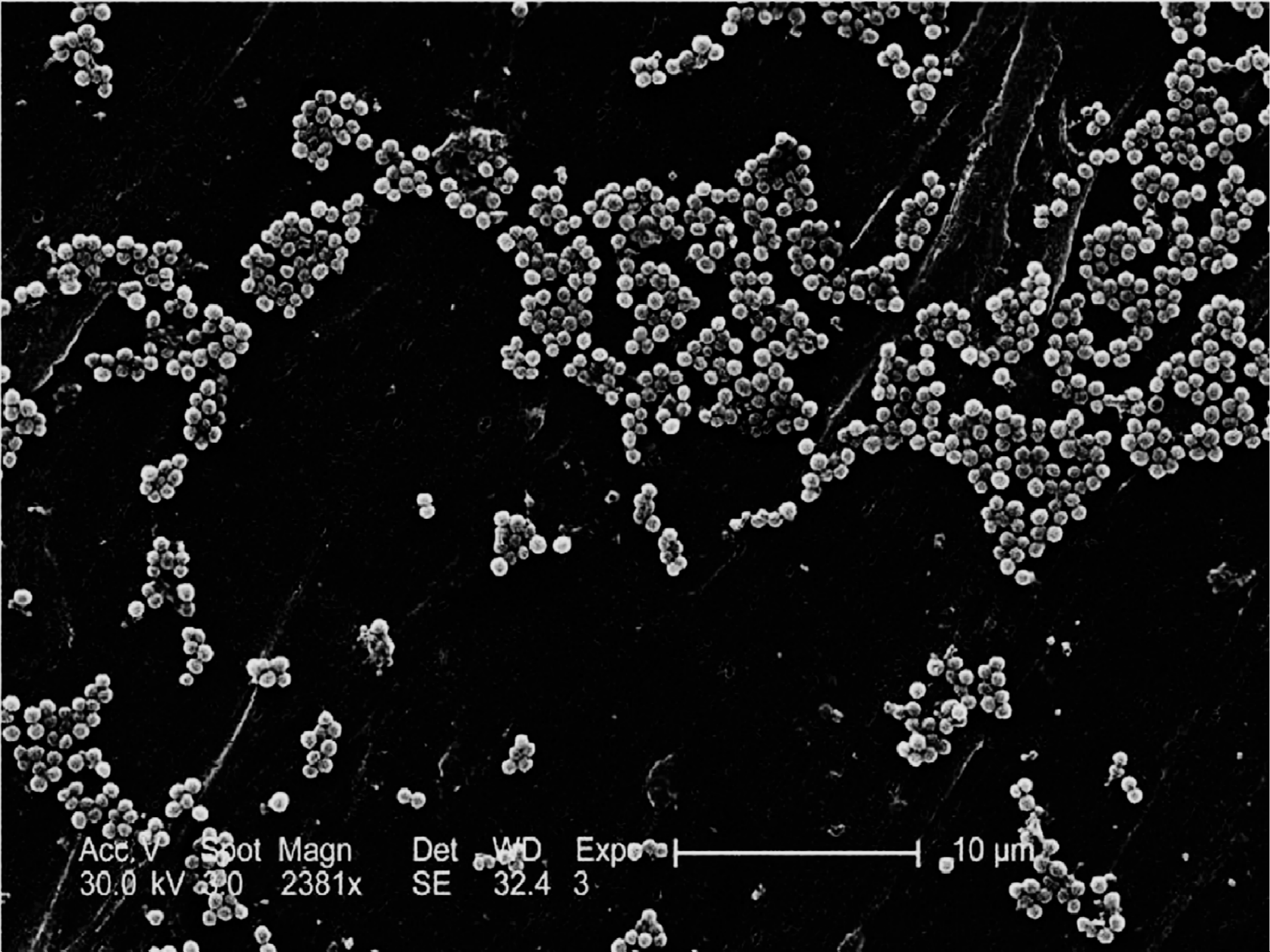
- Factors favoring pathogen
- Factors bringing pathogen to hosts

Staphylococcus aureus

- Most common cause of cultured bacterial infections
- Discovered in pus, 1881, by Ogston, a surgeon in UK
- Garré in 1885 rubbed pure culture of *S. aureus* on arm; developed many abscesses with 17 scars



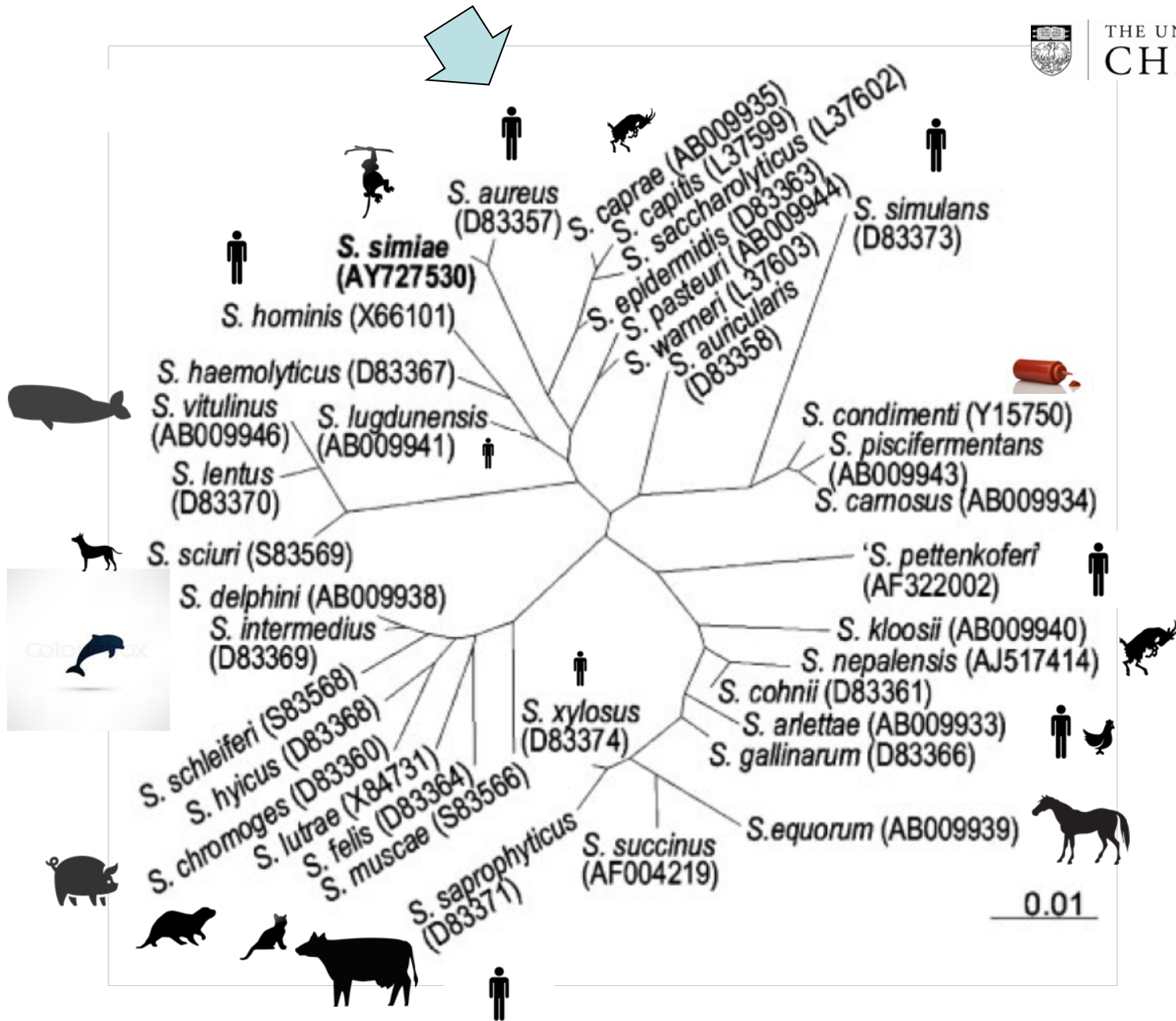
Photo Credit: Gregory Moran, M.D.



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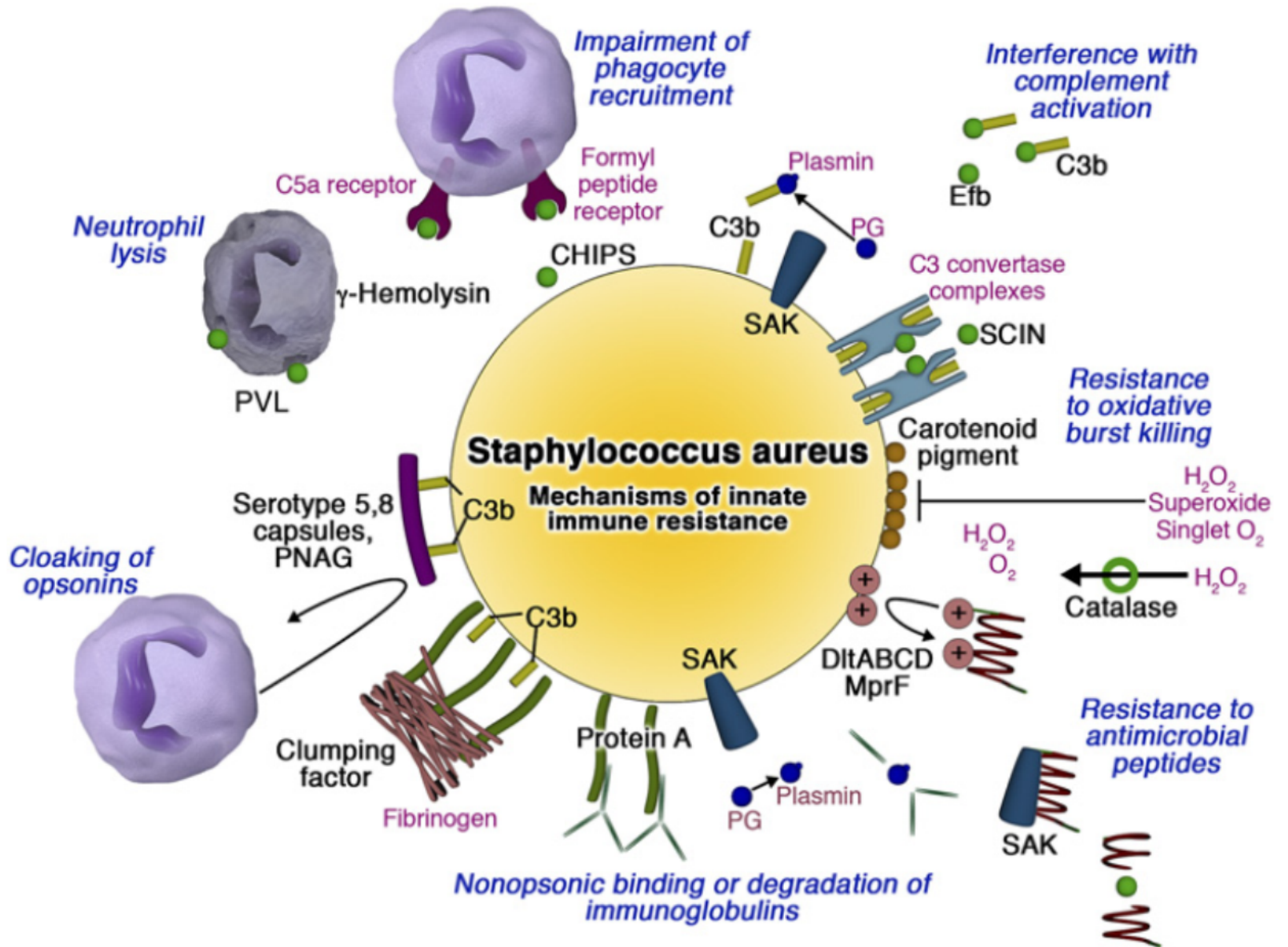
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Staphylococcus aureus

- Skin most common site of infection
 - boils, furuncles, carbuncles, folliculitis
- Abscesses in skin, bone, other organs
- Reservoir: human beings
 - 30-60% of people carry *S. aureus*



Staphylococcus aureus
History of Antibiotic Resistance

- Before 1930s: No antibiotics
 - Case fatality rate of bloodstream infection: 80%
- 1930s: Introduction of sulfa drugs, successfully treated *S. aureus* pneumonia

Howard Florey, 1898-1968

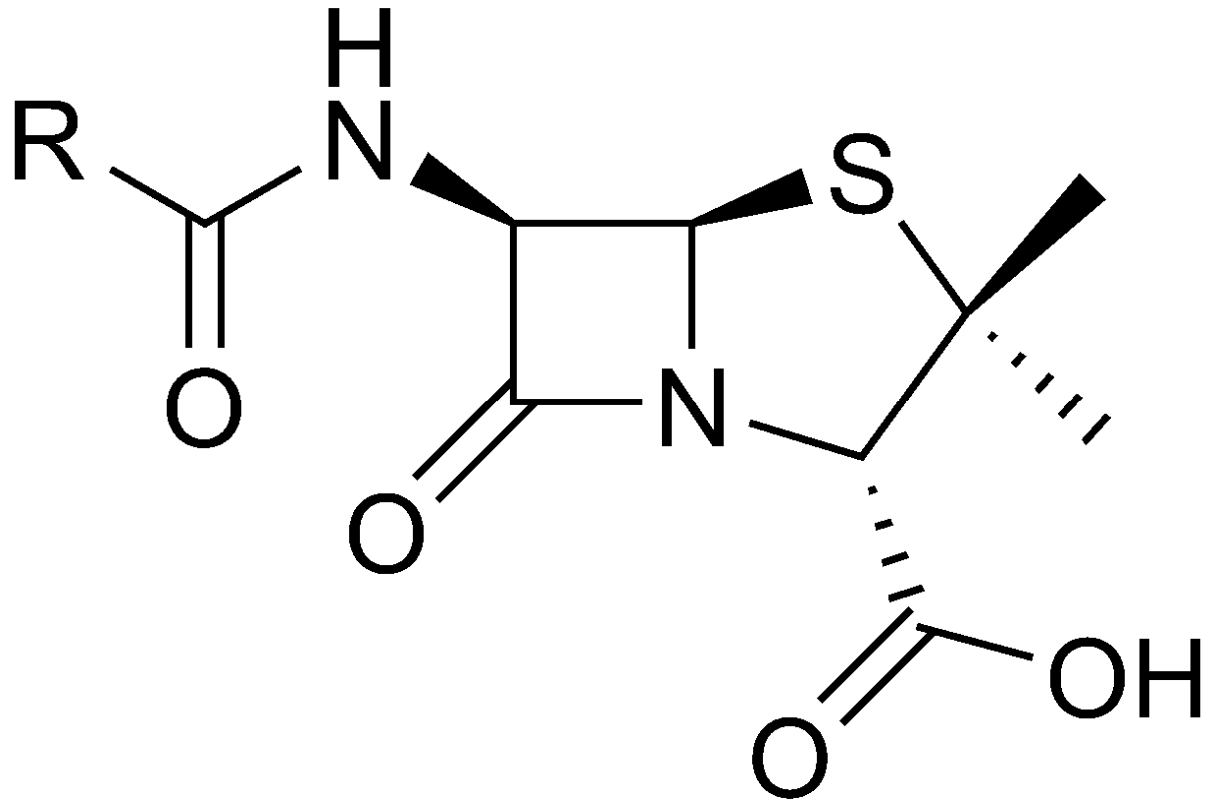


Developed penicillin as a drug

Nobel Prize, 1945



Penicillin core



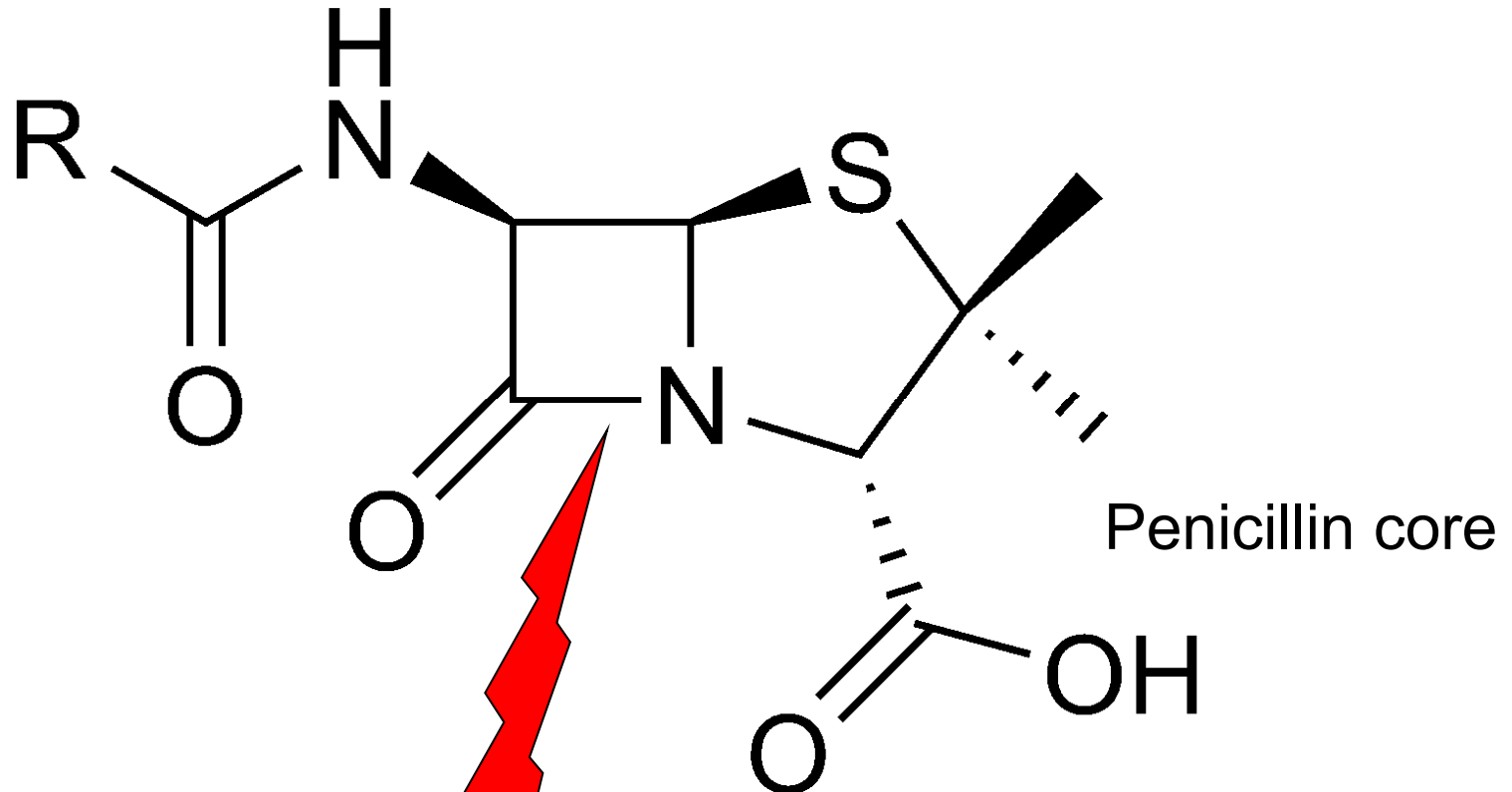
Staphylococcus aureus
History of Antibiotic Resistance

- 1940s: Penicillin, “wonder drug”
 - Reduced mortality dramatically
 - 1945: resistance to penicillin described
- Mechanism:
 - Binds to penicillin-binding proteins (PBP1, 2, 3, and 4) on bacterial membrane → interferes with cell wall synthesis → kills bacteria

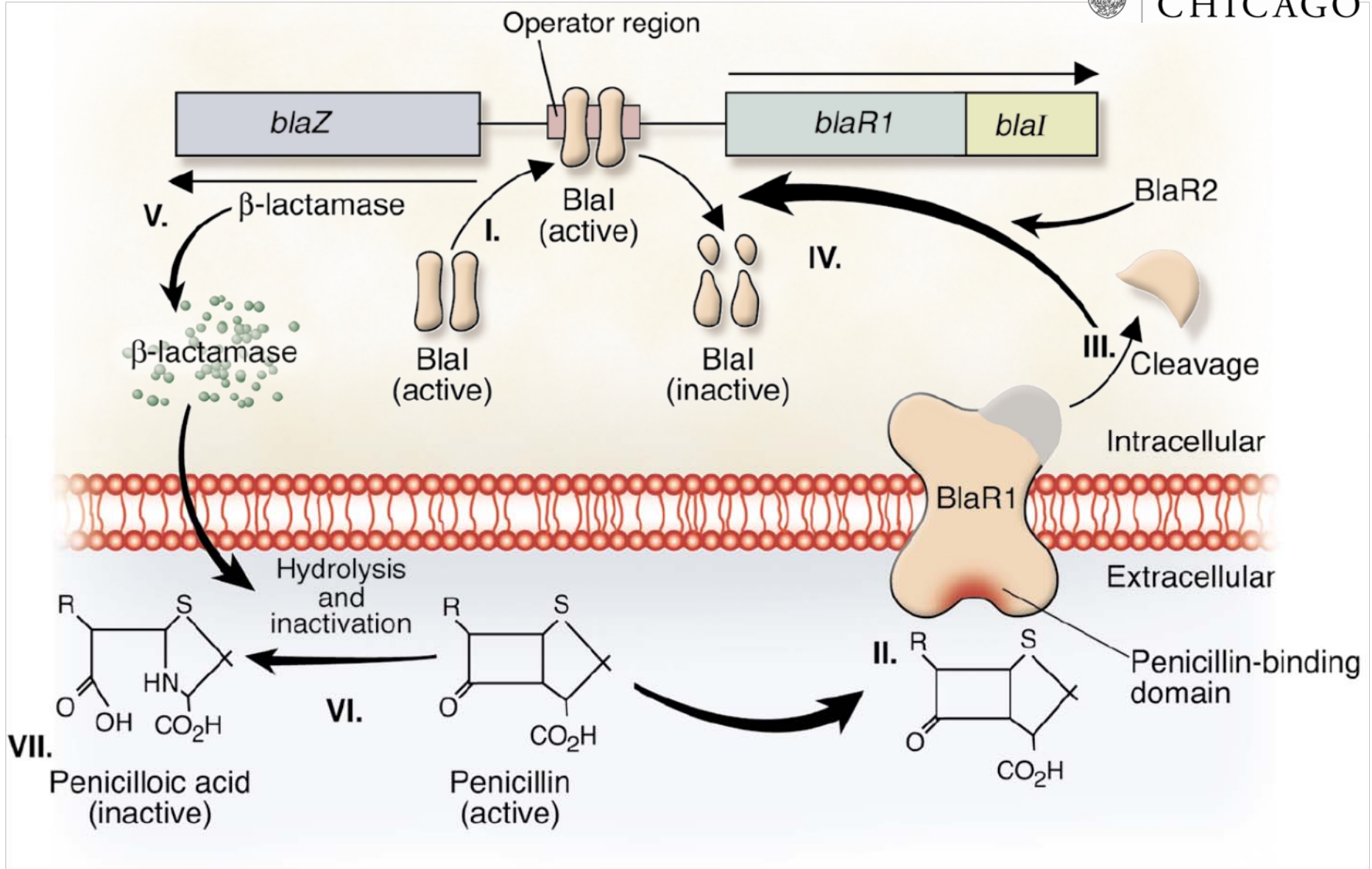
Staphylococcus aureus
History of Antibiotic Resistance

- Penicillin-resistant *S. aureus* = “PRSA”
- β -lactamase: enzyme that “cuts” penicillin
 - Renders it ineffective

β -lactamase: Site of Action



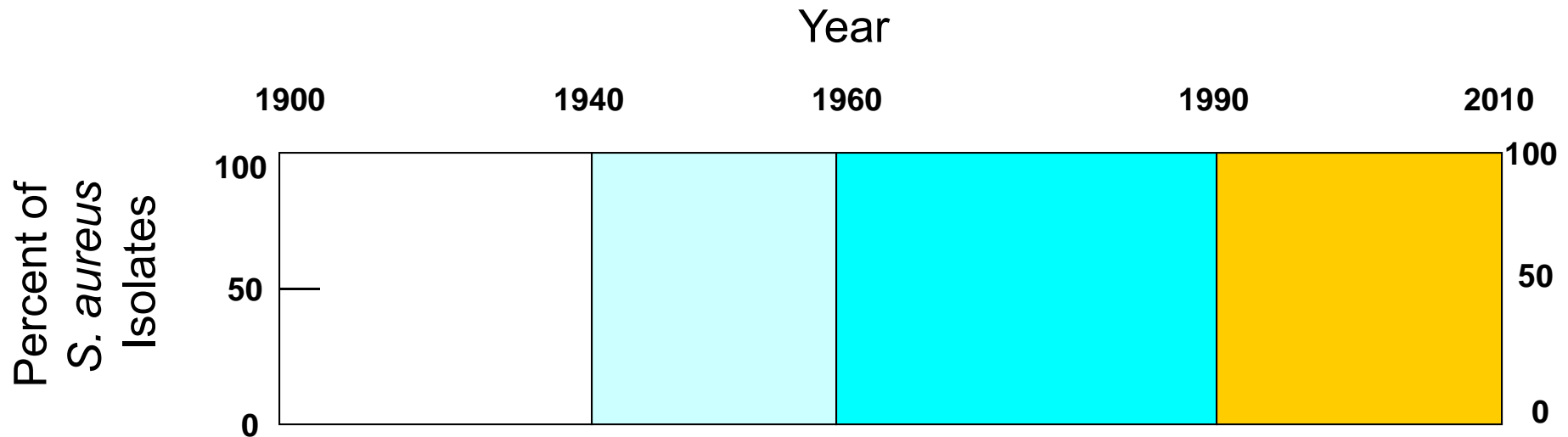
β -lactamase
hydrolyzes this bond,
inactivating penicillin



Staphylococcus aureus
History of Antibiotic Resistance

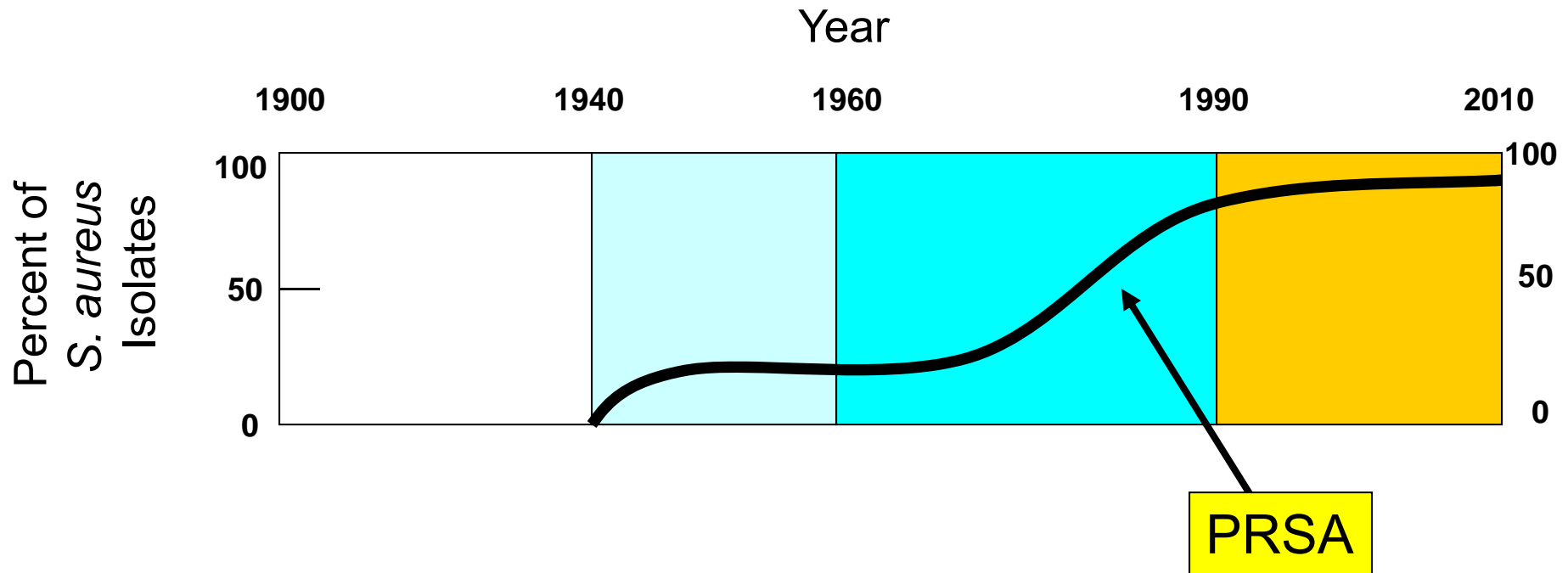
- PRSA predominant among infecting strains in USA by mid-1970s
- Now ~90% of *S. aureus* isolates are PRSA

History of β -lactam Resistance in *S. aureus*





History of β -lactam Resistance in *S. aureus*



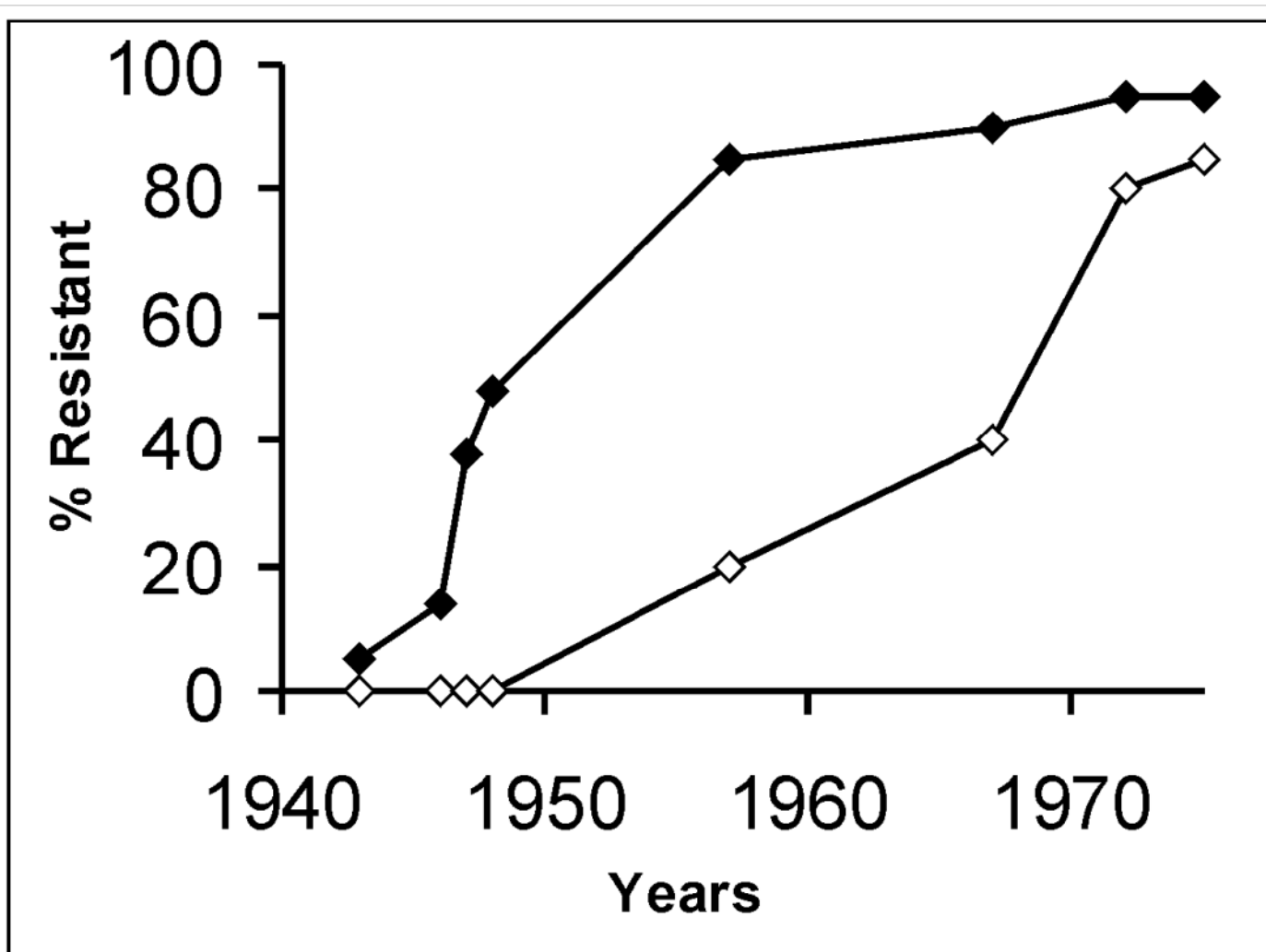


Figure. Secular trends of approximate prevalence rates for penicillinase-producing, methicillin-susceptible strains of *Staphylococcus aureus* in hospitals (closed symbols) and the community (open symbols).

New Drugs for PRSA

- Methicillin: the first β -lactamase-resistant penicillin, developed in 1959
 - Killed PRSA
 - Subsequently many variants (oxacillin, nafcillin)
- **Methicillin-resistant *S. aureus* (MRSA)** arose within 1 year, in 1960!

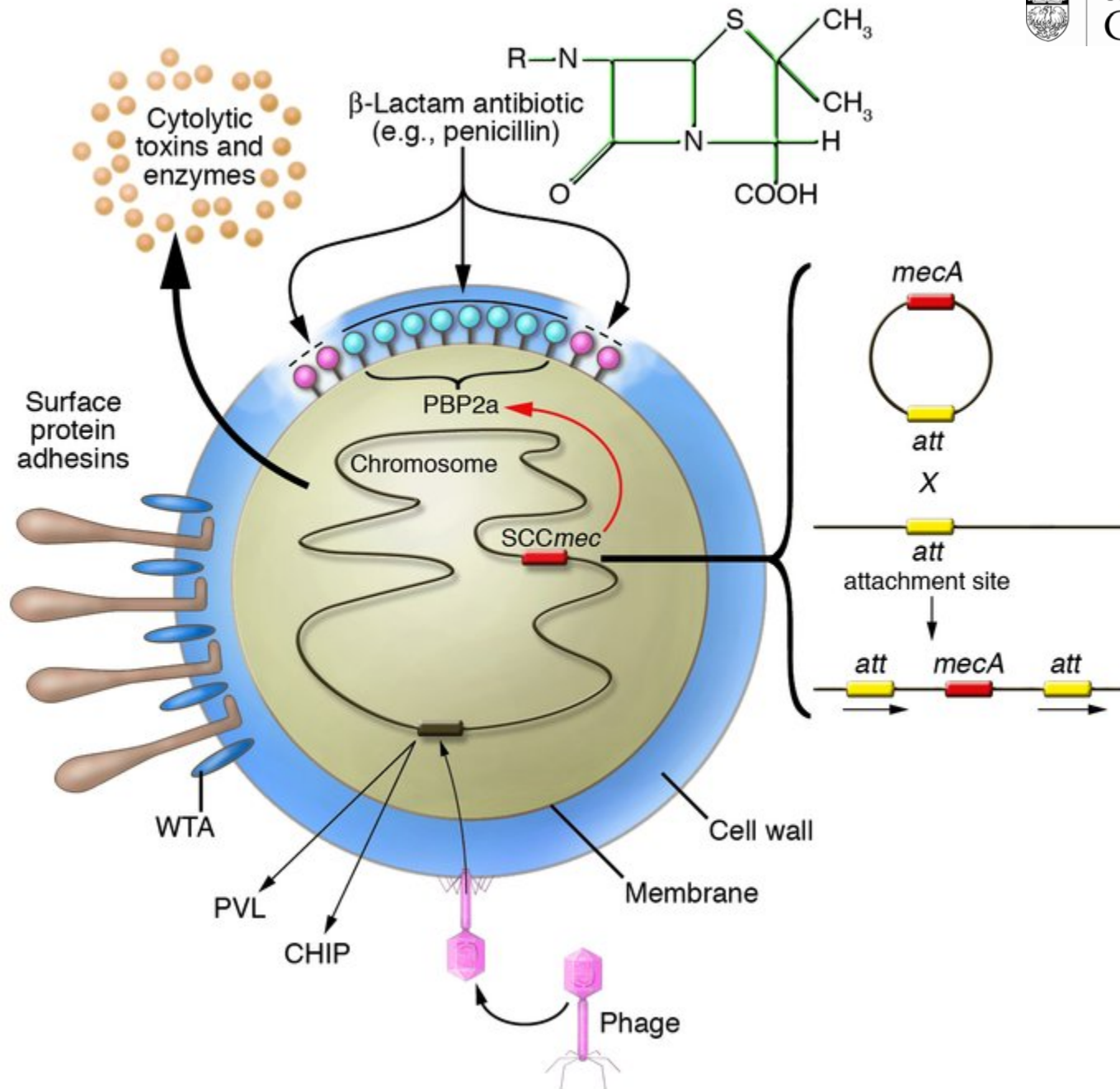


MRSA: Emergence

- MRSA: *S. aureus* resistant to all β -lactam drugs (penicillins, cephalosporins, carbapenems)
- **1960:** MRSA first reported in UK
- **1968:** first reported in US (Boston)

MRSA: Mechanism

- Mechanism: *mecA* gene codes for PBP2a → cell membrane
- No β -lactams can bind the penicillin-binding proteins when PBP2a is present in membrane





MRSA 1970s-80s

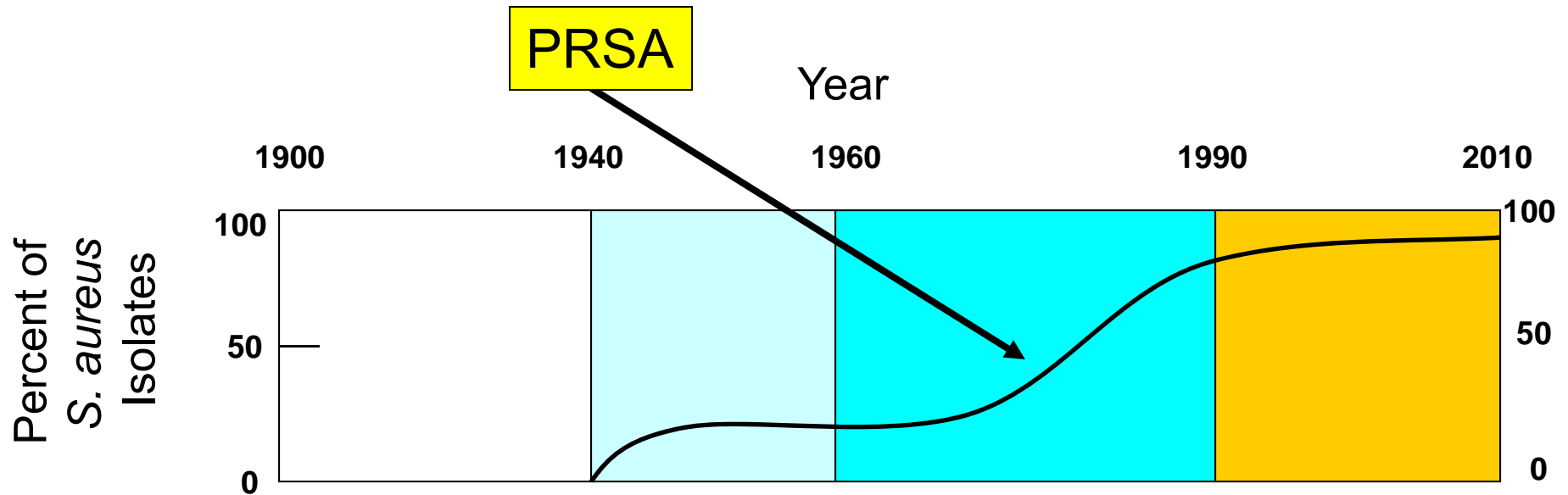
- 1970s: Epidemic outbreaks among hospitalized patients; 2.5% of U. S. hospital isolates in 1975 were MRSA
 - more common in Europe
- 1980s: Gradually became endemic in large American hospitals; 5% in 1981 (NNISS)



HA-MRSA 1990s-2002

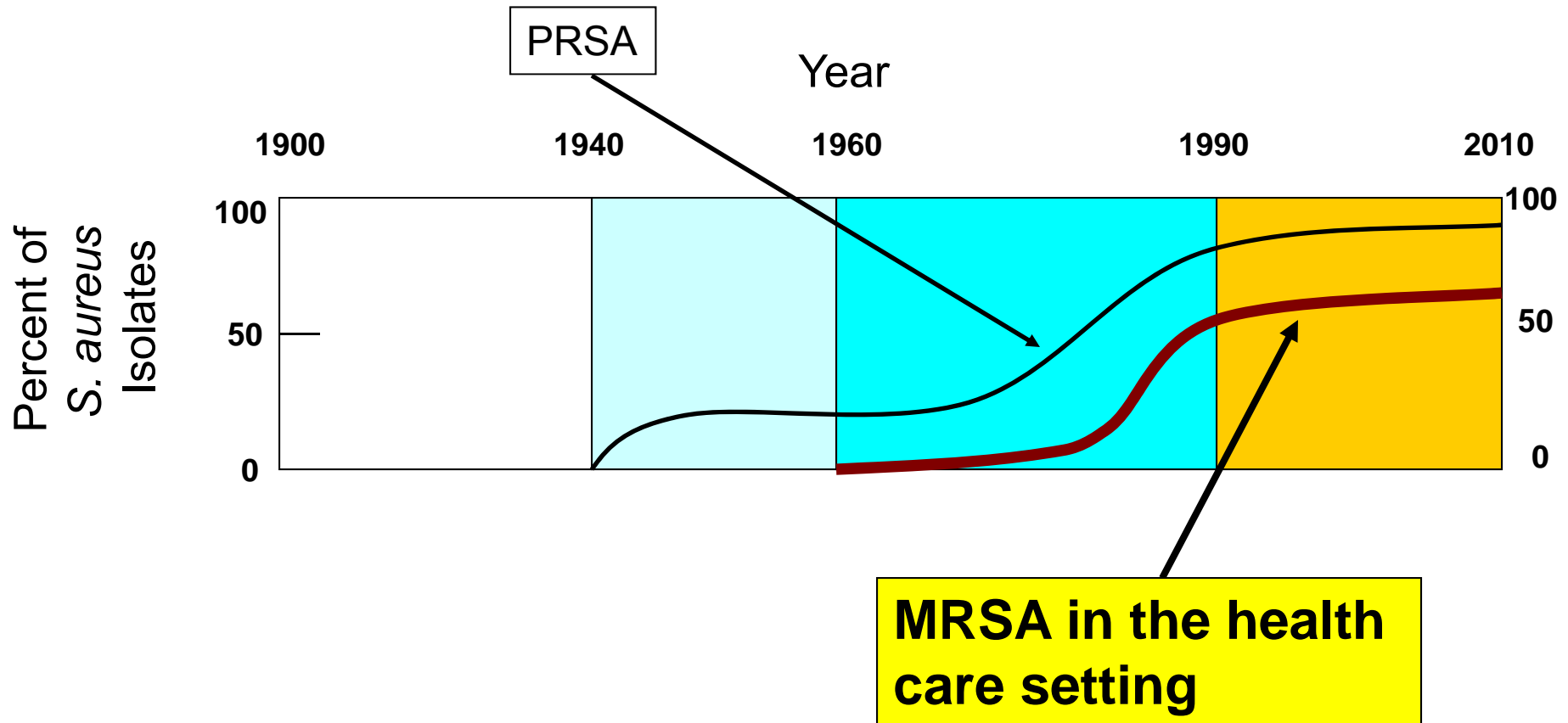
- Early 1990s: Hospital-acquired SA were 29% MRSA 1991
 - MRSA frequently MDR strains

History of β -lactam Resistance in *S. aureus*





History of β -lactam Resistance in *S. aureus*





Methicillin (oxacillin)-resistant *Staphylococcus aureus* (MRSA) Among ICU Patients, 1995-2004



Source: National Nosocomial Infections Surveillance (NNIS) System

SAFER • HEALTHIER • PEOPLE™

Table 1Mechanisms of *S. aureus* resistance to antimicrobials^A

| Antibiotic | Resistance gene(s) | Gene product(s) | Mechanism(s) of resistance | Location(s) |
|---|---|---|--|---------------------|
| β-Lactams | 1) <i>blaZ</i> | 1) β-Lactamase | 1) Enzymatic hydrolysis of β-lactam nucleus | 1) Pl:Tn |
| | 2) <i>mecA</i> | 2) PBP2a | 2) Reduced affinity for PBP | 2) C:SCC <i>mec</i> |
| Glycopeptides | 1) Unknown (VISA) | 1) Altered peptidoglycan | 1) Trapping of vancomycin in the cell wall | 1) C |
| | 2) | 2) D-Ala-D-Lac | 2) Synthesis of dipeptide with reduced affinity for vancomycin | 2) Pl:Tn |
| Quinolones | 1) <i>parC</i> | 1) ParC (or GrlA) component of topoisomerase IV | 1,2) Mutations in the QRDR region, reducing affinity of enzyme-DNA complex for quinolones | 1) C |
| | 2) <i>gyrA</i> or <i>gyrB</i> | 2) GyrA or GyrB components of gyrase | | 2) C |
| Aminoglycosides (e.g., gentamicin) | Aminoglycoside-modifying enzymes (e.g., <i>aac</i> , <i>aph</i>) | Acetyltransferase, phosphotransferase | Acetylating and/or phosphorylating enzymes modify aminoglycosides | Pl, Pl:Tn |
| Trimethoprim-sulfamethoxazole (TMP-SMZ) | 1) Sulfonamide: <i>sulA</i> | 1) Dihydropteroate synthase | 1) Overproduction of <i>p</i> -aminobenzoic acid by enzyme | 1) C |
| | 2) TMP: <i>dfrB</i> | 2) Dihydrofolate reductase (DHFR) | 2) Reduced affinity for DHFR | 2) C |
| Oxazolidinones | <i>rrn</i> | 23S rRNA | Mutations in domain V of 23S rRNA component of the 50S ribosome. Interferes with ribosomal binding | C |
| Quinupristin-dalfopristin (Q-D) | 1) Q: <i>ermA</i> , <i>ermB</i> , <i>ermC</i> | 1) Ribosomal methylases | 1) Reduce binding to the 23S ribosomal subunit | 1) Pl, C |
| | 2) D: <i>vat</i> , <i>vatB</i> | 2) Acetyltransferases | 2) Enzymatic modification of dalfopristin | 2) Pl |

^AExamples of several of the *S. aureus* mechanisms of resistance to selected antibiotics (77, 95–97). Pl, plasmid; C, chromosome; Tn, transposon; QRDR, quinolone resistance-determining region.

Methicillin-resistant *Staphylococcus aureus* (MRSA): **A Shift?**

- 1980s: MRSA infections in healthy people in Australia
- Not clear if new strains
 - Perhaps “feral” strains spread from hospitals?
 - Strains susceptible to gentamicin



Community-Acquired Methicillin-Resistant *Staphylococcus aureus* in Children With No Identified Predisposing Risk

Betsy C. Herold, MD; Lilly C. Immergluck, MD; Melinda C. Maranan, MD; Diane S. Lauderdale, PhD; Ryan E. Gaskin; Susan Boyle-Vavra, PhD; Cindy D. Leitch; Robert S. Daum, MD

In 1998, Dr. Robert Daum noted increased number of kids with no previous exposure to the health care setting with MRSA skin infections in Chicago

Chicago

- Compared nosocomial MRSA & community-associated (CA-) MRSA in children without health care exposure
- Increase in CA-MRSA rate (per 100,000 hospital admissions):
 - **10** in 1988-90
 - **259** in 1993-95



**Four Pediatric Deaths
from Community-Acquired Methicillin-Resistant *Staphylococcus aureus* —
Minnesota and North Dakota, 1997–1999**

Methicillin-resistant *Staphylococcus aureus* (MRSA) is an emerging community-acquired pathogen among patients without established risk factors for MRSA infection (e.g., recent hospitalization, recent surgery, residence in a long-term-care facility [LTCF], or injecting-drug use [IDU]) (1). Since 1996, the Minnesota Department of Health (MDH) and the Indian Health Service (IHS) have investigated cases of community-acquired MRSA infection in patients without established risk factors. This report describes four fatal cases among children with community-acquired MRSA; the MRSA strains isolated from these patients appear to be different from typical nosocomial MRSA strains in antimicrobial susceptibility patterns and pulsed-field gel electrophoresis (PFGE) characteristics.



Comparison of Community- and Health Care–Associated Methicillin-Resistant *Staphylococcus aureus* Infection

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METHICILLIN-RESISTANT *Staphylococcus aureus* (MRSA) was identified as a nosocomial pathogen in the 1960s.¹⁻⁴ Established risk factors for MRSA infection include recent hospitalization or surgery, residence in a long-term care facility, dialysis, and indwelling percutaneous medical devices and catheters.^{5,6} More recently, however, cases of MRSA have been documented among healthy community-dwelling persons without established risk factors for MRSA acquisition. These infections were apparently acquired in the community and have been referred to as either *community-acquired* or *community-associated* MRSA infections. Published reports of community-associated MRSA infections in North America include Minnesota and North Dakota,⁷⁻⁹ Nebraska,¹⁰ Alaska,¹¹ Chicago, Ill,¹²⁻¹⁴ Dallas, Tex,^{15,16} Winnipeg, Manitoba,¹⁷

Context Methicillin-resistant *Staphylococcus aureus* (MRSA) has traditionally been considered a health care–associated pathogen in patients with established risk factors. However, MRSA has emerged in patients without established risk factors (community-associated MRSA).

Objective To characterize epidemiological and microbiological characteristics of community-associated MRSA cases compared with health care–associated MRSA cases.

Design, Setting, and Patients Prospective cohort study of patients with MRSA infection identified at 12 Minnesota laboratory facilities from January 1 through December 31, 2000, comparing community-associated (median age, 23 years) with health care–associated (median age, 68 years) MRSA cases.

Main Outcome Measures Clinical infections associated with either community-associated or health care–associated MRSA, microbiological characteristics of the MRSA isolates including susceptibility testing, pulsed-field gel electrophoresis, and staphylococcal exotoxin gene testing.

Results Of 1100 MRSA infections, 131 (12%) were community-associated and 937 (85%) were health care–associated; 32 (3%) could not be classified due to lack of information. Skin and soft tissue infections were more common among community-associated cases (75%) than among health care–associated cases (37%) (odds ratio [OR], 4.25; 95% confidence interval [CI], 2.97-5.90). Although community-associated MRSA isolates were more likely to be susceptible to 4 antimicrobial classes (adjusted OR, 2.44; 95% CI, 1.35-3.86), most community-associated infections were initially treated with antimicrobials to which the isolate was nonsusceptible. Community-associated isolates were also more likely to belong to 1 of 2 pulsed-field gel electrophoresis clonal groups in both univariate and multivariate analysis. Community-associated isolates typically possessed different exotoxin gene profiles (eg, Panton Valentine leukocidin genes) compared with health care–associated isolates.

Conclusions Community-associated and health care–associated MRSA cases differ demographically and clinically, and their respective isolates are microbiologically distinct. This suggests that most community-associated MRSA strains did not originate in health care settings, and that their microbiological features may have contributed to their emergence in the community. Clinicians should be aware that therapy with β -lactam antimicrobials can no longer be relied on as the sole empiric therapy for severely ill outpatients whose infections may be staphylococcal in origin.

JAMA. 2003;290:2976-2984

www.jama.com

Author Affiliations: Acute Disease Investigation and Control Section (Drs Naimi, O'Boyle, Danila, and Lynfield and Mss LeDell, Como-Sabeti, and Borchardt) and Division of Public Health Laboratories (Mr Boxrud and Ms Johnson), Minnesota Department of Health, Minneapolis; French Reference Centre for Staphylococci, Lyon, France (Drs Etienne and Vandenesch); and Division of Healthcare Quality

Promotion, National Center for Infectious Diseases (Dr Fridkin) and Division of Applied Public Health Training, Epidemiology Program Office (Dr Naimi), National Center for Chronic Diseases, Centers for Disease Control and Prevention, Atlanta, Ga.

Corresponding Author and Reprints: Timothy S. Naimi, MD, MPH, K-67, 4770 Buford Highway NE, Atlanta, GA 30341 (e-mail: tbn7@cdc.gov).

CA-MRSA, Minnesota, 2000

- Prospective cohort
- 12 hospital & clinic labs
- CA-MRSA patients were
 - younger than HA-MRSA
 - more likely skin infections
- **New Strains had emerged!**
 - Resistant to fewer non-beta-lactams

CA-MRSA: New Syndromes

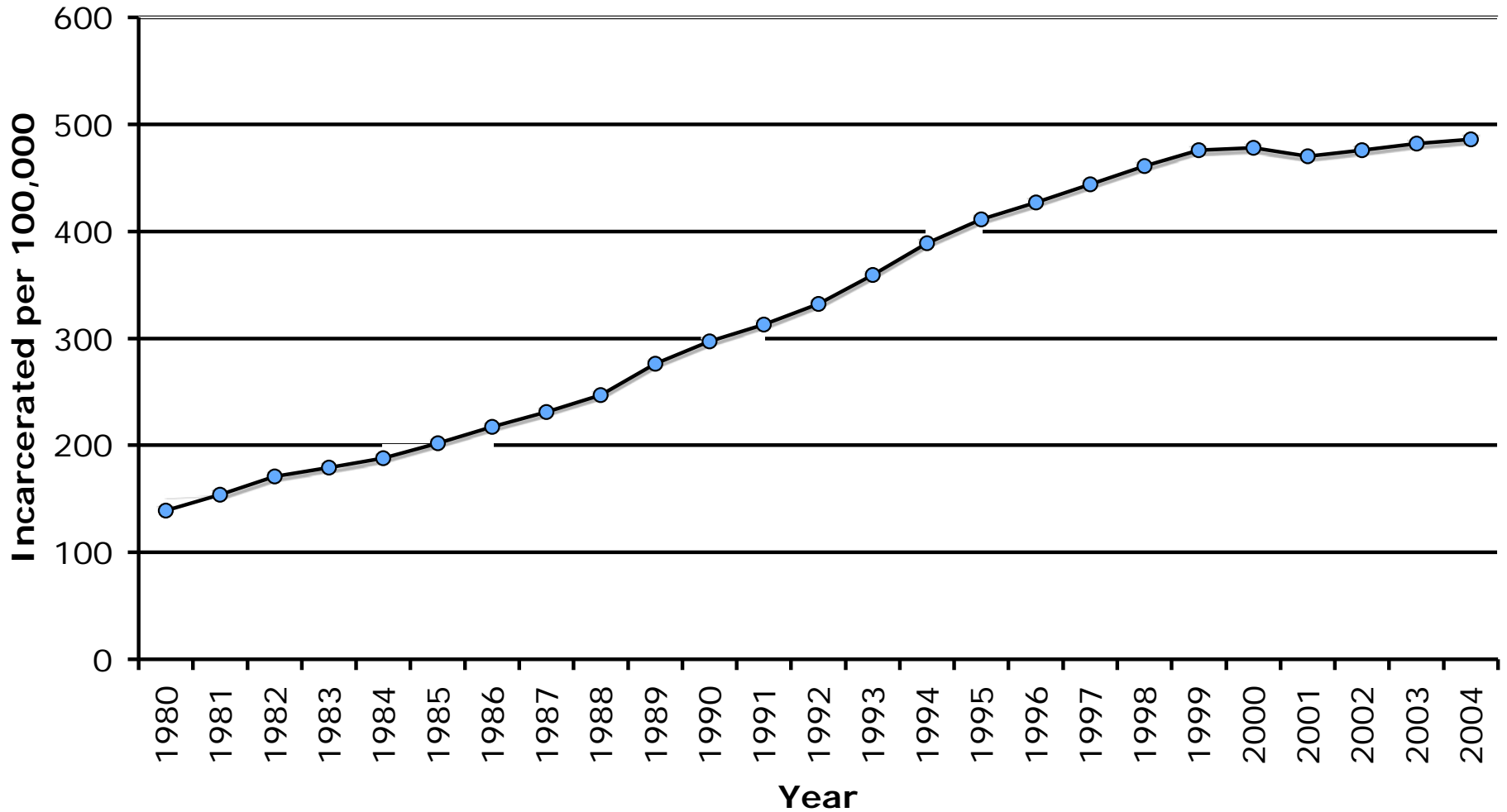
- Severe sepsis
- Pelvic septic thrombophlebitis



FIGURE 2. Characteristic necrotic changes of community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) pneumonia on chest CT scan.

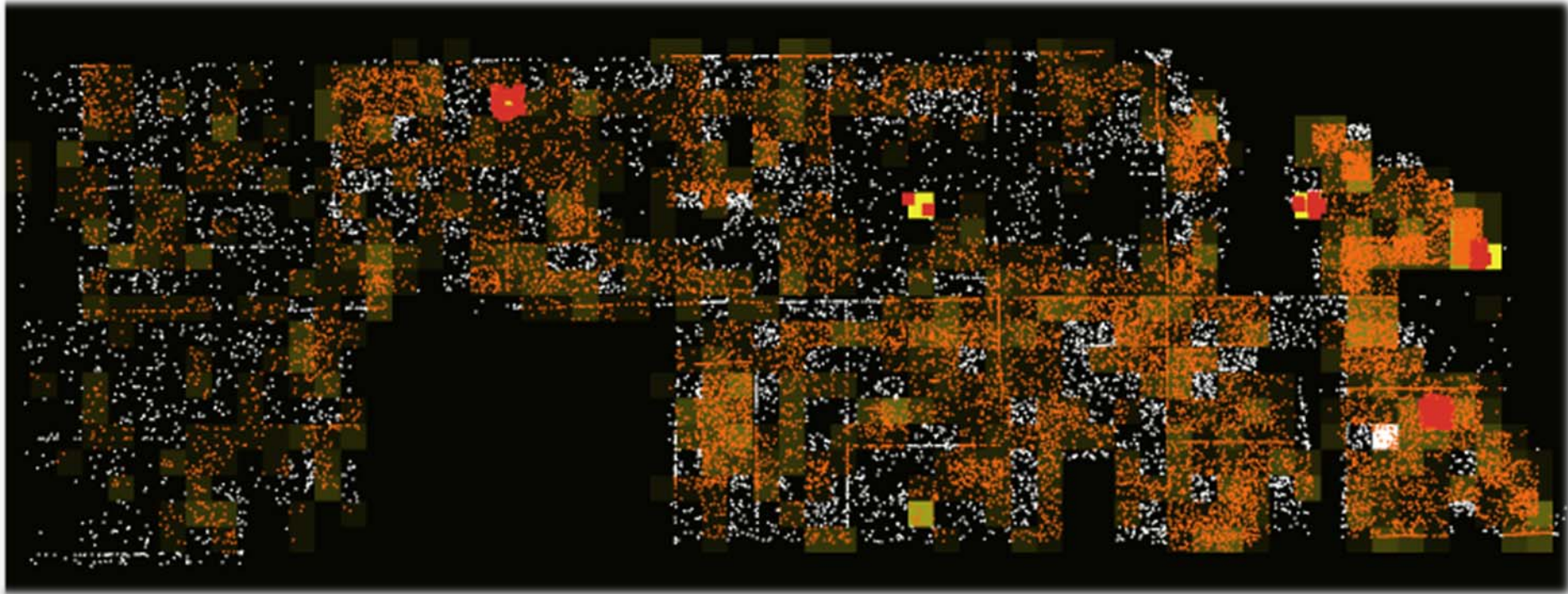


Number of Sentenced Inmates Under Federal or State Jurisdiction per 100,000 Population, 1980-2004



Source: Data from U.S. Department of Justice, Office of Justice Programs, Bureau of Justice Statistics, <http://www.ojp.usdoj.gov/bjs/glance/tables/incrttab.htm>, accessed 11/2/05

The Chicago Agent-Based MRSA Model



Places:

- Infected People
- Colonized People
- Uncolonized People

Areas:

- Many Colonized People
- Place with Uncolonized People

CA-MRSA: An emerging pathogen

- *New high-risk groups*
 - Incarcerated
 - Children
 - Urban underserved
 - Military
 - Athletes
 - Indigenous peoples
 - ER patients with STIs
 - Veterinarians / Animal handlers
 - HIV / MSM
 - Household contacts of the above



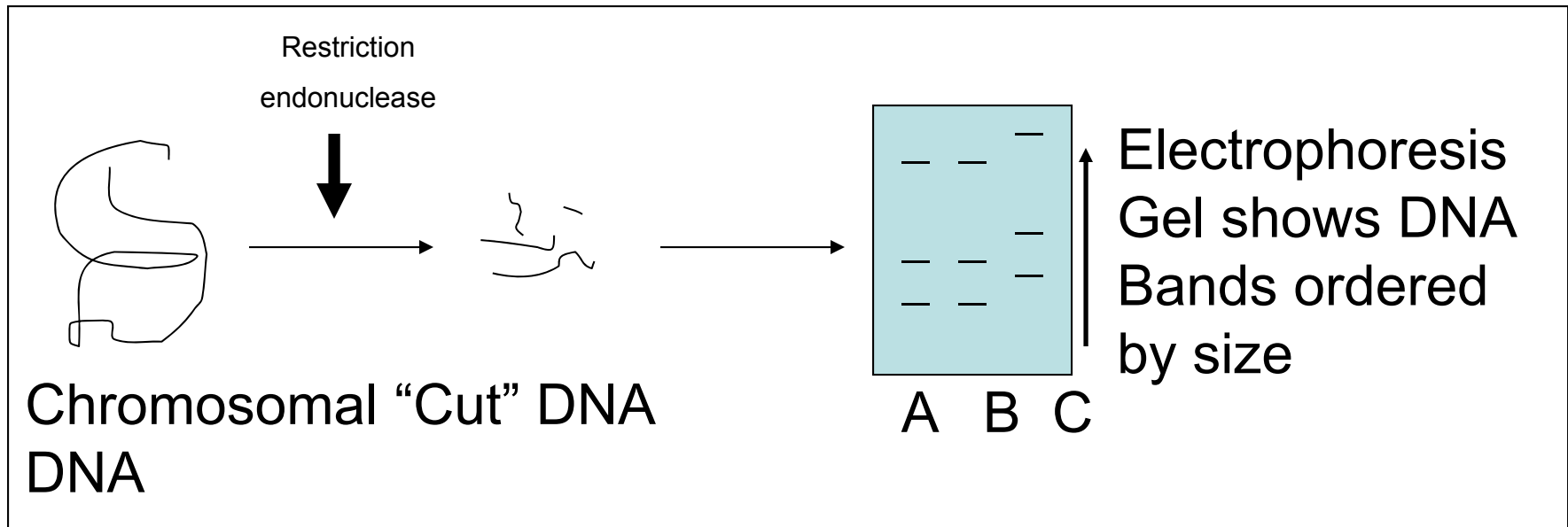
ED MRSA SSTIs, 2004

- 11 urban EDs across the U.S.
- 422 adult skin infection patients
- 79% of SSTIs caused by MRSA
- **97%** of MRSA were **USA300**

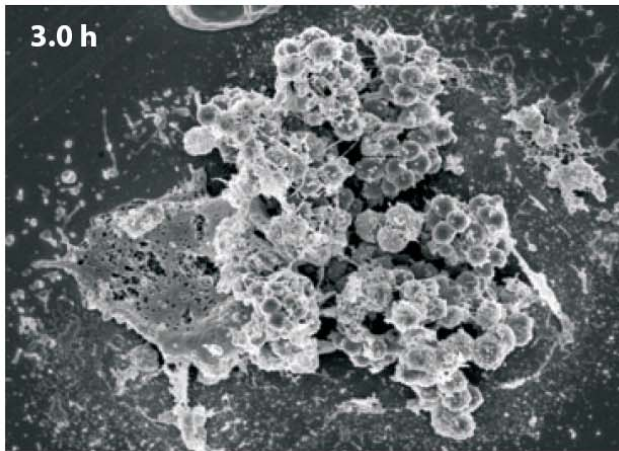
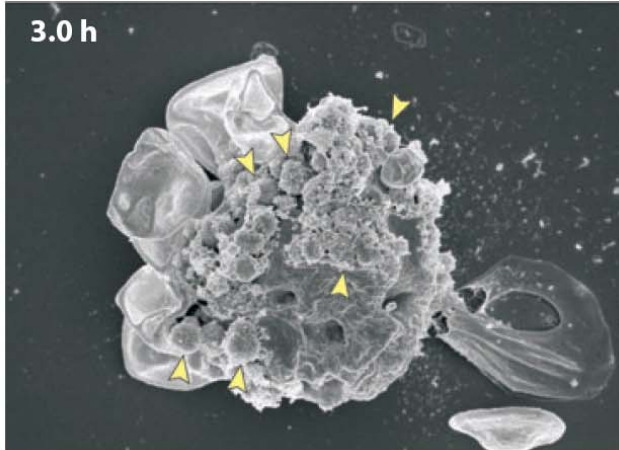
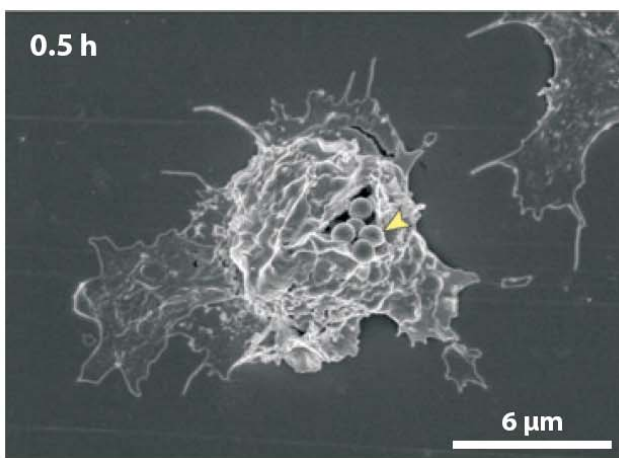
ED MRSA Infections, 2004

- High-risk population, MRSA nearly replaced MSSA in <10 years
- Widespread across the U.S.

- **Pulsed-field electrophoresis (PFGE)**
 - Whole-genome DNA cleaved & run on gel
 - Pattern is reproducible; very fine resolution



Conclusion: Isolates A and B are the same, C is different



Virulence Factor in
USA300 and other CA-MRSA:

**Panton Valentine leukocidin
(PVL)**

Hypothetical virulence factors in USA300 and other CA-MRSA strains.

Chromosomal Genetic Elements

ACME

Uniquely carried by MRSA USA300

SCCmec IV or V

Contain *mecA*, conferring β -lactam resistance

Efflux Pump

NorB

May provide a fitness advantage to USA400

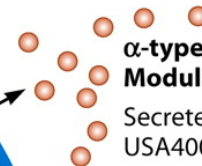
Secreted Toxins and Factors



Panton-Valentine leukocidin (PVL)

Strong epidemiologic association with CA-MRSA strains

Rarely found in MSSA and HA-MRSA strains



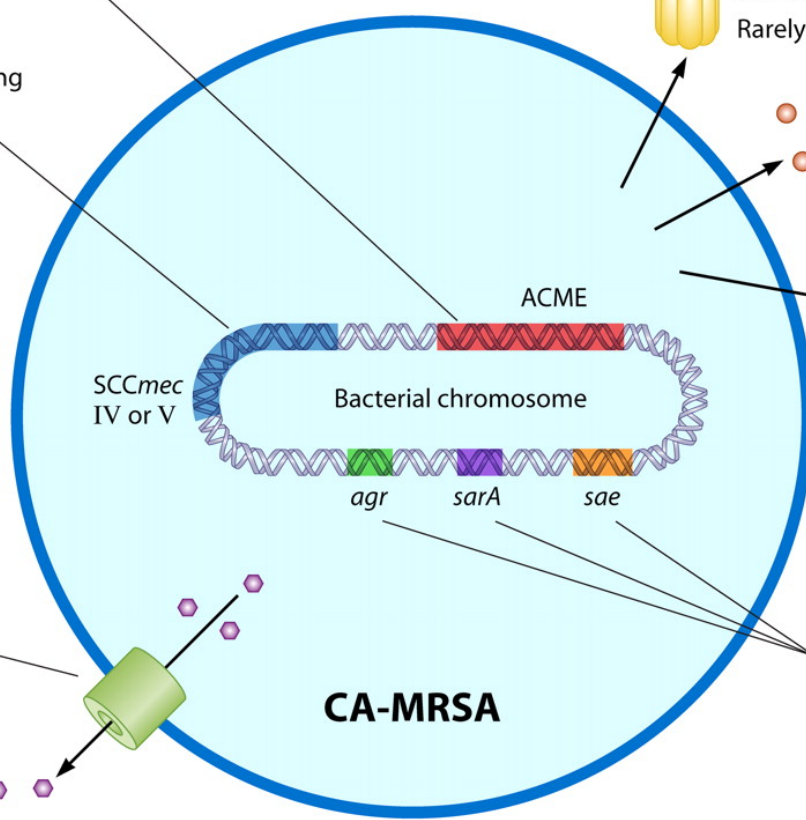
α -type Phenol Soluble Modulins (PSMs)

Secreted by USA300 and USA400 in high concentration



α -toxin

Increased expression in USA300



Global Gene Regulators

agr, *sarA*, and *sae*

Transcription increased in USA300
Upregulate many virulence factors

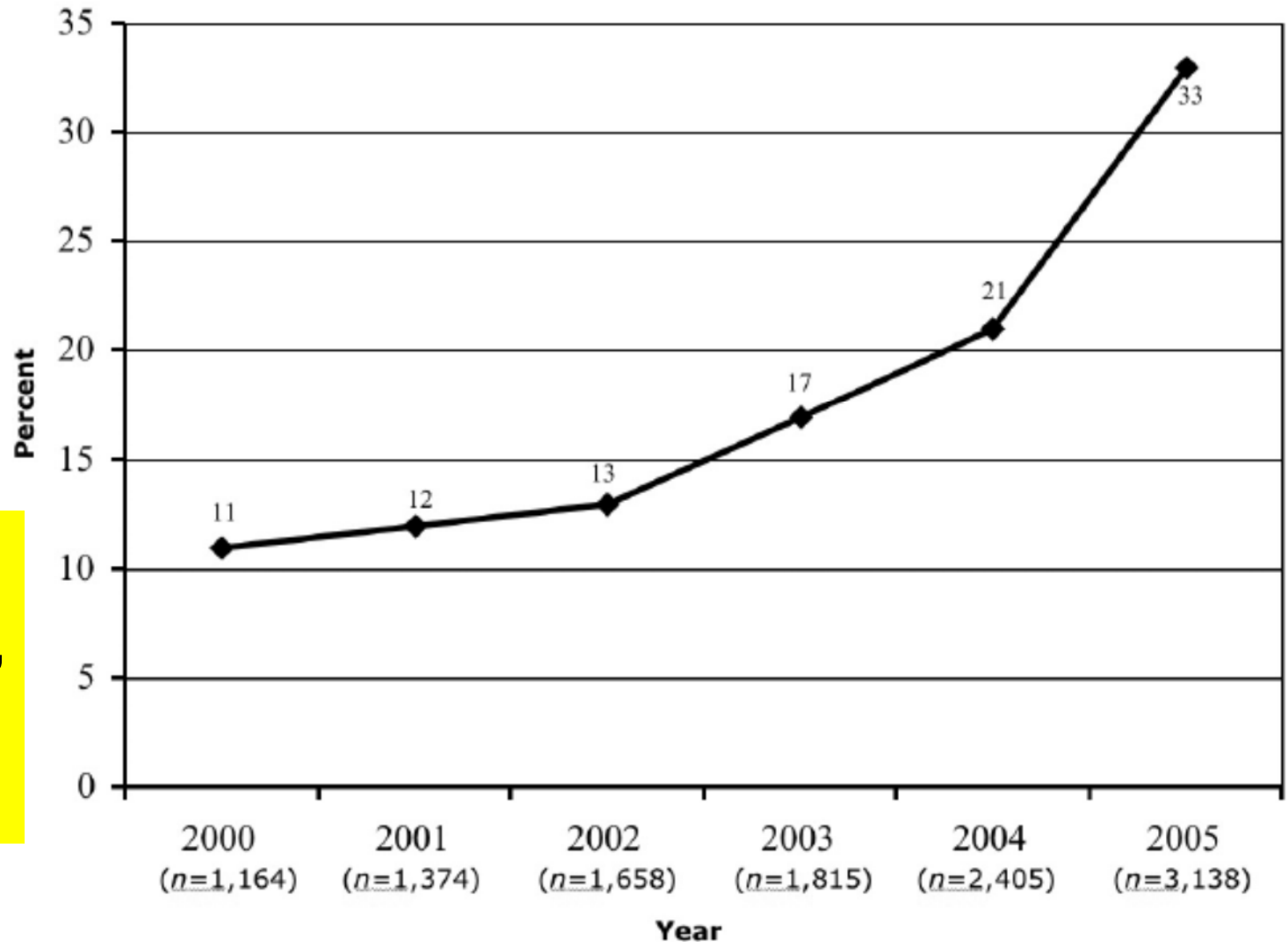
David M Z , and Daum R S Clin. Microbiol. Rev.
2010;23:616-687

Clinical Microbiology Reviews

MRSA: A Shift in Epidemiology

- MRSA spread rapidly as asymptomatic colonizer among healthy people
 - Kids in Nashville, 2001-4: 0.8% → 9.2%
- 13% of U.S. Veterans admitted to a VA hospital were colonized

Figure 1. Percent of methicillin-resistant *Staphylococcus aureus* cases classified as community-associated, 2000–2005^a



MN: 12
Hospitals,
CDC
criteria

MRSA: A Shift in Epidemiology

- All hospitals in San Francisco
- MRSA infection patients, 2005
- Incidence: **313/100,000**
- Excluding those with hospital stay in past 12 mo: **243/100,000**

CA-MRSA Incidence

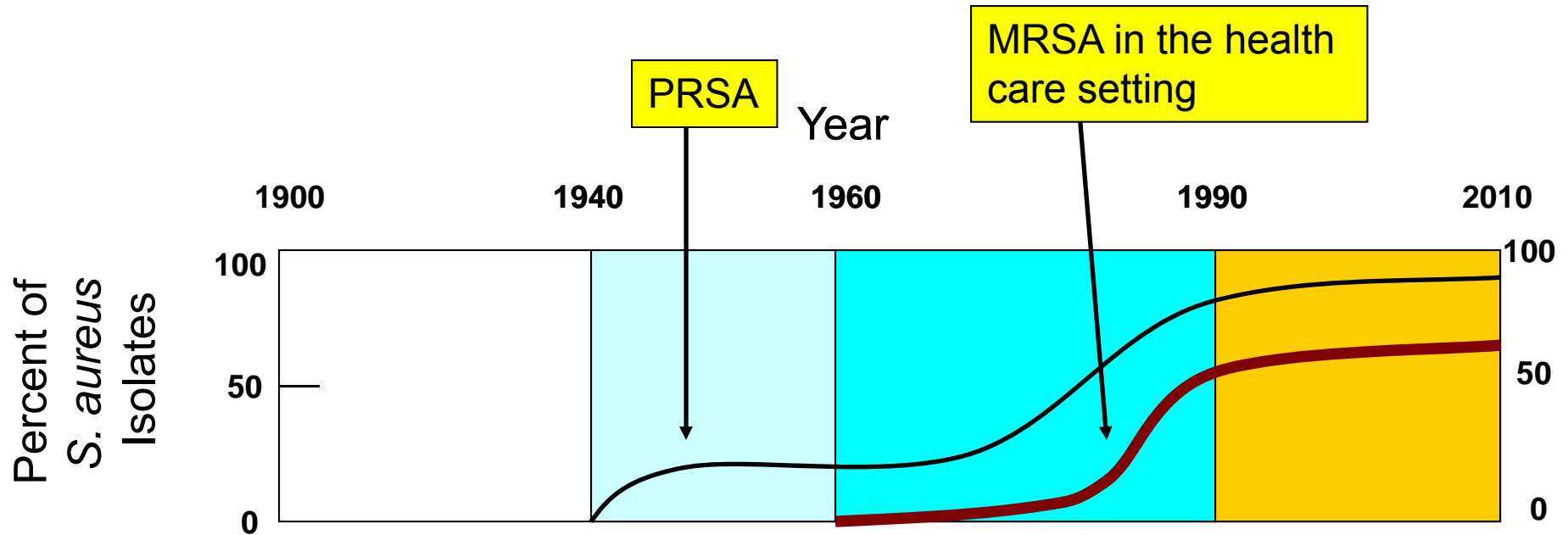
- Veterans in Maryland, 1999-2008: mean annual incidence = **251.8/100,000**
- Soldiers, Army Base in Georgia, 2005-8: **>3500/100,000**
- 3 First Nations communities, Northern Canada, 2006-8: **1460-4820/100,000**

Morrison-Rodriguez, et al. Epidemiol Infect 2010;138:721-9; Tracy LA, et al. EID 2011;17:441-8; Golding GR, et al. EID 2011;17:722-5.

Recurrent MRSA: USA300, Skin Infections

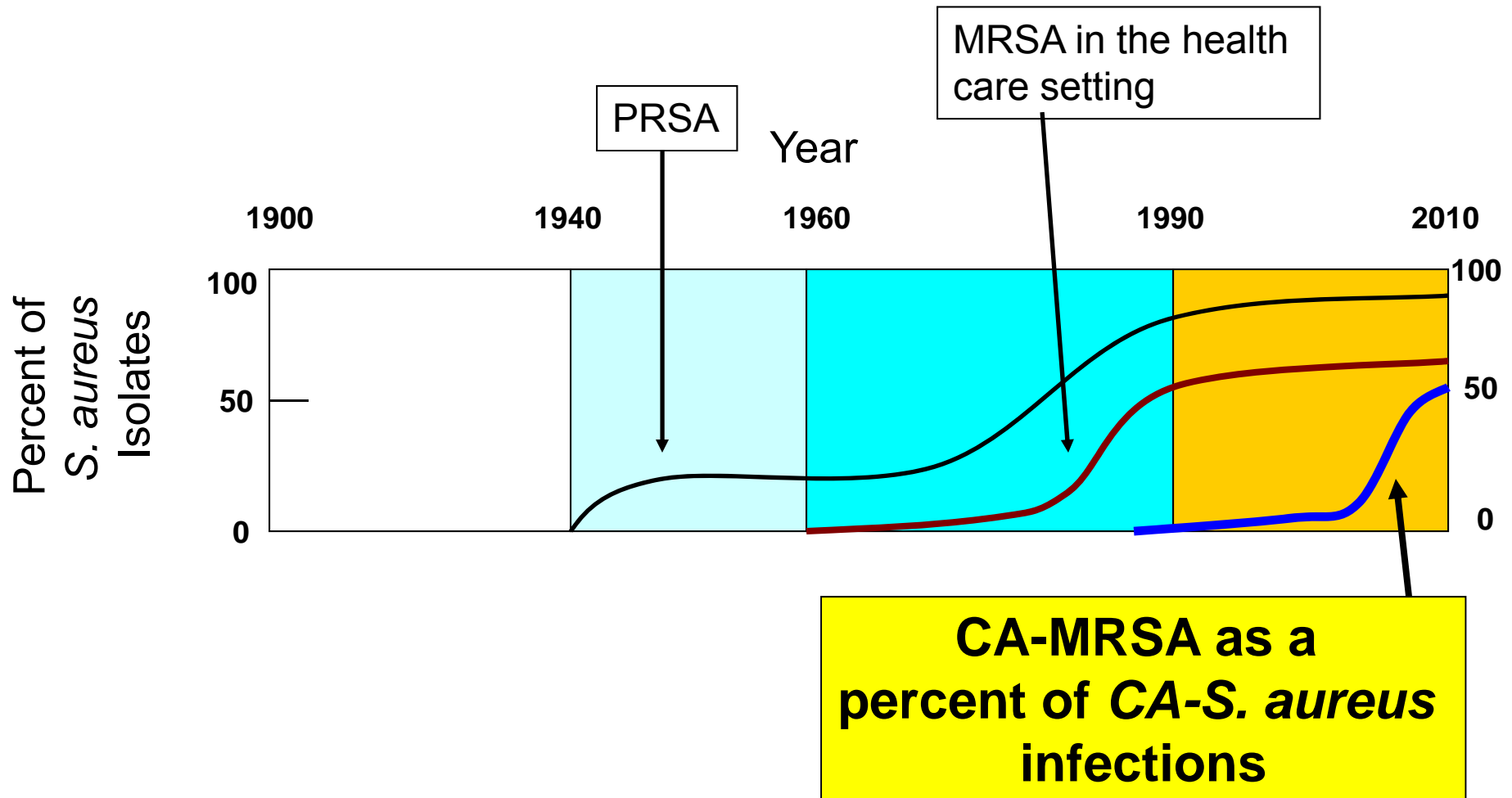
- Detroit: 160 pts w/ USA300 infections: 32.6% had recurrent or relapsed infection
 - Followed mean of 506 ± 134 (s.d.) days
- San Antonio, TX: 253 sequential pts in 1999-2003 w/ CO-MRSA SSTIs requiring surgical treatment
 - Recurrent SSTIs in 21% (53/253); 2-52 weeks after index SSTI

History of β -lactam Resistance in *S. aureus*





History of β -lactam Resistance in *S. aureus*



Methicillin-Resistant
Staphylococcus aureus

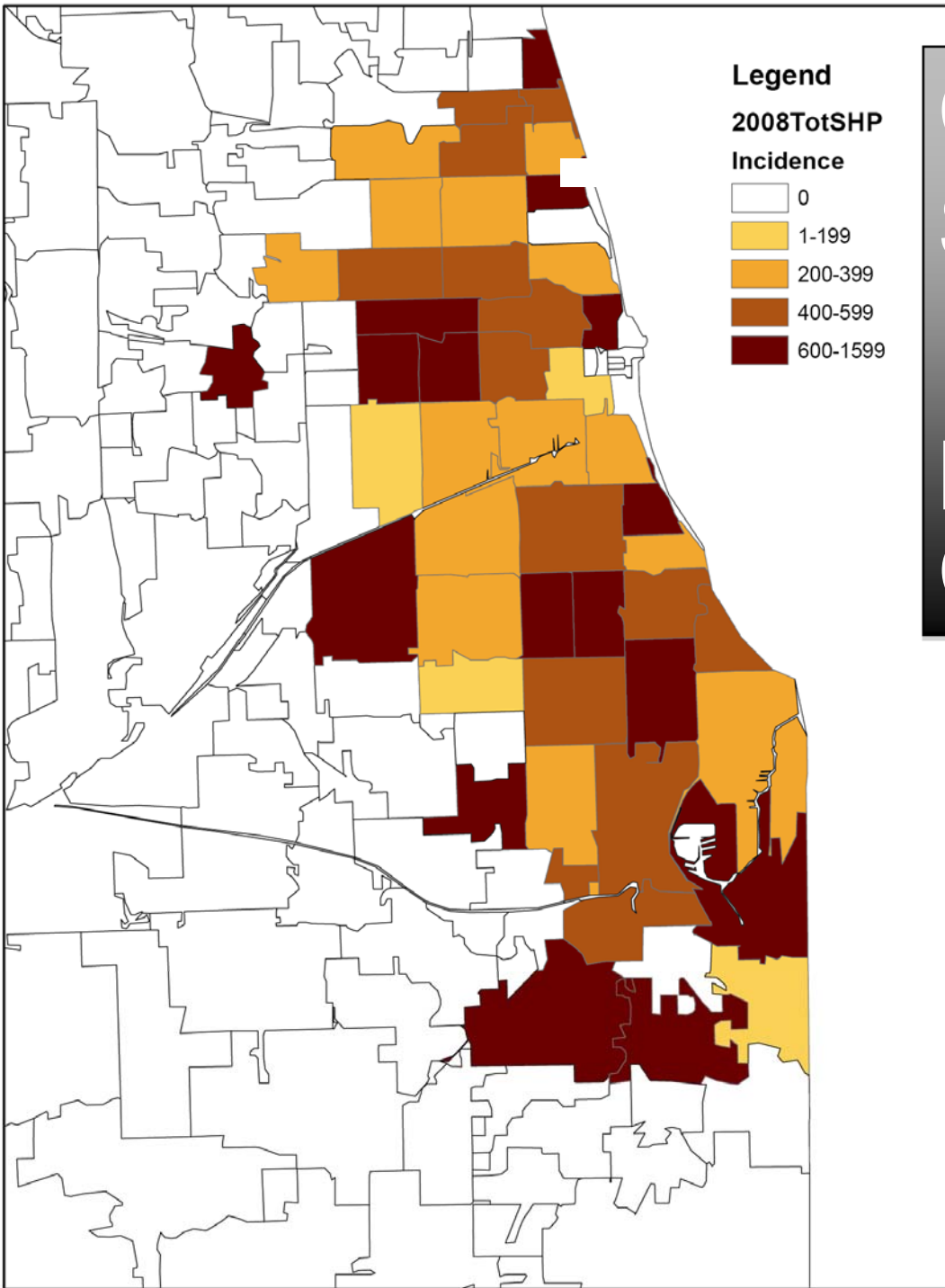
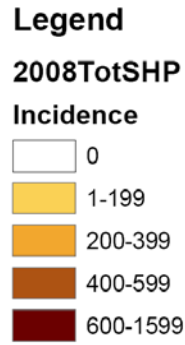


Community-Associated
(Community MRSA)

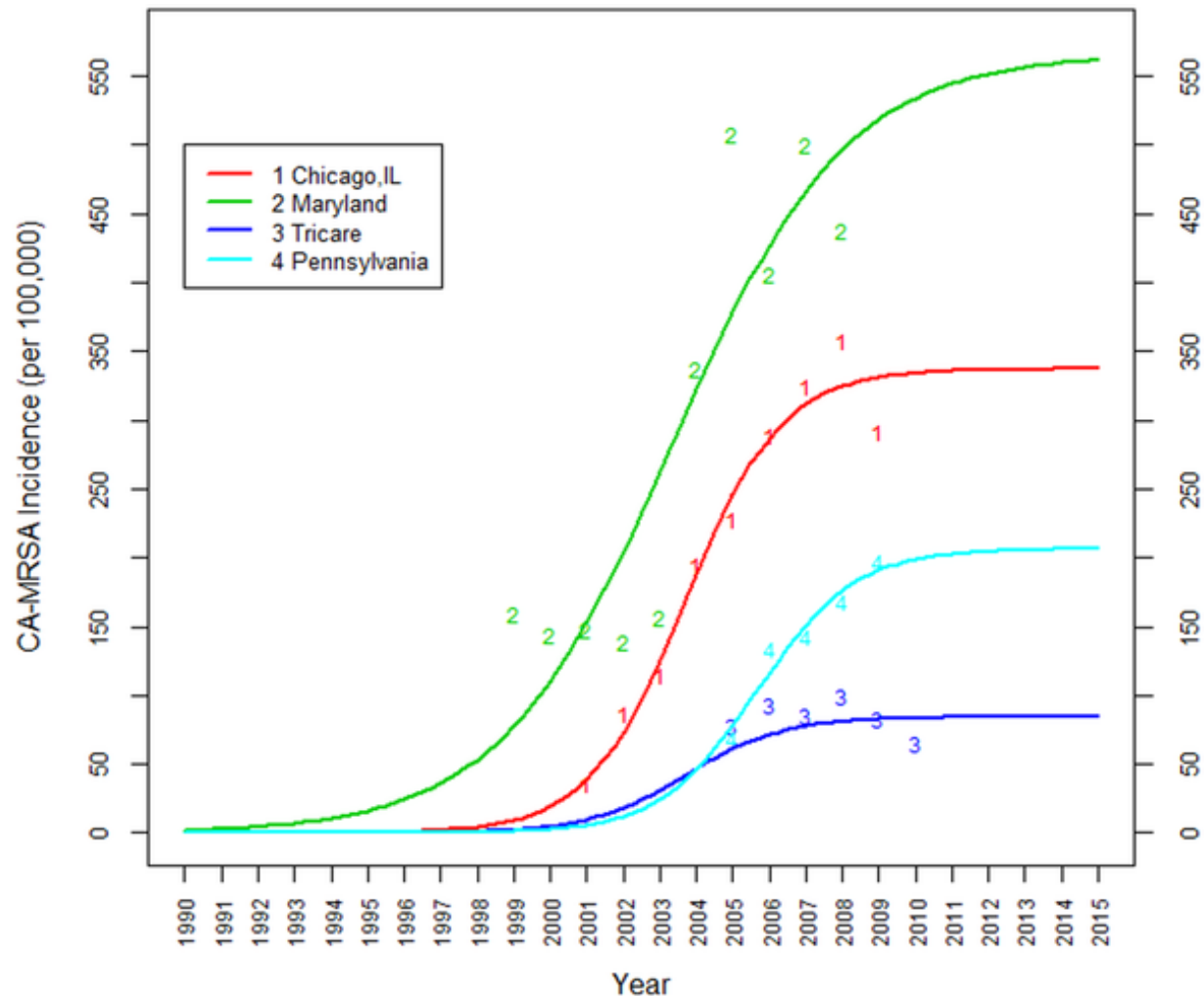
Health Care-Associated
(Hospital MRSA)

| | | |
|-----------------------|---|--|
| Health Care Contact | Yes/No | Yes |
| Sites of Infection | Usually Skin | Diverse |
| Antibiotic Resistance | Rarely Multi-drug | Often Many Classes |
| High-Risk Groups | Urban Children, IVDU, Detainees, Soldiers, Athletes | Sick, Recent Antibiotics, Catheters |

CA-MRSA SSTI Incidence per 100,000, Chicago



Estimated population incidence rate curves, based on the two CA-MRSA population incidence studies from Chicago, IL [9], Maryland [10], the population insured under Tricare [12] and Pennsylvania [11].



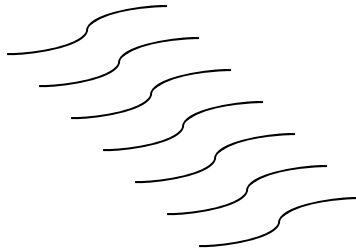
Dukic VM, Lauderdale DS, Wilder J, Daum RS, et al. (2013) Epidemics of Community-Associated Methicillin-Resistant *Staphylococcus aureus* in the United States: A Meta-Analysis. PLoS ONE 8(1): e52722. doi:10.1371/journal.pone.0052722
<http://www.plosone.org/article/info:doi/10.1371/journal.pone.0052722>

Multi-locus sequence typing (MLST)

Genomic Prep: DNA isolation

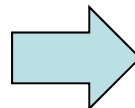


PCR for segments of 7 housekeeping genes



Sequence gene segments

ATACGATCG



Allele numerical assignments together determine a specific sequence type (ST)

ST121



Assign predefined polymorphism designations to the allele of each gene

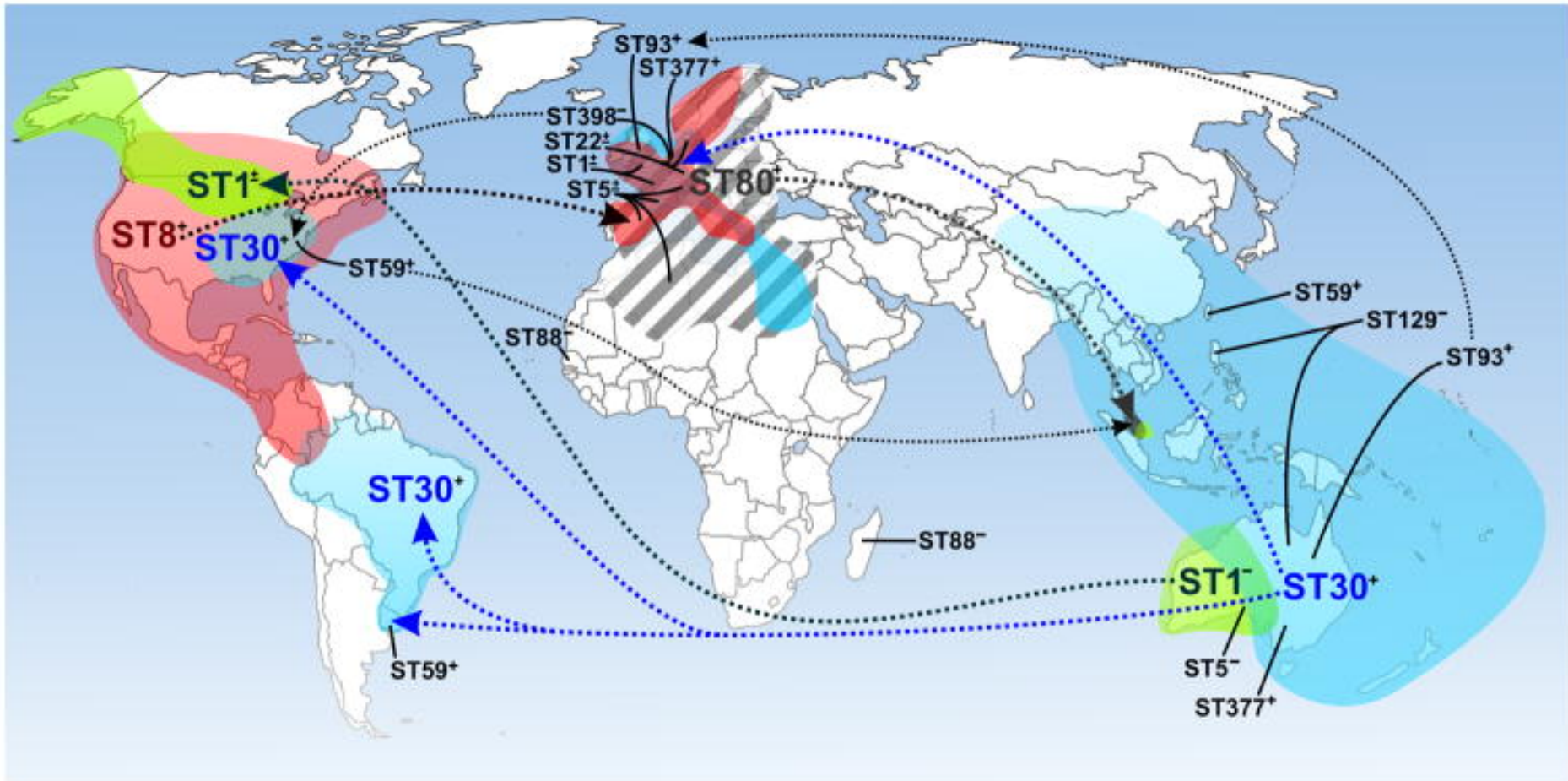
| | |
|-----------|-----|
| ATACGATCG | 21 |
| ATACGATCG | 5 |
| ATACGATCG | 18 |
| ATACGATCG | 2 |
| ATACGATCG | 21 |
| ATACGATCG | 131 |
| ATACGATCG | 21 |
| ATACGATCG | 41 |

MLST: Pros and Cons

- ~2700 types defined
- Common language
- Reproducible
- Stable
- Can relate strains to one another

- Not very high discriminatory index
- e.g., 85% MRSA in USA is ST8 or ST5!
- Difficulties in nomenclature of “clonal clusters”

CA-MRSA: Genetic Diversity



Chambers HF, et al. Lancet 2010;375:1557-68.

The Genomics Revolution, 2010-

- Whole Genome Sequencing
- Ultimate resolution
- Cheaper (\$10,000 → \$240) since 2002
- Functional information
- 80% core genome

- Uses
 - Track evolution
 - Time to common ancestor
 - MGEs
 - Importation
 - Understand transmission
 - Interspecies spread

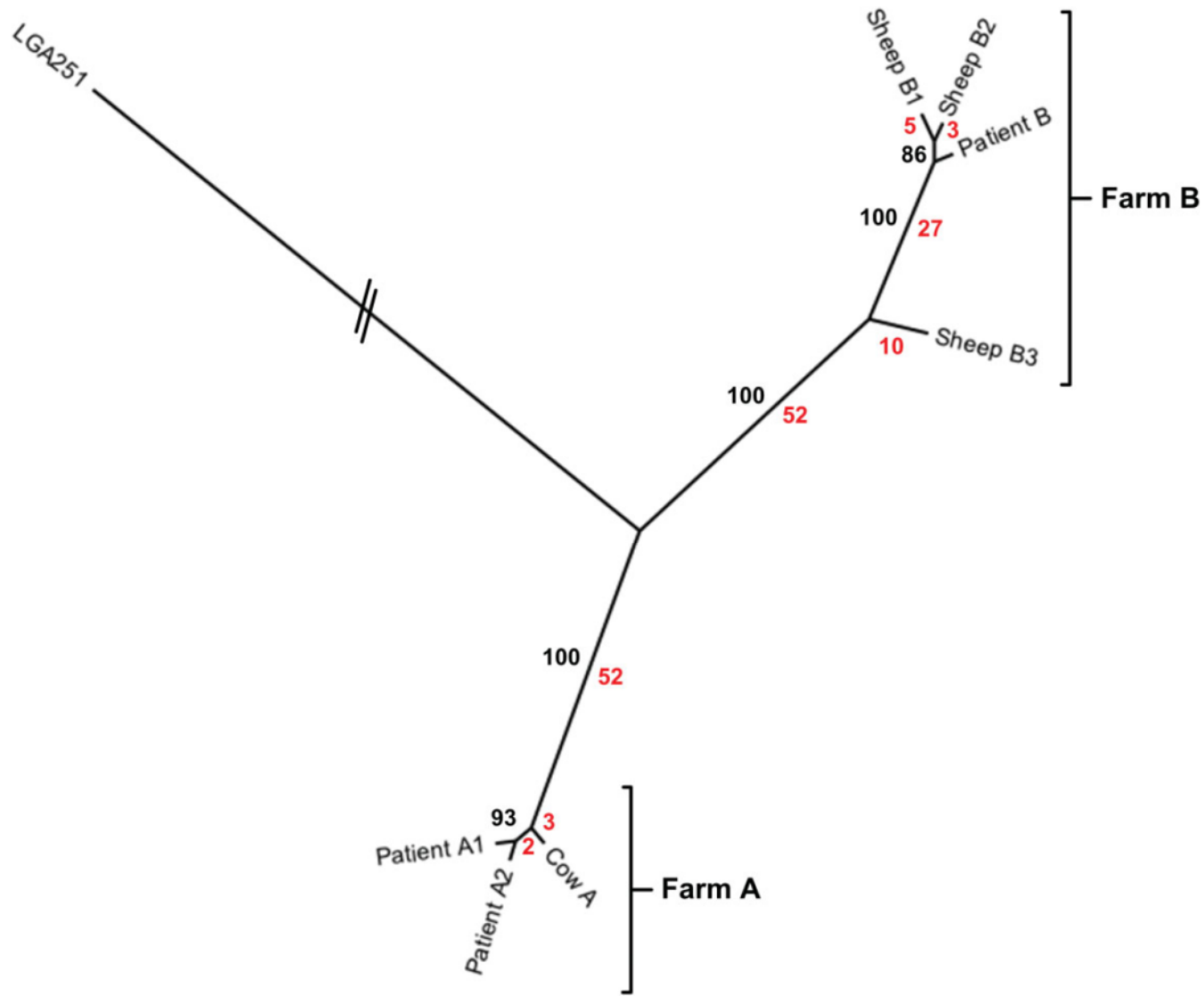


Figure 1. Phylogenetic relationships between human and animal isolates. Figure shows an unrooted maximum likelihood tree generated from SNPs in the core genome. The branch length for LGA251 has been trimmed. Bootstrap values for branches are shown in black. The number of differentiating SNPs for each branch is shown in red.

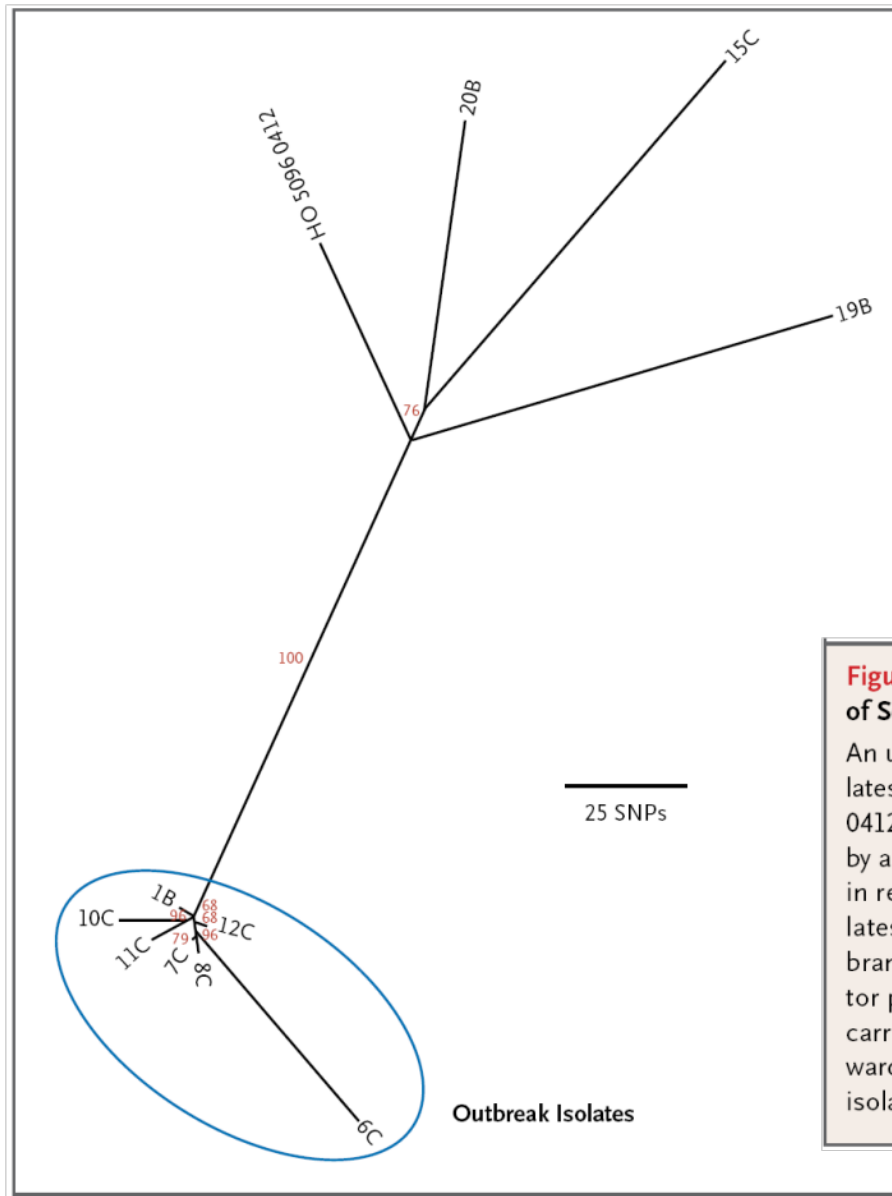
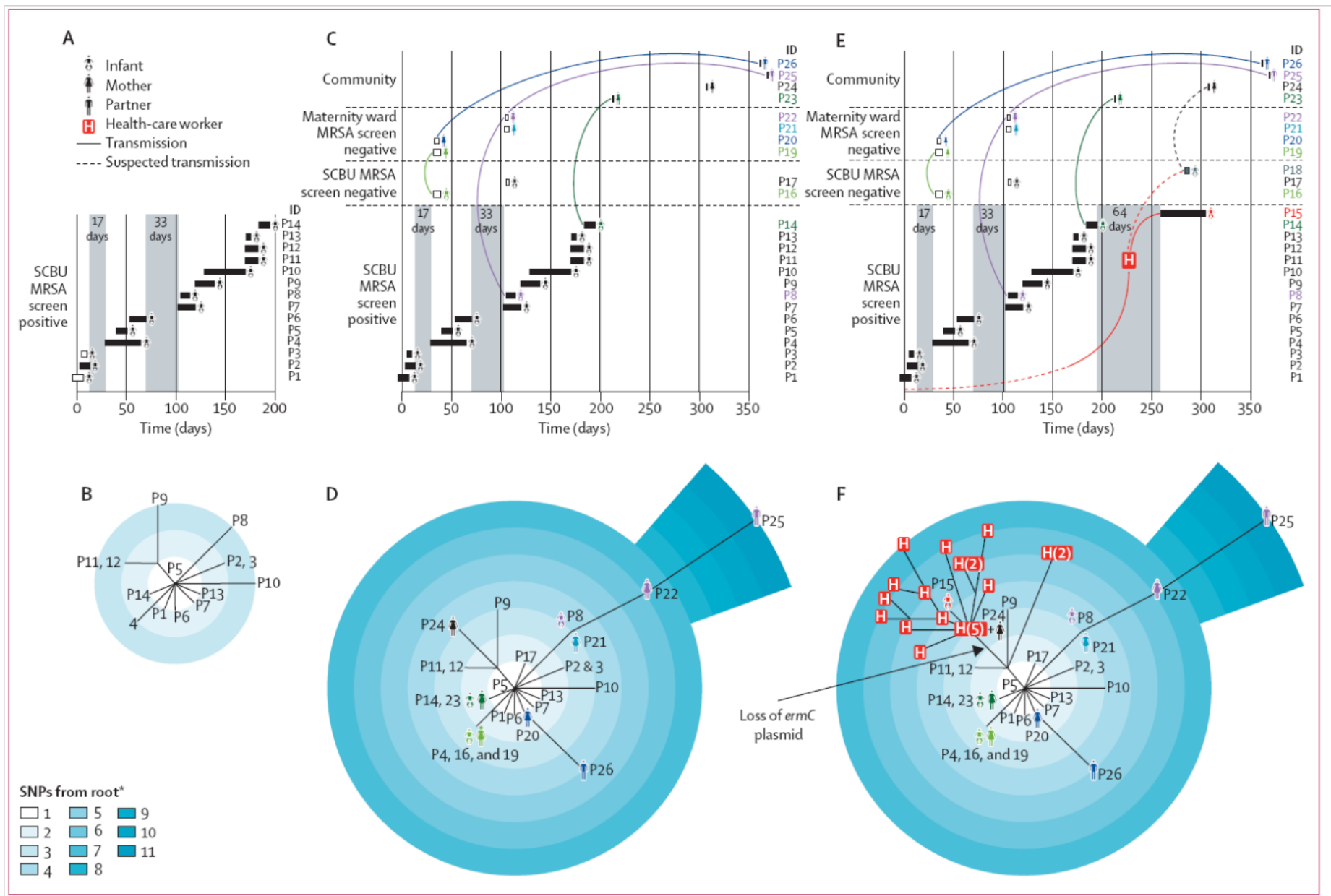


Figure 3. Results of Phylogenetic Analysis of the 10 MRSA Isolates of Sequence Type 22.

An unrooted maximum likelihood tree of the 10 sequence type 22 MRSA isolates identified, together with a reference sequence type 22 isolate (HO 5096 0412), is shown. The isolates are identified with the patient number followed by a B for bacteremia or a C for carriage of MRSA. Bootstrap values are shown in red. Outbreak isolates are circled in blue. Six of the seven outbreak isolates clustered closely together. The seventh isolate (6C) had an extended branch length that could be explained by the fact that it had a hypermutator phenotype. The remaining sequence type 22 isolates associated with carriage by an infant in the NICU (15C) or bacteremia in patients on other wards of the same hospital (19B, 20B) were distantly related to the outbreak isolates and to each other. SNP denotes single-nucleotide polymorphism.



Harris SR, et al. Lancet Infect Dis 2013;13:130-6.

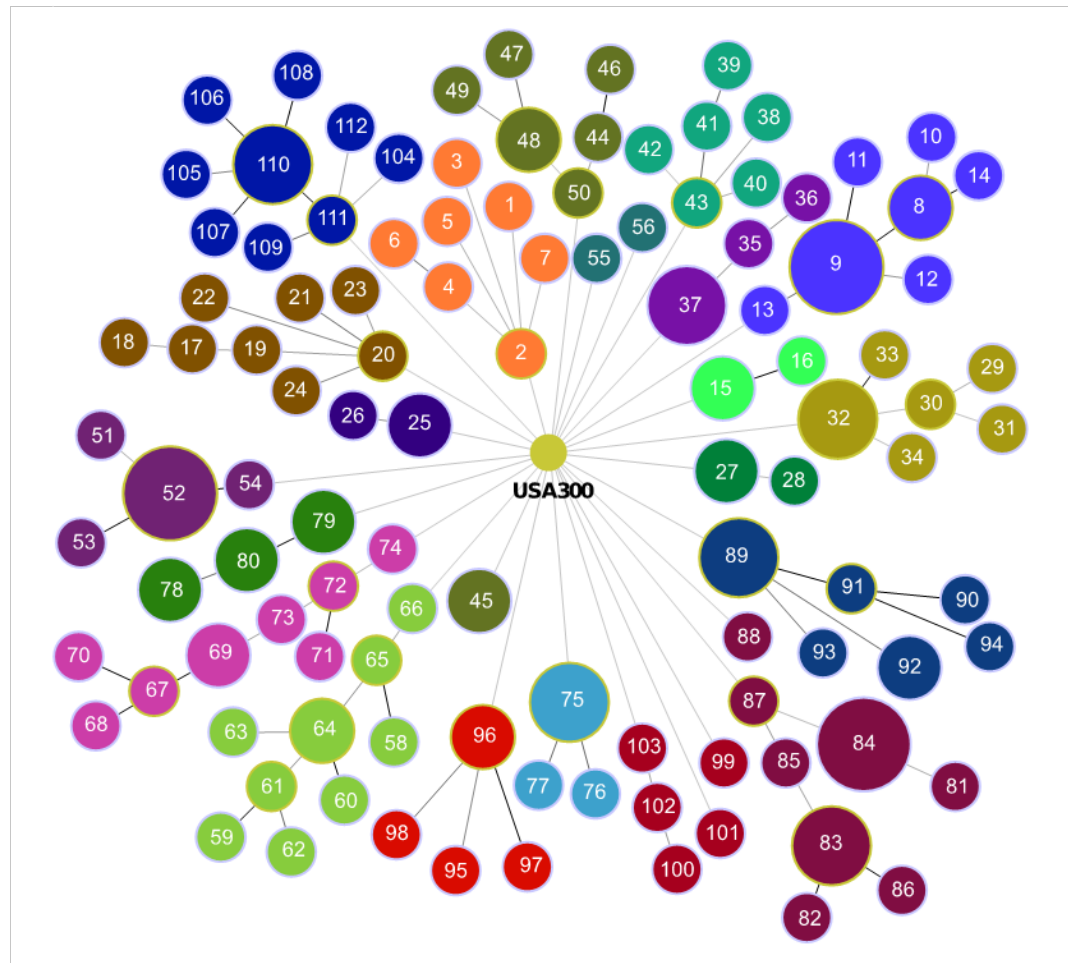


Figure-2: Minimum spanning tree showing genetic relationship among 147 *S. aureus* strains with MLST-8. Each circle represents a haplotype and color represents the household to which it belongs. The haplotypes from 1 to 56 are from UCLA and 57 to 112 are from Chicago households. A solid black connecting line between any two haplotypes indicates that they differ by only a single SNP, whereas grey lines indicate >1 SNP difference.

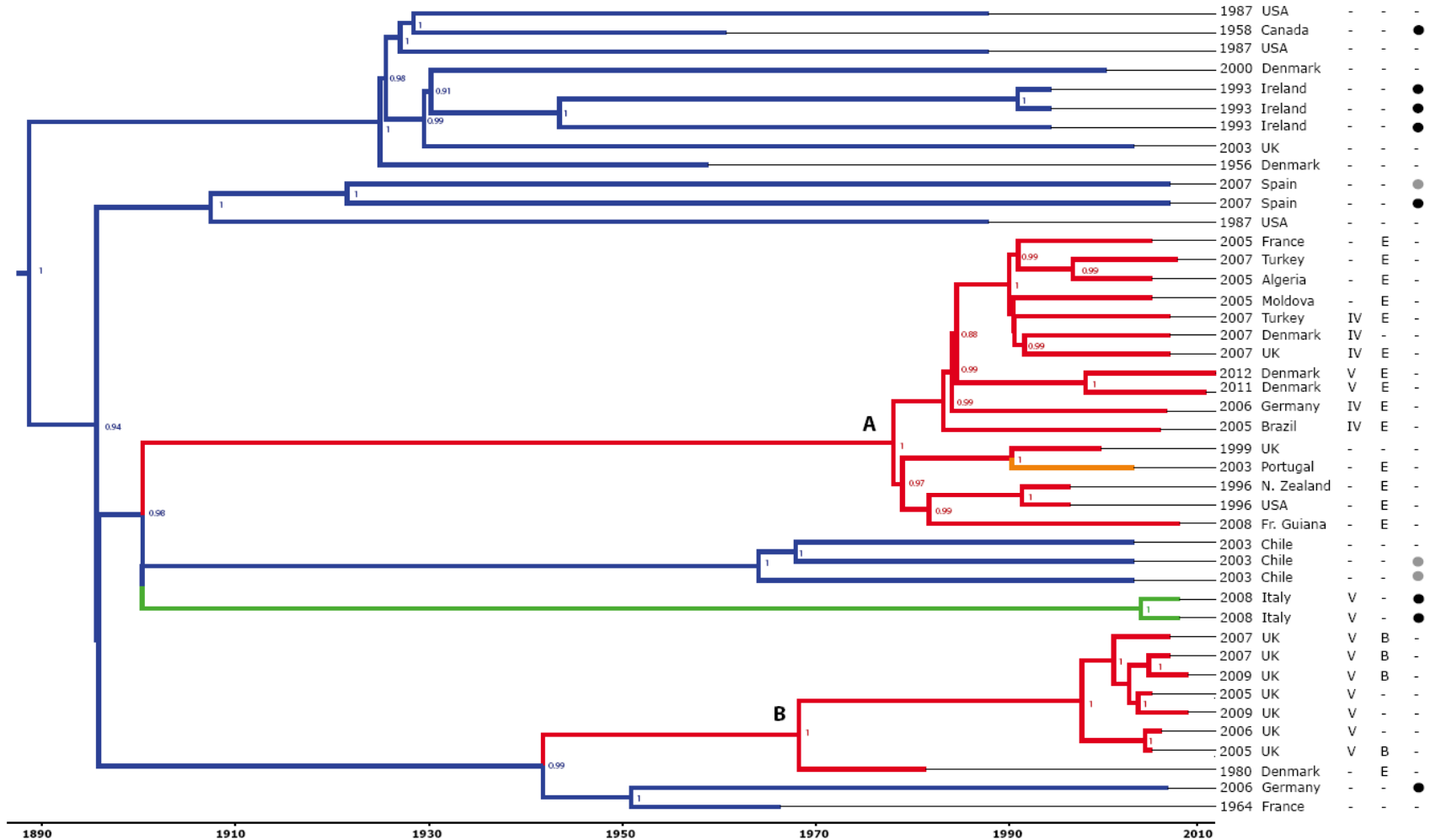


FIG 1 Identification of human epidemic *S. aureus* clones descended from bacteria that made livestock-to-human host jumps. The Bayesian phylogenetic reconstruction of the CC97 lineage is shown. The tree is based on core genome alignment with branches color coded according to the host species association (blue, bovine; green, porcine; orange, caprine; red, human) and date and country of origin of each isolate indicated. The presence or absence (–) of β -toxin phage IEC variants B and E and SCCmec type IV or V is indicated by the appropriate letter, and the presence of *S. aureus* pathogenicity island (SaPI)-encoded *vwb* or phage-encoded *lukM/lukF* is denoted by black and gray circles, respectively. The branch lengths are scaled according to the time scale bar (years) and the posterior probability values are indicated at each node. Clades A and B are shown.

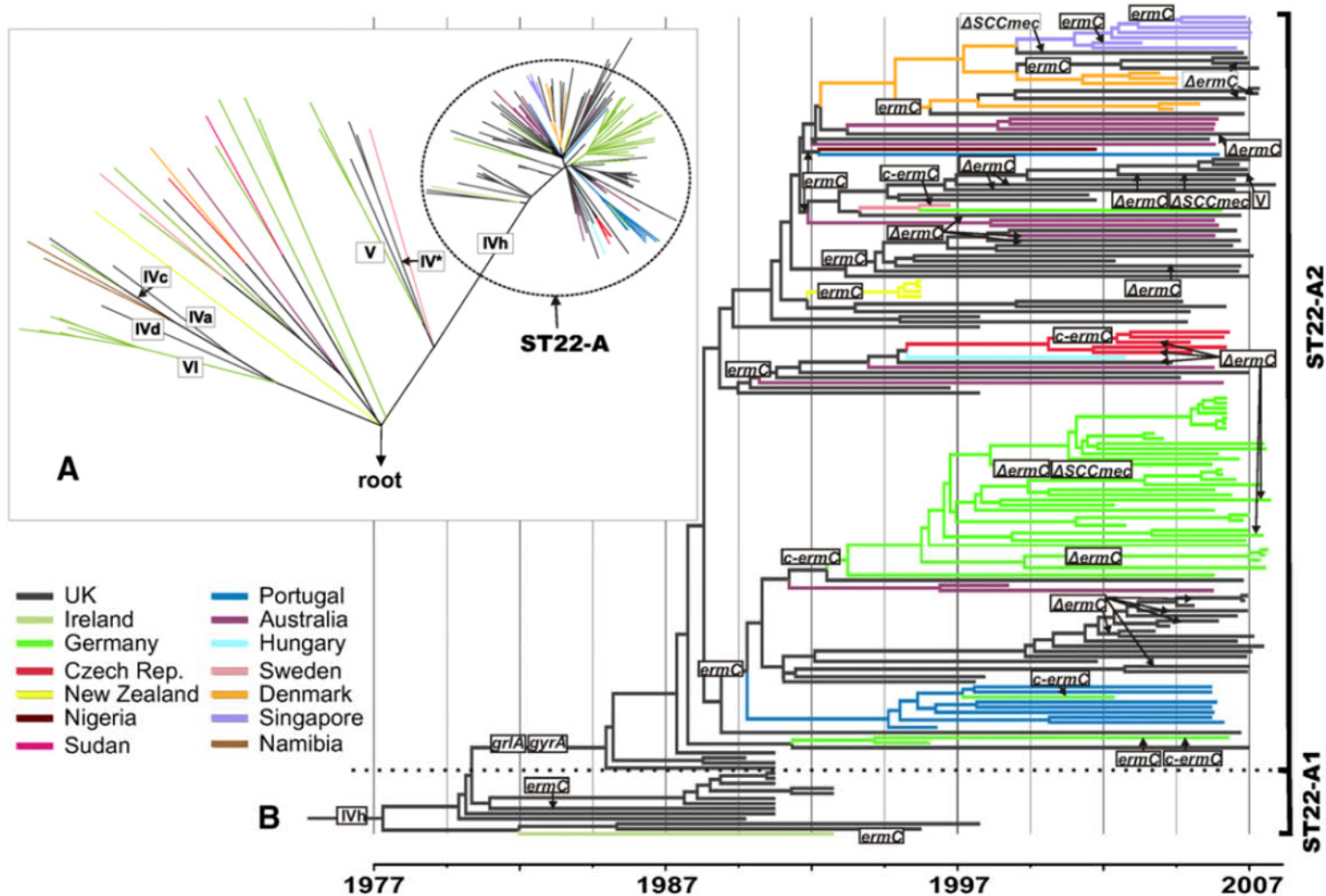
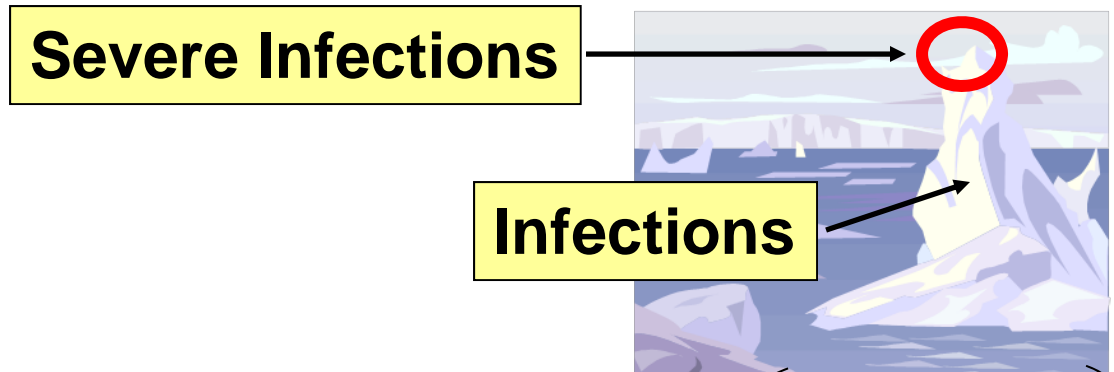


Figure 1. Phylogeny of ST22 and the emergence of MRSA clones. (A) Maximum likelihood phylogenetic tree of ST22 isolates. The tree was rooted by using the distantly related *S. aureus* isolate MSA476 as an outgroup. Colors indicate the isolates' countries of origin. Roman numerals indicate acquisitions of structurally different SCCmec elements, which cause methicillin resistance. (B) Maximum clade credibility tree of the ST22-A clade based on BEAST analysis using a variable clock rate (uncorrelated lognormal) model. Tips of the tree are constrained by isolation dates, the time scale is shown at the bottom. Gains and losses (Δ) of genetic determinants for resistance to methicillin (SCCmec IVh), fluoroquinolones (point mutations in *grlA* and *gyrA*), erythromycin (plasmid-encoded *ermC*), and clindamycin (mutations in *ermC* leader peptide region, *c-ermC*) have been mapped on the tree by applying the parsimony criterion.



MRSA: Where should we intervene?



COLONIZATION RESERVOIR:
Nose?, skin?, environment?

MRSA and the Environment

- Beach Water
 - Agriculture
 - Sewage effluents
 - Lives for weeks on inanimate objects
- Unknowns
 - How common?
 - How important?
 - How to study?
 - Currency of genes or of bacteria?
 - Livestock?
 - Food?

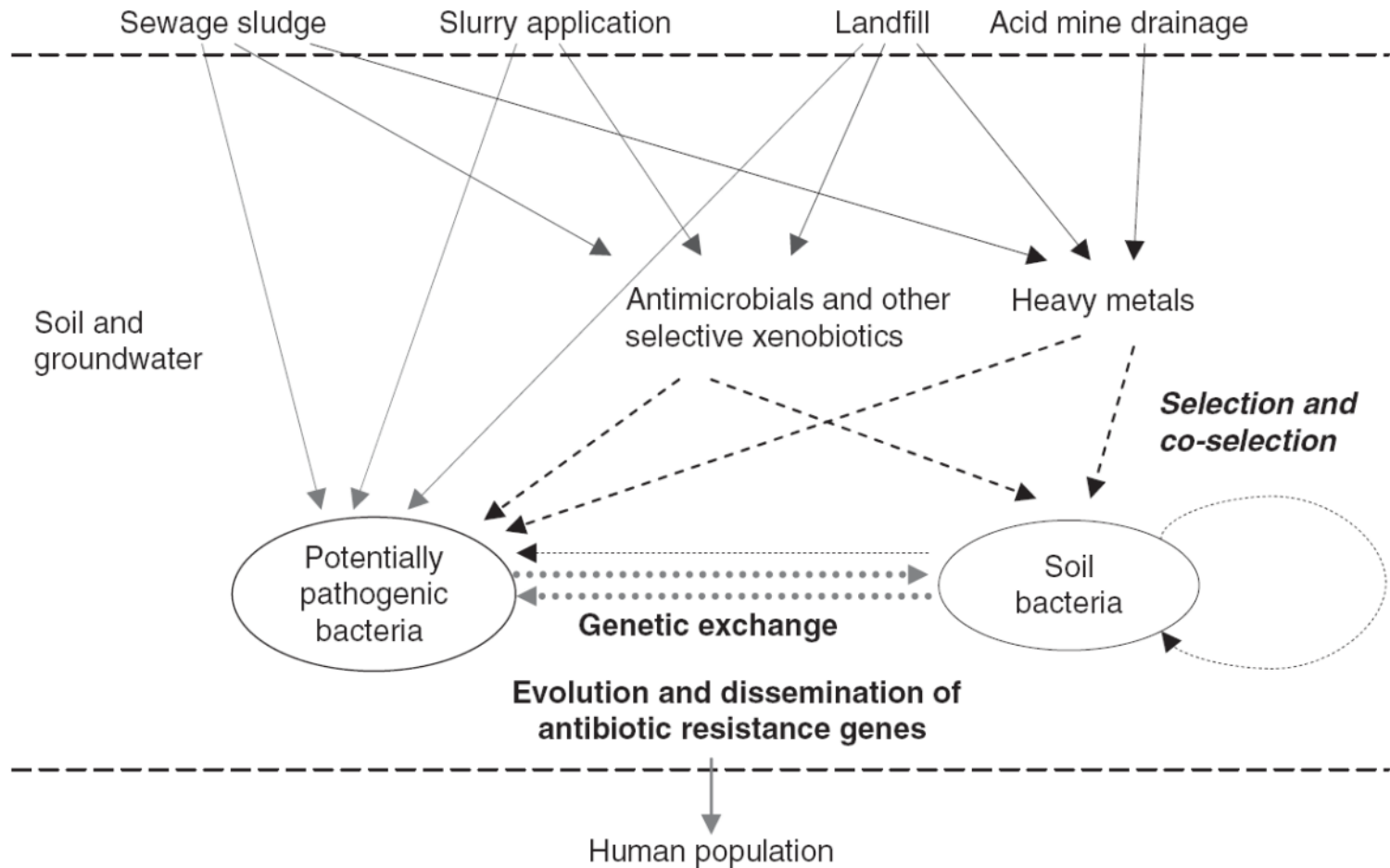


FIGURE 7.1 Anthropogenic sources of bacterial pathogens, pharmaceuticals, and heavy metals, which, in conjunction with indigenous soil bacteria, provide a mixture of genes and selective pressure for selection or coselection of antibiotic resistance.

Sewage, Water and Antibiotic Resistance Genes

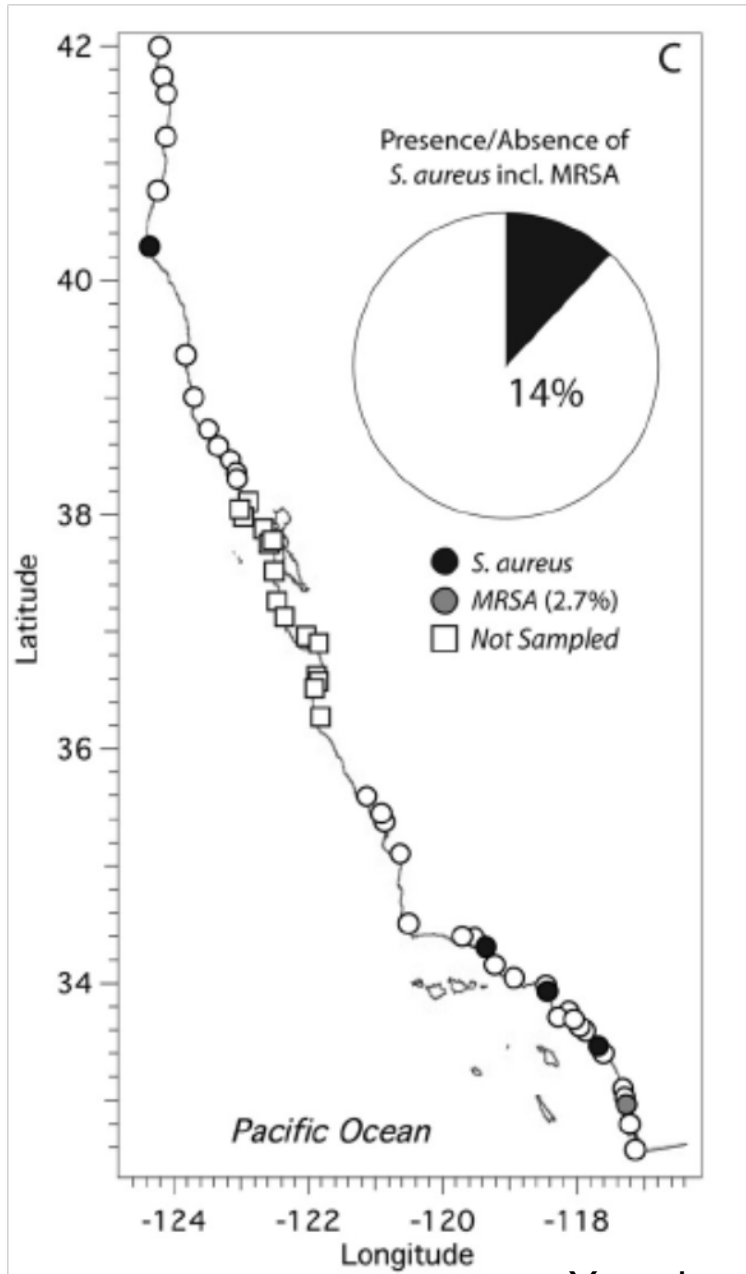
- Dozens of studies recently on gram-negative bacteria in sewage and soil
 - ESBLs
 - Carbapenemases
 - Many studies in Europe
- Less on MRSA
 - Exception is in agricultural settings
 - Pigs are a major reservoir of MRSA

Municipal wastewater

- Sweden, 2008
- 2 month collection, 4 sites at a WWTP
- MRSA identified in inlet and activated sludge only
- Processing removed most MRSA
- May select for more resistant strains and PVL+ strains

Washington Beaches

- Water and intertidal sand samples from 10 beaches in Washington state in 2008
- 5 multidrug-resistant MRSA isolates obtained (among 51 Staph species)
- Genotypes similar to healthcare-associated strains



California beach sand

- 53 beaches tested, Oct. 2009
- MRSA in 3% of samples
- Much less common than *E. coli*

California Beach Seawater & Sand

- 3 Southern California beaches
- MRSA in low concentration in 1.6% of 366 seawater samples & 2.7% of 366 sand samples
- *S. aureus* in water correlated with *S. aureus* in sand, water temp, enterococci in water, & number of swimmers

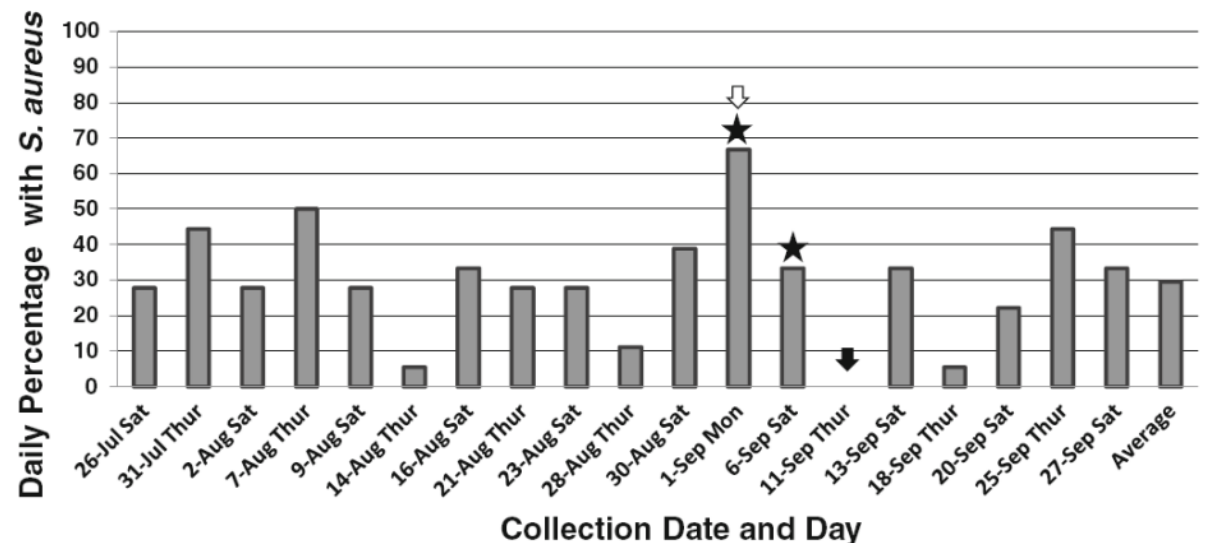


Goodwin KD, et al. Water Res 2012;46:4195-207.

Florida Beaches and Beach Water

- 1001 water and 36 intertidal sand samples
- 2007-2009
- 1334 MSSA & 22 MRSA isolates obtained
- 17/22 MRSA were USA300; 21/22 were PVL+
- More *S. aureus* corr. with more bathers

Fig. 1 Daily percentage of ambient water samples from which MSSA or MRSA were isolated. Stars denote the days that MRSA were isolated. *Open arrow* denotes holiday and *solid arrow* indicates day with inclement weather



Great Lakes Beaches

- Study of 273 beach water and 22 tributary samples in 2010 at 12 beaches (none in IL)
- Isolated *mecA* and *femA* (likely MRSA) from beach water at 8/12 beaches
- MRSA may be more common closer to urban areas in beach water



Summary

- HA-MRSA
- CA-MRSA
 - New risk groups / New syndromes
 - Variable incidence
- Genotyping & Whole Genome Sequencing
- Environmental contamination



Photo Credit: Gregory Moran, M.D.

Photo Credit: Major Kirk Waibel, MD

