SUPER BUGS!

HOW TO TREAT THE WORST OF THE WORST INFECTIOUS PATHOGENS!

Vicky Shah, PharmD, BCPS

Associate Professor of Clinical Sciences

Chair of Service and Clinical Site Relationships

Roosevelt University College of Science, Health and Pharmacy

Disclosures

No financial or other conflicts to disclose

Learning Objectives – Pharmacists

- Discuss the importance of appropriate antibiotic use to prevent multi-drug resistant pathogens
- Review culture & sensitivity reports
- Describe appropriate treatment regimens for KPC, CRE, ESBL, etc.
- Identify the coverage spectrum of newer antibiotic agents which can be utilized for multi-drug resistant pathogens

Learning Objectives – Technicians

- Discuss the importance of appropriate antibiotic use to prevent multi-drug resistant pathogens
- Recognize new antibiotic agents being used

Pre-Test Question 1

Which of the following combinations is the appropriate treatment for NMD-1 Carbapenemase-Producing Enterobacteriaceae? SELECT ALL THAT APPLY

a. Piperacillin/Tazobactam
b. Tigecycline + Amikacin + Colistin
c. Amoxicillin/Clavulanic Acid
d. Vancomycin
e. Ceftolozane/Tazobactam

Pre-Test Question 2

Which of the following is the first line treatment for ESBL E. Coli infections?

a. Carbapenems
b. Fosfomycin
c. Piperacillin/Tazobactam
d. Amoxicillin/Clavulanic Acid

Pre-Test Question 3

Which of the following is considered first line treatment for hospital acquired MRSA infections?

a. Daptomycin
b. Linezolid
c. Clindamycin
d. Vancomycin

Pre-Test Question 4

Which of the following medications can be used for clostridium difficile infections?

a. IV Vancomycin
b. Oral Vancomycin
c. IV Zosyn
d. Oral Augmentin

Infection

Invasion of an organism's body tissues by disease-causing agents, their multiplication, and the reaction of host tissues to these organisms/toxins.

Caused by viruses, prions, bacteria, parasites, fungi, ticks, mites, insects, etc.

The host's immune system will usually provide protection against invasive organisms, which may lead to biological changes within the body (i.e. temperature increase, increased WBC, etc.)

Spellberg B, Guidos R, Gilbert D, et al. Infectious Diseases Society of A. The epidemic of antibiotic-resistant infections: A call to action for the medical community from the Infectious Diseases Society of America. *Clin Infect Dis* 2008;46:155–64.

HOW CAN INFECTIONS BE TRANSMITTED FROM PERSON TO PERSON?

WHAT ARE SOME THINGS THAT CAN BE DONE TO PREVENT THE SPREAD OF INFECTION?

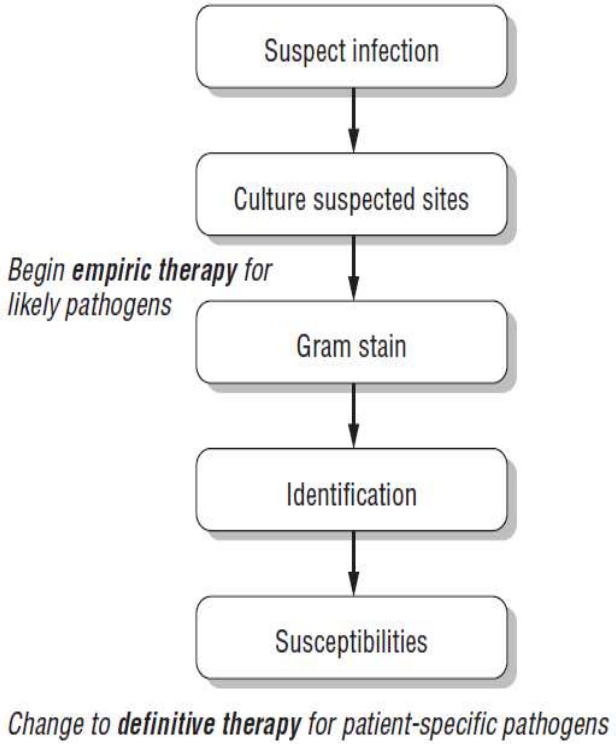
Definitions

Empiric Therapy

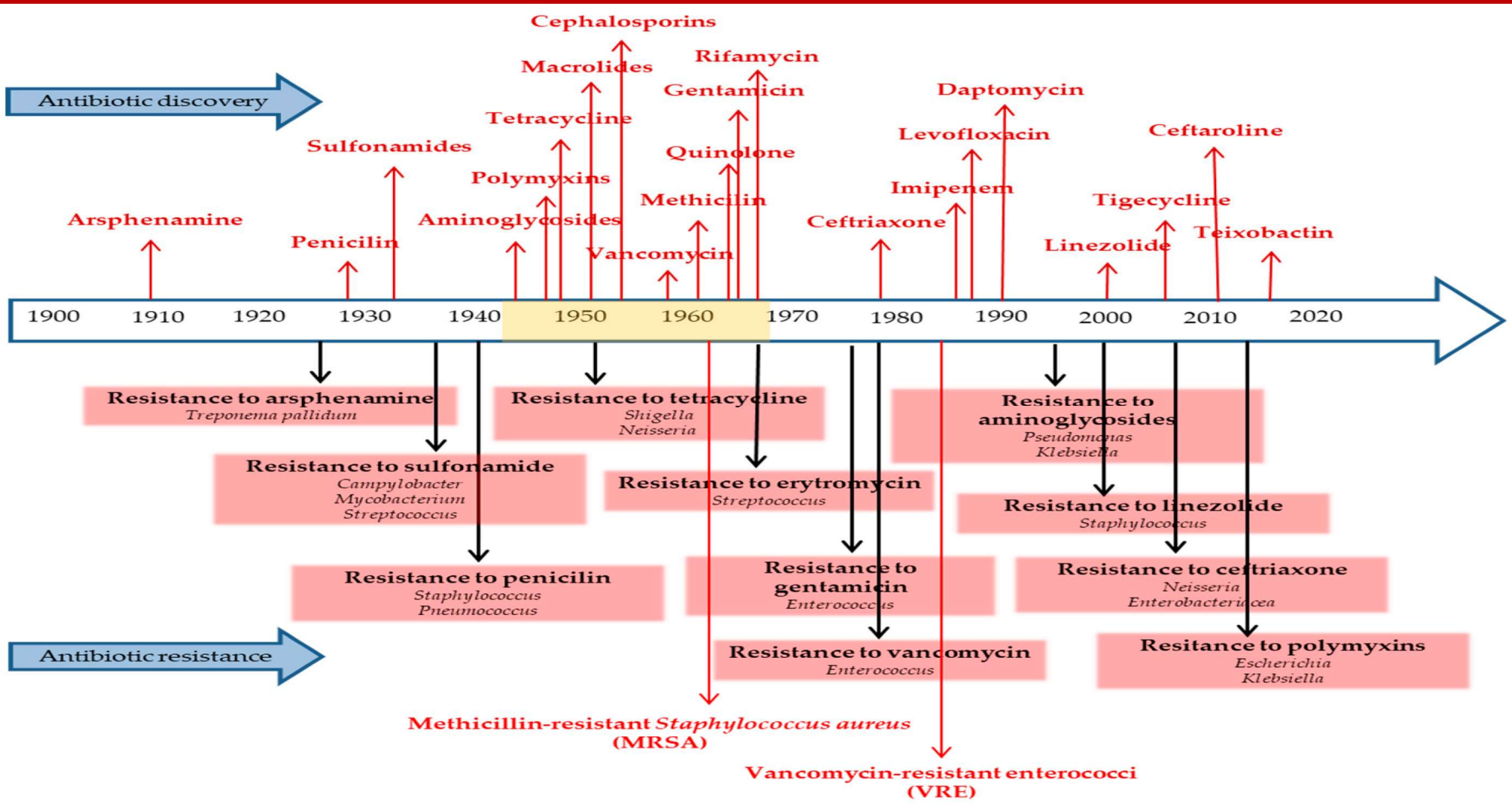
- Infecting organism(s) not yet identified
- More "broad spectrum"

Definitive Therapy

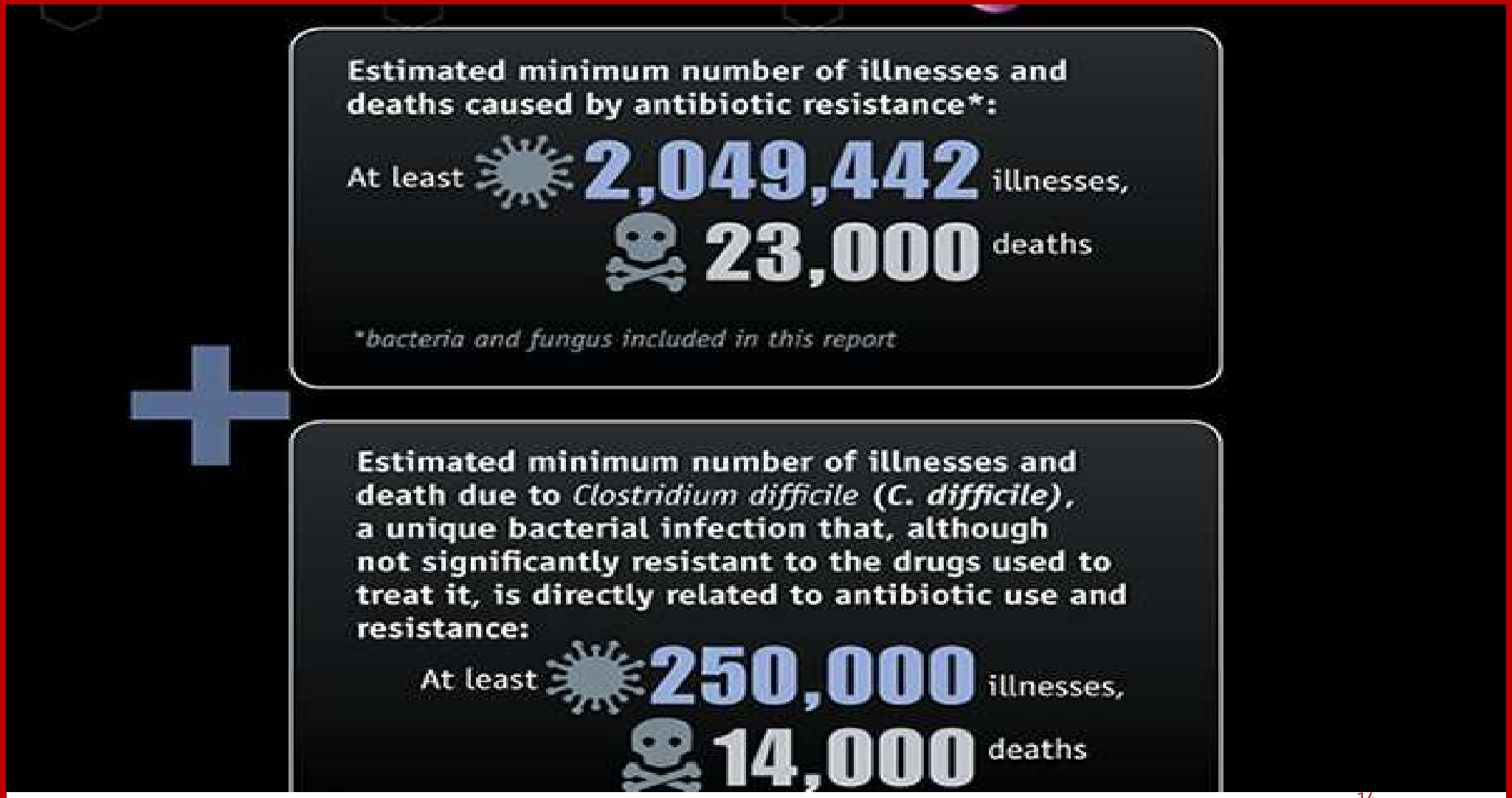
- Organism(s) identified and specific therapy chosen
- More "narrow" spectrum



Spellberg B, Guidos R, Gilbert D, et al. Infectious Diseases Society of A. The epidemic of antibiotic-resistant infections: A call to action for the medical community from the Infectious Diseases Society of America. *Clin Infect Dis* 2008;46:155–64.

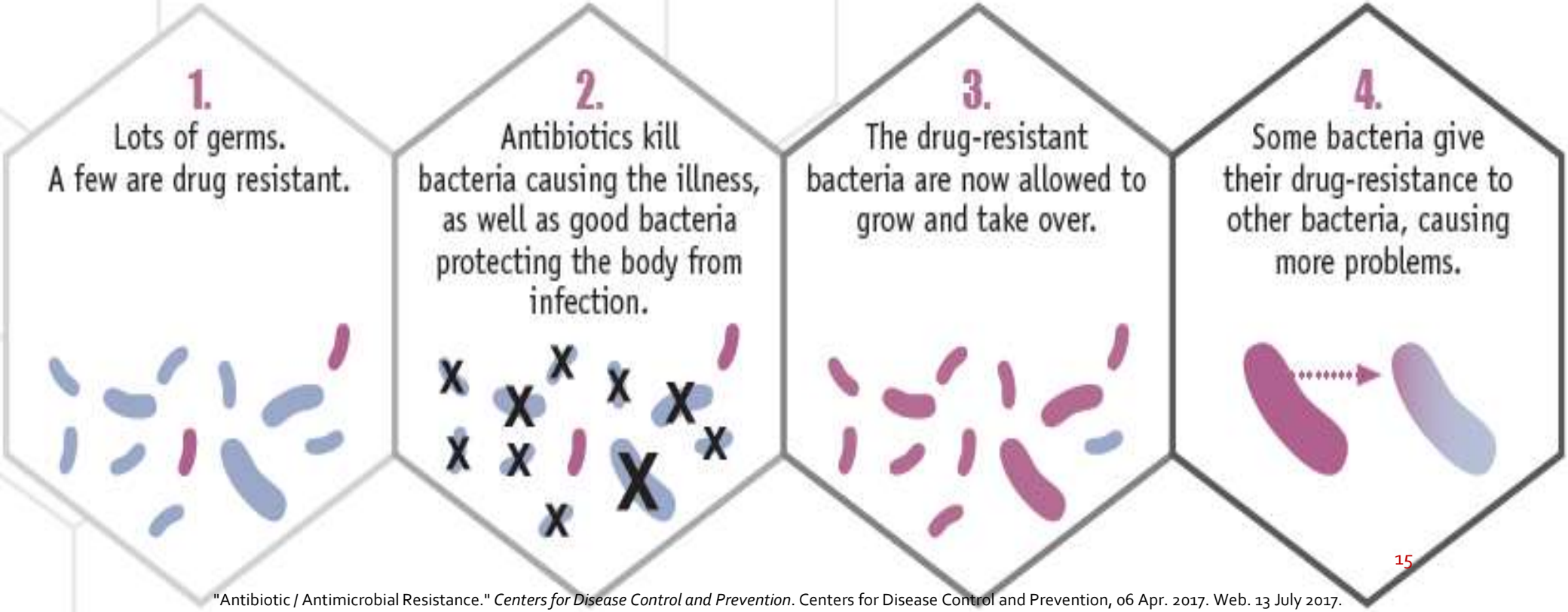


"Antibiotic / Antimicrobial Resistance." *Centers for Disease Control and Prevention*. Centers for Disease Control and Prevention, 2019.

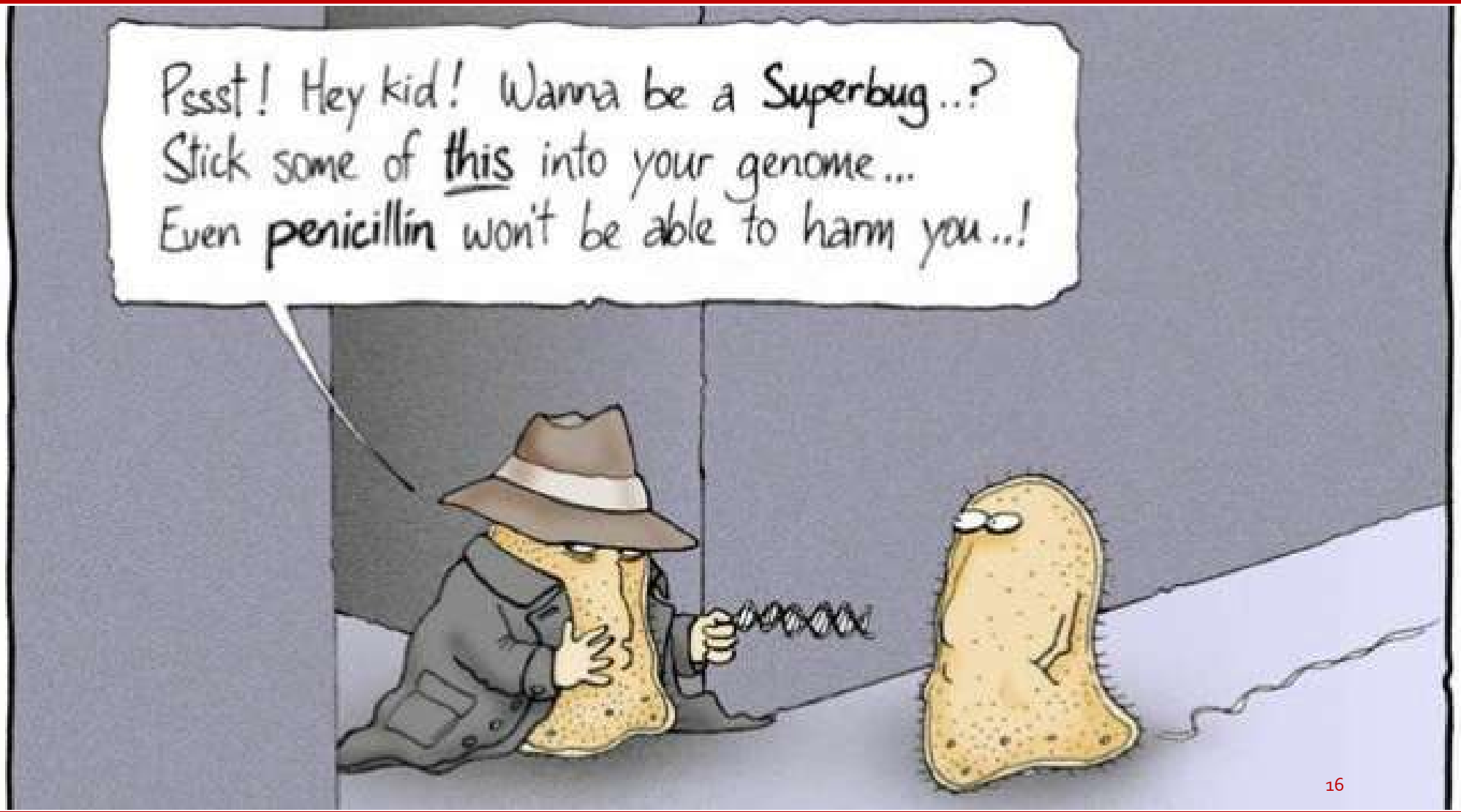


"Antibiotic / Antimicrobial Resistance." *Centers for Disease Control and Prevention*. Centers for Disease Control and Prevention, 06 Apr. 2017. Web. 13 July 2017.

How Antibiotic Resistance Happens

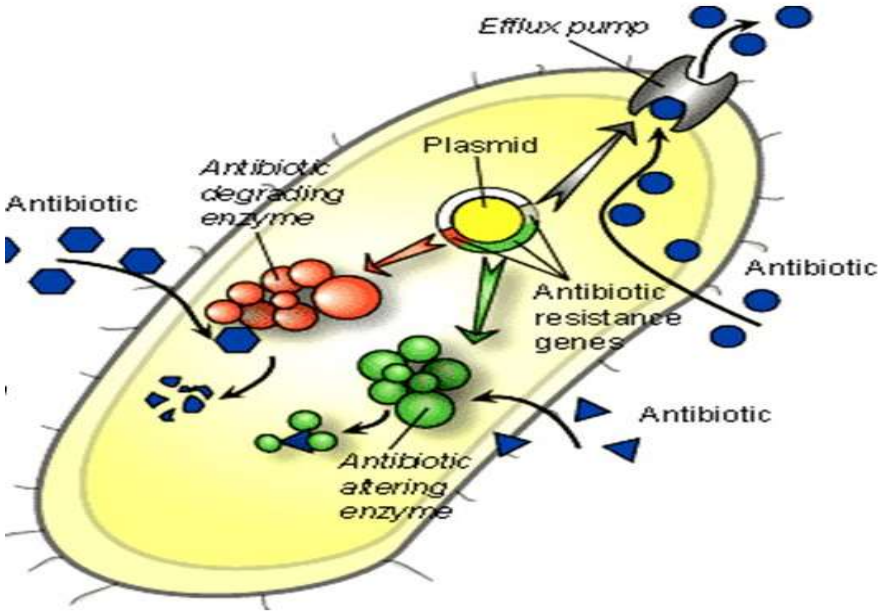


"Antibiotic / Antimicrobial Resistance." *Centers for Disease Control and Prevention*. Centers for Disease Control and Prevention, 06 Apr. 2017. Web. 13 July 2017.

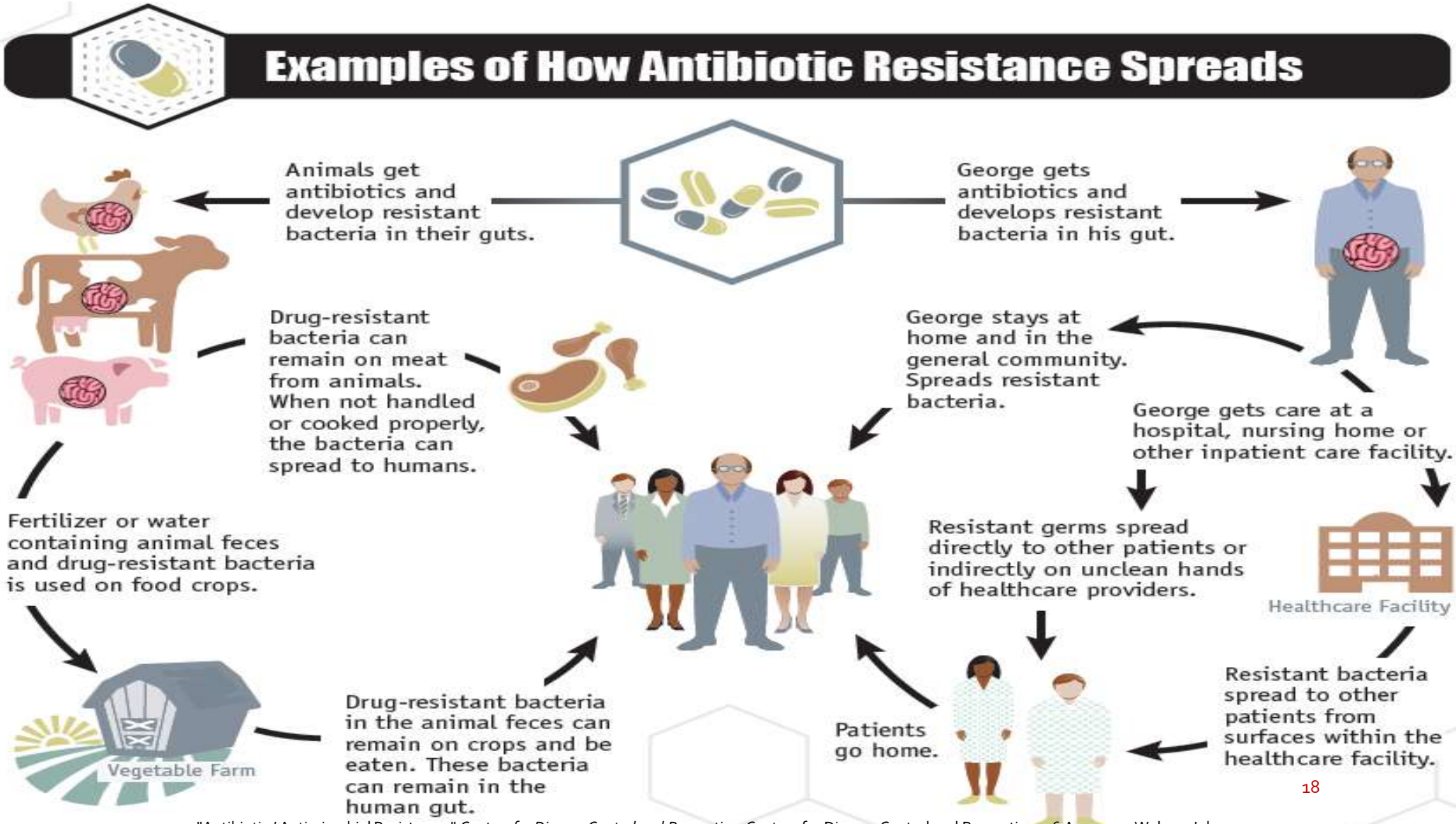

Pssst! Hey kid! Wanna be a Superbug..?
Stick some of this into your genome...
Even penicillin won't be able to harm you..!

Mechanisms of Antibiotic Resistance

- Efflux pumps
- Hydrolysis
- Reduced uptake
- Enzymatic modification



Davies J. (1994). Inactivation of antibiotics and dissemination of resistance genes. Science 264:375-82.



"Antibiotic / Antimicrobial Resistance." *Centers for Disease Control and Prevention*. Centers for Disease Control and Prevention, 06 Apr. 2017. Web. 13 July 2017.

Pharmacist's Role

Education!

- Only use when antibiotics are prescribed by physician
- Always take full prescription even if patient feels better
- Never use left over antibiotics
- Never share antibiotics

Appropriate Antimicrobial Stewardship

- Assist physicians in selecting appropriate antibiotics
- Avoid antibiotics for viral/fungal infections

Avoid inappropriate use of antibiotics

- Recommend narrow spectrum antibiotics after cultures are completed
- Avoid duplicate coverage unless needed
- Utilize hospital specific antibiograms

CDC . Active Bacterial Core Surveillance Methodology (2012) . http://www.cdc.gov/abcs/index .html[Accessed 5/23/2013]

Susceptibility Testing

MIC = minimum inhibitory concentration

- Minimum concentration required to stop bacterial growth

Breakpoint = MIC cutoff for susceptibility

- MIC below breakpoint → susceptible
- MIC above breakpoint → resistant

- Determined by CLSI or EUCAST based on in vitro testing and physiologically achievable concentrations at usual doses

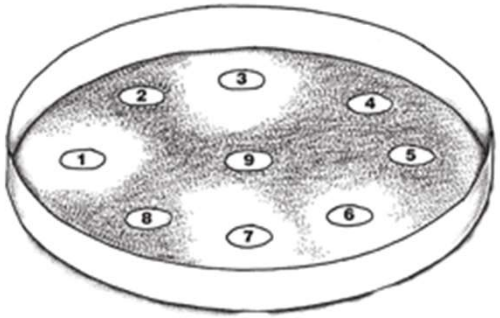
Graman PS, Menegus MA. Microbiology laboratory tests. In: Betts RF, Chapman SW, Penn RL, eds. *A Practical Approach to Infectious Diseases*. Philadelphia, PA: Lippincott Williams & Wilkins, 2003:929–956.

Susceptibility Report

S = susceptible	• Reasonable to expect efficacy
I = intermediate	• Activity likely insufficient • In some cases may have some efficacy at maximum doses • "Susceptible-dose dependent" (S-DD)
R = resistant	• No clinically useful activity • In multidrug resistant organisms, may still have a role for synergy

Graman PS, Menegus MA. Microbiology laboratory tests. In: Betts RF, Chapman SW, Penn RL, eds. *A Practical Approach to Infectious Diseases*. Philadelphia, PA: Lippincott Williams & Wilkins, 2003:929–956.

Cultures



With antibacterials 1, 3, 6 & 7, the bacteria show a sensitivity to an antibiotic. The bacteria are resistant to medications 2, 4, 5, 8 & 9.

Examples of Antibiotic Susceptibility Breakpoints

Organism Antibiotics	Susceptible	Intermediate	Resistant
E. coli			
Cefepime	≤ 8 mcg/ml	16 mcg/ml	≥ 32 mcg/ml
Levofloxacin	≤ 2 mcg/ml	4 mcg/ml	≥ 8 mcg/ml
Trimethoprim/ sulfamethoxazole	≤ 2/38 mcg/ml	—	≥ 4/76 mcg/ml
Streptococcus pneumoniae			
Cefepime (meningitis)	≤ 0.5 mcg/ml	1 mcg/ml	≥ 2 mcg/ml
(Non-meningeal)	≤ 1 mcg/ml	2 mcg/ml	≥ 4 mcg/ml
Levofloxacin	≤ 2 mcg/ml	4 mcg/ml	≥ 8 mcg/ml
Trimethoprim/ sulfamethoxazole	≤ 0.5/9.5 mcg/ml	1–2/19–38 mcg/ml	≥ 4/76 mcg/ml

Colony count greater than 100,000 CFU/ml Escherichia coli (ESBL)
************ISOLATION PRECAUTIONS ARE RECOMMENDED**************

DRUG	MIC	INTERPRETATION
Amikacin	<=2	S
Ampicillin	>=32	R
AmpSulbactam	4	S
Aztreonam	4	S
Cefazolin	>=64	R
Cefepime	2	S
Ceftriaxone	32	R
Ciprofloxacin	>=4	R
Ertapenem	<=0.5	S
Gentamicin	<=1	S
Meropenem	<=0.25	S
Nitrofurantoin	<=16	S
PiperTazobactam	<=4	S
Tobramycin	<=1	S
TrimethSulfa	<=20	S

Graman PS, Menegus MA. Microbiology laboratory tests. In: Betts RF, Chapman SW, Penn RL, eds. *A Practical Approach to Infectious Diseases*. Philadelphia, PA: Lippincott Williams & Wilkins, 2003:929–956.

Assessment

Drug	MIC	Breakpoint	Interpretation
Amikacin	2	4	
Ampicillin	32	16	
Aztreonam	4	2	

Assessment

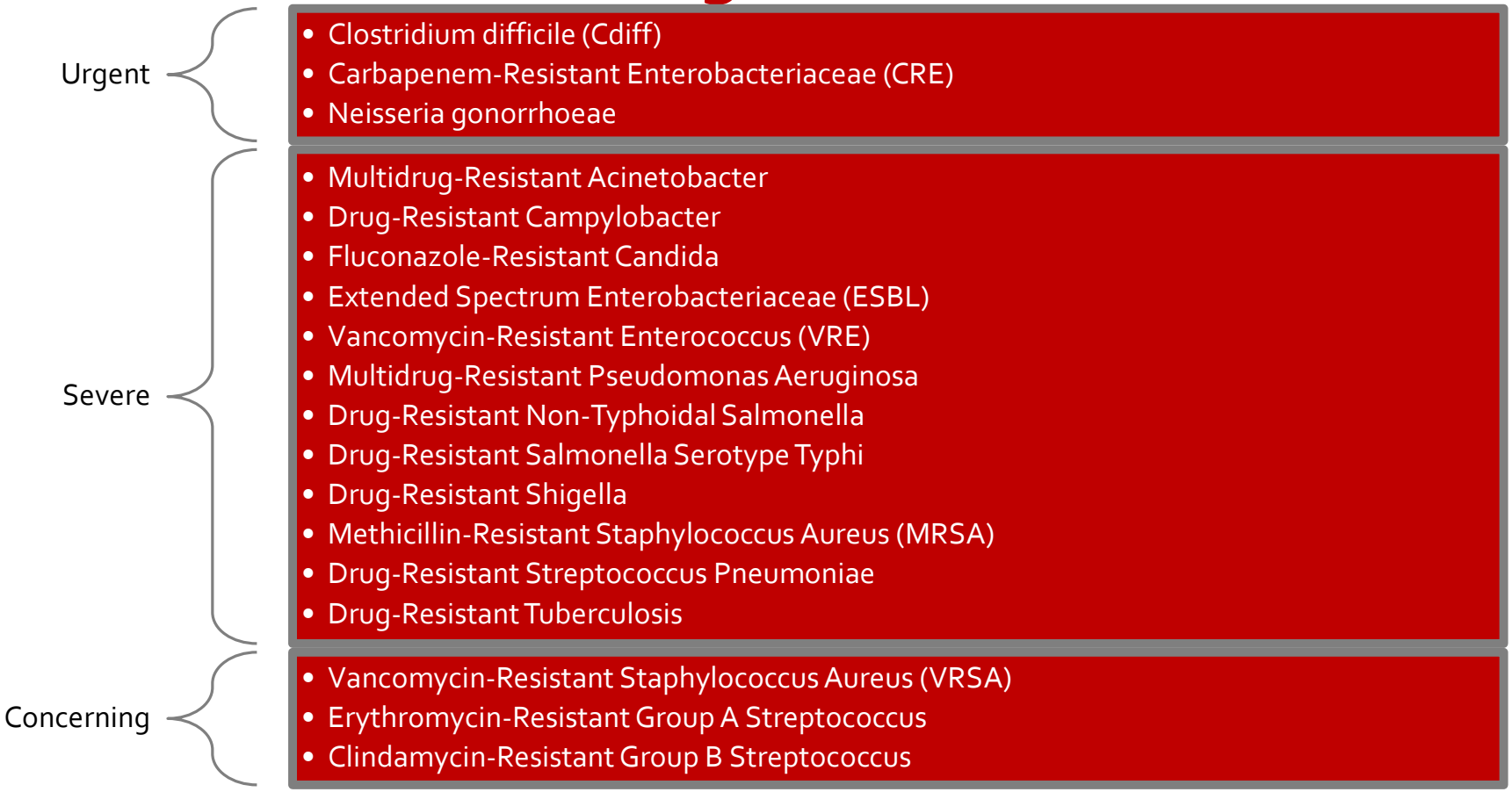
Drug	MIC	Breakpoint	Interpretation
Amikacin	2	4	S
Ampicillin	32	16	R
Aztreonam	4	2	R

SUPER BUGS!

ESKAPE/ESCAPE

E	• Enterococcus faecium
S	• Staphylococcus aureus
K or C	• Klebsiella pneumoniae • Clostridium difficile
A	• Acinetobacter spp
P	• Pseudomonas aeruginosa
E	• Enterobacter spp • Enterbacteriaceae

Big Threats



"Big Threats." *Big Threats - CDC*. Centers for Disease Control and Prevention, n.d. Web.

	Threat	Change in Rates or Number of Infections***			
		2020 vs. 2019	2021 vs. 2020	2022 vs. 2021	2022 vs. 2019
URGENT*	Hospital-onset CRE	▲ Increase	▲ Increase	— Stable	▲ Increase
URGENT*	Hospital-onset Carbapenem-resistant *Acinetobacter*	— Stable	— Stable	— Stable	▲ Increase**
URGENT*	Clinical Cases of *C. auris*	▲ Increase	▲ Increase	▲ Increase	▲ Increase
SERIOUS*	Hospital-onset MRSA	▲ Increase	— Stable	▼ Decrease	— Stable
SERIOUS*	Hospital-onset VRE	▲ Increase	▲ Increase	— Stable	▲ Increase
SERIOUS*	Hospital-onset ESBL-producing Enterobacterales	▲ Increase	— Stable	— Stable	▲ Increase
SERIOUS*	Hospital-onset MDR *Pseudomonas aeruginosa*	▲ Increase	▲ Increase	— Stable	▲ Increase

"Big Threats." *Big Threats - CDC*. Centers for Disease Control and Prevention, n.d. Web.

SUPER BUGS!

Urgent

- Clostridium difficile (Cdiff)
- Carbapenem-Resistant Enterobacteriaceae(CRE)
- Neisseria gonorrhoeae

Severe

- Extended Spectrum Beta-Lactamases (ESBL)
- Vancomycin-Resistant Enterococcus (VRE)
- Methicillin-Resistant Staphylococcus Aureus (MRSA)

"Big Threats." *Big Threats - CDC*. Centers for Disease Control and Prevention, n.d. Web.

CLOSE YOUR EYES

Peggy Lillis

Clostridium Difficile

One of the leading causes of morbidity and mortality in the hospital

Resistance is not an issue but overuse of antibiotics has led to an increase incidence of Cdiff infections

Abou Chakra CN, McGeer A, Labbe AC, et al. Factors associated with complications of *Clostridium difficile* infection in a multicenter prospective cohort. *Clin Infect Dis*. 2015;61(12):1781-1788.

WHAT ANTIBIOTIC HAS THE HIGHEST RISK FOR CAUSING CDFIFF INFECTIONS?

Antibiotic Related Risk

High Risk	Medium Risk	Low Risk
Cephalosporins	Macrolides	Aminoglycosides
Clindamycin	Tetracyclines	Metronidazole
Ampicillin/Amoxicillin		Anti-pseudomonal Penicillin
Fluoroquinolones		Rifampin
		Vancomycin

Abou Chakra CN, McGeer A, Labbe AC, et al. Factors associated with complications of *Clostridium difficile* infection in a multicenter prospective cohort. *Clin Infect Dis*. 2015;61(11):1781-1788.

Clostridium difficile

Mechanism of Infection:

- Gram-positive, spore-forming anaerobic bacillus
- Produces two major toxins: **Toxin A (TcdA) and Toxin B (TcdB)**
- Toxins cause **inflammation, mucosal damage, and fluid secretion** in the colon

Pathogenesis Steps:

- Disruption of gut microbiota (due to antibiotic use)
- Colonization of **C. difficile** spores in the gut
- Toxin production leads to **colitis and diarrhea**

Severe cases may progress to **pseudomembranous colitis, toxic megacolon, or sepsis**

Kelly CR, Fischer M, Allegretti JR, et al. ACG Clinical Guidelines: Prevention, Diagnosis, and Treatment of Clostridioides difficile Infections. *Am J Gastroenterol.* 2021;116(6):1124-1147. doi:10.14309/ajg.0000000000001278

Clostridium difficile Resistance

Hypervirulent & Common Strains

1.NAP1/BI/027: Highly virulent, produces 16-23x more toxin A & B
- Fluoroquinolone-resistant
- Associated with severe disease, outbreaks, and higher mortality

NAP2/078 Strain

- Found in livestock (pigs, cattle)
- Can cause severe community-acquired CDI

NAP4/017 Strain

- Lacks Toxin A but still highly pathogenic
- Fluoroquinolone resistance reported

Patients infected with NAP1 strain have:

- More severe colitis, higher recurrence rates, increased ICU admissions
- Higher mortality risk compared to other strains
- More resistant ciprofloxacin, levofloxacin

Kelly CR, Fischer M, Allegretti JR, et al. ACG Clinical Guidelines: Prevention, Diagnosis, and Treatment of Clostridioides difficile Infections. *Am J Gastroenterol.* 2021;116(6):1124-1147. doi:10.14309/ajg.0000000000001278

Treatment

Initial CDI Episode:

- Preferred: Fidaxomicin 200 mg BID x 10 days
- Alternative: PO Vancomycin 125 mg QID x 10 days
- If above unavailable (non-severe cases): Metronidazole 500 mg TID x 10-14 days

First Recurrence:

- Preferred: Fidaxomicin 200 mg BID x 10 days or BID x 5 days then QOD x 20 days
- Alternative: PO Vancomycin in tapered/pulsed regimen

Fulminant CDI:

- Vancomycin 500 mg QID orally or NG tube
- Consider rectal vancomycin if ileus is present
- IV metronidazole 500 mg q8h with vancomycin

Multiple Recurrences:

- Fidaxomicin (standard or extended-pulsed)
- PO Vancomycin tapered and pulsed regimen
- PO Vancomycin x 10 days followed by rifaximin
- Fecal Microbiota Transplantation (FMT)
- Adjunctive: Bezlotoxumab IV once discontinued effective January 31, 2025

Kelly CR, Fischer M, Allegretti JR, et al. ACG Clinical Guidelines: Prevention, Diagnosis, and Treatment of Clostridioides difficile Infections. *Am J Gastroenterol.* 2021;116(6):1124-1147. doi:10.14309/ajg.0000000000001278

Treatment Resistance/Recurrence

Bezlotoxumab (Monoclonal Antibody to Toxin B):

- Reduces recurrence in high-risk patients
- Use in patients with prior CDI episode in last 6 months

FMT (Fecal Microbiota Transplantation):

- For multiple CDI recurrences after standard treatments fail
- FDA warning: Risk of transmission of pathogens

Rifaximin:

- Used after vancomycin for 20 days to prevent recurrence
- Limited data, but promising in some recurrent CDI cases

Kelly CR, Fischer M, Allegretti JR, et al. ACG Clinical Guidelines: Prevention, Diagnosis, and Treatment of Clostridioides difficile Infections. *Am J Gastroenterol.* 2021;116(6):1124-1147. doi:10.14309/ajg.0000000000001278

CLOSE YOUR EYES

David Ricci

CRE Vs. CPE

Carbapenem-Resistant Enterobacteriaceae (CRE)

A bacterium that is a member of the enterobacteriaceae family resistant to the carbapenem class of antibiotics by any means

Carbapenemase-Producing Enterobacteriaceae (CPE)

A bacterium that is a member of the enterobacteriaceae family resistant to the carbapenem class of antibiotics through the production of a carbapenemase

Humphries RM, Yang S, Hemarajata P, et al.First report of ceftazidime-avibactam resistance in a KPC-3-expressing *Klebsiella pneumoniae* isolate.*Antimicrob Agents Chemother*. 2015;59(10):6605-6607.
Kumarasamy KK, Toleman MA,Walsh TR, et al. Emergence of a new antibiotic resistance mechanism in India, Pakistan, and the UK:a molecular, biological, and epidemiological study. *Lancet Infect Dis*. 2010;10(9):597-602.

Types of Carbapenemases

Klebsiella-Producing Carbapenemases (KPC)

- Inhibit all beta-lactams

New Delhi Metallo-Beta Lactamases (NMD-1)

- Inhibit all beta-lactams
- Susceptible to Aztreonam

Verona Integron Encoded Metallobetalacamase (VIM)

- Inhibit all beta-lactams

Oxacillin Hydrolyzing (OXA)

- Inhibit all beta-lactams but weak inhibition against carbapenems

Humphries RM, Yang S, Hemarajata P, et al.First report of ceftazidime-avibactam resistance in a KPC-3-expressing *Klebsiella pneumoniae* isolate.*Antimicrob Agents Chemother.* 2015;59(10):6605-6607.
Kumarasamy KK, Toleman MA,Walsh TR, et al. Emergence of a new antibiotic resistance mechanism in India, Pakistan, and the UK:a molecular, biological, and epidemiological study. *Lancet Infect Dis.* 2010;10(9):597-602.

Carbapenemase-Producing Enterobacteriaceae Treatments

Tigecycline + Polymyxins + Aminoglycosides

- Tigecycline has limited efficacy and numerous adverse effects

Ceftolozane-Tazobactam (Zerbaxa)

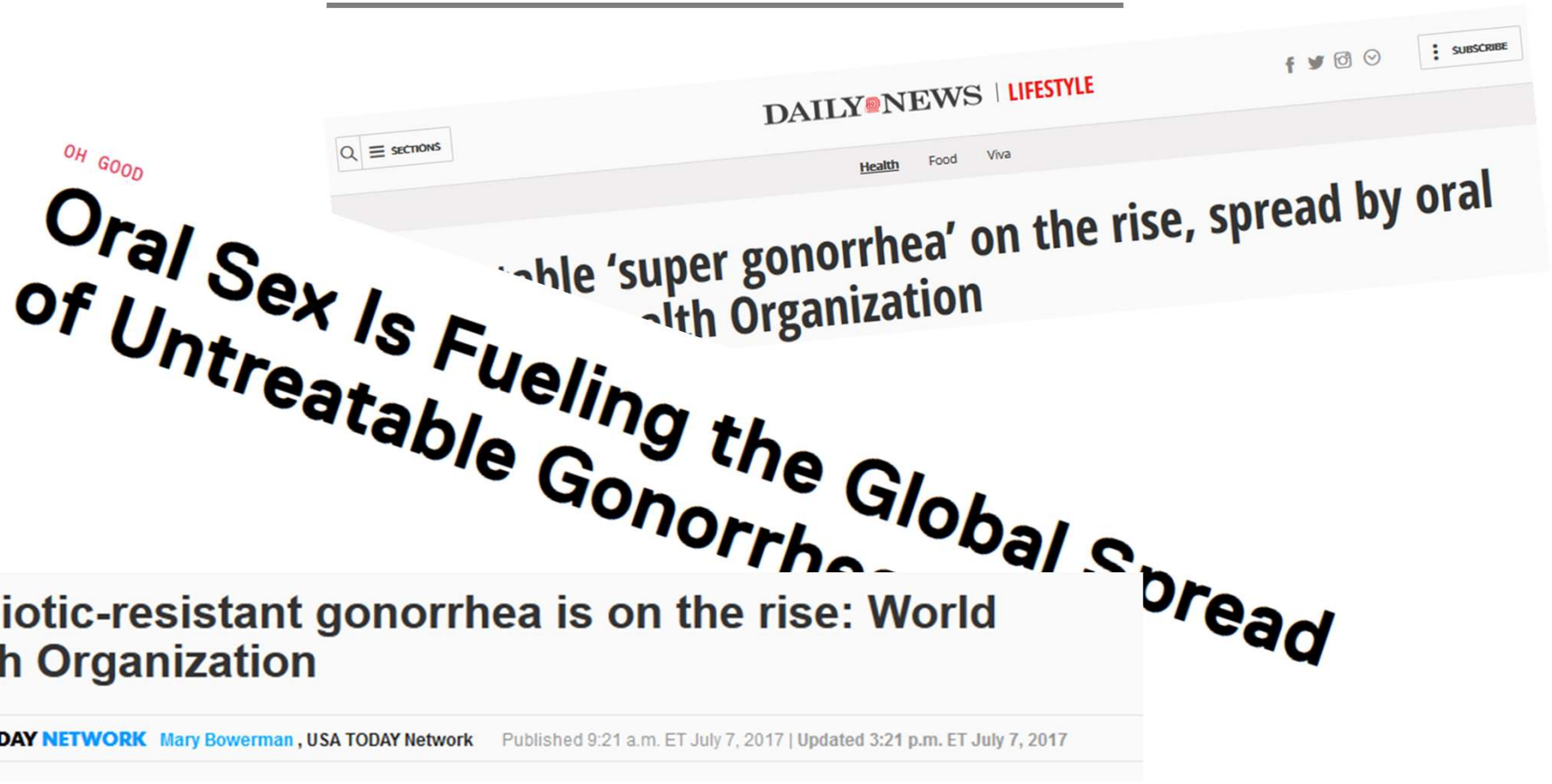
Ceftazidime-Avibactam (Avycaz)

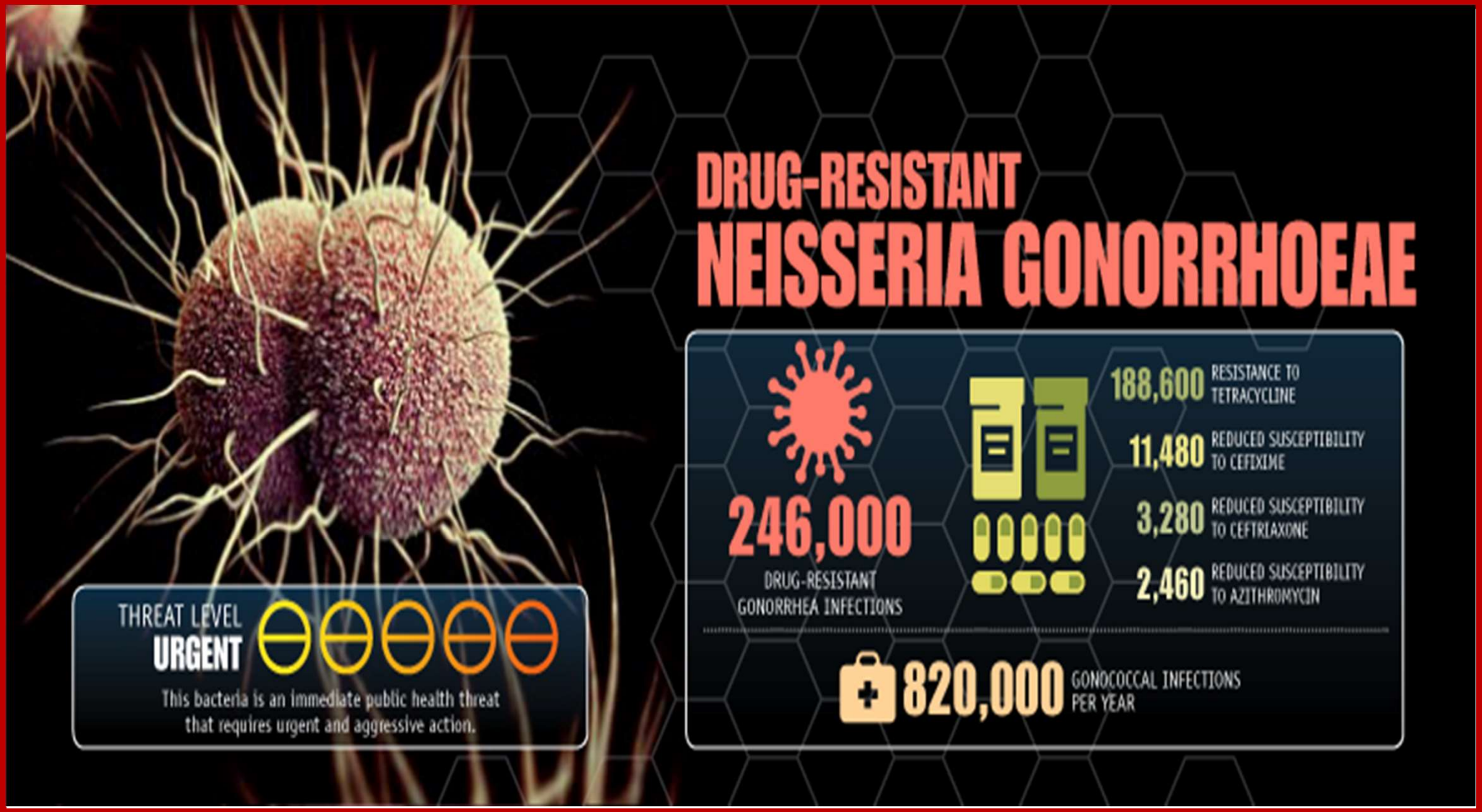
- No activity against NDM-1 and VIM

Imipenem/Cilastatin + Relebactam (Recarbrio)

Humphries RM, Yang S, Hemarajata P, et al.First report of ceftazidime-avibactam resistance in a KPC-3-expressing *Klebsiella pneumoniae* isolate.*Antimicrob Agents Chemother.* 2015;59(10):6605-6607.
Kumarasamy KK, Toleman MA,Walsh TR, et al. Emergence of a new antibiotic resistance mechanism in India, Pakistan, and the UK:a molecular, biological, and epidemiological study. *Lancet Infect Dis.* 2010;10(9):597-602.

Neisseria Gonorrhoeae




DRUG-RESISTANT
NEISSERIA GONORRHOEAE
THREAT LEVEL
URGENT
This bacteria is an immediate public health threat that requires urgent and aggressive action.
246,000
DRUG-RESISTANT GONORRHEA INFECTIONS
188,600 RESISTANCE TO TETRACYCLINE
11,480 REDUCED SUSCEPTIBILITY TO CEFIXIME
3,280 REDUCED SUSCEPTIBILITY TO CEFTRIAXONE
2,460 REDUCED SUSCEPTIBILITY TO AZITHROMYCIN
820,000 GONOCOCCAL INFECTIONS PER YEAR

Neisseria Gonorrhoeae

- Second most reported sexually transmitted infection
- Increased resistance to Azithromycin, Fluoroquinolones and oral Cefixime has created massive crisis
- Resistance to Ceftriaxone has also been noted

CDC (14 July 2014). "Gonorrhea - CDC Fact Sheet". Retrieved 17 October 2014.

Neisseria Gonorrhoeae

Pathogen – *Neisseria gonorrhoeae*

- Gram Negative Diplococci

Common sites of infections

- Cervix, urethra, rectum, pharynx, conjunctiva

Common co-infection – *Chlamydia trachomatis*

- **Routine treatment covers both** *N. gonorrhoeae* & *C. trachomatis*

CDC (14 July 2014). "Gonorrhea - CDC Fact Sheet". Retrieved 17 October 2014.

Neisseria Gonorrhoeae – Treatment

Preferred Treatment:

- **Ceftriaxone 500mg IM x 1 PLUS doxycycline 100mg PO daily x 7 days**
  (if *Chlamydia trachomatis* infection is not excluded)
- In patients **≥150 kg, ceftriaxone dose should be 1 gram**

Alternative Treatments (FYI):

- Cefixime 800mg PO x 1
- Gentamicin 240mg IM plus 2 grams PO azithromycin
- Fluoroquinolones no longer recommended due to resistance

St. Cyr S, Barbee L, Workowski KA, et al. Update to CDC's Treatment Guidelines for Gonococcal Infection, 2020. MMWR Morb Mortal Wkly Rep 2020;69:1911–1916.
DOI: http://dx.doi.org/10.15585/mmwr.mm6950a6

Neisseria Gonorrhoeae

Susceptibility testing is recommended for all individuals for who Azithromycin + Ceftriaxone is not effective

Treatments in the Pipeline!

- Solithromycin – Completed Phase III Clinical Trial
- Zoliflodacin – Currently in Phase II
- Gepotidacin – Currently in Phase II

CDC (14 July 2014). "Gonorrhea - CDC Fact Sheet". Retrieved 17 October 2014.
Putnam, Shannon D.; Sader, Helio S.; Farrell, David J.; Biedenbach, Douglas J.; Castanheira, Mariana (2011). "Antimicrobial characterisation of solithromycin (CEM-101), a novel fluoroketolide: activity against staphylococci and enterococci". International Journal of Antimicrobial Agents. **37** (1): 39–45. PMID 21075602. doi:10.1016/j.ijantimicag.2010.08.021

Extended Spectrum Beta-Lactamase (ESBL)

Nearly 900 different β-lactamases have been found (penicillinases, cephalosporinases, etc.)

Some β-lactam antibiotics are resistant to some β-lactamases, but sensitive to others

- *Staphylococcus aureus*, *Haemophilus influenza*, and *Escherichia coli* possess β-lactamases that prefer **penicillins,** but not **cephalosporins**
- *Psuedomonas aeruginosa* and *Enterobacter* species destroy **penicillins** and **cephalosporins**
- Penicillinases and cephalosporinases do not destroy **carbapenems**, but carbapenemases and metallo-β-lactamases exist that do destroy **carbapenems**

Centers for Disease Control and Prevention. *Antibiotic Resistance Threats in the United States,* 2013. http://www.cdc.gov/drugresistance/threat-report-2020/.

Beta Lactamases – Carbapenems

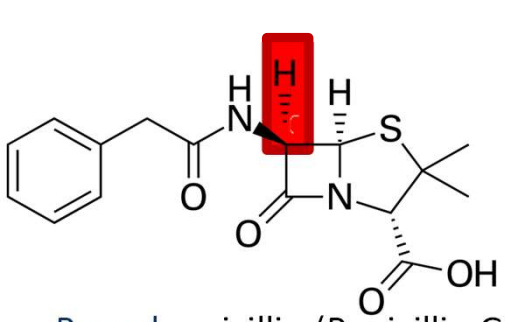
Carbapenems are more β-lactamase-resistant than other drugs

Resistance is conferred by the different stereochemistry in the β-lactam ring

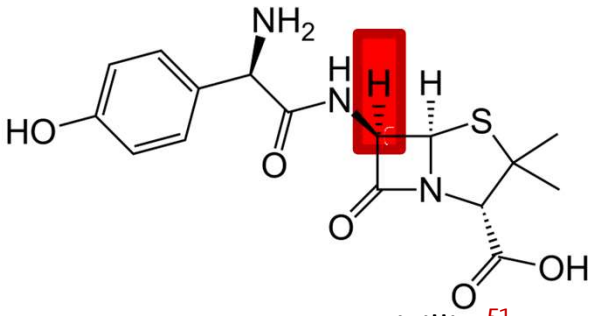
Imipenem

Meropenem

Sensitive to β-lactamase



Benzylpenicillin (Penicillin G)



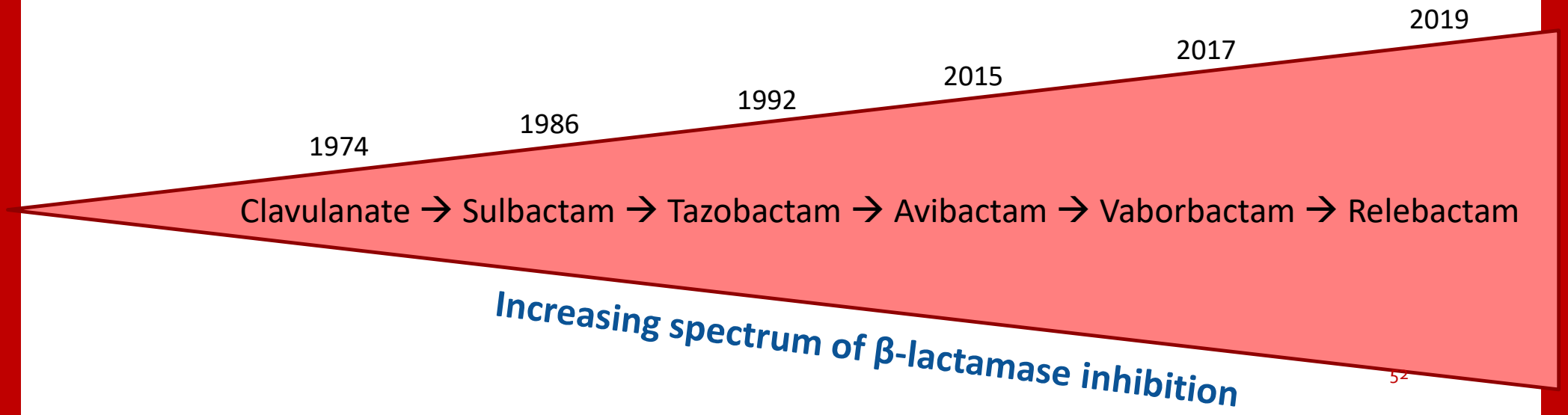
Amoxicillin

Beta Lactamase Inhibitors

Not all β-lactamase inhibitors are created equally

In general, the newer ones have greater spectra of activity, but older ones are still in use to stabilize drugs against certain bacteria

β-lactamase inhibitors cannot be mixed with any random β-lactam drug, but are instead combined with drugs that are sensitive to the β-lactamase that the inhibitor targets



Extended Spectrum Beta-Lactamase (ESBL)

Carbapenems	Fosfomycin	Ceftolozane/ Tazobactam (Zerbaxa)	Ceftazidime/Avibactam (Avycaz)
Meropenem/ Vaborbactam (Vabomere)	Tigecycline	Imipenem/Cilastatin/ Relebactam (Recarbrio)	Omadacycline/ Evaracycline

Centers for Disease Control and Prevention. *Antibiotic Resistance Threats in the United States,* 2013. http://www.cdc.gov/drugresistance/threat-report-2020/.

Vancomycin – Concerns for Resistance

Vancomycin Resistant Enterococcus (VRE)

Vancomycin Intermediate Staph aureus (VISA)

Vancomycin Resistant Staph aureus (VRSA)

Centers for Disease Control and Prevention. *Antibiotic Resistance Threats in the United States,* 2013. http://www.cdc.gov/drugresistance/threat-report-2020/.

Resistance to Vancomycin

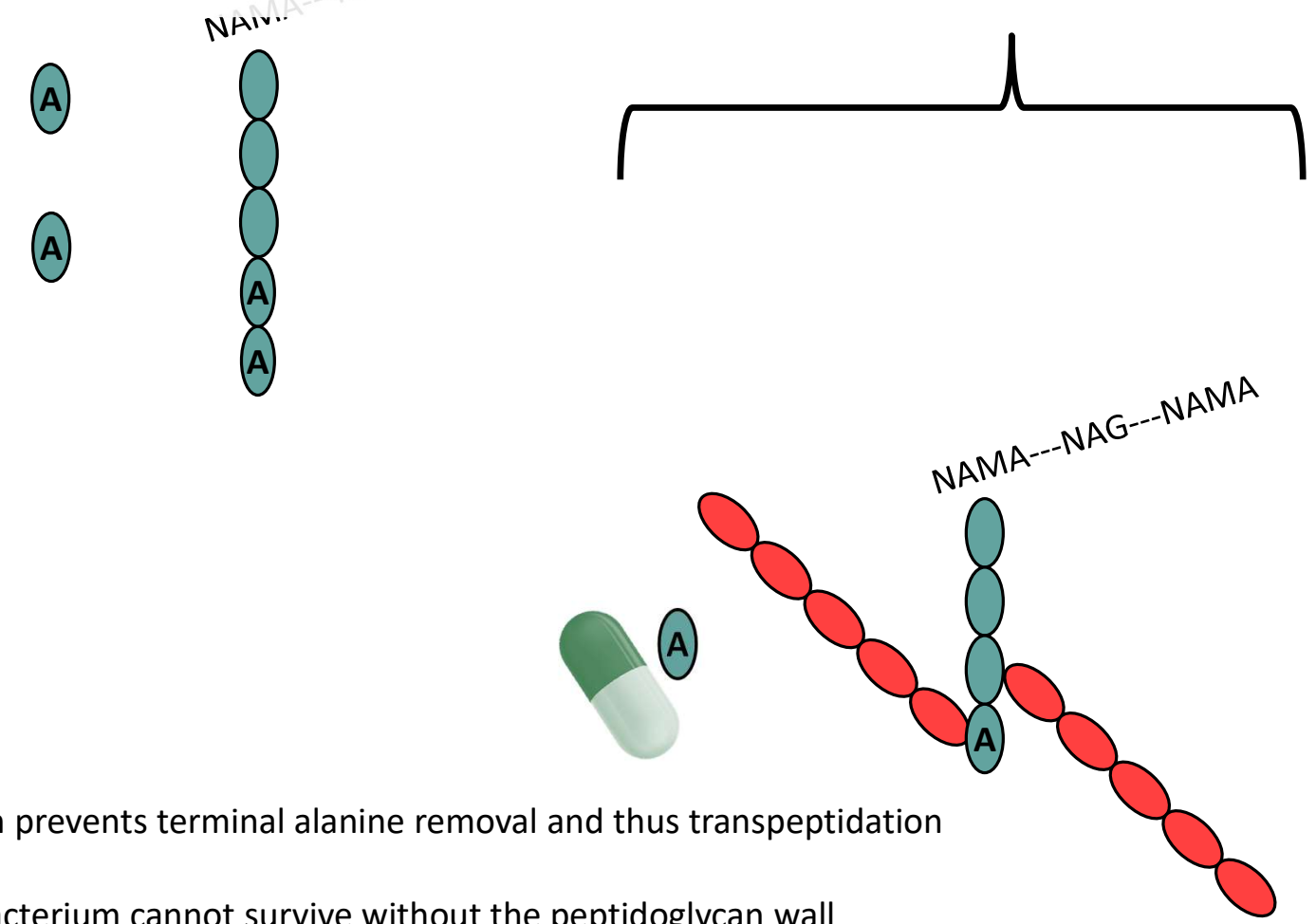
Enterococci and *Staph. Aureus* can resist vancomycin by a simple trick

They replace the terminal D-alanine with a D-lactate or D-serine, which prevents vancomycin from binding altogether

Lactate is not even an amino acid, highlighting the fact that resistance-generating bacterial evolution is extremely flexible, and perhaps unpredictable with respect to mechanism

Centers for Disease Control and Prevention. *Antibiotic Resistance Threats in the United States,* 2013. http://www.cdc.gov/drugresistance/threat-report-2020/.

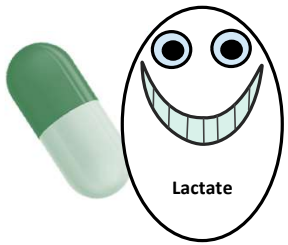
Vancomycin-Inhibited Peptidoglycan Synthesis

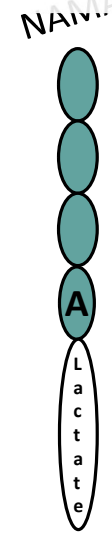


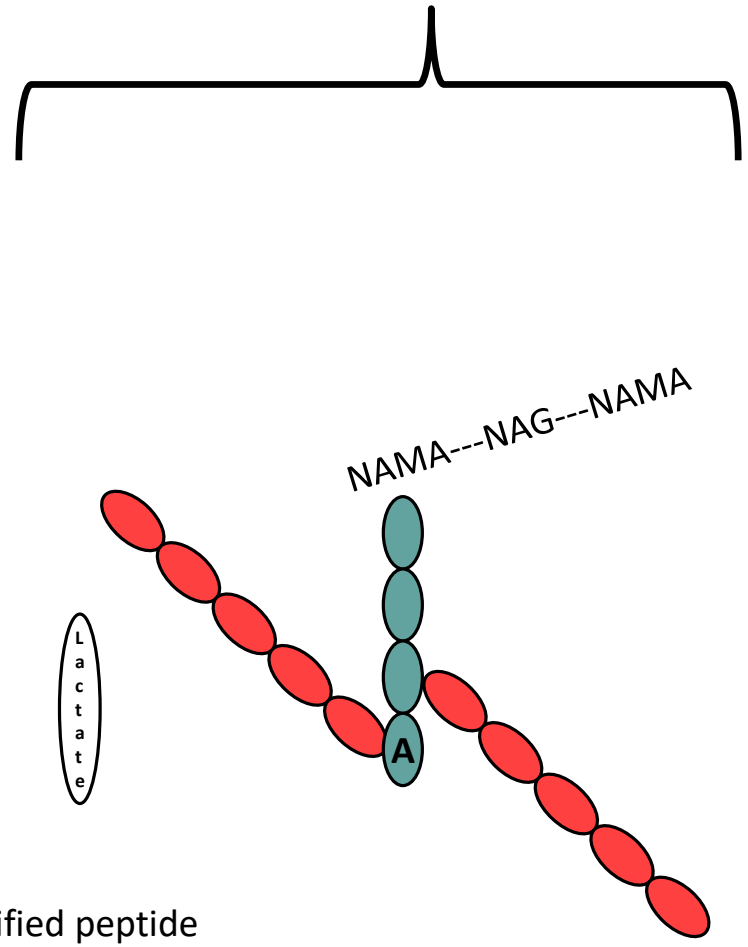
Vancomycin prevents terminal alanine removal and thus transpeptidation

Bacterium cannot survive without the peptidoglycan wall

Peptidoglycan Synthesis Resistant to Vanco







Vancomycin has no affinity for the lactate-modified peptide

Terminal alanine-lactate bond breakage promotes peptide attachment

Vancomycin-Resistant Enterococcus (VRE)

NON-VRE

- Ampicillin/Sulbactam
- Vancomycin
- Piperacillin/Tazobactam
- Penicillin
- Quinupristin/Dalfopristin

VRE

- E. faecalis
  - Penicillin G
  - Ampicillin
  - Linezolid
  - Daptomycin
  - Tigecycline
  - Nitrofurantoin
  - Fosfomycin
  - Doxycycline
- E. faecium
  - Daptomycin
  - Linezolid
  - Tigecycline
  - Quinupristin/Dalfopristin
  - Nitrofurantoin
  - Fosfomycin
  - Doxycycline

Centers for Disease Control and Prevention. *Antibiotic Resistance Threats in the United States,* 2013. http://www.cdc.gov/drugresistance/threat-report-2013/. Accessed January 12, 2016.

CLOSE YOUR EYES

Ricky Lannetti

Methicillin-Resistant Staphylococcus Aureus (MRSA)

Hospital Acquired

- Vancomycin
- Daptomycin
- Linezolid/Tedizolid
- Ceftaroline
- Telavancin
- Dalbavancin/Oritavancin
- Tigecycline
- Quinupristin/Dalfopristin

Community Acquired

- Sulfamethoxazole/Trimethoprim
- Doxycycline/Minocycline
- Clindamycin
- Linezolid/Tedizolid
- Moxifloxacin

Centers for Disease Control and Prevention. *Antibiotic Resistance Threats in the United States,* 2013. http://www.cdc.gov/drugresistance/threat-report-2013/. Accessed January 12, 2016.

Pharmacy Company

Years of research

New drug

Microbes

New Antibiotics…?
Oh… I got to evolve again!!!

Resources for Pharmacists

https://www.cdc.gov/

http://www.who.int/en/

Post-Test Question 1

Which of the following combinations is the appropriate treatment for NMD-1 Carbapenemase-Producing Enterobacteriaceae? SELECT ALL THAT APPLY

a. Piperacillin/Tazobactam
b. Tigecycline + Amikacin + Colistin
c. Amoxicillin/Clavulanic Acid
d. Vancomycin
e. Ceftolozane/Tazobactam

Post-Test Question 2

Which of the following is the first line treatment for ESBL E. Coli infections?

a. Carbapenems
b. Fosfomycin
c. Piperacillin/Tazobactam
d. Amoxicillin/Clavulanic Acid

Post-Test Question 3

Which of the following is considered first line treatment for hospital acquired MRSA infections?

a. Daptomycin
b. Linezolid
c. Clindamycin
d. Vancomycin

Post-Test Question 4

Which of the following medications can be used for clostridium difficile infections?

a. IV Vancomycin
b. Oral Vancomycin
c. IV Zosyn
d. Oral Augmentin

QUESTIONS?

Vicky Shah, PharmD, BCPS

Associate Professor of Clinical Sciences

Chair of Service and Clinical Site Relationships

vshah09@roosevelt.edu