

Occurrence of *vanB* vancomycin resistant enterococci (VRE) in a patient

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Antibiotic susceptibility



Enterococcus faecium

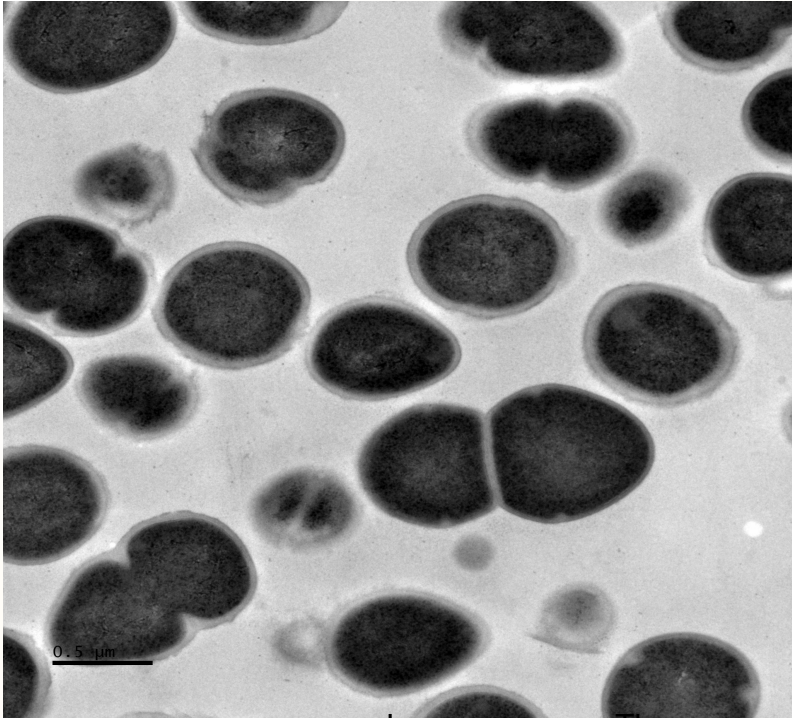
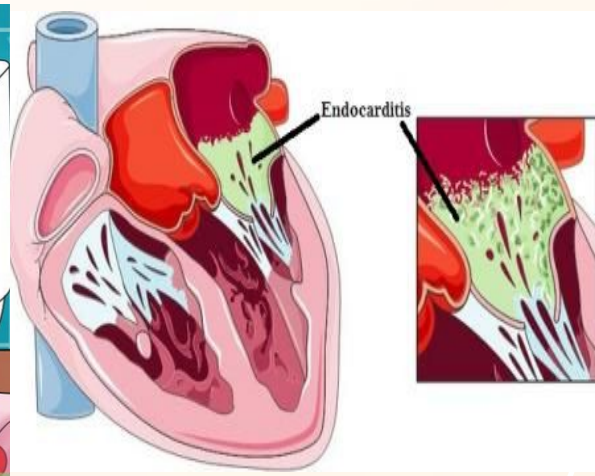
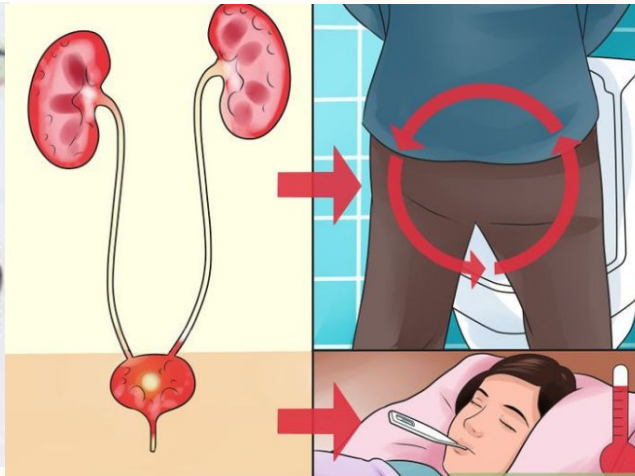


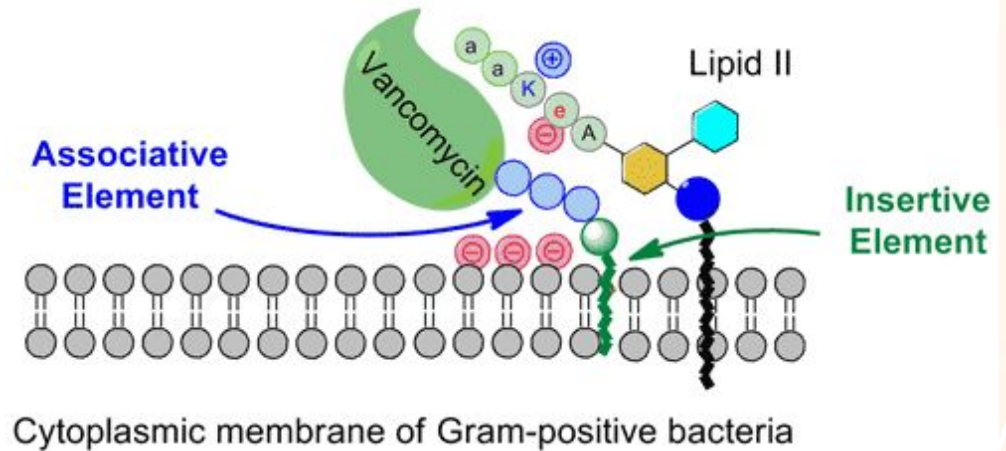
Image source – Theresa
Wagner



E. faecium infections



Treatment for *E. faecium* infections



Vancomycin Resistance *Enterococcus* - VRE

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Chemotherapy**

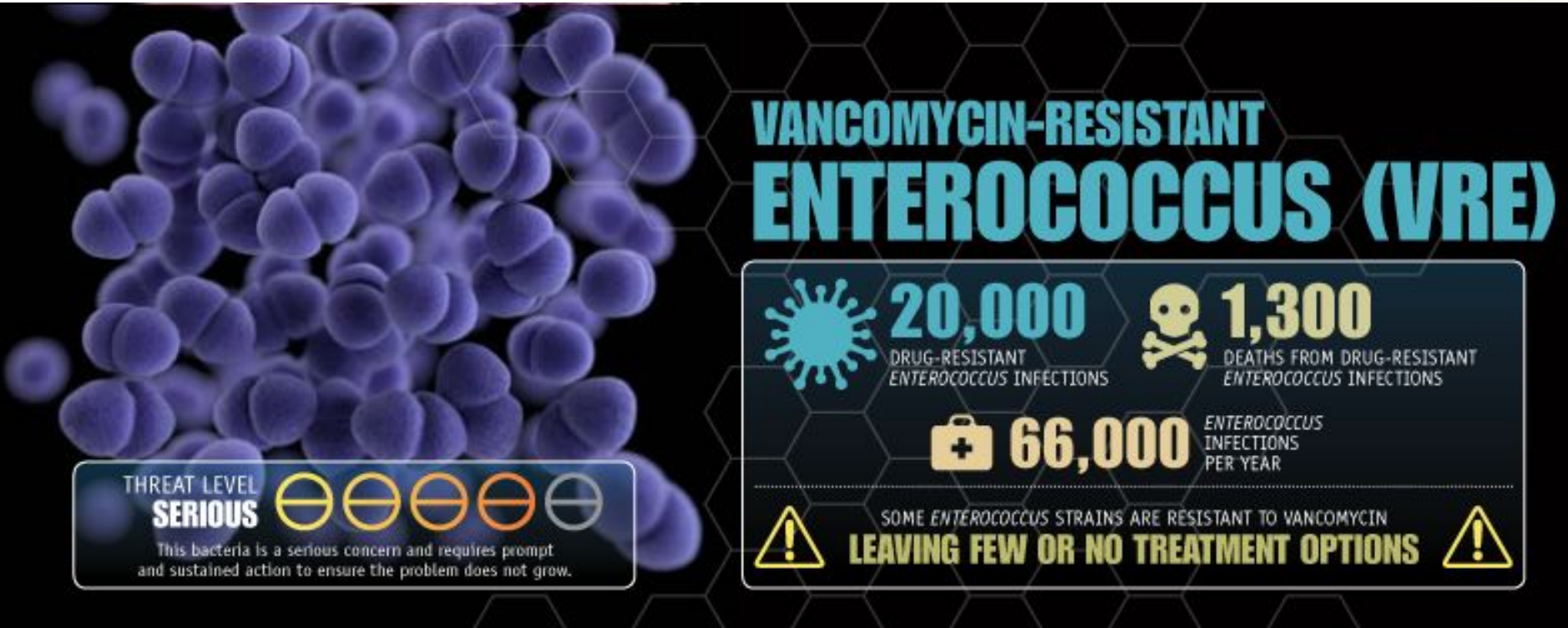
Twenty-five years of shared life with vancomycin-resistant enterococci: is it time to divorce?

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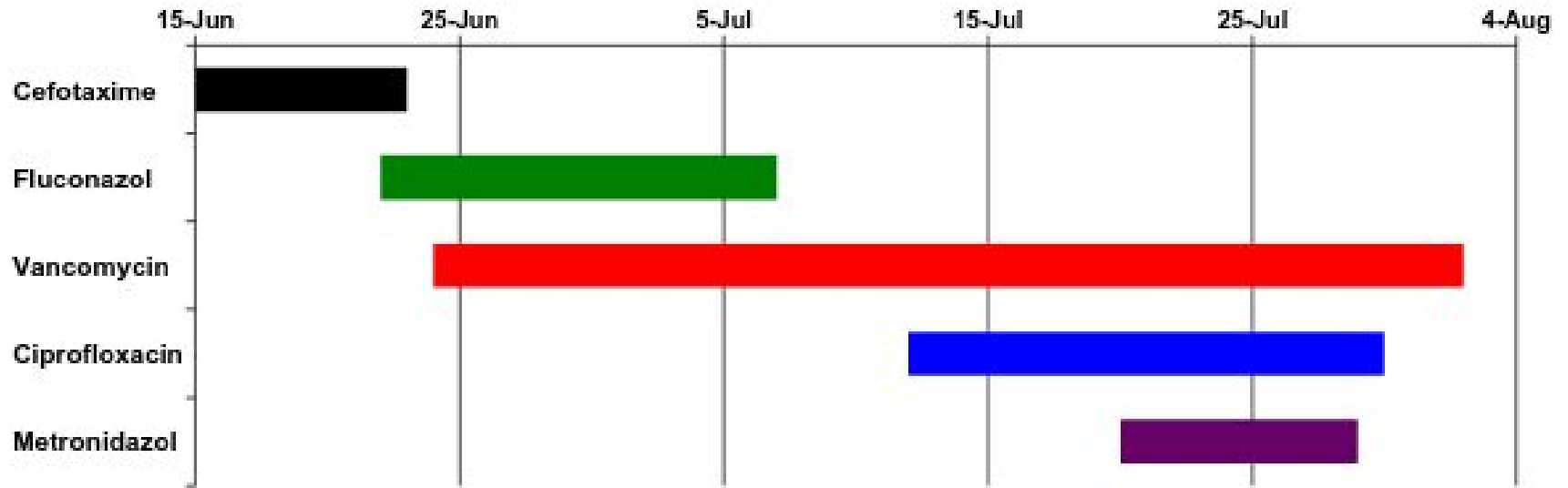
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Vancomycin Resistance *Enterococcus* - VRE

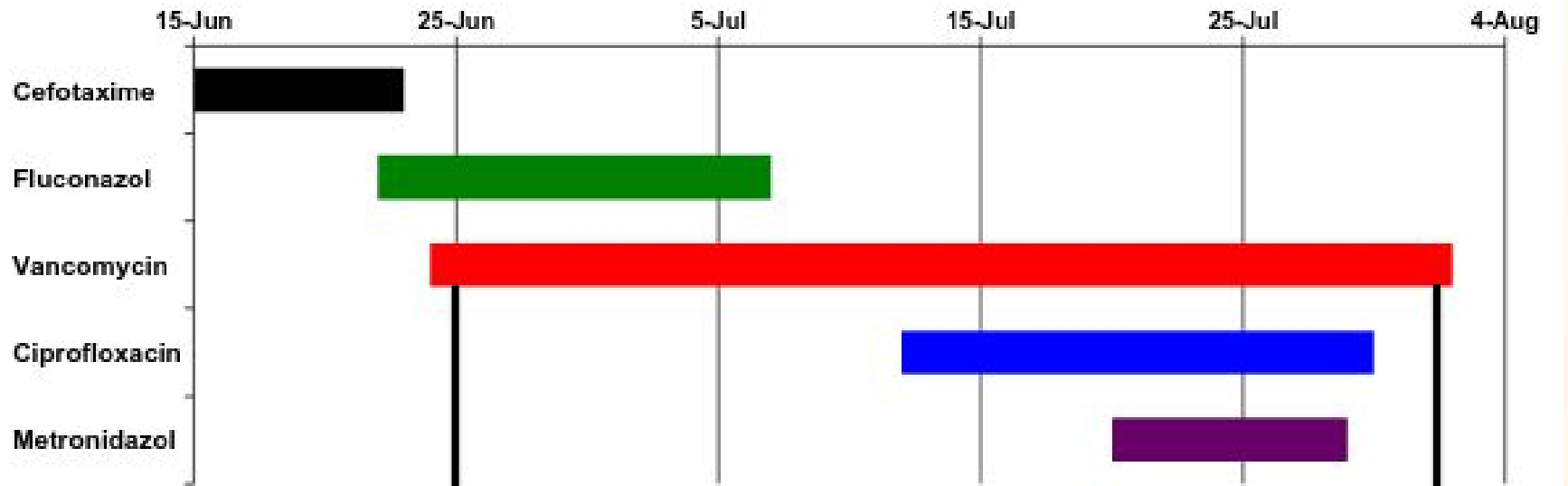


Case background





- Ventilator-associated pneumonia
- Candida albicans* septicemia
- Enterococcus faecium* septicemia
- Acinetobacter* sp. and *Enterobacter* sp. wound infections
- Bacteriodes* sp. wound infections



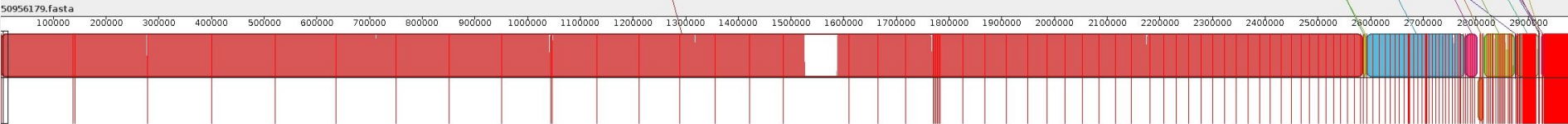
Van Susceptible

Van Resistant

Comparison – whole genome



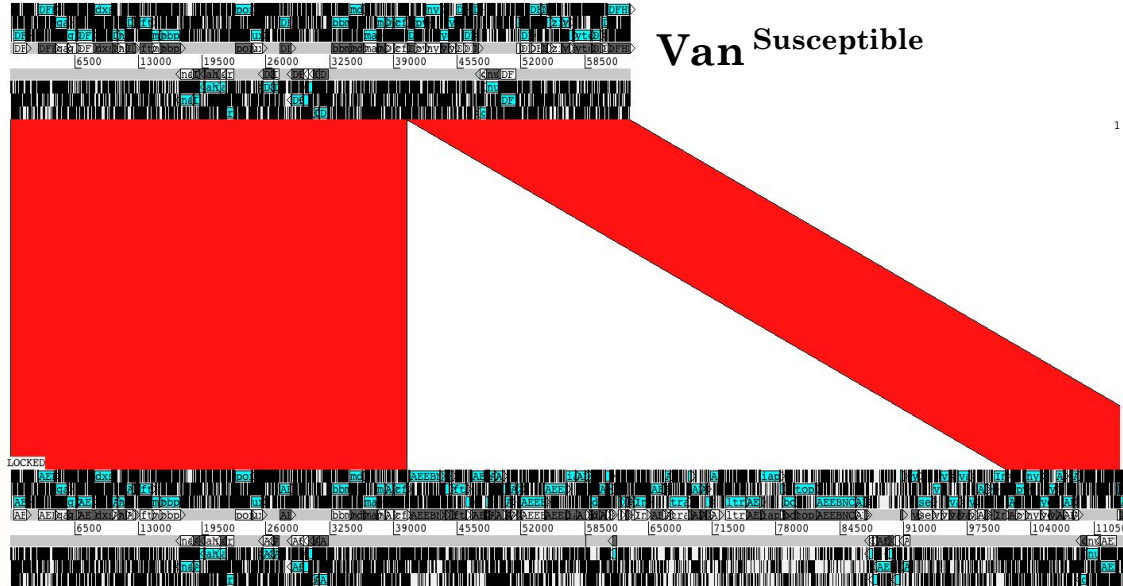
Van Susceptible



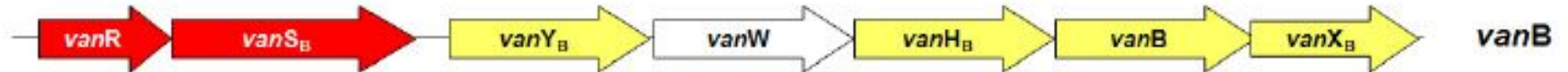
Van Resistant



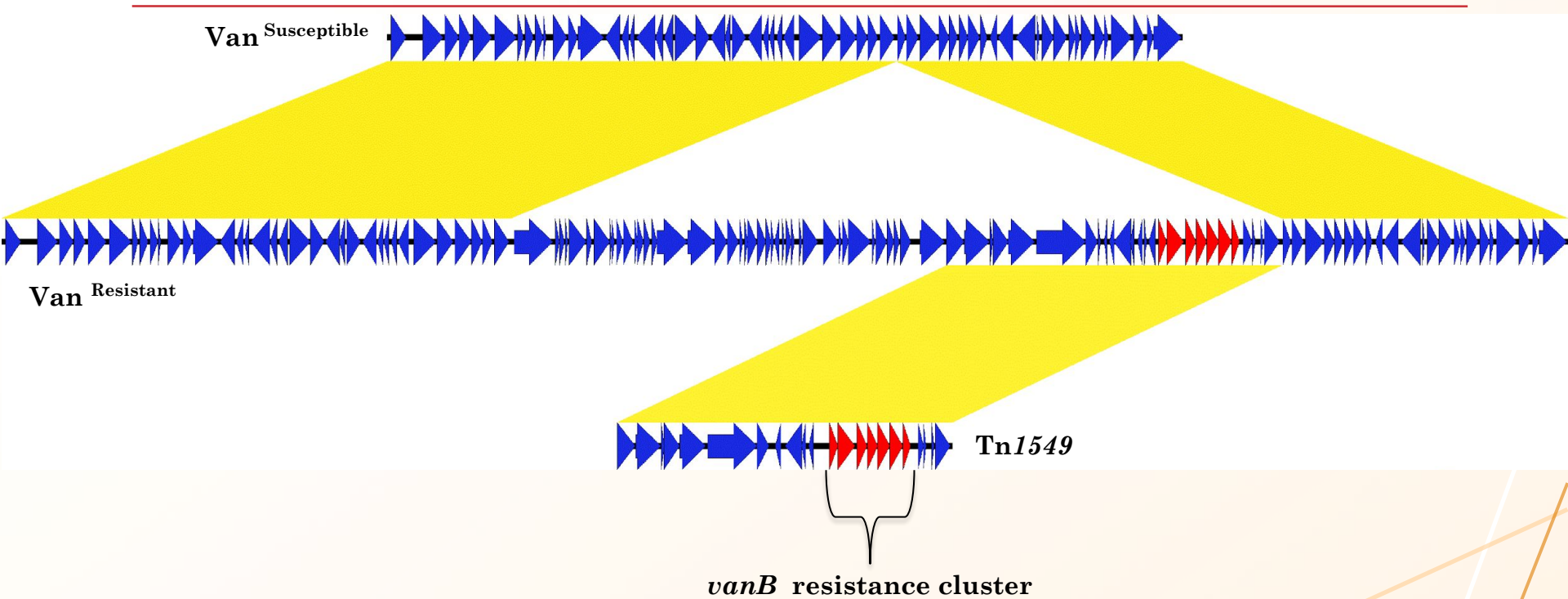
Comparison



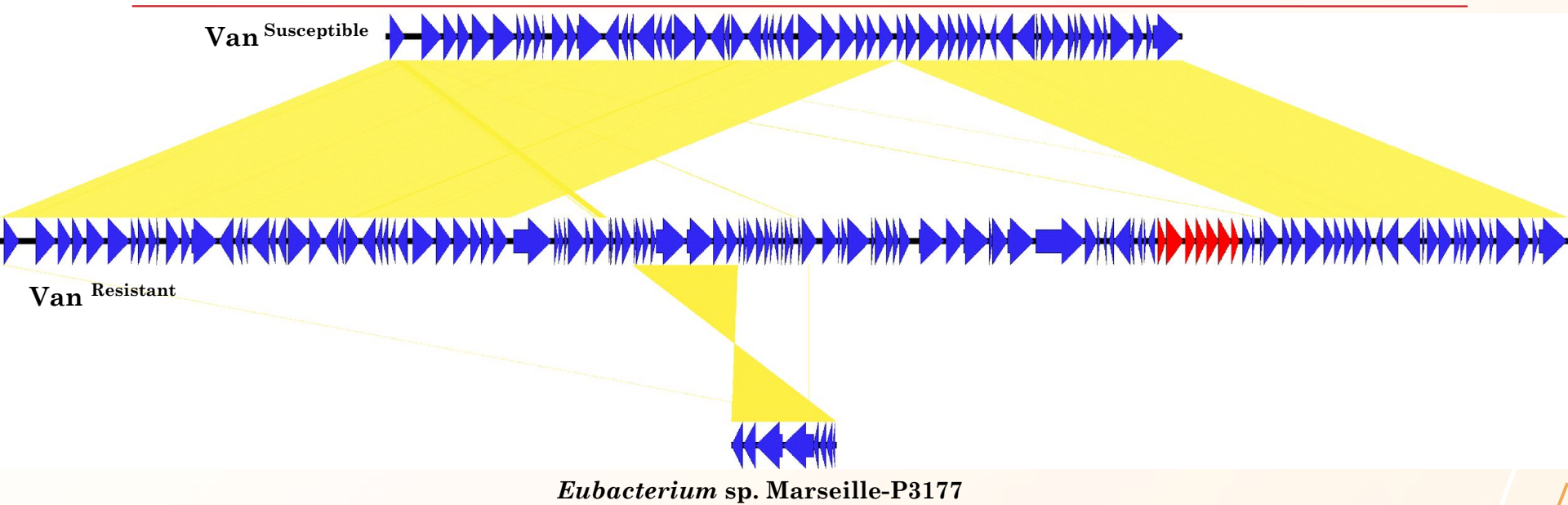
Van Resistant



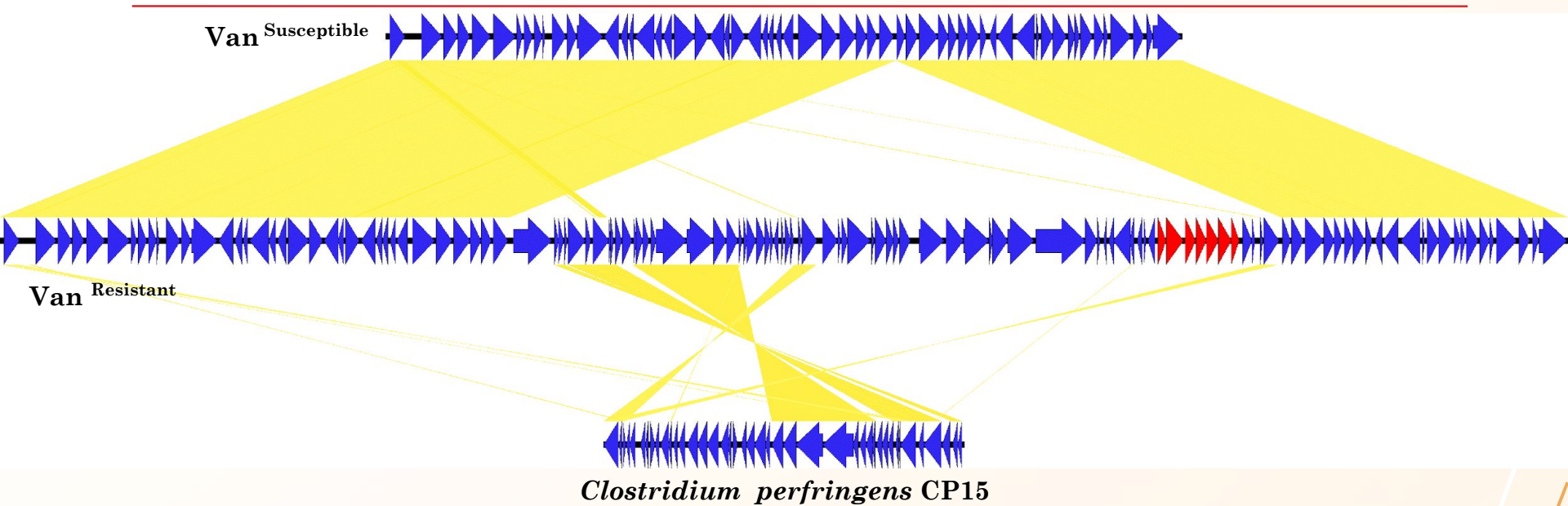
Comparison against bacterial genomes



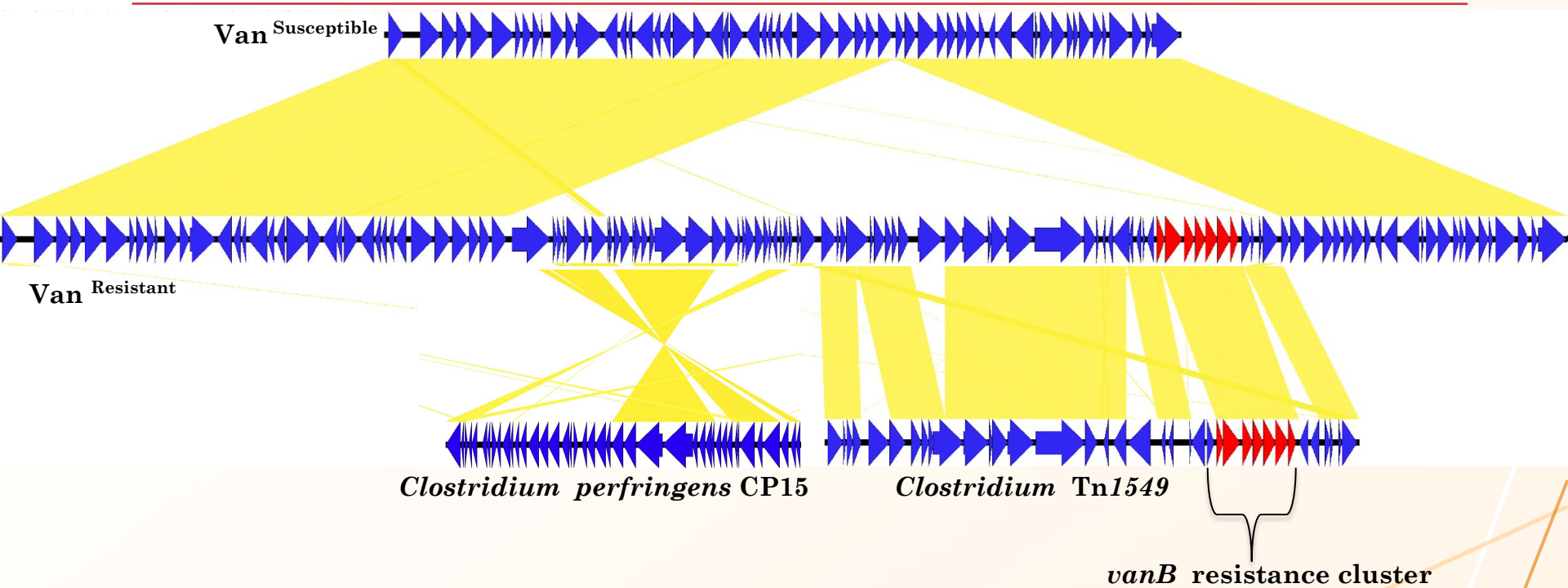
Comparison against bacterial genomes



Comparison against bacterial genomes



Comparison against bacterial genomes



Gene transfer

nature
genetics

The multidrug-resistant human pathogen *Clostridium difficile* has a highly mobile, mosaic genome

Mohammed Sebaihia¹, Brendan W Wren², Peter Mullany³, Neil F Fairweather⁴, Nigel Minton⁵, Richard Stabler², Nicholas R Thomson¹, Adam P Roberts³, Ana M Cerdeño-Tárraga¹, Hongmei Wang³, Matthew TG Holden¹, Anne Wright⁴, Carol Churcher¹, Michael A Quail¹, Stephen Baker¹, Nathalie Bason¹, Karen Brooks¹, Tracey Chillingworth¹, Ann Cronin¹, Paul Davis¹, Linda Dowd¹, Audrey Fraser¹, Theresa Feltwell¹, Zahra Hance¹, Simon Holroyd¹, Kay Jagels¹, Sharon Moule¹, Karen Mungall¹, Claire Price¹, Ester Rabbinoiwitsch¹, Sarah Sharp¹, Mark Simmonds¹, Kim Stevens¹, Louise Unwin¹, Sally Whithead¹, Bruno Dupuy⁶, Gordon Dougan¹, Bart Barrell¹ & Julian Parkhill¹

We determined the complete genome sequence of *Clostridium difficile* strain 630, a virulent and multidrug-resistant strain. Our analysis indicates that a large proportion (11%) of the genome consists of mobile genetic elements, mainly in the form of conjugative transposons. These mobile elements are putatively responsible for the acquisition by *C. difficile* of an extensive array of genes involved in antimicrobial resistance, virulence, host interaction and the production of surface structures. The metabolic capabilities encoded in the genome show multiple adaptations for survival and growth within the gut environment. The extreme genome variability was confirmed by whole-genome microarray analysis; it may reflect the organism's niche in the gut and should provide information on the evolution of virulence in this organism.

Gene transfer



Antimicrobial Agents
and Chemotherapy

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Transfer of Vancomycin Resistance Transposon Tn1549 from *Clostridium symbiosum* to *Enterococcus* spp. in the Gut of Gnotobiotic Mice

[Aline Launay](#),¹ [Susan A. Ballard](#),² [Paul D. R. Johnson](#),² [M. Lindsay Grayson](#),² and [Thierry Lambert](#)^{1,*}

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- Karina Olsen
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- National expertise service for the detection of antibiotic resistance (K-res)



BLAST result



Functions of the genes in this regions

- Hypothetical protein – 50
- Van genes
- DNA translocase FtsK
- putative endopeptidase p60
- **Daunorubicin/doxorubicin** resistance ATP-binding protein DrrA
- Conjugal transfer protein TraG
- Group II intron-encoded protein LtrA
- putative endopeptidase p60
- **Bacteriocin BCN5**
- DNA topoisomerase 3
- putative transcriptional regulatory protein YedW
- Signal-transduction histidine kinase senX3
- D-alanyl-D-alanine carboxypeptidase
- D-specific alpha-keto acid dehydrogenase
- D-alanyl-D-alanine dipeptidase
- Transposase from transposon Tn916