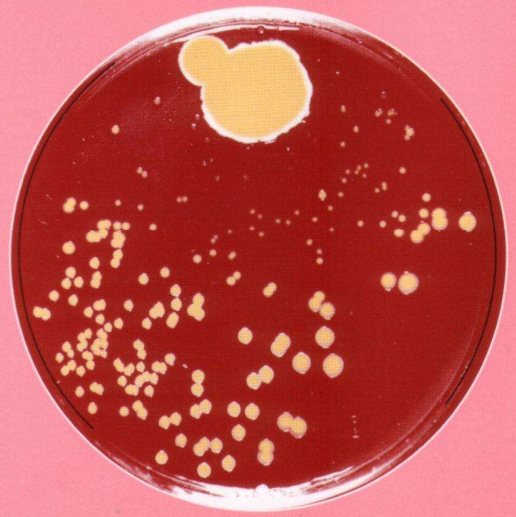
# Wound Infection

Dr Andrew Jones

VERSION 12

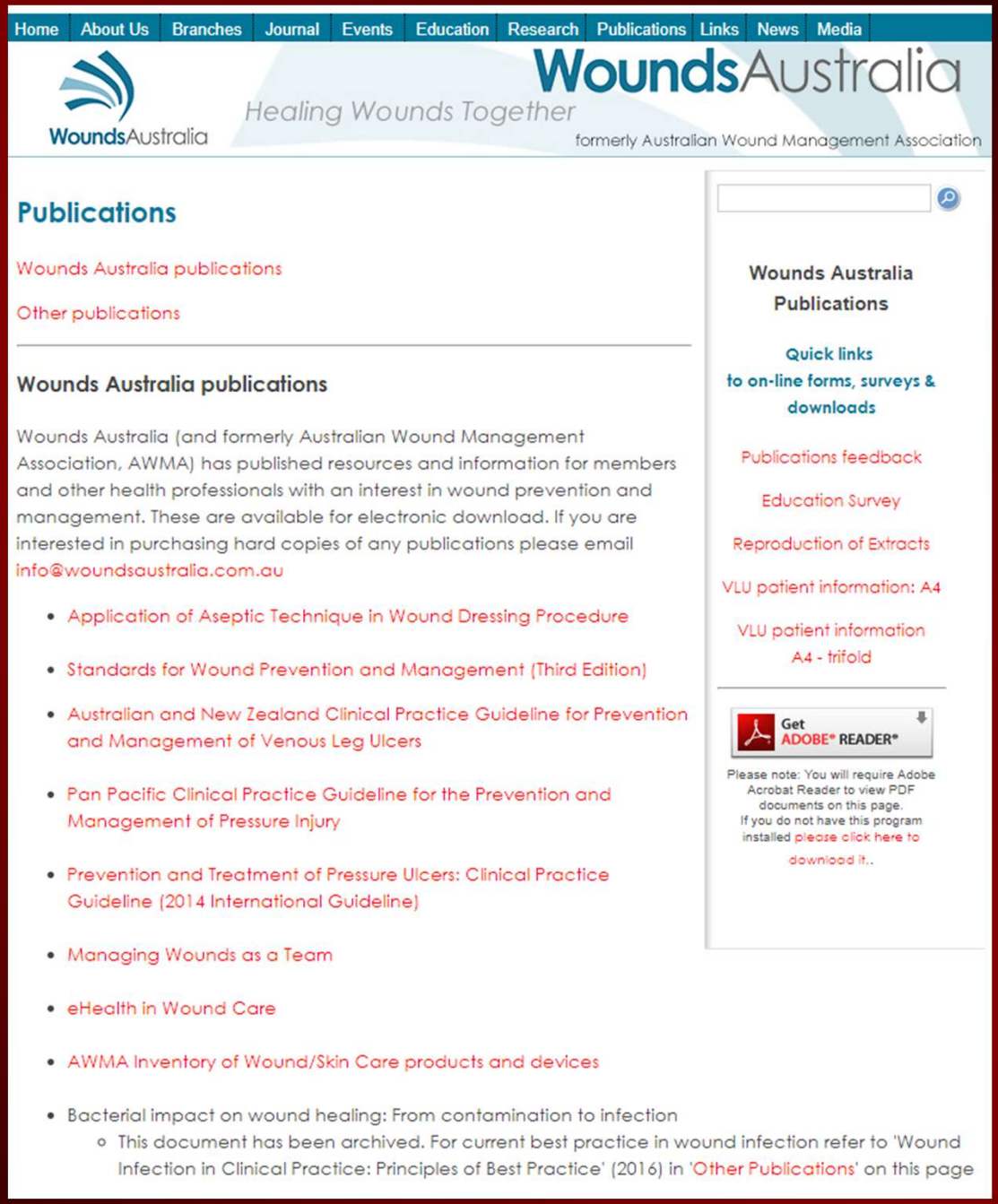
2003

# Therapeutic Guidelines
# **Antibiotic**



# TPG (antibiotics)

- For uninfected ulcers, cultures and antibiotic therapy are unnecessary. Topical antibiotics should not be used. For cellulitis, treat with systemic antibiotics—see Diabetic foot infection for antibiotic therapy.
- For detailed information on the management of venous leg ulcers, see the Australian and New Zealand clinical practice guideline [URL].



Home | About Us | Branches | Journal | Events | Education | Research | Publications | Links | News | Media

WoundsAustralia

Healing Wounds Together

WoundsAustralia

formerly Australian Wound Management Association

# Publications

Wounds Australia publications

Other publications

## Wounds Australia publications

Wounds Australia (and formerly Australian Wound Management Association, AWMA) has published resources and information for members and other health professionals with an interest in wound prevention and management. These are available for electronic download. If you are interested in purchasing hard copies of any publications please email info@woundsaustralia.com.au

- Application of Aseptic Technique in Wound Dressing Procedure
- Standards for Wound Prevention and Management (Third Edition)
- Australian and New Zealand Clinical Practice Guideline for Prevention and Management of Venous Leg Ulcers
- Pan Pacific Clinical Practice Guideline for the Prevention and Management of Pressure Injury
- Prevention and Treatment of Pressure Ulcers: Clinical Practice Guideline (2014 International Guideline)
- Managing Wounds as a Team
- eHealth in Wound Care
- AWMA Inventory of Wound/Skin Care products and devices
- Bacterial impact on wound healing: From contamination to infection
  - This document has been archived. For current best practice in wound infection refer to 'Wound Infection in Clinical Practice: Principles of Best Practice' (2016) in 'Other Publications' on this page

Wounds Australia Publications

Quick links to on-line forms, surveys & downloads

Publications feedback

Education Survey

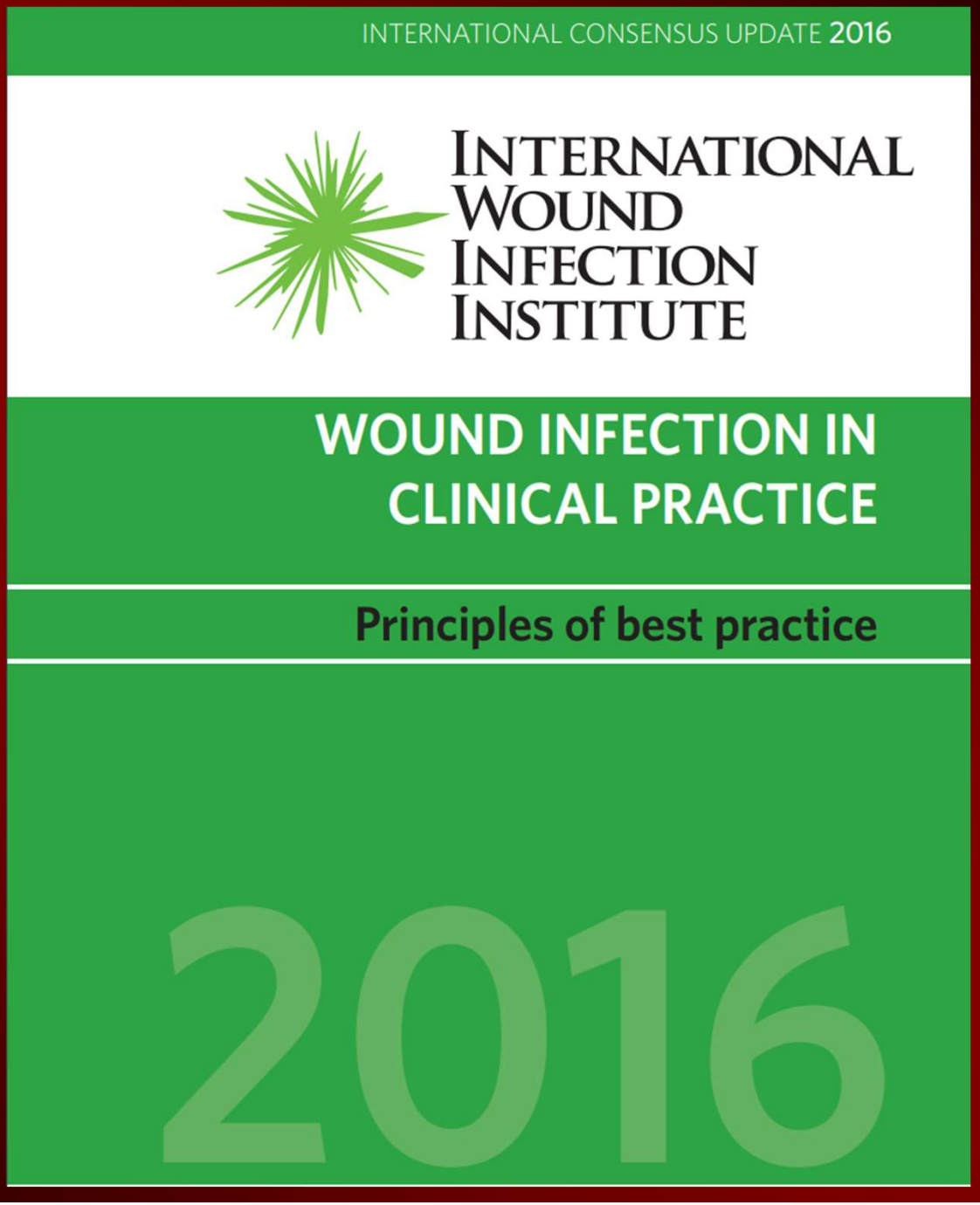
Reproduction of Extracts

VLU patient information: A4

VLU patient information A4 - trifold

Get ADOBE® READER®

Please note: You will require Adobe Acrobat Reader to view PDF documents on this page. If you do not have this program installed please click here to download it..


INTERNATIONAL CONSENSUS UPDATE 2016
INTERNATIONAL WOUND INFECTION INSTITUTE
WOUND INFECTION IN CLINICAL PRACTICE
Principles of best practice
2016

# Bacterial presence

Wounds usually contain bacteria – often without detrimental effect

**DEFINITIONS**

The presence of bacteria in a wound may result in:

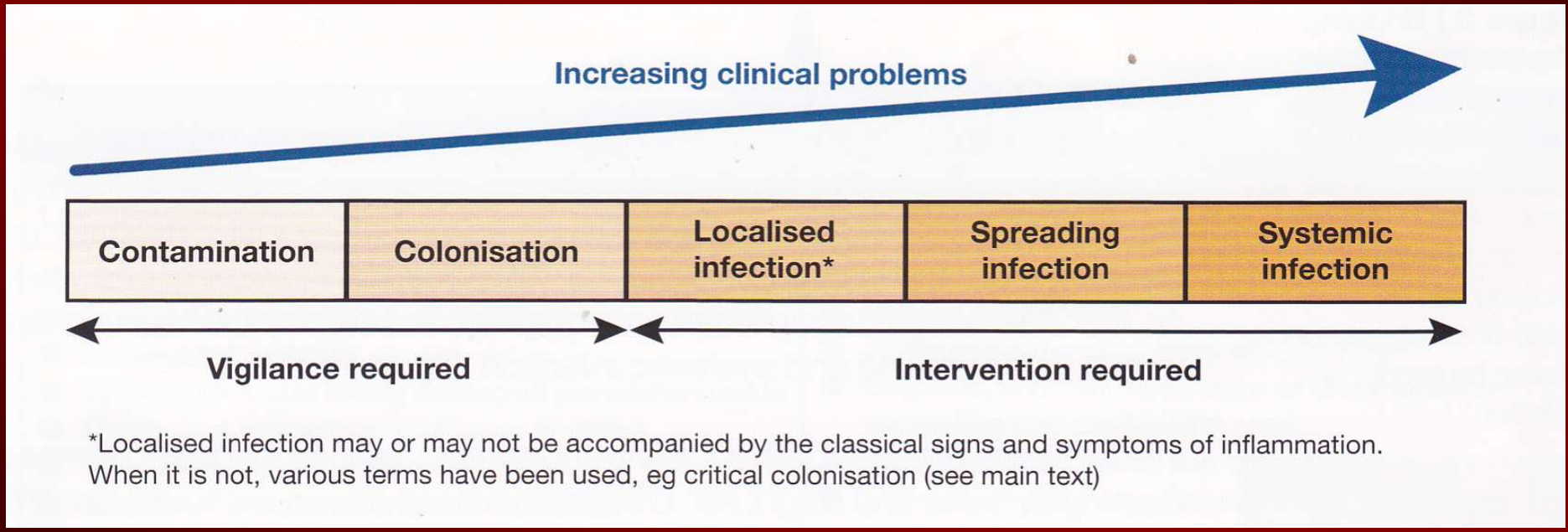
- **contamination** – the bacteria do not increase in number or cause clinical problems
- **colonisation** – the bacteria multiply, but wound tissues are not damaged
- **infection** – the bacteria multiply, healing is disrupted and wound tissues are damaged (local infection). Bacteria may produce problems nearby (spreading infection) or cause systemic illness (systemic infection) (Figure 1).


WAY OUT


CARLSBERG
TURNER
6
2


PA

# Infection




*Localised infection may or may not be accompanied by the classical signs and symptoms of inflammation. When it is not, various terms have been used, eg critical colonisation (see main text)

# Risk factors

**RISK OF INFECTION**

The risk of wound infection is increased by:

- any factor that debilitates the patient, impairs immune resistance or reduces tissue perfusion, eg:
  - comorbidities – diabetes mellitus, immunocompromised status, hypoxia/poor tissue perfusion due to anaemia or arterial/cardiac/respiratory disease, renal impairment, malignancy, rheumatoid arthritis, obesity, malnutrition
  - medication – corticosteroids, cytotoxic agents, immunosuppressants
  - psychosocial factors – hospitalisation/institutionalisation, poor personal hygiene, unhealthy lifestyle choices
- certain wound characteristics (Box 1) or poor standards of wound care related hygiene.

**Clinicians must maintain a high level of clinical suspicion for wound infection, particularly in patients with diabetes mellitus, autoimmune disorders, hypoxia/poor tissue perfusion, or immunosuppression**

**BOX 1 | Wound characteristics that may increase the risk of infection**

**Acute wounds**

- Contaminated surgery
- Long operative procedure
- Trauma with delayed treatment
- Necrotic tissue or foreign body*

**Chronic wounds**

- Necrotic tissue or foreign body*
- Prolonged duration
- Large in size and/or deep
- Anatomically situated near a site of potential contamination, eg anal area

*Particularly in the presence of hypoxia

**Figure 4 |** Triggers for suspecting wound infection (adapted from[2-4])

**NB:** Evidence is continuing to accumulate that in different wound types infection may produce specific characteristic signs and symptoms.

## ACUTE WOUNDS

eg surgical or traumatic wounds, or burns

| Localised infection | Spreading infection |
|---|---|
| ■ Classical signs and symptoms:<br>– new or increasing pain<br>– erythema<br>– local warmth<br>– swelling<br>– purulent discharge<br>■ Pyrexia – in surgical wounds, typically five to seven days post-surgery<br>■ Delayed (or stalled) healing (Box 5, see page 10)<br>■ Abscess<br>■ Malodour | As for localised infection PLUS:<br>■ Further extension of erythema<br>■ Lymphangitis (Box 5, see page 10)<br>■ Crepitus in soft tissues (Box 5, see page 10)<br>■ Wound breakdown/dehiscence |

**Notes**

- Burns – also skin graft rejection; pain is not always a feature of infection in full thickness burns
- Deep wounds – induration (Box 5, see page 10), extension of the wound, unexplained increased white cell count or signs of sepsis may be signs of deep wound (ie subfascial) infection
- Immunocompromised patients – signs and symptoms may be modified and less obvious

## SYSTEMIC INFECTION (adapted from[7-9])

Sepsis – documented infection with pyrexia or hypothermia, tachycardia, tachypnoea, raised or depressed white blood cell count

↓

Severe sepsis – sepsis and multiple organ dysfunction

↓

Septic shock – sepsis and hypotension despite adequate volume resuscitation

↓

Death

NB: Other sites of infection should be excluded before assuming that systemic infection is related to wound infection

## CHRONIC WOUNDS

eg diabetic foot ulcers, venous leg ulcers, arterial leg/foot ulcers or pressure ulcers

| Localised infection | Spreading infection |
|---|---|
| ■ New, increased or altered pain*<br>■ Delayed (or stalled) healing* (Box 5, see page 10)<br>■ Periwound oedema<br>■ Bleeding or friable (easily damaged) granulation tissue<br>■ Distinctive malodour or change in odour<br>■ Wound bed discoloration<br>■ Increased or altered/purulent exudate<br>■ Induration (Box 5, see page 10)<br>■ Pocketing (Figure 2)<br>■ Bridging (Figure 3) | As for localised infection PLUS:<br>■ Wound breakdown*<br>■ Erythema extending from wound edge<br>■ Crepitus, warmth, induration or discoloration spreading into periwound area<br>■ Lymphangitis (Box 5, see page 10)<br>■ Malaise or other non-specific deterioration in patient's general condition |

**Notes**

- In patients who are immunocompromised and/or who have motor or sensory neuropathies, symptoms may be modified and less obvious. For example, in a diabetic patient with an infected foot ulcer and peripheral neuropathy, pain may not be a prominent feature[4]
- Arterial ulcers – previously dry ulcers may become wet when infected
- Clinicians should also be aware that in the diabetic foot, inflammation is not necessarily indicative of infection. For example, inflammation may be associated with Charcot's arthropathy

*Individually highly indicative of infection. Infection is also highly likely in the presence of two or more of the other signs listed


2. Cutting KF, Harding KG. Criteria for identifying wound infection. *J Wound Care* 1994; 3(4): 198-201.
3. Gardner SE, Frantz RA, Doebbeling BN. The validity of the clinical signs and symptoms used to identify localized chronic wound infection. *Wound Repair Regen* 2001; 9(3): 178-86.
4. European Wound Management Association. Position Document: *Identifying criteria for wound infection*. London: MEP Ltd, 2005.
5. Wilson AP, Treasure T, Sturridge MF, Grüneberg RN. A scoring method (ASEPSIS) for postoperative wound infections for use in clinical trials of antibiotic prophylaxis. *Lancet* 1986; 1(8476): 311-13.
6. Horan TC, Gaynes RP, Martone WJ, et al. CDC definitions of nosocomial surgical site infections 1992: a modification of CDC definitions of surgical wound infections. *Infect Control Hosp Epidemiol* 1992; 13(10): 606-8.
7. Remick DG. Pathophysiology of sepsis. *Am J Path* 2007; 170(5): 1435-44.
8. Lever A, Mackenzie I. Sepsis: definition, epidemiology and diagnosis. *BMJ* 2007; 335: 879-83.
9. Levy MM, Fink MP, Marshall JC, et al. 2001 SCCM/ESICM/ACCP/ATS/SIS International sepsis definitions conference. *Crit Care Med* 2003; 31(4): 1250-56.

# Microbiology

**BOX 2 | Indications for wound specimen collection for microbiological analysis**

- Acute wounds with signs of infection*
- Chronic wounds with signs of spreading or systemic* infection† (Figure 4, see page 3)
- Infected chronic wounds that have not responded to or are deteriorating despite appropriate antimicrobial treatment
- As required by local surveillance protocols for drug resistant micro-organisms

*In patients showing signs of sepsis, blood cultures are important, and cultures of other likely sites of infection should be considered

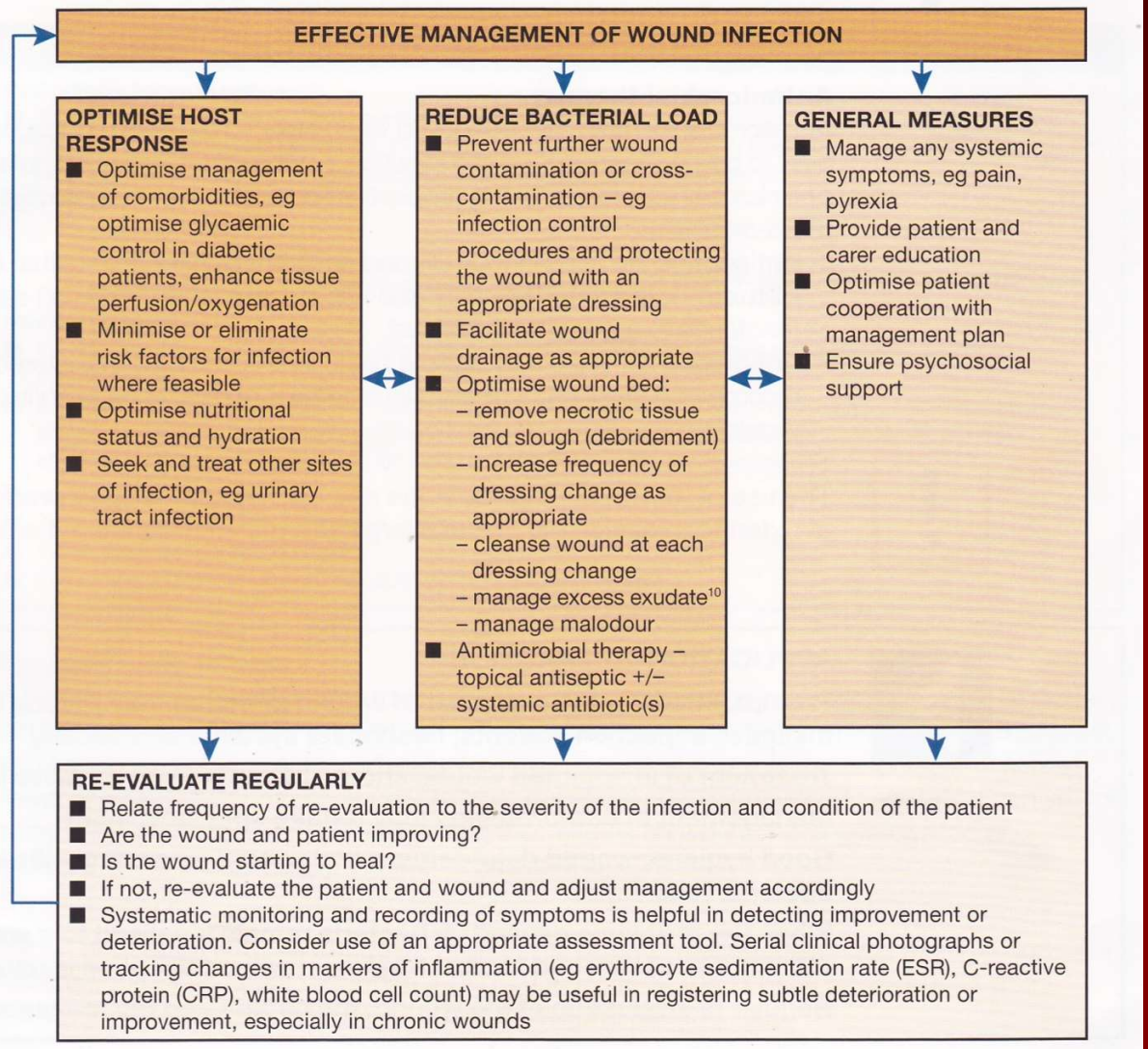
†Also consider for high-risk chronic wounds with signs of localised infection, eg delayed (or stalled) healing, in patients who have diabetes mellitus or peripheral arterial disease, or who are taking immunosuppressants or corticosteroids

Beware of interpreting a microbiology report in isolation – consider the report in the context of the patient and the wound and, if appropriate, consult a microbiologist or infectious disease specialist

## REDUCING BACTERIAL LOAD

### Effective hygiene and preventative measures

Infection control procedures should be followed to prevent further contamination of the wound and cross-contamination. Good hygiene practice includes paying particular attention to thorough hand cleansing/disinfection and suitable protective working clothes, including gloves.

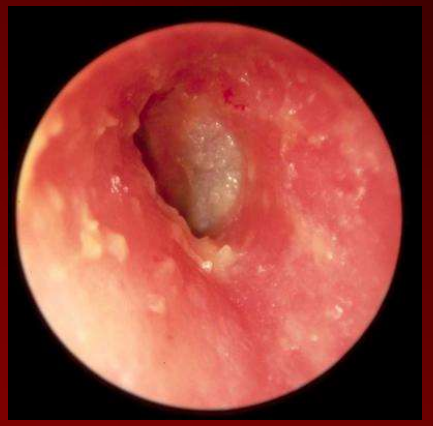


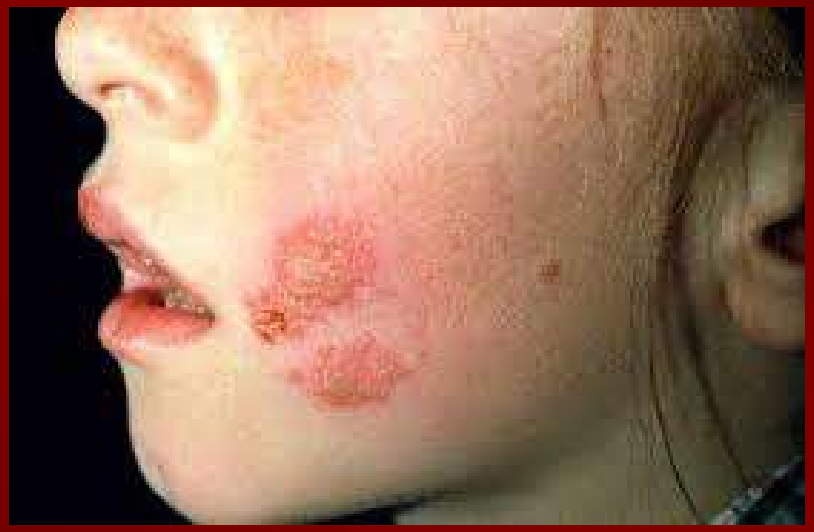
# Topical antibiotics

The use of topical antibiotics in the management of infected wounds should generally be avoided to minimise the risk of allergy and the emergence of bacterial resistance

Topical antibiotics should only be used in infected wounds under very specific circumstances by experienced clinicians (eg topical metronidazole might be used for the treatment of malodour in fungating wounds)

# Topicals





# Systemic Rx

**BOX 4 | Using systemic antibiotics in wound infection**

**Indications for systemic antibiotics**

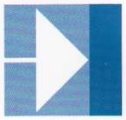
- Prophylaxis where risk of wound infection is high, eg contaminated colonic surgery or 'dirty' traumatic wounds
- Spreading or systemic wound infection
- When culture results reveal β-haemolytic streptococci, even in the absence of signs of infection

**Review antibiotic regimen**

- If there is no improvement of systemic or local signs and symptoms, re-evaluate the patient and the wound; consider microbiological analysis and changing antibiotic regimen
- If the patient has an antibiotic-related adverse event; discontinue causative antibiotic

**Discontinue/review systemic antibiotics**

- At the end of the prescribed course (according to type of infection, wound type, patient comorbidities and local prescribing policy)



**APPLICATION TO PRACTICE**

**Use systemic antibiotics in the context of a management plan that incorporates optimising host immune response and local methods of reducing bacterial load (Figure 5, see page 5)**

**Clearly define reasons for use, treatment goals and duration of antibiotic therapy**

**In chronic wounds, unless the patient is systemically unwell or a limb is in danger, microbiological results should usually be awaited before commencing systemic antibiotics**

**Seek local expert advice to determine the most appropriate antibiotic(s) to use**

**If empirical treatment is necessary, start with an appropriate broad-spectrum antibiotic. When antibiotic susceptibilities become available follow local microbiological/infectious disease advice, possibly switching to a narrower-spectrum agent**

**Table 1 |** Antiseptics that may be used in the management of wound infection

| Antiseptic | Formulation(s) | Notes |
|---|---|---|
| **Acetic acid** | Solution | ■ Considered for its effect against *Pseudomonas aeruginosa*<br>■ Consider protecting periwound skin during use |
| **Chlorhexidine** | Solution, powder, impregnated dressings | ■ May be used as an alternative in patients allergic to iodine preparations |
| **Honey** | Available for direct application, impregnated dressings | ■ Antimicrobial effects have been attributed to some components and physical properties. However, composition (and hence antibacterial activity) is highly variable, making comparison of clinical trials difficult |
| **Hydrogen peroxide** | Solution, cream | ■ Caution is advised when using the solution because cases of gas embolism have been described |
| **Iodine** | PVP-I: solution, cream, ointment, spray, impregnated dressings<br>Cadexomer iodine: ointment, paste, powder, impregnated dressings | ■ Modern products slowly release relatively low levels of iodine, reducing the likelihood of toxicity and staining<br>■ Povidone iodine (polyvinylpyrrolidone iodine – PVP-I) is an iodine–surfactant complex<br>■ Cadexomer iodine releases iodine from highly absorbent beads |
| **Potassium permanganate** | Solution, tablets for dissolving in water | ■ Used as a soak to reduce wound bacterial load<br>■ Has astringent effect; may be useful in 'weepy' wounds |
| **Polyhexamethyl biguanide (PHMB)** | Solution, impregnated dressings | ■ Also known as polyhexanide and polyaminopropyl biguanide; related to chlorhexidine<br>■ Currently used mainly for burns |
| **Silver** | Silver sulfadiazine: cream, impregnated dressings<br>Ionic silver: impregnated dressings, nanocrystalline silver | ■ Available in several forms including silver sulfadiazine (silver–antibiotic combination)<br>■ More recently, dressings have become available that release charged silver atoms (ionic silver – $Ag^+$) on contact with wound fluid<br>■ The amount/rate of ionic silver released from different dressings is variable. Initial release of high levels followed by sustained release appears to aid reduction in bacterial numbers and a wide spectrum of activity<br>■ Staining of the wound bed or surrounding skin by ionic silver dressings may occur occasionally and is usually reversible |
| **Sodium hypochlorite** | Solution | ■ Not usually recommended unless suitable alternatives are unavailable |
| **Triclosan** | Solution, impregnated dressings | ■ Mainly used as a skin disinfectant or surgical scrub |

# High Usage of Topical Fusidic Acid and Rapid Clonal Expansion of Fusidic Acid–Resistant *Staphylococcus aureus*: A Cautionary Tale

**Deborah A. Williamson,[1,2,3] Stefan Monecke,[4,5] Helen Heffernan,[2] Stephen R. Ritchie,[1] Sally A. Roberts,[6] Arlo Upton,[7] Mark G. Thomas,[1] and John D. Fraser[1,3]**

[1]Department of Molecular Medicine and Pathology, University of Auckland, [2]Institute of Environmental Science and Research, Wellington, and [3]Maurice Wilkin Centre for Molecular Biodiscovery, Auckland, New Zealand; [4]Alere Technologies, Jena, and [5]Institute for Medical Microbiology and Hygiene, Dresden, Germany; [6]Department of Clinical Microbiology, Auckland District Health Board, and [7]LabTest Auckland, New Zealand

**Our aim was to assess national prescribing trends and deter mine longitudinal resistance patterns for topical antimicrobi als in New Zealand. We observed a dramatic increase in fusidi acid (FA) resistance, and clonal expansion of FA-resistan *Staphylococcus aureus*. This increase was concurrent with significant national increase in topical FA dispensing.**

***Keywords.*** antimicrobial use; antimicrobial resistance epidemiology; *fusC*; MRSA.

Although evidence-based prescribing supports the use of topi cal antimicrobials for only a few dermatological indications, in cluding impetigo, studies suggest that topical antimicrobials ar often prescribed for a number of other, nonindicated skin con ditions [1]. Importantly, previous work has demonstrated tha injudicious use of topical antimicrobials, such as fusidic aci (FA) or mupirocin, is associated with the rapid developmen of resistance. For example, a study in 1999 in New Zealan (NZ) suggested that unrestricted usage of mupirocin contribut ed to a high rate (28%) of mupirocin resistance in communit isolates of *Staphylococcus aureus* [2]. The same study als

Received 1 May 2014; accepted 7 August 2014; electronically published 18 August 2014.
Correspondence: Deborah A. Williamson, BSc, MBChB, MRCP, FRCPA, Department of Mole ular Medicine and Pathology, University of Auckland, Private Bag 92019, Auckland, NZ (debbi williamson@esr.cri.nz).

**Clinical Infectious Diseases® 2014;59(10):1451–4**

DOI: 10.1093/cid/ciu658

# AAAAARRGHHHH!

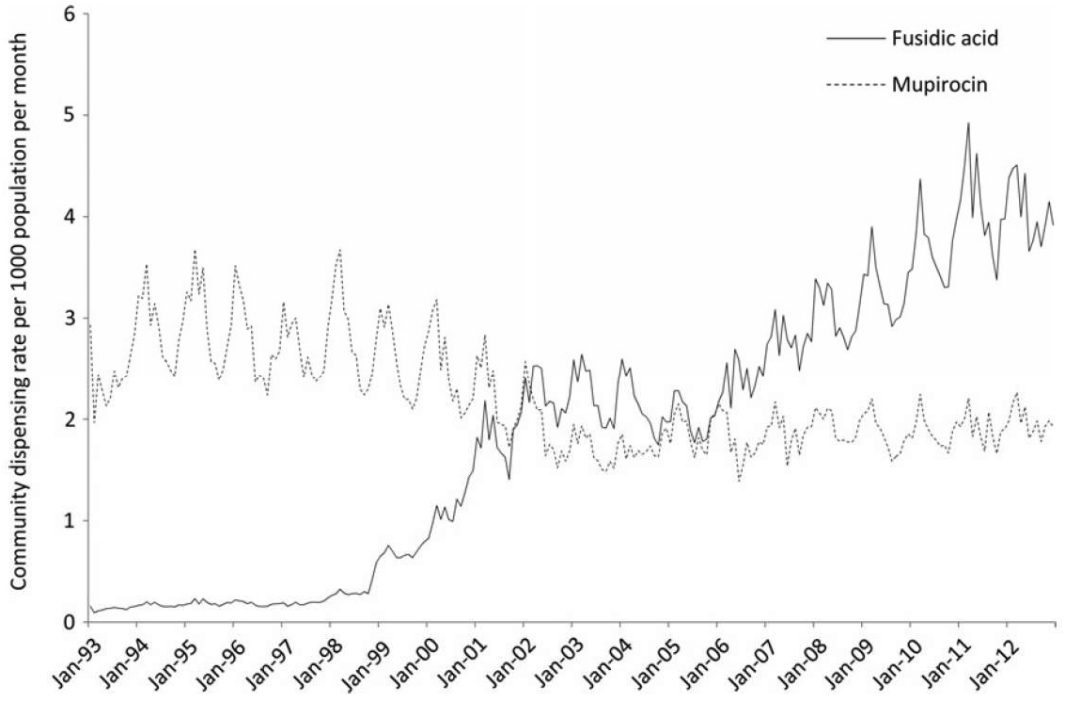
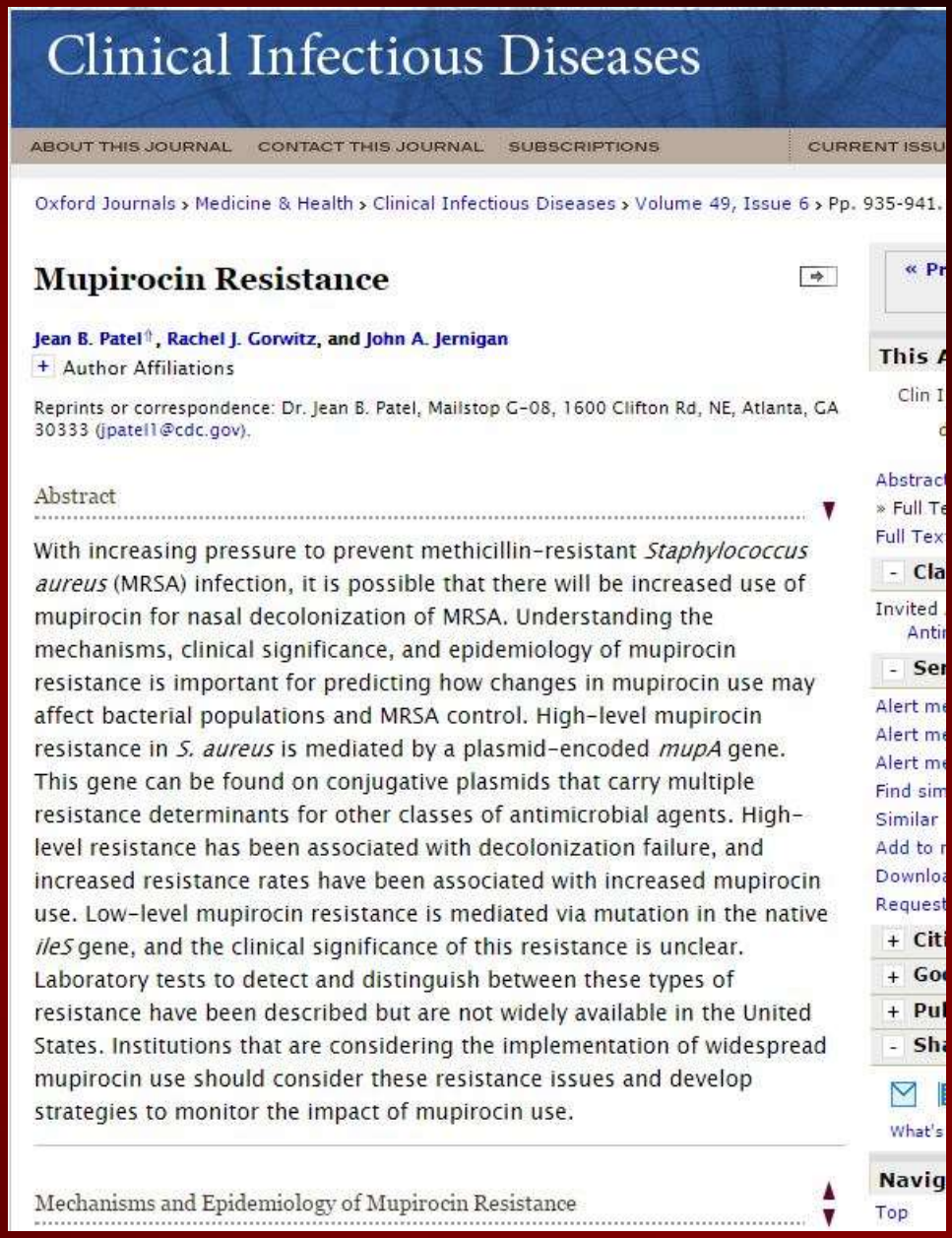



**Figure 1.** Community dispensing rates for topical fusidic acid and mupirocin in New Zealand, January 1993 to December 2012.

# More Aaaarrgghh



Clinical Infectious Diseases

ABOUT THIS JOURNAL | CONTACT THIS JOURNAL | SUBSCRIPTIONS | CURRENT ISSU

Oxford Journals › Medicine & Health › Clinical Infectious Diseases › Volume 49, Issue 6 › Pp. 935-941.

## Mupirocin Resistance

Jean B. Patel, Rachel J. Gorwitz, and John A. Jernigan

+ Author Affiliations

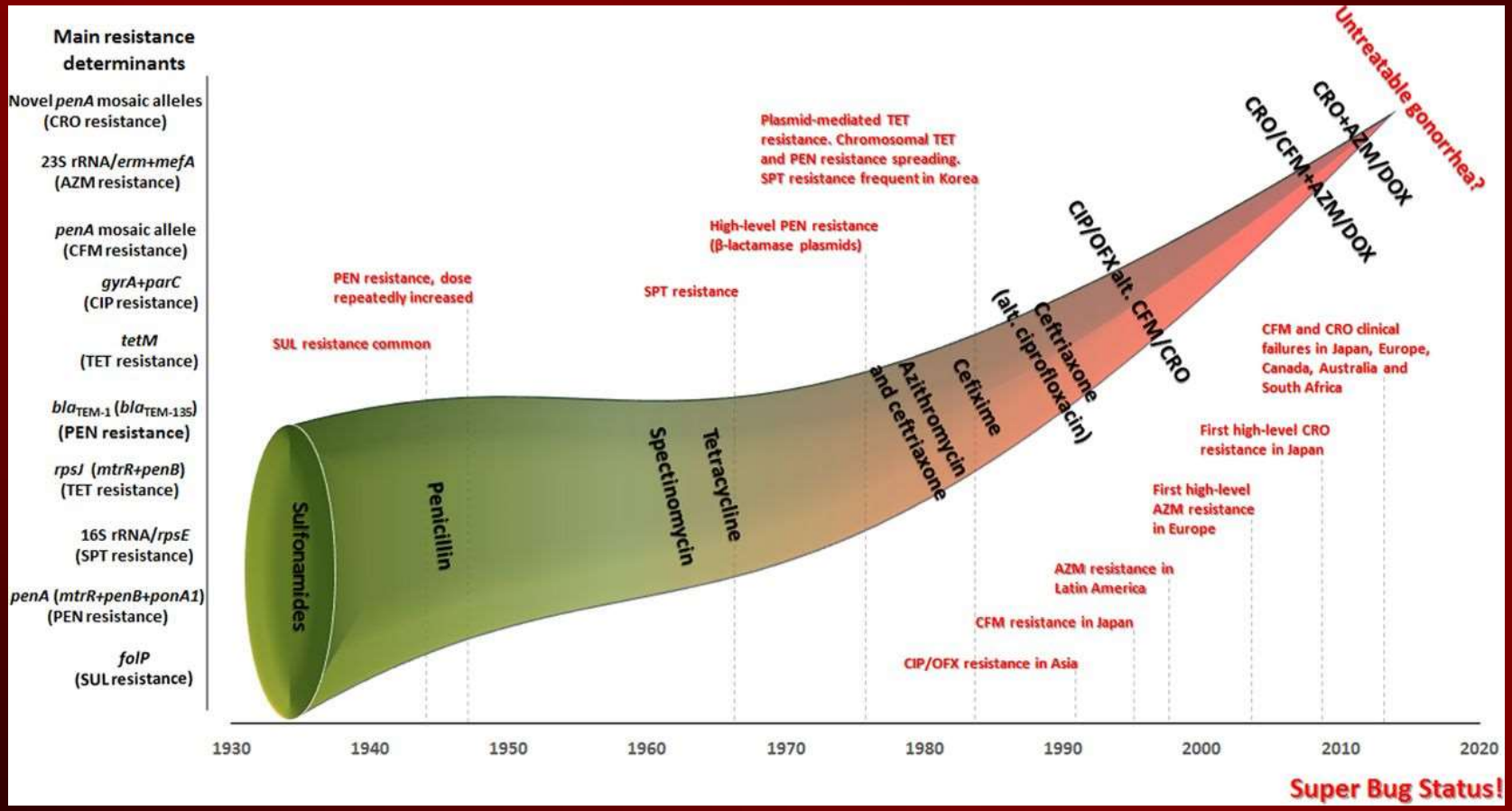
Reprints or correspondence: Dr. Jean B. Patel, Mailstop G-08, 1600 Clifton Rd, NE, Atlanta, GA 30333 (jpatel1@cdc.gov).

Abstract

With increasing pressure to prevent methicillin-resistant *Staphylococcus aureus* (MRSA) infection, it is possible that there will be increased use of mupirocin for nasal decolonization of MRSA. Understanding the mechanisms, clinical significance, and epidemiology of mupirocin resistance is important for predicting how changes in mupirocin use may affect bacterial populations and MRSA control. High-level mupirocin resistance in *S. aureus* is mediated by a plasmid-encoded *mupA* gene. This gene can be found on conjugative plasmids that carry multiple resistance determinants for other classes of antimicrobial agents. High-level resistance has been associated with decolonization failure, and increased resistance rates have been associated with increased mupirocin use. Low-level mupirocin resistance is mediated via mutation in the native *ileS* gene, and the clinical significance of this resistance is unclear. Laboratory tests to detect and distinguish between these types of resistance have been described but are not widely available in the United States. Institutions that are considering the implementation of widespread mupirocin use should consider these resistance issues and develop strategies to monitor the impact of mupirocin use.

Mechanisms and Epidemiology of Mupirocin Resistance

# *Neisseria gonorrhoeae*




COCAINE
TOOTHACHE DROPS
Instantaneous Cure!
PRICE 15 CENTS.
Prepared by the
LLOYD MANUFACTURING CO.
219 HUDSON AVE., ALBANY, N. Y.
For sale by all Druggists.
(Registered March 1885.)
See other side.