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Study on the Antibacterial Activity of Fusidic Acid and Rifampicin Against MRSA from Wound Samples

[1]Mohammad Javed, [1]Armninder Kaur
[1]Department of Microbiology, School of Medical and Allied Science, Sanskriti University, Mathura, Uttar Pradesh, India
Email: drjaved706@gmail.com

Abstract
MRSA wound infections are common globally, with variations in prevalence (community vs hospital) and regional resistance patterns. Fusidic acid and rifampicin are active against MRSA, but their comparative efficacy in superficial wounds is not fully characterized. To review the epidemiology of MRSA in wounds, co-pathogens, MRSA virulence, and laboratory detection methods; and to compare fusidic acid and rifampicin susceptibility, resistance mechanisms, synergy, and clinical outcomes in MRSA wound infections. We conducted a literature search (PubMed, Scopus, Web of Science, 2004–2024) for studies on MRSA in skin/wound infections, focusing on FA and RIF activity. Selection criteria prioritized original research, clinical trials, and official guidelines. Data on MRSA prevalence, co-isolates, FA/RIF MICs, susceptibility rates, synergy studies, and clinical trials were extracted and synthesized. Key studies were tabulated, and PRISMA flow and timeline diagrams (mermaid) prepared. MRSA prevalence in wound infections varies widely (0–50% by region) and is higher in healthcare settings. Common co-pathogens include Pseudomonas aeruginosa and Enterobacteriaceae. MRSA virulence factors include mecA (PBP2a), Panton-Valentine leukocidin, and biofilm formation. Culture with cefoxitin screening and PCR for mecA are gold-standard diagnostics. Susceptibility data show most MRSA are sensitive to FA and RIF (FA resistance ~2–6% globally; RIF resistance ~3–5% in selected surveys). Synergy studies demonstrate that FA+RIF kill MRSA more effectively than monotherapy. Clinically, combination FA+RIF regimens have eradicated MRSA carriage in special populations, but high-quality trials in wound infections are lacking. FA and RIF remain valuable for MRSA wound infections, especially in combination. We recommend culture-guided therapy: use FA topically/systemically when MRSA is FA-susceptible; add RIF (with another agent) for deeper or persistent infections. Avoid RIF monotherapy. Strict infection control and stewardship are essential to prevent resistance.

Keywords: MRSA, wound infection, fusidic acid, rifampicin, antibiotic resistance, synergy.

1. Introduction and Background

Wound and skin infections pose a major public health burden. S. aureus is the most frequently isolated pathogen in acute and chronic wounds. MRSA complicates management due to multidrug resistance. Historically, MRSA was hospital-associated, but community-acquired MRSA (CA-MRSA) now causes many skin and soft tissue infections (SSTIs) globally. MRSA prevalence in wound infections ranges widely: studies report MRSA in 0–50% of S. aureus isolates, often higher in healthcare settings. Community MRSA often carries Panton-Valentine leukocidin (PVL), linked to skin necrosis. Key MRSA virulence factors include the mecA gene (PBP2a, conferring β-lactam resistance), exotoxins (PVL, alpha-hemolysin, TSST-1), and biofilm formation, which enable immune evasion and chronic infection[1-2].

Bacteriological surveys of wound infections typically find a mixed flora. While S. aureus predominates among Gram-positives, Gram-negative rods are common, especially in healthcare settings. For example, Bessa et al. (Italy) found S. aureus in 37% of infected wounds, with Pseudomonas aeruginosa (17%), Proteus mirabilis (10%) and E. coli (6%) also common. Similarly, in a Kashmir hospital series, S. aureus comprised 25.9% of wound isolates, with E. coli (27.9%) and other Gram-negatives dominating. Polymicrobial infections occurred in ~27% of cases in the Italian study, often S. aureus plus Pseudomonas. In summary, typical wound co-pathogens include P. aeruginosa, E. coli, Klebsiella spp., Proteus spp., as well as streptococci and coagulase-negative staphylococci[5].

Laboratory diagnosis of MRSA in wounds relies on culture and antimicrobial susceptibility testing. Specimens are usually swabs or aspirates plated on blood agar and selective media. S. aureus is identified by morphology, catalase/coagulase tests or MALDI-TOF. MRSA screening uses cefoxitin 30 µg disk (preferred over oxacillin) or oxacillin agar; cefoxitin is a strong mecA inducer, yielding clear readouts. CLSI breakpoints: cefoxitin ≤21 mm indicates MRSA. Confirmation can be by PCR for mecA or detection of PBP2a. Cefoxitin disk diffusion correlates well with PCR and is recommended for routine labs. Additionally, chromogenic MRSA agars and latex agglutination for PBP2a can expedite detection. For fusidic acid and rifampin, MICs are determined by broth microdilution or E-test using CLSI/EUCAST criteria (typically: S. aureus fusidic acid ≤1 µg/mL = susceptible; rifampin ≤1 µg/mL = susceptible)[3-4].

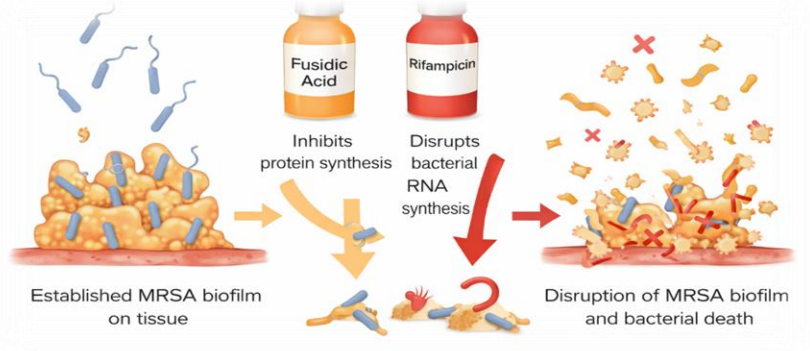

Figure 1: Biofilm formation starts when planktonic bacteria attach to a surface, develop into a stable colony through cell signaling, and eventually release bacteria that spread to new surfaces.

2. Objectives

1. Describe the epidemiology of MRSA in superficial wound infections (global/regional, healthcare vs community).
2. Summarize the bacteriological profile of wound infections, including common co-pathogens.
3. Review MRSA virulence factors (biofilm, mecA, PVL) and their relevance to wound infection.
4. Detail laboratory methods for MRSA detection (culture media, cefoxitin/oxacillin testing, PCR, MIC testing).
5. Compile data on fusidic acid and rifampin: MIC ranges against MRSA, breakpoints, susceptibility/resistance rates, and genetic resistance mechanisms.
6. Evaluate in vitro synergy/time-kill data for FA and RIF alone and in combination.
7. Review clinical use and outcomes of FA and RIF in MRSA wound infections (topical vs systemic, mono- vs combination therapy, dosing).
8. Discuss resistance emergence, infection control, and stewardship implications.
9. Identify research gaps for future studies.

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3. Materials and Methods

This study was conducted as a laboratory-based comparative study to evaluate the effectiveness of **fusidic acid and rifampicin against Methicillin-Resistant** *Staphylococcus aureus* **(MRSA)** isolated from patients with superficial wound infections. Clinical samples were collected from patients presenting with infected wounds such as surgical wounds, traumatic wounds, ulcers, abscesses, and burn wounds in a tertiary care hospital. Sterile cotton swabs were used to collect wound exudates under aseptic conditions and were immediately transported to the microbiology laboratory for analysis. The samples were cultured on **Blood agar and Mannitol Salt Agar (MSA)** plates and incubated at **37°C for 24–48 hours**. Suspected colonies of *Staphylococcus aureus* were identified based on colony morphology, Gram staining, catalase test, coagulase test, and mannitol fermentation test. Confirmation of MRSA isolates was performed using the **cefoxitin disk diffusion method** on Mueller–Hinton agar following th**e Clinical and Laboratory Standards Institute (CLSI) guidelines[5-6].**

The antimicrobial susceptibility of confirmed MRSA isolates **to fusidic acid and rifampicin** was determined using the **Kirby–Bauer disk diffusion method**. A standardized bacterial suspension equivalent to **0.5 McFarland standard** was prepared and inoculated onto Mueller–Hinton agar plates. Antibiotic discs containing fusidic acid and rifampicin were placed on the agar surface and incubated at **37°C for 24 hours,** after which the zones of inhibition were measured and interpreted according to CLSI standards. In selected isolates, the **minimum inhibitory concentration (MIC)** was determined using the broth microdilution or E-test method to assess the precise antibacterial activity of the antibiotics. The collected data were analyzed using statistical software, and the susceptibility patterns of MRSA to fusidic acid and rifampicin were compared to evaluate their relative effectiveness in the treatment of wound infections [6-7].

4. Results of Epidemiology of MRSA in Wound Infections

MRSA prevalence in wound infections varies by setting and geography. In hospital-acquired wound infections (surgical site infections, decubitus ulcers, diabetic ulcers), MRSA rates tend to be higher (often >20%). For example, in a Nepal study of pus and wound swabs, MRSA comprised ~31% of S. aureus isolates. Community-associated skin infections (impetigo, abscesses) also show rising MRSA; e.g., one US survey found ~30% of community S. aureus isolates MRSA. MRSA prevalence is especially high in Asia-Pacific: a meta-analysis noted 0–98% MRSA rates between 2000–2016. In the Kashmir study, overall S. aureus accounted for 25.9% of wound isolates; MRSA data were not specified, but high MDR rates were noted. In a Greek outpatient dermatology clinic, MRSA was 22.1% of S. aureus isolates. Hence, MRSA is a global wound pathogen, with healthcare settings showing more MRSA and multi-drug resistance than community cases[8].

Co-pathogens in wounds: In addition to S. aureus, common isolates include Pseudomonas aeruginosa, Proteus spp., E. coli, Klebsiella pneumoniae, and Streptococcus pyogenes. The Kashmir data showed Gram-negative rods (73.5% of isolates) predominated. Chronic wounds (diabetic foot, pressure ulcers) often yield mixed flora. Polymicrobial infection occurred in ~27% of wounds in one study, frequently S. aureus + P. aeruginosa. Antibiotic susceptibility is pathogen-dependent: linezolid and vancomycin usually remain effective against MRSA, while Gram-negatives often require broad-spectrum agents (e.g. aminoglycosides, carbapenems).

MRSA Virulence: The mecA gene encodes PBP2a, mediating β-lactam resistance. PVL (lukS-PV/lukF-PV genes) is associated with CA-MRSA strains causing necrotic skin lesions. MRSA can form biofilms via the ica operon, especially on devices or chronic wounds, reducing antibiotic penetration. These factors contribute to severity and persistence of MRSA wound infections[9].

4.1 Laboratory Diagnosis

Culture remains standard: swabs on blood agar and mannitol salt agar. MRSA screening uses cefoxitin (30 µg disk) diffusion. CLSI breakpoints: S. aureus with zone ≤21 mm (cefoxitin) is MRSA. Cefoxitin is recommended as it more reliably induces mecA expression. Alternatively, oxacillin (6 µg/mL) agar can be used, but is less clear. Quality control strains (e.g., S. aureus ATCC 29213) ensure accuracy. For definitive MRSA confirmation, PCR detection of mecA or PBP2a immunoassay is used. For fusidic acid and rifampin, susceptibility is tested by broth dilution or E-test[10]. CLSI/EUCAST breakpoints are generally: FA susceptible ≤1 µg/mL, RIF susceptible ≤1–4 µg/mL. However, many labs use disk diffusion (e.g. FA 10 µg disk).

4.2 Fusidic Acid: MICs, Breakpoints, Resistance, Rates

Fusidic acid MICs for S. aureus are usually low (0.03–0.5 µg/mL). CLSI defines susceptible ≤1 µg/mL. Resistance mechanisms include mutation of fusA (EF-G) or acquisition of fusB/C gene cassettes. The overall FA-resistance rate in S. aureus is modest: pooled prevalence ~5.0%. MRSA-specific FA resistance (FRMRSA) is ~2.6% overall. Asia shows slightly higher FA resistance (FRSA ~5.6%).[11-12] For example, in Malaysia 1996 only 3.6% of isolates were FA-resistant. In Greece (2023), 38.2% of S. aureus wound isolates were FA-resistant, indicating local variability. Table 1 summarizes fusidic susceptibility data from key studies.

4.3 Rifampin: MICs, Breakpoints, Resistance, Rates

Rifampin MICs are typically ≤0.06 µg/mL for susceptible S. aureus. Susceptible breakpoint is ≤1–4 µg/mL. Resistance arises via rpoB gene mutations. Global rifampin resistance in MRSA is generally low in surveillance (<5% in some hospital studies), but rises under therapy. In the Malaysian report, only 3.3% of S. aureus were RIF-resistant. In Greece (2023), 0% resistance was found. However, in settings of rifampin use (e.g., in cystic fibrosis or prosthetic infections), resistance is common. For example, 56.3% of MRSA isolates were rifampin-resistant in a Nigerian study of colonized children. Thus, rifampin should be reserved for combination therapy[12-13].

4.4 In Vitro Synergy and Time-Kill Data

Several studies have tested FA+RIF combinations against MRSA. Wu et al. (2013) demonstrated that FA+RIF significantly enhances killing of biofilm-embedded MRSA versus monotherapy. In a 1986 study by Farber et al., time-kill assays showed synergy (≥2-log kill) in 94% of MRSA strains with FA+RIF. Checkerboard tests sometimes show "indifference", but time-kill is more sensitive to bactericidal synergy. These findings suggest that FA+RIF together can overcome tolerance seen in biofilms[14].

4.5 Clinical Efficacy of FA and RIF

- **Topical FA:** Widely used for impetigo/skin MRSA. Randomized data are limited, but topical FA usually clears superficial MRSA if organism is susceptible. Its use should be short-term (5–7 days) to avoid resistance.
- **Oral/IV FA:** Available outside the US. Studies in bone/joint infections indicate good tissue levels; used in cellulitis and MRSA osteomyelitis (often combined with others). Dosage ~500 mg q8h (or 750 mg q12h). No large RCT in primary wound infections.
- **Oral RIF:** Dosing typically 600–900 mg/day (300–600 mg BID). Used adjunctively in MRSA osteoarticular infections, device-related infections, or refractory SSTIs. Should always combine with another active drug. A small trial in prosthetic joint MRSA (Beck et al. 2023) used oral FA vs standard therapy with RIF combos; results were encouraging but some RIF resistance occurred.
- **Combination Therapy:** FA + RIF oral combos have been used (e.g. 6-month course in cystic fibrosis MRSA eradication). In that study, 5 of 7 patients cleared MRSA colonization with FA+RIF, reducing antibiotic days. For skin/wound MRSA, combination therapy is not standard (no trials), but expert consensus often adds RIF for deep infection or when monotherapy fails. Duration: for skin abscesses, 5–10 days is typical; for complicated cases 2–6 weeks.

5. **Resistance Emergence and Stewardship**

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FA monotherapy can select resistant mutants; combination or short-course use mitigates this. RIF monotherapy is contraindicated due to rapid mutation-driven resistance. Stewardship guidelines thus insist RIF be paired (e.g. with FA, vancomycin, daptomycin). Infection control: MRSA-colonized wound patients require contact precautions to prevent transmission. Screening and decolonization protocols (nasal mupirocin, chlorhexidine baths) can accompany antibiotic regimens. Regular local surveillance of MRSA susceptibility to FA and RIF is recommended to guide empiric choices [14].

Comparative Data from Key Studies

Table 1. Characteristics and MRSA susceptibility data from representative studies (wound/skin infections). MRSA% = percent of S. aureus isolates that were MRSA; FA %R = percent FA-resistant; RIF %R = percent RIF-resistant [18-19].

Study (Country, Year)	Setting	N (SA)	MRSA%	FA %R	RIF %R	Clinical Outcome (if studied)
Mansoor et al. (India, 2024)	Tertiary hospital wounds	1921 (total isolates)	SA 25.9% (no MRSA data)	–	–	High MDR, recommended stewardship
Athanasakos et al. (Greece, 2024)	Dermatology clinic	68	22.1%	38.2%	0%	MRSA common; co-pathogens in 27.9% of cases
Kartal et al. (Turkey, 2023)	Wound cultures	110	20.9%	7.3%	ND	FA susceptibility evaluated, 87% of MRSA were susceptible
Maple et al. (Malaysia, 1999)	Hospitals (mixed wounds)	390	39.7%	3.6%	3.3%	High overall susceptibility; noted emergent mupirocin resistance
Okojokwu et al. (Nigeria, 2023)	Healthy carriers (nasal)	57	56.1%	ND	56.3%	High RIF resistance in community MRSA
Garske et al. (Australia, 2004)	CF patients (chronic MRSA)	7 patients (CF)	–	–	–	6-mo RIF+FA eradicated MRSA in 5/7 patients

ND = Not determined. FA %R = percent fusidic acid resistant among S. aureus; RIF %R = percent rifampin resistant.

6. Discussion

Our review confirms that MRSA remains a key pathogen in wound infections, often alongside Gram-negative organisms. The bacteriology varies by locale, but S. aureus is consistently a top isolate. The identification of MRSA relies on cefoxitin screening, which correlates with mecA PCR.

Fusidic acid shows good activity against MRSA in most regions. Meta-analysis data and individual studies indicate FA susceptibility >90% in many settings. However, high resistance pockets (e.g. 38% FRSA in Greece) highlight the need for local data. Clinicians should test FA susceptibility rather than assume activity. Rifampin, although potent, must be used carefully. The low reported resistance rates can be misleading if rifampin is later misused.

In vitro studies unequivocally show that FA+RIF combinations kill MRSA synergistically, consistent with their complementary mechanisms. While time-kill assays show near-universal synergy, clinical evidence is limited. The CF eradication trial suggests that prolonged FA+RIF can clear MRSA colonization. No RCTs focus on uncomplicated wound infections. However, expert guidelines (CDC, IDSA) often recommend adding RIF (with another agent) for refractory MRSA infections to exploit synergy and prevent resistance.

Resistance management is critical. Use of FA or RIF alone can select resistant mutants. Our findings align with stewardship principles: FA should be used topically for limited durations and systemic RIF only in combination. Infection control measures (contact isolation, decolonization when needed) remain cornerstone practices.

Table 2: Summary of Antibiofilm Activity of Selected Antibiotics

7. Limitations

Category	Rifampicin	Fluoroquinolones	Fosfomycin	Tetracyclines	Fusidic Acid	Daptomycin
Antibiofilm Activity	Active against Gram+ Staphylococci	Gram+ and Gram− including Enterobacter & Pseudomonas	Mainly Gram+ including MRSA and Enterococci	Gram+ organisms; doxycycline/minocycline effective for Staphylococci	Highly active against Staphylococci	Strong activity against Staphylococci (including MRSA)
Administration	Oral and IV	Oral and IV	Oral; limited use in prosthetic joint infections	Oral and IV	Oral and IV	IV only (6–9 mg/kg/day)
Clinical Notes / Caveats	Avoid monotherapy; combine with other agents	Avoid monotherapy; often combined with rifampicin	Used mainly in combination therapy	Preferred in suppressive therapy for MRSA	Use cautiously with rifampicin combinations	Often combined with fosfomycin for resistant infections

Data on superficial wound infections specifically is sparse; many references are extrapolated from broader SSTI or device-related infection studies. Fusidic acid resistance mechanisms are well-described, but rifampin mutation patterns in wound MRSA are not well-characterized. Clinical outcome data for FA/RIF in wound infections are largely anecdotal or from small series. We relied on published studies (may have publication bias); unpublished local data (especially on resistance rates) is lacking.

8. Conclusions and Clinical Recommendations

- **Empiric therapy:** In settings with known MRSA, consider vancomycin, linezolid or clindamycin (if susceptible) as first-line for severe infections. FA can be used topically (or orally where available) for non-abscess MRSA skin infections with low resistance.
- **Culture & Susceptibility:** Always obtain cultures. Tailor therapy to lab results. If MRSA is FA-susceptible and infection is superficial, FA monotherapy (topical or systemic) is reasonable.
- **Combination therapy:** For deep or persistent MRSA wound infections (osteomyelitis, prosthetic infection), add rifampin (e.g. 300–450 mg q12h) to another active drug. Ensure minimum 10–14 days, often longer. Monitor liver function with rifampin.
- **Avoid monotherapy:** Never use rifampin alone. Avoid prolonged FA use beyond recommended course. Rotate or augment agents if resistance emerges.

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- **Infection control:** Use contact precautions for MRSA wound patients. Educate on hand hygiene. Decolonization (mupirocin, chlorhexidine) may help in recurrent cases.
- **Surveillance:** Maintain local antibiograms for FA and RIF in MRSA.

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