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Review Article

Gepotidacin: Development, Mechanism of Action, Clinical Trials, and Potential in Antimicrobial Therapy

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ABSTRACT

The continued spread of antibiotic resistance has made treating simple urinary tract infections (UTIs) and urogenital gonorrhoea more difficult. New oral therapy alternatives are urgently required. Gepotidacin (formerly GSK2140944) is a new, bactericidal, oral, 'first-in-class' triazaacenaphthylene antibiotic that inhibits bacterial DNA replication by inhibiting two key topoisomerase enzymes. Mutations in both enzymes are likely required for resistance to develop, raising optimism that the medicine may be able to retain long-term efficacy. Phase III trials are now being conducted, and data from Phase II clinical trials of Gepotidacin in urogenital gonorrhoea and UTIs seem encouraging. We provide an overview of gepotidacin's history and talk about its possible applications in medicine in this review. Gepotidacin will be the first novel oral antibiotic for UTIs in almost two decades if it is approved. (1)

INTRODUCTION

The public health of the world is seriously threatened by the on-going spread of antimicrobial resistance (AMR) in Gram-negative bacteria. Urogenital gonorrhoea and urinary tract infections (UTIs) are two of the most prevalent Gram-negative infections in the globe. In 2019, the second most frequent cause of bacterial death globally was the uropathogen Escherichia coli. (2) With an estimated 50% to 60% of adult women experiencing at least one UTI in their lifetime, UTIs are among the most common illnesses seen in clinical practice. (3) The on-going rise of AMR makes treating UTIs more challenging.

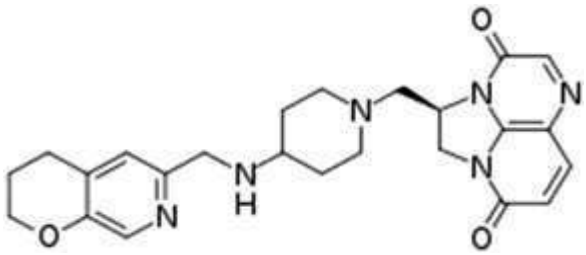

Figure 1. Structure of gepotidacin

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This is particularly true given the rise in the prevalence of MDR strains of Gram-negative uropathogen in ambulatory settings. (4) These include Klebsiella pneumoniae, Klebsiella oxytoca, Pseudomonas aeruginosa, Proteus mirabilis, and E. coli. More than 75% of all simple and catheter-associated UTIs are caused by Uropathogenic E. Coli (UPEC). (5) Parenteral antibiotics, which are less practical and typically more expensive than oral medicines, are typically used to treat MDR infections. Therefore, it is necessary to address the clinical demand for new antibiotics that are both safe and effective against MDR uropathogen. The US FDA has acknowledged this need and has been pushing for the creation of novel antibiotics to fight bacterial resistance. (6)

Serious side effects, including infertility and pelvic inflammatory disease, can result from untreated urogenital gonorrhoea. Similar to carbapenem-resistant Acinetobacter, Candida auras, C. difficile, and carbapenem-resistant Enterobacteriaceae, the CDC has designated MDR Neisseria gonorrhoeae as an urgent hazard due to its recent development. Another alternate regimen is to take cefixime orally once, however this does not produce as high or long-lasting bactericidal drug levels as ceftriaxone. Ceftriaxone-resistant isolates of N. gonorrhoeae have also been reported. (7) (8)

The development of the new antibiotic gepotidacin has been fueled by the need for more oral medications to treat gonorrhea and MDR UTIs (figure.1). The primary features of gepotidacin are covered in this study, along with its mode of action, antibacterial spectrum of activity, pharmacodynamic (PD) and pharmacokinetic (PK) characteristics, clinical trial outcomes, and potential applications in clinical practice. To find pertinent English-language publications on gepotidacin published between 2016 and 2023, we used PubMed.

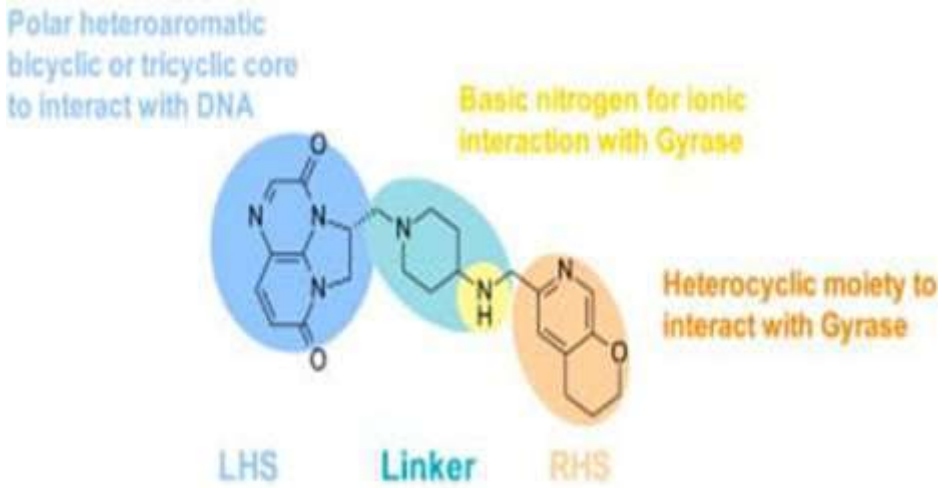


Figure 2. Chemical Structure of Gepotidacin

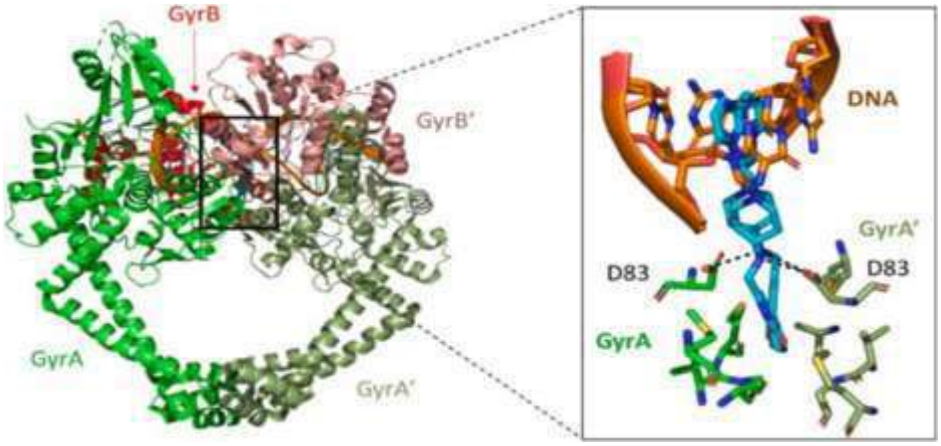
Develoment of Gepotidacin

Research into various drugs that target bacterial topoisomerases has been prompted by the evolution of resistance to quinolones. Efflux pumps, alterations in cell wall permeability, and point mutations in the genes encoding DNA gyrase and DNA topoisomerase IV are the primary causes of quinolone resistance. (9) described the first member of a novel class of type IIA topoisomerase inhibitors, which have a new method of action that gets beyond quinolone resistance and broad-spectrum antibacterial efficacy. Since then, this agent—designated GSK2140944—has advanced

through numerous PK/PD and in vitro activity tests as well as Phase I, II, and III clinical trials. Mesylate salt for an IV solution, tablets, capsules, and the free base for tablets and capsules were among the gepotidacin formulations employed in the PK assessments. (10)

The formulation of mesylate salt oral tablets was chosen for possible commercialization as clinical development advanced. Since most current medications are parenteral, oral versions may be helpful in treating gonorrhea and MDR UTIs. For both patients and healthcare organizations, oral antibiotics offer a number of benefits over numerous parenteral formulations, such as lower total costs, shorter hospital stays, fewer problems, and greater convenience. (11) Furthermore, one of the main objectives of antibiotic stewardship is to reduce the development of AMR by switching hospitalized patients from parenteral antibiotics to an efficient oral medication.

Mechanism of action



Temporary single-strand (type I) or double-strand (type II) DNA breaks are caused by topoisomerases, which are significant and common enzymes that control DNA topology (12). There are two subtypes of type II DNA topoisomerases: IIA and IIB. DNA gyrase and topoisomerase IV are the two type IIA topoisomerases found in the majority of bacteria. Topoisomerase IV and the B component of DNA gyrase are specifically inhibited by gepotidacin, a new (or non-fluoroquinolone) bacterial topoisomerase inhibitor (13) High amounts of gyrase-mediated single-stranded breaks are induced by gepotidacin. (14) This mechanism produces a low rate of spontaneous single-step resistance and is bactericidal. Gepotidacin has a unique binding mechanism that sets it apart from fluoroquinolones, according to structural evidence using type II topoisomerase and DNA gyrase (15).

Additionally, gepotidacin exhibits in vitro efficacy against target bacteria that possess fluoroquinolone resistance determinants. Gepotidacin MICs of 256 mg/L and >256 mg/L, respectively, were caused by (16)selected gepotidacin single-step mutations in K. pneumoniae and E. coli. They came to the conclusion that ciprofloxacin exposure over time co-selected for decreased susceptibility to gepotidacin. Additionally, continued use of antibiotics may favor mutations that act as precursors to resistance to newly developed antimicrobials.

During Phase I and II clinical studies, the PK properties of gepotidacin were clearly characterized in healthy adult subjects. (17) It was demonstrated that the PK/PD index linked to the effectiveness of gepotidacin was comparable to

that of fluoroquinolone drugs (18). For E. Coli isolates in Mueller-Hinton broth, the minimum inhibitory concentration (MIC) of gepotidacin varied between 1 and 4 mg/L. The median gepotidacin free-drug AUC/MIC ratios linked to net bacterial stasis and 1- and 2-log10 cfu decreases were 33.9, 43.7, and 60.7, according to VanScoy et al. (19). Additionally, 275 and higher free-drug AUC/MIC ratios were adequate to inhibit microbial resistance.

(20) With a single dose, gepotidacin plasma concentrations peak at 3.00 hours. However, after two doses of gepotidacin were administered at intervals of 12 or 6 hours, two maxima in plasma concentration were seen at 1.5 and 2.25 hours. The mean terminal elimination time (t½) was 11.8 hours, the mean Cmax was 4.37 µg h/mL, and the mean AUC0–∞ value was 15.8 for a single dose of 1500 mg mesylate salt capsule of gepotidacin while fasting. Both a free-base roller-compacted tablet (AUC0–∞17.5 µg h/mL; Cmax 4.49; t1/2 10.2 h) and a free-base high-shear wet granulation tablet (AUC0–∞ 18.6 µg h/mL; Cmax 5.35; t½ 10.2 h) showed comparable results during fasting (21).

A free-base 750 mg tablet formulation showed sufficient plasma levels and a manageable risk-safety profile in a Phase IIa PK experiment involving female participants with an uncomplicated UTI. (22) After 1500 mg of gepotidacin was administered and sustained for 24 hours, one study showed urine concentrations of more than 4 mg/L. (23) Another discovered that roughly half of the oral dosage is absorbed and mostly excreted as unaltered medication in the urine (approximately 20% of the dose). (24)

Previous clinical studies supported the choice of dosage and time interval for Phase III clinical trials of gepotidacin, which called for two doses of 3000 mg spaced 10–12 hours apart to treat urogenital gonorrhea and 1500 mg twice day for 5 days to treat uncomplicated UTI. (25) (26) While lowering the chance of N. gonorrhoeae developing a resistance to gepotidacin, the longer time interval is thought to help with gastrointestinal tolerance and limiting Cmax-related side effects such QT prolongation. (27) With MICs of 1 mg/L, the systemic exposure was also doubled when the two doses were spaced 10–12 hours apart as opposed to a single 3000 mg dosage. (28)

The frequency and severity of gepotidacin's gastrointestinal adverse effects, which include nausea, emesis, diarrhea, abdominal discomfort, and flatulence, are known to increase with dosage. Both adult and teenage participants in one trial experienced emesis shortly after receiving gepotidacin; nevertheless, the Cmax and AUC values for those who experienced emesis and those who did not were comparable, indicating that emesis had minimal effect on plasma concentration. (29) Although acetylcholinesterase inhibition-related adverse effects, such as cardiovascular side effects, have been documented, they were uncommon in this trial. (30)

Antimicrobial Susceptibility and the Range of Microbiological Activity

In vitro, gepotidacin exhibits strong efficacy against several common bacterial infections. Biedenbach et al. (31) tested it against Streptococcus pneumoniae (n = 549), Haemophilus influenzae (n = 981), Moraxella catarrhalis (n = 158), Streptococcus pyogenes (n = 199), Staphylococcus aureus (n = 1008), E. coli (n = 1010), Shigella spp. (n = 21), and Clostridium perfringens (n = 101) using isolates from a global collection that was established between 2010 and 2012 (Table 1). The MIC90 for E. Coli was 2 mg/L (range: ≤0.03 to >32), while the MIC90 for isolates that were not susceptible to levofloxacin rose to 4

mg/L (range: 0.06 to >32). MIC90 values against nitrofurantoin-non-susceptible increased from 2 to 4 mg/L. isolates that are ESBL screen-positive and not sensitive to fosfomycin. Levofloxacin resistance was found in almost 75% of the E. ssColi isolates that tested positive on the ESBL screen; the MIC90 against this subset was 4 mg/L. These results are consistent with a publication on the effectiveness of gepotidacin against E. coli urine isolates from outpatient clinics in Germany. (32) Another study examined the effectiveness of gepotidacin against 145 isolates of N. gonorrhoeae and the impact of in vitro test conditions on the susceptibility of gepotidacin and two additional antibacterials (ceftriaxone and ciprofloxacin) to a different set of nine isolates of N. gonorrhoeae and a quality control strain. (33) A number of variables that might affect gepotidacin MIC calculations were discovered. When agar plates were incubated in 10% CO2, ceftriaxone MICs likewise tended to be marginally lower than the reference MICs, taking into account factors such media type, inoculum concentration, and media pH. Using agar dilution, gepotidacin has also been tested against Gram-positive (n = 225) and Gram-negative (n = 333) anaerobes. At doses of 4 and 2 mg/L, respectively, it inhibited 90% of isolates. Rebound to pre-dosing levels is visible during the first month after treatment, suggesting that a 5-day course of gepotidacin has no lasting impact on the human microbiota. (35)

MIC data for gepotidacin[a]

Organism	MIC_{50} (mg/L)	MIC_{90} (mg/L)	Range
MRSA	0.25	0.5	≤0.06 to 1
MSSA	0.5	0.5	0.12 to 2
S. pneumoniae	0.12	0.25	0.03 to 1
S. pyogenes	0.25	0.25	0.03 to 0.5
H. influenzae	0.5	1	≤0.015 to 8
M. catarrhalis	≤0.06	≤0.06	≤0.06 to 0.12
E. coli	2	2	≤0.03 to >32
Shigella spp.	0.5	1	not provided
C. perfringens	0.12	0.5	not provided
N. gonorrhoeae	0.25	0.5	≤0.015 to 1

Clinical trial

Doses of 1000 and 1800 mg of IV gepotidacin resulted in a moderate increase (7–10 beats/min) in resting heart rate and a minor lengthening of the QT in a Phase I investigation including healthy volunteers (36) In order to attain supratherapeutic plasma levels, the IV formulation was selected. 1500 mg of oral gepotidacin was assessed in three distinct hepatic settings (normal, mild impairment, and severe impairment) in a second Phase I non-randomized, open-label, multicenter clinical trial (37) In all participants, gepotidacin was found to be safe and generally well tolerated. Considering these Therefore, patients with mild to moderate hepatic impairment probably won't need to change their dosage. On the other hand, significant hepatic impairment might necessitate a dose reduction or an increase in the interval between doses. Gepotidacin's effectiveness and safety in treating adult patients with suspected or confirmed Gram-positive acute bacterial skin and skin structure infections (ABSSSIs) were assessed in a Phase II clinical research (38) The average duration of exposure to oral and intravenous gepotidacin was 7.5 and 3.4 days, respectively. The modified ITT and safety populations, which used three doses of gepotidacin, comprised 122 patients. The majority

had either cellulitis (24%), a cutaneous abscess (32%), or a wound infection (44%). The major goal of the trial, which was a composite of safety (withdrawal rate) and efficacy (cure rate), was achieved in accordance with the protocol.

Gastrointestinal side effects were the most frequent, with nausea accounting for 20% and diarrhea for 13%. An adverse event occurred in four patients (3%) and resulted in either one patient withdrawing from the trial or three patients permanently stopping their therapy. Microbiological efficacy was also assessed as a secondary endpoint in this trial; S. aureus accounted for 76% of isolates (69% MRSA, 31% MSSA), with the remaining isolates consisting of other Gram-positive aerobes (11%), Gram-negative aerobes (12%), and anaerobes (1%). For S. aureus, post-therapy microbiological success (as measured by the pretreatment pathogen's eradication confirmed by culture or assumed based on clinical success) was 90% in the 750 mg q12h dose and 89% in the 1000 mg q12h dosing. and 73% in the groups that received 1000 mg every 8 hours. (39) Other Gram-positive pathogens showed a similar pattern.
69 adult patients with urogenital gonorrhea were recruited for a second Phase II clinical trial, and they were randomized 1:1 to receive either a 1500 mg or 3000 mg oral dose of gepotidacin. (40) For the 1500 and 3000 mg dosage groups, 97% and 95% of subjects, respectively, achieved microbiological eradication of N. gonorrhoeae. Each of the three individuals with microbiological failures had isolates of Gonorrhoeae with a MIC of 1 µg/mL for gepotidacin. The incidence of adverse events was 34 out of 53 (64%) in the 3000 mg treatment group and 27 out of 52 (52%) in the 1500 mg group. The most commonly reported adverse effects were nausea (13%), abdominal pain (15%), flatulence (23%), and diarrhea (27%). Neither dose resulted in treatment-limiting adverse effects. In a third Phase II clinical trial, 22 women with a UTI were recruited and administered 1500 mg of oral gepotidacin twice a day for five days. They then returned to the clinic for follow-up visits on days 28±3 and test-of-cure (TOC) visits on days 10 to 13. (41) Clinical success was noted for 19 out of 22 individuals (86%), at TOC. Almost all had gastrointestinal side effects, primarily vomiting (n = 5, 23%), nausea (n = 17, 77%), and diarrhea (n = 18, 82%). No changes from baseline or clinically significant ECG abnormalities were found. Furthermore, according to Fridericia, none of the subjects exhibited a QT interval corrected for heart rate of ≥480 ms or an increase of >30 ms. (42)

Possible use of gepotidacin

UTI

In clinical practice Gepotidacin may be able to address an unmet demand for an additional oral drug against E. coli and a treatment option for MDR strains, given the prevalence of UTIs and the rise of AMR. Despite the high clinical success rate of 86% in a Phase II clinical trial for uncomplicated UTI, nearly all patients experienced gastrointestinal side effects. (43) In a

recent Phase I experiment, it was observed that food intake considerably increased the intestinal tolerance of gepotidacin. (44)

Urogenital Gonorrhoea

In recent years, the treatment options for urogenital gonorrhea have been severely limited due to the global spread of MDR N. gonorrhoeae. For urogenital gonorrhea, a Phase II clinical trial using oral gepotidacin as a single dosage showed >95% microbiological eradication. (44) The majority of adverse events, which affected more than 50% of participants, were gastrointestinal in nature. In order to increase efficacy and reduce the establishment of resistance, the Phase III study prescribed two oral doses of 3000 mg spaced 10–12 hours apart due to the higher gepotidacin MIC in the three patients who failed microbiological eradication in this trial. (45). More details about the effectiveness and tolerability of oral gepotidacin in comparison to oral azithromycin and IM ceftriaxone will be available from this ongoing Phase III EAGLE-1 clinical trial.

Importantly, there is currently no proof that gepotidacin works for gonorrhea at other locations, such as the pharynx or the rectal cavity. (46)

Numerous other clinical uses for gepotidacin are likely due to its extensive in vitro activity against anaerobic, Gram-positive, and Gram-negative bacteria. Treatment of ABSSSIs looks to be clinically and microbiologically effective, as previously observed in Phase II trials; nevertheless, Phase III studies are required to compare with existing conventional regimens. (47) In a primate animal model, gemotidacin has Other demonstrated effectiveness against Yersinia pestis. In vitro research indicates potential against drug-resistant Mycoplasma genitalium and other Mycoplasma and Ureaplasma infections. Gepotidacin has demonstrated efficacy against mycobacterial pathogens, such as drug-resistant non-tuberculous mycobacteria and Mycobacterium tuberculosis, in both in vitro and in vivo animal models. Although more research, including clinical trials, is required for this use, gepotidacin's antibacterial spectrum may also make it useful in treating pneumonia, particularly against MDR nosocomial infections. Stenotrophomonas maltophilia is a nosocomial infection that is becoming more and more well-known and frequently resistant to several antibiotics. A recent study showed good effectiveness against this disease. (48).

CONCLUSION

New antibiotics, especially those with an oral formulation, are desperately needed to treat MDR infections. Gepotidacin's safety and effectiveness in early trials seem promising, however the high frequency of gastrointestinal side effects is alarming. Hopefully, gepotidacin's development will continue to advance since it would address a significant unmet clinical need. Gepotidacin would be the first novel antibiotic for UTIs in almost two decades if it were authorized.

REFERENCES

1. Richard R Watkins, Dipendra Thapaliya , Tracy L Lemonovich , Robert A Bonomo.
2. Global mortality associated with 33 bacterial pathogens in 2019: a systematic analysis for the Global Burden of Disease Study 2019.
3. Castillo-Pino, Martha Medina and Edgardo.
4. Richard R. Watkins MD, MS a b.
5. MD, Thomas A. Waller.
6. Aneela Majeed, Sumaiah Alarfaj,Rabih Darouiche &Mayar Mohajer.
7. Michaela Day1, Rachel Pitt1 ORCID icon, Nisha Mody1 ORCID icon, John Saunders1 , Rupa Rai1 , Achyuta Nori1 ORCID icon,

Hannah Church1 , Sarah Mensforth1 , Helen Corkin1 , Jacqueline Jones2 , Preneshni Naicker3 , Wazirzada M Khan1 , Rebecca Thomson Glover.

8. Xiaomian Lin https://orcid.org/0000-0002-7780-0458 18819360127@163.com, Wentao Chen https://orcid.org/0000-0002-4062-3865, Yuqi Yu, Yinyuan Lan, Qinghui Xie, Yiwen Liao, Xingzhong Wu, Sanmei Tang, Xiaolin Qin, Heping Zheng zhengheping@smu.edu.cn.
9. by Marcin Rozwadowski 1, 2,*ORCID andDamian Gawel 2,*ORCID.
10. Aline Barth https://orcid.org/0000-0002-4788-6051 alinebbarth@hotmail.com, Mohammad Hossain, Darin B. Brimhall, Caroline R. Perry Caroline.R.Perry@gsk.com, Courtney A. Tiffany, Sherry Xu, Etienne F. Dumont.
11. Watkins, Richard R.
12. 1, Benjamin D. Bax.
13. uilia Sellarès-Nadal, Joaquin Burgos, Vicenç Falcó, Benito Almirante.
14. Gibson, Elizabeth G.
15. Benjamin D. Bax, Pan F. Chan, Drake S. Eggleston, Andrew Fosberry, Daniel R. Gentry, Fabrice Gorrec, Ilaria Giordano, Michael M. Hann, Alan Hennessy, Martin Hibbs, Jianzhong Huang, Emma Jones, Jo Jones, Kristin Koretke Brown, Ceri J. Lewis, Earl W. May, M.
16. Petra Szili, Gábor Draskovits, Tamás Révész, Ferenc Bogár, Dávid Balogh, Tamás Martinek, Lejla Daruka,.
17. Aline Barth https://orcid.org/0000-0002-4788-6051 alinebbarth@hotmail.com, Mohammad Hossain, Darin B. Brimhall, Caroline R. Perry Caroline.R.Perry@gsk.com, Courtney A. Tiffany, Sherry Xu, Etienne F. Dumont.
18. Paul G. Ambrose, Sujata M. Bhavnani , Christopher M. Rubino , Arnold Louie , Tawanda Gumbo , Alan Forrest , George L. Drusano.
19. Brian D. VanScoy https://orcid.org/0000-0001-6527-1475, Elizabeth A. Lakota, Haley Conde, Steven Fikes, Sujata M. Bhavnani https://orcid.org/0000-0003-4891-6467, Philippa B. Elefante, Nicole E. Scangarella-Oman https://orcid.org/0000-0003-4211-8261, Paul.
20. Brian D. VanScoy https://orcid.org/0000-0001-6527-1475, Elizabeth A. Lakota, Haley Conde, Steven Fikes, Sujata M. Bhavnani https://orcid.org/0000-0003-4891-6467, Philippa B. Elefante, Nicole E. Scangarella-Oman https://orcid.org/0000-0003-4211-8261, Paul.
21. Aline Barth https://orcid.org/0000-0002-4788-6051 alinebbarth@hotmail.com, Mohammad Hossain, Darin B. Brimhall, Caroline R. Perry Caroline.R.Perry@gsk.com, Courtney A. Tiffany, Sherry Xu, Etienne F. Dumont.
22. Stephanie N Taylor, David H Morris , Ann K Avery , Kimberly A Workowski , Byron E Batteiger , Courtney A Tiffany , Caroline R Perry , Aparna Raychaudhuri , Nicole E Scangarella-Oman , Mohammad Hossain ..
23. J. Scott Overcash, Courtney A. Tiffany, Nicole E. Scangarella-Oman, Caroline R. Perry, Yu Tao, Mohammad Hossain, Aline Barth, Etienne F. Dumont.
24. Kitaw Negash, Clara Andonian,Clive Felgate,Cathy Chen,Igor Goljer.
25. Aline Barth https://orcid.org/0000-0002-4788-6051 alinebbarth@hotmail.com, Mohammad Hossain, Darin B. Brimhall, Caroline R. Perry Caroline.R.Perry@gsk.com, Courtney A. Tiffany, Sherry Xu, Etienne F. Dumont.
26. Nicole E. Scangarella-Oman, Mohammad Hossain, Paula B. Dixon, Karen Ingraham, Sharon Min, Courtney A. Tiffany, Caroline R.

Perry, Aparna Raychaudhuri, Etienne F. Dumont, Jianzhong Huang, Edward W. Hook III, Linda A. Miller.
27. Nicole E. Scangarella-Oman, Mohammad Hossain, Paula B. Dixon, Karen Ingraham, Sharon Min, Courtney A. Tiffany, Caroline R. Perry, Aparna Raychaudhuri, Etienne F. Dumont, Jianzhong Huang, Edward W. Hook III, Linda A. Miller.
28. Nicole E. Scangarella-Oman, Mohammad Hossain, Paula B. Dixon, Karen Ingraham, Sharon Min, Courtney A. Tiffany, Caroline R. Perry, Aparna Raychaudhuri, Etienne F. Dumont, Jianzhong Huang, Edward W. Hook III, Linda A. Miller.
29. Aline Barth https://orcid.org/0000-0002-4788-6051 alinebbarth@hotmail.com, Mohammad Hossain, Darin B. Brimhall, Caroline R. Perry Caroline.R.Perry@gsk.com, Courtney A. Tiffany, Sherry Xu, Etienne F. Dumont.
30. Aline Barth https://orcid.org/0000-0002-4788-6051 alinebbarth@hotmail.com, Mohammad Hossain, Darin B. Brimhall, Caroline R. Perry Caroline.R.Perry@gsk.com, Courtney A. Tiffany, Sherry Xu, Etienne F. Dumont.
31. D. J. Biedenbach, S. K. Bouchillon, M. Hackel, L. A. Miller, N. E. Scangarella-Oman, C. Jakielaszek, D. F. Sahm.
32. Michael Kresken, Esther Wohlfarth , Chase Weikel , Deborah Butler , Yvonne Pfeifer , Guido Werner ,.
33. a, Nicole E. Scangarella-Oman.
34. Andrea Nuzzo, Stephanie Van Horn, Christopher Traini, Caroline R. Perry, Etienne F. Dumont, Nicole E. Scangarella-Oman, David F. Gardiner & James R. Brown.
35. Mohammad Hossain, Meijian Zhou, Courtney Tiffany, Etienne Dumont, Borje Darpo.
36. Mohammad Hossain, Courtney Tiffany, Yu Tao, Aline Barth, Thomas C. Marbury, Richard A. Preston, Etienne Dumont.
37. William O'Riordan, Courtney Tiffany, Nicole Scangarella-Oman, Caroline Perry, Mohammad Hossain, Teri Ashton, Etienne Dumont.
38. Nicole E. Scangarella-Oman, Karen A. Ingraham, Courtney A. Tiffany, Lynn Tomsho, Stephanie F. Van Horn, David N. Mayhew, Caroline R. Perry, Theresa C. Ashton, Etienne F. Dumont, Jianzhong Huang, James R. Brown, Linda A. Miller.
39. Brian D. VanScoy https://orcid.org/0000-0001-6527-1475, Elizabeth A. Lakota, Haley Conde, Steven Fikes, Sujata M. Bhavnani https://orcid.org/0000-0003-4891-6467, Philippa B. Elefante, Nicole E. Scangarella-Oman https://orcid.org/0000-0003-4211-8261, Paul.
40. Stephanie N Taylor, David H Morris , Ann K Avery , Kimberly A Workowski , Byron E Batteiger , Courtney A Tiffany , Caroline R Perry ,.
41. Caroline Perry, Mohammad Hossain, Marcy Powell, Aparna Raychaudhuri, Nicole Scangarella-Oman, Courtney Tiffany, Sherry Xu, Etienne Dumont & Salim Janmohamed.
42. Aline Barth, Mohammad Hossain, Caroline R. Perry, Annette S. Gross, Hirofumi Ogura, Shaila Shabbir, Sebin Thomas, Etienne F. Dumont, Darin B. Brimhall, Meenakshi Srinivasan, Brandon Swift.
43. William O'Riordan, Courtney Tiffany, Nicole Scangarella-Oman, Caroline Perry, Mohammad Hossain, Teri Ashton, Etienne Dumont.
44. Meredith A. Hackel https://orcid.org/0000-0003-3339-2701 mhackel@ihma.com, James A. Karlowsky, Michele A. Canino, Daniel F. Sahm, Nicole E. Scangarella-Oman https://orcid.org/0000-0003-4211-8261.

45. Nicole E Scangarella-Oman1, Mohammad Hossain1, Caroline R Perry1, Courtney Tiffany1, Marcy Powell2, Brandon Swift2, Etienne F Dumont1.
46. Mohammad Naiyaz Ahmad https://orcid.org/0000-0002-9300-6107, Tanu Garg https://orcid.org/0000-0002-1997-6279, Shriya Singh https://orcid.org/0000-0003-0397-5498, Richa Shukla, Pradip Malik, Ramya V. Krishnamurthy, Parvinder Kaur https://orcid.org/0000-000.
47. William O'Riordan, Courtney Tiffany, Nicole Scangarella-Oman, Caroline Perry, Mohammad Hossain, Teri Ashton, Etienne Dumont.
48. : Ken B. Waites, Donna M. Crabb, Li Xiao, Lynn B. Duffy.
49. a, Nicole E. Scangarella-Oman.
50. Hoover JL, Singley CM, Elefante Pet al. Efficacy of human exposures of gepotidacin (GSK2140944) against Escherichia coli in a rat pyelonephritis model. Antimicrob Agents Chemother 2019 and https://doi.org/10.1128/AAC.00086-19, 63: e00086-19.
51. Hackel MA, Karlowsky JA, Canino MAet al. In vitro activity of gepotidacin against Gram-negative and Gram-positive anaerobes. Antimicrob Agents Chemother 2022 and https://doi.org/10.1128/aac.02165-21, 66: e0216521.

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