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EXPECTED (INTRINSIC) ANTIMICROBIAL RESISTANCE OF CLINICALLY IMPORTANT HUMAN PATHOGENIC BACTERIA

A QUICK REFERENCE FOR CLINICAL MICROBIOLOGY LABORATORIES

No.	ORGANISM (Alphabetical)	GRAM STAIN	INTRINSIC (EXPECTED) RESISTANCE TO (Representative Antimicrobials / Classes)	PRINCIPAL MECHANISM(S) OF INTRINSIC RESISTANCE	CLINICAL / LABORATORY IMPLICATION
1	*Acinetobacter baumannii complex*	GNB	Aminopenicillins, Amoxicillin–clavulanate, Aztreonam, Ertapenem, Glycopeptides, Macrolides, Lincosamides, Oxazolidinones, Rifampicin, Trimethoprim	Low outer membrane permeability, efflux pumps, absence / modification of target sites	Do not report susceptible to above agents. Verify results if susceptible reported.
2	*Citrobacter freundii complex*	GNB	Aminopenicillins (Ampicillin, Amoxicillin), 1st generation cephalosporins, Macrolides	Constitutive AmpC β-lactamase, efflux pumps	Third generation cephalosporins may be active but monitor for AmpC overproduction.
3	*Enterobacter cloacae complex*	GNB	Aminopenicillins, 1st generation cephalosporins, Ampicillin–sulbactam, Cephamycins (e.g., Cefoxitin)	Chromosomal AmpC β-lactamase	Avoid cephamycins; risk of derepressed AmpC.
4	*Enterococcus faecalis*	GPC	Cephalosporins (all generations), Clindamycin, Trimethoprim–sulfamethoxazole, Low-level resistance to Aminoglycosides (streptomycin)	Low affinity PBPs, inherent target insensitivity, active efflux	Aminoglycosides only with cell wall active drug for synergistic effect.
5	*Enterococcus faecium*	GPC	Cephalosporins (all), Clindamycin, Trimethoprim–sulfamethoxazole, Low-level resistance to Aminoglycosides	Low affinity PBPs (PBP5), efflux, target modification	Frequently shows acquired resistance in addition to intrinsic resistance.
6	*Escherichia coli*	GNB	Macrolides, Lincosamides, Glycopeptides, Streptogramins, Rifampicin, Fosfomycin (systemic use)	Outer membrane impermeability, target absence / modification, efflux	Intrinsic resistance does not predict outcome with acquired resistance.
7	*Haemophilus influenzae*	GNB	Glycopeptides (Vancomycin, Teicoplanin), Clindamycin, Daptomycin, Rifampicin	Lack of cell wall targets or target modification	β-lactams and macrolides usually active (unless β-lactamase positive).
8	*Klebsiella pneumoniae*	GNB	Aminopenicillins (Ampicillin, Amoxicillin)	Chromosomal SHV-1 β-lactamase	Do not report susceptible to ampicillin.
9	*Morganella morganii*	GNB	Colistin, Polymyxin B, Tigecycline, Nitrofurantoin	Altered LPS / outer membrane	Report as resistant to polymyxins and tigecycline.
10	*Proteus mirabilis*	GNB	Colistin, Polymyxin B, Nitrofurantoin, Tigecycline	Altered LPS, intrinsic efflux	Avoid polymyxins, tigecycline and nitrofurantoin.
11	*Providencia spp.*	GNB	Colistin, Polymyxin B, Tigecycline, Nitrofurantoin	Altered LPS / decreased outer membrane permeability	Do not report susceptible to polymyxins and tigecycline.
12	*Pseudomonas aeruginosa*	GNB	Aminopenicillins, Cephalosporins (e.g., Cefazolin, Cefoxitin), Ertapenem, Tigecycline, Trimethoprim–sulfamethoxazole, Aztreonam (often), Nitrofurantoin, Fosfomycin (systemic use)	Low outer membrane permeability, efflux pumps, AmpC, target modifications	Carbapenems (except ertapenem) and anti-pseudomonal agents active.
12	*Serratia marcescens*	GNB	Colistin, Polymyxin B, Nitrofurantoin	Altered LPS (poor binding of polymyxins)	Report as resistant to polymyxins and nitrofurantoin.
14	*Staphylococcus spp. (S. aureus & CoNS)*	GPC	Aztreonam, Colistin, Polymyxins, Fosfomycin (systemic use), Cephalosporins (for CoNS variable)	Outer membrane barrier (absent), lack of target	β-lactams (penicillins, cephs) may be active (unless β-lactamase/MRSA).
15	*Stenotrophomonas maltophilia*	GNB	Carbapenems (all), Aminoglycosides, Colistin, Polymyxins, Trimethoprim–sulfamethoxazole (variable), Cephalosporins (most)	Multiple efflux pumps, low permeability, β-lactamases	TMP–SMX is drug of choice; many agents intrinsically ineffective.
16	*Streptococcus spp. (β-haemolytic & Viridans group)*	GPC	Aminoglycosides (as single agent), Aztreonam, Colistin, Polymyxins, Rifampicin	Target modifications, absence of uptake systems	Penicillin and related β-lactams remain drugs of choice.

IMPORTANT: NEVER REPORT AS SUSCEPTIBLE

- Ampicillin / Amoxicillin → *Klebsiella pneumoniae*
- Aminopenicillins → *Acinetobacter spp., Pseudomonas aeruginosa*
- Ertapenem → *Pseudomonas aeruginosa, Acinetobacter spp.*
- Aztreonam → *Acinetobacter baumanii, Stenotrophononas maltophilia, many Streptococcus spp.*
- Colistin / Polymyxins → *Proteus mirabilis, Morganella morganii, Providencia spp., Serratia marcescens*
- Nitrofurantoin → *Proteus mirabilis, Morganella morganii, Providencia spp., Serratia marcescens*
- Tigecycline → *Proteus mirabilis, Morganella morganii, Providencia spp.*

INTRINSIC vs ACQUIRED RESISTANCE

- Intrinsic (expected) resistance is natural and predictable due to inherent structural or functional traits.
- Acquired resistance develops due to mutation or gene acquisition.
- Do not assume all resistance is acquired.
- Always interpret AST with clinical context and current breakpoints.

When resistance is unexpected, repeat test and confirm identification.

PRACTICAL LABORATORY NOTES (KEY POINTS)

- These resistances are predictable and should guide reporting and therapy.
- Do not report combinations listed here as susceptible, even if in vitro results appear susceptible.
- If an unexpected susceptible result is obtained for an intrinsic resistance, verify the test (method, inoculum, medium, organism identification).
- Consider organism identification carefully.
- Apply current CLSI and EUCAST breakpoints.
- Be alert for acquired resistance in addition to intrinsic resistance.
- Communicate critical results promptly.
- Use infection site, host factors and PK/PD to guide therapy.

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6. Humphries RM, Simner PJ, et al. Recent CLSI breakpoint updates and implementation guidance. J Clin Microbiol. 2025;63(1):e0225-24.