Chromosomal Locations of *mcr-1* and $bla_{CTX-M-15}$ in Fluoroquinolone-Resistant *Escherichia coli* ST410

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To the Editor: Recently, Yi-Yun Liu et al. reported on the discovery of *mcr-1*, a plasmidborne resistance gene mediating resistance to colistin, in isolates obtained from humans and animals (*1*). Since the original publication, *mcr-1* with or without the insertion element IS*Apl1* has been detected on plasmids of different incompatibility groups, including IncI2, IncHI2, and IncX4, and in many different countries (*1–3*). Because colistin is a last-resort parenteral antimicrobial drug, the transfer of *mcr-1* by

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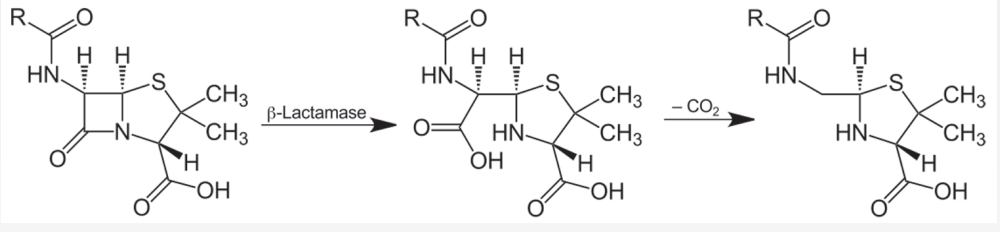
etymologia

β-Lactamase [ba'tə lak'tə-mās]

Enzymes that catalyze the cleavage of β-lactam rings in penicillins, cephalosporins, monobactams, and carbapenems were first described by Abraham and Chain in 1940. These enzymes confer resistance to β-lactam antibiotics on bacteria that produce them. β-lactamases are ancient, theorized to have evolved 1–2 billion years ago, but the emergence and spread of penicillin-resistant staphylococci in hospitals in the 1950s showed how penicillin use could select producers from a population of nonproducers. "Lactam" is a portmanteau of "**lac**tone" (from the Latin *lactis*, "milk," since lactic acid was isolated from soured milk) and "**am**ide." The "β" refers to the nitrogen's position on the second carbon in the ring. The suffix "-ase," indicating an enzyme, is derived from "diastase" (from the Greek *diastasis*, "separation"), the first enzyme discovered in 1833 by Payen and Persoz.



Action of β-lactamase and decarboxylation of the β-lactam ring. Equation by Jü, own work, public domain, https://commons.wikimedia.org/w/index.php?curid=11204303

Sources

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