PHENOXYPEN ®

TECHNICAL BULLETIN 2026

The molecule penicillin V

Penicillin V is a closely related analogue of penicillin G (benzylpenicillin) and their antibacterial spectra are very similar. The reactive 4-ring explains why most penicillins are not effective after oral admistration.

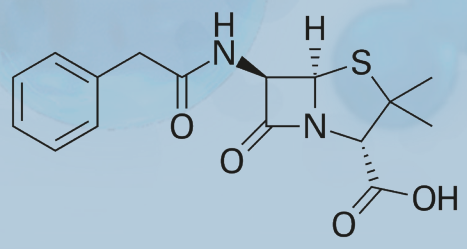
The ß-lactam ring reacts with the acid contents of the stomach losing their activity. However, due to chemical manipulation of a part of the structure, pencillin V is acid stable and appropriate for oral administration.[1,2]

Penicillin V is also known as phenoxymethylpenicillin or penicillin VK.

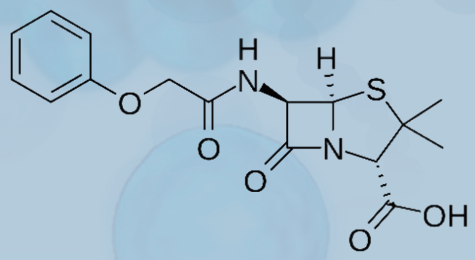
Formula: $C_{16}H_{18}N_2O_5S$

Molar mass 350.39 g mol^{-1}

First prepared/formula established in 1948 by Behrens, Corse and Edwards



Penicillin G



Penicillin V

The method of preparation

By definition, the natural penicillins are those produced biosynthetically. A high yielding ultraviolet mutant strain of *Penicillium chrysogenum* is added to fermenters. After days of carefully controlled agitation and fermentation, a final concentration of penicillin in the crude culture medium is formed. The principal yield of this process is benzyl penicillin G. The addition of precursors to the fermentation medium resulted in penicillins whose antibacterial and intrinsic properties differ from those of penicillin G. Addition of side chain precursor phenoxyacetic acid results in the fungus producing phenoxymethyl penicillin (penicillin V).[3]

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Mechanism of action

Penicillins target the bacteria during its active multiplication stage, by interfering with bacterial cell wall peptidoglycan synthesis. It inhibits the biosynthesis of cell wall mucopeptide by binding to specific penicillin-binding proteins (PBPs) located inside the bacterial cell wall, which are critical in the cell wall synthesis and maintenance, as well as cell division. This disrupts the third and last stage of bacterial cell wall synthesis. This subsequently leads to abnormal cell growth and cell lysis, resulting in a bactericidal effect. The earlier mentioned ß-lactam ring plays an important role in the whole process, because the mechanism of action depends on the reactivity of the ß-lactam ring. [2]

Penicillin V has bactericidal and time-dependent effects. Optimal killing occurs if bacteria are exposed to an antimicrobial concentration exceeding 1-4 times the MIC for sufficient time between the dosing intervals. Thus, for time-dependent drugs, a time above the MIC (T>MIC) is the best pharmacokinetic/pharmacodynamic (PK/PD) parameter predicting microbiological and clinical efficacy.[4]

Minimum Inhibitory Concentrations (MICs) were determined against *Streptococcus suis* isolates from diseased pigs in Europe (2019-2024). Most of the isolates showed a MIC of ≤ 0.0625 µg/ml. [5]

Pharmacokinetics in swine

Upon oral administration, phenoxymethylpenicillin is rapidly but incompletely absorbed.6 The absolute bioavailability of phenoxymethylpenicillin showed to be approximately 21,1%. Phenoxymethylpenicillin is well distributed over most of the tissues, leading to a high concentration in the kidneys and the liver. Phenoxymethylpenicillin is partially decomposed in the gastrointestinal tract. A small portion of the absorbed amount is metabolised in the body. For the most part, phenoxymethylpenicillin is excreted in unaltered active form in urine and faeces.[5]

References

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