Flavo-8 and Flavo-80

The only Flavophospholipol registered
for use at levels up to 20 ppm and as an aid in

reducing

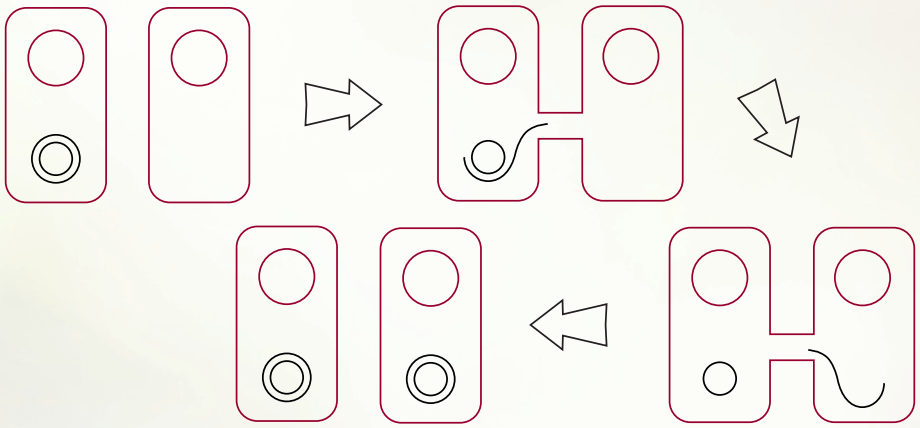
Antibiotic resistance

How resistance is acquired

Antibiotic resistance can be acquired via transfer of extrachromosal DNA located on plasmids. Plasmids pass from one bacterium to another through bacterial conjugation.

Conjugation is common means by which antibiotic resistance spreads between bacterial species, genera and even families.

Plasmid-mediated conjugation occurs in gram-negative bacteria and in some gram-positive bacteria such as Streptococcus spp, Staphylococcus spp and Enterococcus.spp.



During conjugation a sex pilus is constructed from the donor bacterium and ensnares the recipient bacterium merging the cytoplasm of both bacteria via a controlled pore. This pore allows the transfer of plasmids.

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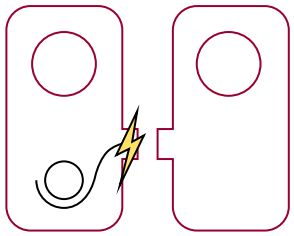
Flavo-80 Reg.no. G4149 (Act 36 of 1947)

Flavo-8 Reg.no. G4151(Act 36 of 1947)

Flavo and resistance reduction



Flavo prevents the formation of the pili and subsequent plasmid transfer.

Over the past 40 years of use no transferable form of resistance against flavophospholipol has been observed.

Flavophospholipol (Flavo) feed supplementation also did not select for resistance to available therapeutic agents.

Flavo selectively inhibits the growth of Salmonella typhimurium isolates containing plasmids.

Flavo shows a decrease in conjugation transfer of plasmids in strains of E. Coli, Salmonella spp., S. aureus and E. faecium Flavo decrease conjugation of E.Coli, resistant to broad spectrum β-lactams and therefore a reduction in the horizontal spread of resistance plasmid occurs.

Flavo and human use

Flavo is used as a feed additive growth promotor in farm animals but has no therapeutic use in humans. The mechanism of action of Flavo is not shared by any agent used in human medicine.

Flavo-8 and Flavo-80 should be
your growth promotor of choice

Flavo reduces the transfer of antibiotic resistance between bacteria and, consequently, decreases resistance levels in bacterial populations by two mechanisms: enhanced activity against plasmid containing gram-negative bacilli and decrease of the conjugation transfer frequency of plasmids.

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