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IN VITRO ANTIBACTERIAL EFFECT OF LAURIC ACID ON *PAENIBACILLUS LARVAE*, CAUSAL AGENT OF AMERICAN FOULBROOD

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American foulbrood (AFB) is a serious disease that affects the larvae of honeybee *Apis mellifera*. It is caused by *Paenibacillus larvae*, a resistant spore forming bacterium, which remains viable through long periods of time and survives environmental adversities¹. This plague is usually controlled with antibiotics which could leave toxic residues in honey and by products. Laboratory and field trials were conducted to evaluate the effectiveness of lauric acid, an innocuous saturated fatty acid, in the prevention and control of AFB in honeybee colonies. The antimicrobial activity of lauric acid against ten *P. larvae* strains was determined by the broth microdilution method. *In vitro* assays showed the lowest concentration of lauric acid that cause the inhibition of the bacterial strains, The minimal inhibitory concentration (MIC) and the minimal bactericide concentration (MBC) mean values were 20,33 µg mL⁻¹ (range: 13,54 - 27,08 mg mL⁻¹). Lauric acid toxicity to adult honeybees was tested by two different methods and registered after 24h, 48h and 72h. The LD₅₀ (Lethal Dose 50) mean values obtained were 216,6 mg/bee (24h); 166,9 mg/bee (48h); 137,6 mg/bee (72h) by the oral administration method; and 1.352 mg/plate (24h), 1.255 mg/plate (48h) and 1.214 mg/plate (72h) by the complete exposure method. The LD₅₀ indicated that complete exposure method would be the safer one to administer the fatty acid on beehives. The results obtained for lauric acid in the present study contribute to the screening of alternative natural compounds to control AFB in the apiaries. This way, toxicological risks and other undesirable effects, such as resistances due to the indiscriminate use of antibiotics, would be avoided.

¹Genersch E et al. (2006) International Journal Systematic Evolution of Microbiol, 56:501-511.

ANTIPARASITIC HYBRIDS OF *CINCHONA* ALKALOIDS AND BILE ACIDS

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A series of 16 hybrids of *Cinchona* alkaloids and bile acids was prepared by means of a Barton-Zard decarboxylation reaction. Quinine, quinidine, cinchonine and cinchonidine were functionalized at position C-2 of their quinoline nucleus by radical attack of a *nor*-cholane substituent. The compounds were evaluated *in vitro* for their antitrypanosomal, antileishmanial and antiplasmodial activities, along with their cytotoxicity against normal human fibroblast cells. Seven compounds showed promising trypanocidal activity with IC₅₀ in the same range as the commercial drug suramine. Moreover all the 16 hybrids showed antiplasmodial activity (IC₅₀ ≤ 6 µg/ml), particularly those containing a *nor*-chenodeoxycholane moiety with IC₅₀ comparable to those of the natural alkaloids, and good selectivity indexes.

