RESEARCH NOTE

INTERACTIONS BETWEEN ANTIPLASMODIAL 3,6-DIAMINO-1′-DIMETHYL-9-ANILINOACRIDINE AND HEMATIN AND CONCANAMYCIN A

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Abstract. Antiplasmodial 9-anilinoacridine derivatives exert their effects either by inhibiting DNA topoisomerase (topo) II or by interfering with heme crystallization within the parasite acidic food vacuole. Previous studies have shown that analogs of 9-anilinoacridine containing 3,6-diamino substitutions (in the acridine ring) inhibit *Plasmodium falciparum* DNA topo II *in situ,* whereas those with a 3,6-diCl substitution act by inhibiting beta-hematin formation, a property also seen with 3,6-diamino-1′-dimethyl-9-anilinoacridine (DDAA). To understand this seemingly anomalous property of DDAA, studies of its interaction with hematin and localization within the parasite food vacuole were undertaken. A weak interaction with hematin was demonstrated spectroscopically. Antagonism of DDAA inhibition of *Plasmodium falciparum* growth in culture by concanamycin A, a macrolide antibiotic inhibitor of vacuolar H^+-ATPase derived from *Streptomyces* sp, was equivocal.

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