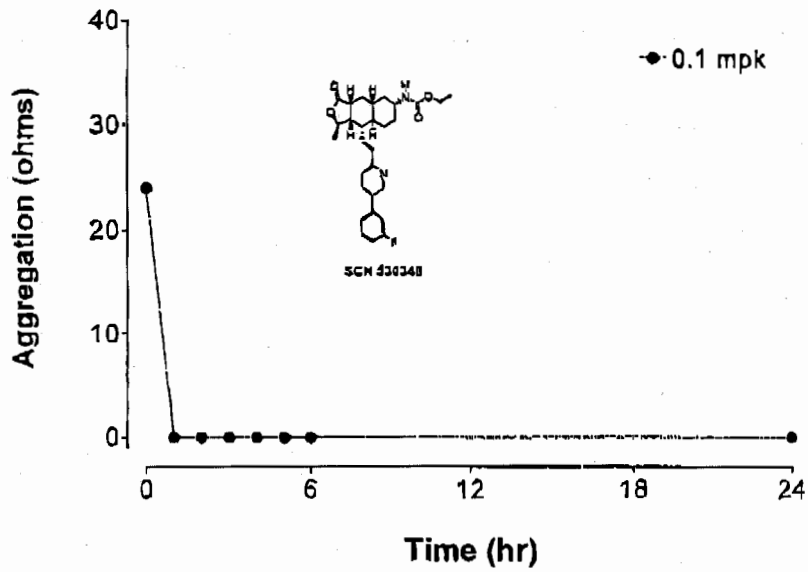


Figure 1. Compound A, incorporating a secondary amine on the right-hand ring, shows substantially 100% inhibition of platelet aggregation (0 ohms of signal) for 24 hours in an *ex-vivo* cynomolgus monkey model after oral administration of 0.1 mg/Kg of the drug.

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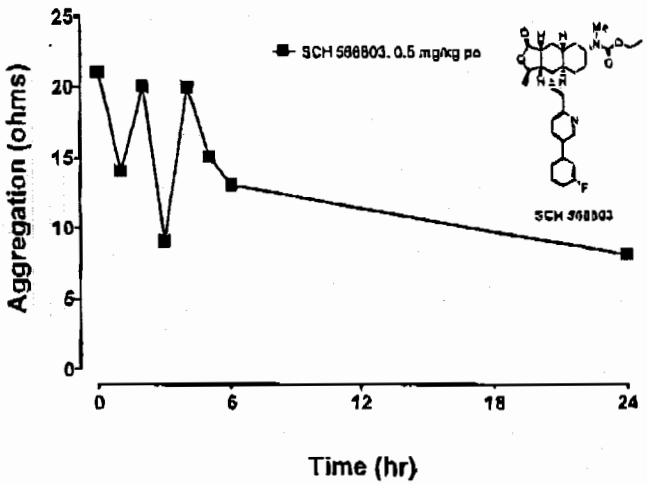


Figure 2. Compound B, incorporating a tertiary amine on the right-hand ring, shows poor inhibition of platelet aggregation in the ex-vivo cynomolgus monkey model after oral administration of 0.5 mg/Kg of the drug. Note that Compound A of Figure 1 was dosed at only 0.1 mg/Kg.

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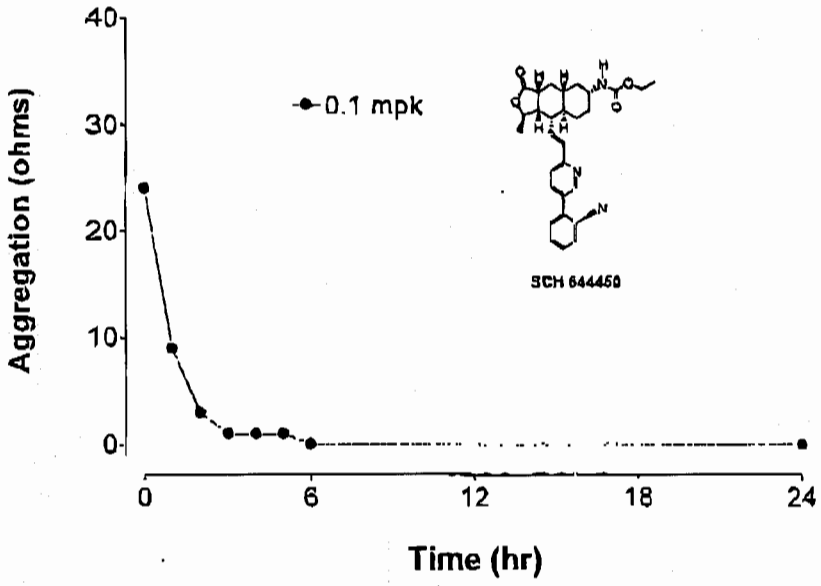


Figure 3. Compound C shows substantially 100% inhibition of platelet aggregation for 24 hours in the *ex-vivo* cynomolgus monkey model after oral administration of 0.1 mg/Kg of the drug. This study demonstrates that the position and choice of phenyl substituents can be varied simultaneously without significant loss of platelet aggregation (ortho-cyano for Compound C in lieu of meta-fluoro for Compound A).

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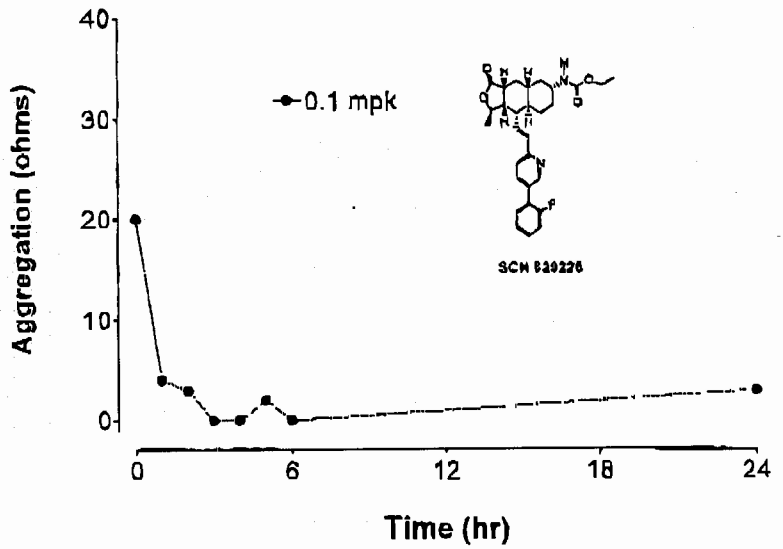


Figure 4. Compound D shows short term near 100% inhibition of platelet aggregation for 24 hours in the *ex-vivo* cynomolgus monkey model after oral administration of 0.1 mg/Kg of the drug.

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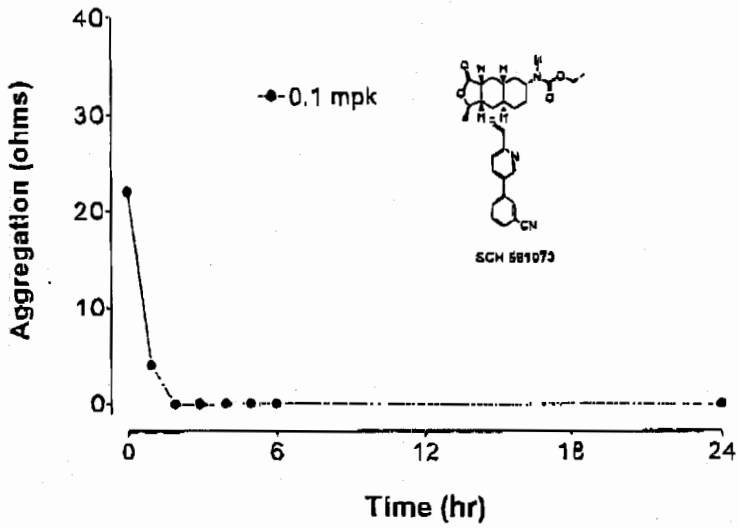


Figure 5. Compound E shows substantially 100% inhibition of platelet aggregation for 24 hours in the *ex-vivo* cynomolgus monkey model after oral administration of 0.1 mg/Kg of the drug.