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Plant polysaccharide antiviral agents from Astragalus and Sterculia species. D.F. Smee[1] and A.J. Verbiscar.[2] [1]Institute for Antiviral Research, Utah State University, Logan, Utah and [2]Anver Bioscience Design, Inc., Sierra Madre California USA.

Certain species of Astragalus and Sterculia plants produce gums comprised primarily of polysaccharide components. These substances were purified and evaluated for antiviral activity in models of cytomegalovirus (murine) and picornavirus (encephalomyocarditis virus) infections in mice. The most active polysaccharide species included Astragalus echidnaeformis, Astragalus brachycentrus, and Sterculia urens. Intraperitoneal doses ranging from 3 to 100 mg/kg/day were effective in dramatically reducing mortality, and no toxicity was evident at the high dose. Treatments had to be given before or soon after virus challenge in order to be effective. The compounds were devoid of in vitro antiviral activity, indicating that immunopotentiation was responsible for conferring protection to the animals. The serum of uninfected animals treated with polysaccharides were assayed for interferon, and approximately 1000 units of interferon activity were present within 2 hours of treatment. The interferon level persisted through 24 hours. Thus, the antiviral activity may be attributable to interferon induction. The polysaccharides represent a new class of immunopotentiating agents that demonstrate antiviral activity in mice.

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DRUG INTERACTIONS WITH MOPYRIDONE

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Mopyridone is a new compound with a strong antiviral effect and low toxicity. The combination of mopyridone and rimantadine manifested a synergistic effect against influenza viruses with sufficient high antiviral activity in vivo and in vitro. The combination mopyridone (1/10 of LD 50) and aspirin demonstrated significantly increased analgetic effect in mice, in comparison to the effect of aspirin alone. According both acetic acid test and Hot plate test, mopyridone alone had significant analgetic effect comparable with the effect of aspirin. Mopyridone had an inducing effect on aniline hydroxylase and cytochrome P-450 and had not significant effect on mouse liver N-demethylase activity (with substrates ethylmorphine, amidopyrine and benzphetamine). These changes are not related with any changes in acute toxicity of some known monooxygenase substrates- amidopyrine, acetaminophen, rimantadin and strychnin. Phenobarbital induction did not alter mopyridone oral acute toxicity in mice, while methylcholanthrene and dexamethasone induction increased it. The participation of some cytochrome P-450 isozymes induced by methylcholanthrene and by dexamethasone in mopyridone biotransformation to more toxic product(s), probably determines the higher mopyridone toxicity in methylcholanthrene and dexamethasone induced mice.