
It has been reported that although ASA may have some beneficial effects in males, no differences from placebo were detected in females in clinical trials on venous thrombosis, TIA and stroke. Since ASA inhibits platelet function, any differences measured in these clinical studies may be related to placebo function, we measured bleeding time and platelet aggregation induced by collagen or arachidonic acid following oral administration of ASA (650 mg) or flurbiprofen (100 mg) in normal male and female volunteers (10 per group, total of 40 subjects) between the age of 50 and 70.

No sex-associated differences in response with either drug were observed in bleeding time or platelet function. Thus, we were unable to confirm that the observation of beneficial effect of ASA in males was due to its effect on platelets. However, more interestingly there was a statistically shorter bleeding time in males (p = 0.026) before administration of drug. It was also found that despite the normal response to collagen, PRP prepared from pre-drug blood samples from 6 individuals (15%) did not aggregate when stimulated with arachidonic acid at concentrations as high as 1 μM. These results suggest that, at least in some individuals, collagen-induced aggregation may proceed by a pathway independent of the arachidonic acid pathway.


This study was designed to determine whether the differential inhibition of prostacyclin(PGI,) production by vessel wall and malondialdehyde(MDA) production by platelet might be possible by oral administration of aspirin(ASA).

Rabbits weighing 2-3kg were used. MDA production by platelet was measured by the Moncada's method with minor modification. The PGI2 production by vessel wall was assessed by the Moncada's method with minor modification.

The PGI2 production by caval vein, pulmonary artery, pulmonary vein, femoral artery, femoral vein and coronary artery was 148-55%, 136-56%, 153-55%, 134-56%, 123-64%, 103-55% to that of aorta, respectively. The PGI2 production by these vessels was inhibited to 20-10% to their initial level 6h after the single oral administration of 0.3g ASA, and restored to the initial level by 24h, while the MDA production was inhibited more conspicuously and remained at less than 50% of the initial level even 48h.

The effect of daily oral administration of ASA on production of PGI2 by aorta and MDA by platelet was investigated. PGI2 production was suppressed to about 10% of the initial level 24h after the last dose of 3 to 7 daily administration of 0.3g of ASA. This indicates that the daily ASA administration results in the cumulative inhibition of PGI2 production. On the other hand, when ASA was administered every other day, the same amount of ASA exerted significantly less inhibition of PGI2 production, being at about 50% of initial level 24h after the last dose. MDA production was nearly completely inhibited over the observation periods.

The results suggest that the differential inhibition of vascular PGI2 production and platelet aggregation might be possible by the administration of ASA at an appropriate amount and proper interval.