

study, we have compared the anti-platelet activity and mechanism of epigallocatechin gallate (EGCG) and epigallocatechin(EGC), which are two major components of GTC. We investigated the inhibitory effects of rabbit platelet aggregation by EGCG and EGC. EGCG inhibited collagen- and arachidonic acid-induced platelet aggregation, with  $IC_{50}$  values of 80 and 100  $\mu$ M, respectively. However EGC without an additional galloyl group on C-3, weakly inhibit collagen- and arachidonic acid-induced aggregation of rabbit platelets at the concentration of 200  $\mu$ M. To investigate the anti-platelet mechanism of EGCG and EGC, we tested the effects of EGCG and EGC on arachidonic acid liberation and thromboxane  $B_2$  conversion. EGCG potently inhibited the arachidonic acid liberation from membrane phospholipids in a dose-dependent manner, while EGC did not show any inhibitory effect. EGCG showed weak inhibition of  $TxB_2$  conversion from arachidonic acid, while EGC significantly inhibited  $TxB_2$  conversion. EGCG and EGC did not alter such coagulation parameters as activated partial thromboplastin time and prothrombin time. Taken together, these observations suggest that the anti-platelet activity of EGCG may be mediated mainly by inhibition of arachidonic acid liberation and that the anti-platelet effect of EGCG is enhanced by the presence of a gallate moiety at C-3 position.

[PA3-10] [ 04/17/2003 (Thr) 14:00 - 17:00 / Hall P ]

### The sphingoid base 1-phosphate as an endogenous marker for Myocardial Infarction

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The purpose of this study was to determine the possibility of sphingolipid as a diagnostic marker for Myocardial Infarction(MI), atherosclerosis-related cardiovascular disease. Sphingolipids are known to play a role in the occurrence of atherosclerosis in human blood vessels. Platelet-poor plasma(PPP) and washed platelets were prepared from healthy volunteers and MI patients, and sphingolipids analyzed. Sphingoid base 1-phosphate(S1P) was decreased in PPP from MI patients by 30-50% compared with normal persons, while MI patients showed the statistically different concentrations of S1P in plasma among stable, unstable and variable MI. Washed platelets from MI patients showed approximately 30 pmol and 18 pmol of sphingosine 1-phosphate and sphinganine 1-phosphate, while 270 pmol and 45 pmol per 100 $\mu$ g protein in normal volunteers, respectively. The sphingolipid profile in human platelets are very similar to the one in plasma. The phytosphingosine 1-phosphate was also detected in 20pmol/200 $\mu$ l normal human plasma. These results suggested that S1P may be a sensitive and specific biomarker for human cardiovascular diseases.

[PA3-11] [ 04/17/2003 (Thr) 14:00 - 17:00 / Hall P ]

### The altered sphingolipid metabolism in rats following fumonisin B1 exposure

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Fumonisin is a specific inhibitor of ceramide synthase in sphingolipid metabolism. The objective of this study was to investigate whether the elevation of free sphingoid bases 1-phosphate (S1P) are related to the fumonisin exposure. Sprague Dawley rats were injected i.p.