In vitro Effects of Vitamin E on Hydrogen Peroxide—induced Platelet Aggregation

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— It was demonstrated that both α -tocopherol acetate and α -tocopherol nicotinate, especially the latter, could inhibit the H_2O_2 -induced platelet aggregation to some extent in vitro. — H_2O_2 ; platelet aggregation; vitamin E acetate; vitamin E nicotinate

It has been reported previously that platelets can be aggregated by hydrogen peroxide (H_2O_2) in vitro (Higashi et al. 1973). This phenomenon might have an in vivo counterpart, because it has recently been demonstrated that H_2O_2 could be released from phagocytes to affect other cells (Baehner et al. 1971). It is well known that vitamin E acts as a natural antioxidant and the H_2O_2 hemolysis of vitamin E deficient erythrocytes can be prevented by the preincubation of the cells with vitamin E (Rose and György 1952). It is reasonable therefore to think that the H_2O_2 -induced platelet aggregation might be inhibited by vitamin E in vitro. The present investigation was undertaken to test this hypothesis.

MATERIALS AND METHODS

Blood was obtained from apparently healthy adolescent volunteers, 2 to 3 hours after breakfast. Methods were described in previous papers (Higashi et al. 1973) unless otherwise indicated.

α -Tocopherol acetate (Eisai Co. and Nakarai Chemicals) and α -tocopherol nicotinate (Eisai Co.) were dissolved in ethanol to make 70/3 mM to 3.5/3 M solutions, and 0.003 ml of the solution was added to 0.6 ml of platelet rich plasma (PRP) by the use of a microsyringe. In control tubes, 0.003 ml of the imidazole buffer solution (IBS) or ethanol was added instead of reagents above described. The mixture was left at room temperature for one hour. Then 0.027 ml of IBS was added to each tube to make a total amount of mixture 0.63 ml respectively. The tube was transferred to the aggregation meter apparatus. After the incubation period of 1 minute at 37°C, 0.07 ml of the aggregating agent, namely 0.3% H_2O_2 in IBS, was added.

RESULTS AND DISCUSSION

Parts of the results obtained were summarized in Tables 1 and 2. As shown in Table 1, α -tocopherol acetate may inhibit H_2O_2 -induced platelet aggregation to some extent at the final concentration of 1 to 2.5 mM in most cases examined.

The inhibitory effect of α -tocopherol nicotinate was apparent already at the final concentration of 0.1 to 0.25 mM as shown in Table 2 (cf. Table 2).

Thus the hypothesis mentioned above proved to be correct.

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TABLE 1. *Effects of vitamin E acetate on H₂O₂-induced platelet aggregation (Maximum aggregation in percentage)*

Case No.	IBS	Ethanol	Final concentrations of vitamin E acetate			
			0.1 mM	0.5 mM	1 mM	2.5 mM
1	28	32	—	—	23	—
2	30	30	—	—	—	24
3	55	55	—	—	45	44
4	51	36	—	—	—	24
5	34	30	—	—	27	18
6	40	37	37	33	37	—
7	56	50	53	46	43	43
8	35	34	26	19	16	17

—, not determined

TABLE 2. *Effects of vitamin E nicotinate on H₂O₂-induced platelet aggregation (Maximum aggregation in percentage)*

Case No.	IBS	Ethanol	Final concentrations of vitamin E nicotinate				
			0.1 mM	0.25 mM	0.5 mM	1 mM	2.5 mM
1	28	32	—	—	—	20	—
2	52	54	—	—	—	—	47
3	46	40	—	—	—	25	24
4	54	52	—	—	30	38	—
5	60	55	—	—	39	39	—
6	50	50	42	32	31	—	—
7	56	43	27	25	26	—	—
8	53	53	35	28	32	—	—

—, not determined

An investigation of compounds capable of inhibiting platelet aggregation could have a potential value for the prevention of occlusive vascular disease (Marcus 1969). From such a point of view and also for the purpose of more fundamental study to clarify the mechanism underlying the platelet aggregation by H₂O₂, further investigation of the present subject is warranted.

References

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