

Dehydroepiandrosterone prevents the aggregation of platelets obtained from postmenopausal women with type 2 diabetes mellitus through the activation of the PKC/eNOS/NO pathway

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The steroid hormone dehydroepiandrosterone (DHEA), suggested to be a cardioprotector, prevents platelet aggregation in healthy humans. This hormone is reduced in postmenopausal women by 60% of its normal value. Platelets in patients with type 2 diabetes (T2D) are more sensitive to aggregation, which has been attributed to a reduced ability to produce nitric oxide (NO). In light of these precedents and considering that DHEA is able to increase the production of NO in cultured endothelial cells, we suggest that DHEA prevents the aggregation of platelet from postmenopausal women with T2D through the activation of PKC/eNOS/NO/cGMP pathway. To determine the effect of DHEA in platelet aggregation, platelet-rich plasma (PRP) obtained from postmenopausal women with T2D was preincubated with DHEA, and aggregation induced by ADP was determined in the presence or absence of L-NNA (LNG-nitroarginine), Rottlerin, NOS, or PKC delta inhibitors, respectively. Platelet NO production was measured with t