

ESTROGEN, INFLAMMATION, AND PLATELET PHENOTYPE.

Miller VM, Jayachandran M, Hashimoto K, Heit JA, Owen WG.

Department of Surgery, Mayo Clinic College of Medicine, Rochester, Minnesota;
Department of Physiology and Biomedical Engineering, Mayo Clinic College of Medicine,
Rochester, Minnesota; Department of Internal Medicine, Mayo Clinic College of Medicine,
Rochester, Minnesota; Department of Biochemistry and Molecular Biology Mayo Clinic
College of Medicine, Rochester, Minnesota

Background: Although exogenous estrogenic therapies increase the risk of thrombosis, the effects of estrogen on formed elements of blood are uncertain.

Objective: This article examines the genomic and nongenomic actions of estrogen on platelet phenotype that may contribute to increased thrombotic risk.

Methods: To determine aggregation, secretion, protein expression, and thrombin generation, platelets were collected from experimental animals of varying hormonal status and from women enrolled in the Kronos Early Estrogen Prevention Study.

Results: Estrogen receptor beta predominates in circulating platelets. Estrogenic treatment in ovariectomized animals decreased platelet aggregation and adenosine triphosphate (ATP) secretion. However, acute exposure to 17beta-estradiol did not reverse decreases in platelet ATP secretion invoked by lipopolysaccharide. Thrombin generation was positively correlated to the number of circulating microvesicles expressing phosphatidylserine.

Conclusion: Assessing the effect of estrogen treatments on blood platelets may lead to new ways of identifying women at risk for adverse thrombotic events with such therapies.