Featured Paper February 2010

Felicitas Müller, Nicola J. Mutch, Wolfdieter A. Schenk, Stephanie A. Smith, Lucie Esterl, Henri M. Spronk, Stefan Schmidbauer, William A. Gahl, James H. Morrissey, and Thomas Renné. 2009. Platelet polyphosphates are proinflammatory and procoagulant mediators in vivo. Cell 139: 1143-1156.

Thrombosis may occur in the venous or arterial circulation, causing PE or myocardial infarction and stroke, the most common causes of death in the developed world. Platelets play a central role in thrombosis, hemostasis, and inflammation. These anucleate cells contribute to fibrin formation and inflammation leading to the concept of “procoagulant platelet activity.” In this study the scientists Felicitas Müller and Thomas Renné from the Department of Molecular Medicine and Surgery and Center for Molecular Medicine, Karolinska Institutet, Stockholm, Sweden (formerly Institute of Clinical Biochemistry and Pathobiochemistry, University of Würzburg) show that the inorganic polymer, polyP, which is secreted upon platelet activation, is responsible for platelet-driven fibrin formation and vascular leakage. PolyP mediates its effects by activating the FXII-driven contact activation system that is conserved in humans and mice. PolyP links platelet plug formation (primary hemostasis) and fibrin generation (secondary hemostasis). PolyP functions are not limited to thrombus formation but also contribute to platelet-driven capillary leakage, which is a hallmark of inflammatory reactions.

The data identify polyP as a new class of mediator having fundamental roles in platelet-driven proinflammatory and procoagulant disorders.