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Arteriosclerosis, Thrombosis, and Vascular Biology

Gas6 Promotes Inflammatory (CCR2^{hi}CX3CR1^{lo}) Monocyte Recruitment in Venous Thrombosis

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Anticoagulants have been the standard of care for venous thromboembolism (VTE), however their association with severe bleeding makes them an imperfect remedy. The Blostein lab is interested in delving down into the cellular components of VTE in order to discover alternative treatments that will minimize the complications of VTE and the use of blood thinners. It is exploring the potential of manipulating the protein Gas6 to better control blood clots. This paper shows that Gas6 is required for the recruitment of inflammatory white cells in the clot, and that reducing Gas6 undermines this process.

The hypothesis is that the development of an inhibitor to Gas6 will interfere with the accumulation of white cells that are contributors to the damage caused by VTE, including post-thrombotic syndrome, chronic lung disease, and death from pulmonary embolism. It has been shown in animal models that targeting Gas6 does not cause bleeding, making it promising for adjuvant therapy to anticoagulants that may have a positive impact on the long-term complications of VTE. The paper concludes, “Deciphering the role of Gas6 and precise function of inflammatory monocytes in venous thrombosis occurrence can be a powerful tool for the identification of new targets for future antithrombotic therapy.”

An accompanying editorial cites several of Dr. Blostein’s earlier publications as “milestones” in the discoveries of Gas6’s signaling function in thrombosis.

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