



## Barry S. Collier, M.D.

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**When blood vessels break, platelets stop the bleeding by adhering to the damaged vessel walls. Collier's research focuses on molecular interactions between blood cells and blood vessels, and on new therapies for thrombotic diseases such as heart attack and stroke.**

Because platelets play a vital role in blood coagulation, deficiencies in their numbers or function can result in excessive bleeding. But when platelets adhere to and aggregate on blood vessels narrowed by atherosclerosis, they can close off the blood vessel and cause a myocardial infarction (heart attack) or stroke.

Current research in Collier's lab focuses on multiple areas of platelet physiology. Among them is the genetic disorder Glanzmann thrombasthenia, which produces hemorrhage as a result of an abnormality of the platelet  $\alpha\text{IIb}\beta_3$  receptor. Collier and his lab members are studying the precise genetic and protein abnormalities responsible for the disease, as well as variants in the genes for the receptor (ITGA2B and ITGB3) identified in the general population by next-generation sequencing.

Other areas of blood and platelet physiology that the lab studies include:

Monoclonal antibody-based therapeutic for myocardial infarction. By studying the receptors responsible for platelet aggregation and patients who genetically lack the receptors, Collier established the platelet  $\alpha\text{IIb}\beta_3$  (GPIIb/IIIa) receptor as an important target for antithrombotic therapy. This led him to develop monoclonal antibodies to the platelet  $\alpha\text{IIb}\beta_3$  receptor that inhibit platelet aggregation. Working with scientists at Centocor, Collier helped develop a derivative of one of these antibodies into the drug abciximab, which was approved in 1994 to prevent ischemic complications of percutaneous coronary interventions, such as stent placement in patients with myocardial infarction and related conditions. More than five million patients worldwide have been treated with abciximab.

High-throughput screening and structure-guided design of a novel antiplatelet drug. Starting with a high-throughput screen for small molecule inhibitors of  $\alpha\text{IIb}\beta_3$ , followed by extensive medicinal chemistry, the Collier lab developed the compound RUC-4 (now zalunfiban) for first point of contact therapy of ST-segment Elevation Myocardial Infarction (STEMI). Zalunfiban met its primary efficacy and safety endpoints in a 2,467 patient Phase 3 study. Current plan is to request approval of the drug by the FDA.

Integrin structure and activation. Integrins, including platelet  $\alpha\text{IIb}\beta_3$  and  $\alpha\text{V}\beta_3$ , are transmembrane glycoprotein receptors. Through site-directed mutagenesis, molecular dynamics, cryo-electron microscopy, and x-ray crystallography studies, the lab studies the mechanisms by which the receptors undergo a transition from an inactive to an active conformation with high affinity for ligands. Most recently, cryo-EM has led to atomic-level, three-dimensional reconstructions of the complex between  $\alpha\text{IIb}\beta_3$  and abciximab and a monoclonal antibody that activates the receptor.

Platelet TGF- $\beta_1$ . The Collier lab discovered that platelet TGF- $\beta_1$  can be activated by shear forces, and studies are under way to assess the biological significance of this finding in several model systems.

Integrin  $\alpha\text{V}\beta_3$ . The Collier lab is developing pure antagonist anti- $\alpha\text{V}\beta_3$  drugs with the goal of studying their impact on a variety of pathological processes, including sickle cell disease, osteoporosis, and herpes virus infection.

Platelet Fc $\gamma$ RIIIa. The Collier lab is developing inhibitors of the binding of immunoglobulin Fc domains in immune complexes to platelet Fc $\gamma$ RIIIa as improved therapy of thrombosis associated with autoimmune disorders.

### EDUCATION

B.A., 1966  
Columbia University  
M.D., 1970  
New York University School of Medicine

### MEDICAL TRAINING

Internship in medicine, 1970–1971  
Residency in medicine, 1971–1972  
Bellevue Hospital

### POSITIONS

Clinical Associate, 1972–1974  
Staff Physician, 1974–1976  
National Institutes of Health  
Assistant Professor, 1976–1978  
Associate Professor, 1978–1982  
Professor, 1982–1993  
Associate Director for Biomedical Research, 1992–1993  
Distinguished Service Professor, 1993  
State University of New York at Stony Brook  
Professor, 1993–2001  
Mount Sinai School of Medicine  
Professor, 2001–  
Vice President for Medical Affairs, 2001–  
Director, Maurice R. and Corinne P. Greenberg Center for Studies in Inflammation, Microbiome, and Metabolism, 2019–  
Co-director for Clinical Studies, Stavros Niarchos Foundation  
Institute for Global Infectious Disease Research, 2023–  
The Rockefeller University  
Physician in Chief, 2001–  
Co-director, Center for Clinical and Translational Science, 2001–  
The Rockefeller University Hospital

### AWARDS

National Research Achievement Award, American Heart Association, 1998  
Warren Alpert Foundation Award, 2001  
Pasarow Award, 2005  
Karl Landsteiner Memorial Award, 2013  
Gill Award, University of Kentucky Gill Heart Institute, 2016  
Grant Medal, International Society on Thrombosis and Haemostasis, 2021

### HONORARY SOCIETIES

National Academy of Sciences  
National Academy of Medicine  
National Academy of Inventors  
American Academy of Arts and Sciences

### SELECTED PUBLICATIONS

Van't Hof, A.W. et al. Zalunfiban at first medical contact for ST-elevation myocardial infarction. *NEJM Evid.* (2025).  
Wang, J. et al. An  $\alpha\text{IIb}\beta_3$  ligand-mimetic murine monoclonal antibody that produces platelet activation by engaging the Fc $\gamma$ IIa receptor. *Blood Adv.* 9, 3518–3529 (2025).  
Buitrago, L. et al. Platelet binding to polymerizing fibrin is avidity driven and requires activated  $\alpha\text{IIb}\beta_3$  but not fibrin cross-linking. *Blood Adv.* 5, 3986–4002 (2021).  
Nešić, D. et al. Cryo-electron microscopy structure of the  $\alpha\text{IIb}\beta_3$ -abciximab complex. *Arterioscler. Thromb. Vasc. Biol.* 40, 624–637 (2020).  
Li, J. et al. Novel pure  $\alpha\text{IIb}\beta_3$  integrin antagonists that do not induce receptor extension, prime the receptor, or enhance angiogenesis at low concentrations. *ACS Pharmacol Transl Sci.* 2, 387–401 (2019).