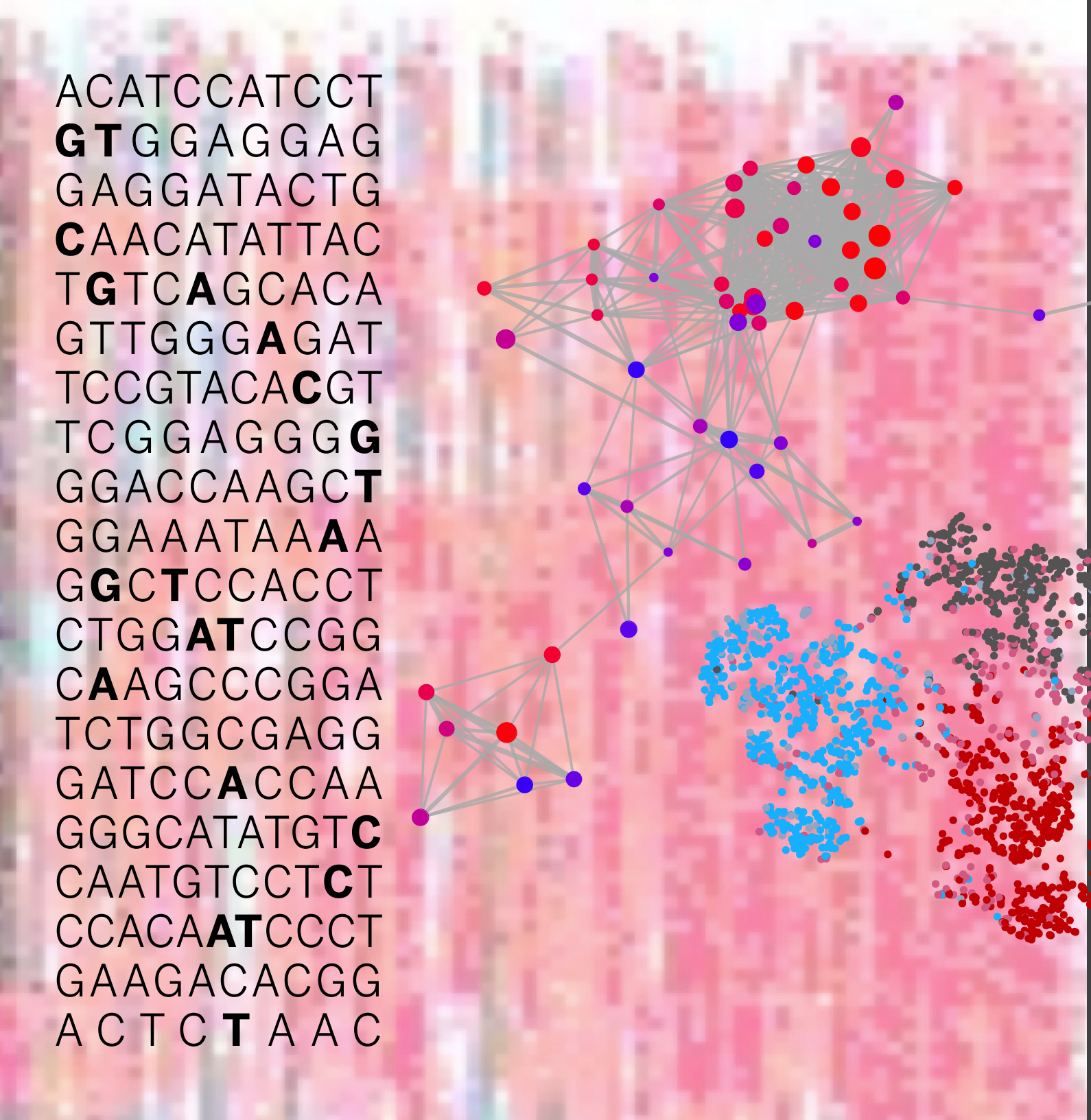


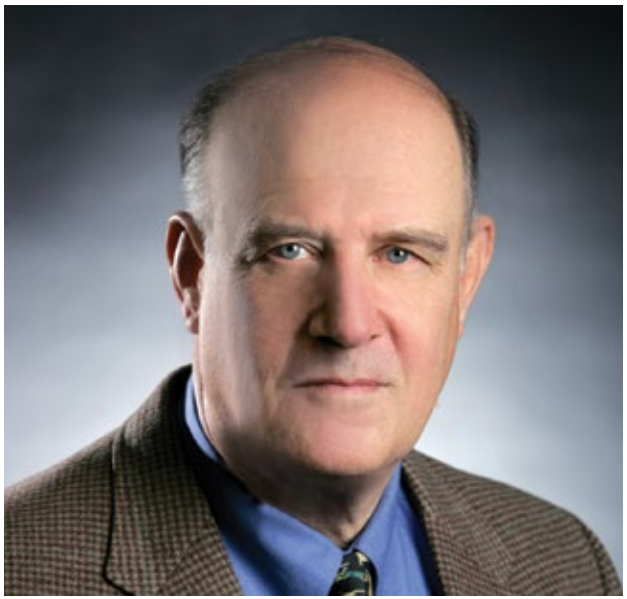
Research Report 2019

versiti™
Blood Research Institute

The cover image illustrates "Big BioData" research conducted by the Computational Biology Core at the Blood Research Institute (see page 51). The nucleotide sequence on the left is part of a synthetic CAR-T cell receptor developed by Dr. Weiguo Cui for treatment of solid tumors. The cluster of dots on the lower right illustrates how individual CD8 cells differ in their tumor-fighting ability, with each cell (dot) colored according to its pattern of gene activity. The background shows gene activity visualized in a so-called heatmap, where the color of each pixel indicates the activity of one of ~25,000 genes. Fun fact: The nucleotides lettered in boldface spell out **Versiti** in the one-letter code for amino acids! (Figure Courtesy of Dr. Hartmut Weiler)

versiti™
Blood Research Institute

A Message from Executive Vice President Gilbert White, II, MD, Chief Scientific Officer



2019 was the start of a transition to new leadership as I announced my intention to step aside from administrative responsibilities and go half-time for two years. We are in the middle of a search for a new Executive Vice President for Research as I write this.

Grant revenues for the year were $15.5 M, down 4% from the previous year, but exceeding our 2019 budget by $1.1 M. New NIH R-type grants or competitive renewals were obtained by Roy Silverstein, Qizhen Shi, Alan Mast, Weiguo Cui, and Renren Wen. Nan Zhu received a 1st %ile score for her R01 and was therefore given a 7-year MERIT award from NCI. Bob Montgomery's Zimmerman Program Project Grant (PPG) had to be resubmitted as a new PPG due to new NIH guidelines and was funded for years 1-5, actually years 11-15. Alan Mast renewed our long-standing REDS contract (REDS-IV-P) for an additional 7 years (years 14-21). A new F-award went to Katelyn Heimbruch in Sid Rao's lab, and also to Paytsar Topchyan in Weiguo Cui's lab. Altogether, there were 15 new NIH grants in 2019 that totaled $34.9 M. Nan Zhu received the second R. Douglas Ziegler Innovation Award for her work related to the pathogenesis of acute myelogenous leukemia.

Promotions during the year included Qizhen Shi, Sandy Haberichter, Brian Curtis, and Josh Field to Senior Investigator and Renren Wen and Anand Padmanabhan to Investigator. William Drobyski, Professor of Medicine at the Medical College of Wisconsin, was appointed as adjunct Senior Investigator at the Blood Research Institute (BRI). Anand Padmanabhan accepted a position at the Mayo Clinic and left Versiti in December.

Individual accomplishments during the year were numerous. Dick Aster received the Coulter Award from the American Society of Hematology, the organization's Lifetime Achievement Award. Roy Silverstein served as President of ASH. Hervé Falet also serves on the Megakaryocyte and Platelet Scientific Committee and Lisa Baumann-Kreuziger is a member of the Thrombosis and Hemostasis Working Group and the ASH Quality Measure Oversight Subcommittee. She also serves on the ASH Thrombophilia Guidelines Panel. Peter Newman continues to serve on the Investment and Audit Committee of ASH. The newly created Hauske Family Chair was conferred on Karin Hoffmeister. Bob Montgomery was invited to give a lecture at the 13th annual Earl W. Davie symposium at the Centre for Blood Research in Vancouver. Karin Hoffmeister and Deb Newman continue to serve on NIH review panels. Alan Mast and Peter Newman continue to serve as Associate Editors of the *Journal of Thrombosis and Haemostasis and Arteriosclerosis, Thrombosis, and Vascular Biology* (ATVB), respectively. Alan is also on the Editorial Board of *Blood Advances*. Subra Malarkannan is Associate Editor of *Frontiers in Immunology*. Hardy Weiler and Roy Silverstein serve on the *ATVB* Editorial Board. Magda Chrzanowska is on the Editorial Board of *Plos One*. Demin Wang is on the Editorial Board of *Blood*. Bonnie Dittel is on the Editorial Boards of the *Journal of Neuroimmunology*

and *Brain, Behavior, and Immunity* and serves as Associate Editor of *Autoimmunity*. Qizhen Shi is on the Editorial Board of *Molecular Therapy, Methods and Clinical Development*. Peter Newman is Co-Editor of the 3rd Edition of *Platelets* and Gil White is Co-Editor of the 6th Edition of *Thrombosis and Hemostasis*. Four students completed their PhD degrees in 2019: Kate Dixon, Zachary Gerbec, Jason Siebert, and Chao Yang in Subra Malarkannan's lab. We celebrated Deb Newman's 30th year and Mark Zogg's, Sandy Holzhauer's, and Irene Hernandez's 20th years with the organization.

The 17th Annual Aster Lecture was delivered on August 13th by Dr. Katherine High, Chief Science Officer at Spark Therapeutic and former Howard Hughes Investigator at the University of Pennsylvania. Her talk was titled "Gene Therapy for Hemophilia: The Long and Winding Road". The 18th Annual Mosesson Lecture was delivered on February 28th by Dr. David Ginsburg, Professor of Genetics and Medicine at the University of Michigan and a Howard Hughes Investigator. His talk was on "Cargo Receptors in the ER: From Clotting Factors to Cholesterol Regulation". The 3rd Annual Jacqueline Fredrick Lecture, selected and hosted by the graduate students and postdoctoral fellows at the BRI, was given on April 23rd by Susan Kaech, Professor and Director of the NOMIS Center for Immunological and Microbial Pathogenesis at the Salk Institute. The talk was titled "Making Immunological Memories". The 3rd Annual R. Douglas Ziegler Innovation Lecture was by Dr. Joseph Miletich, Senior Vice President of Research and Discovery at Merck and Head of Merck Research Laboratories, on November 5th. His talk was titled "Inventing the Future of Drug Discovery and Early Development: Promise and Progress". All four lectures were outstanding examples of cutting-edge science and translation.

The 13th Annual Center for Human Immunology Symposium was held October 30th at the BRI. The symposium, titled "Pathogen Recognition in Innate Immunity," featured renowned speakers from Washington University at St. Louis, Cedars-Sinai Medical Center, Harvard Medical School, University of Massachusetts Medical School, and the University of North Dakota. This year's meeting was hosted by Weiguo Cui, Bonnie Dittel and Jack Gorski. The 2nd Annual Great Lakes Translational Glycomics Symposium was held May 31st at the BRI and was organized by Karin Hoffmeister. Featuring speakers from the University of Alabama at Birmingham, the University of California at San Diego, the Sanford Burnham Institute, Virginia Commonwealth University, and Emory University, it was attended by more than 90 individuals.

The Scientific Advisory Board reviewed the Stem Cell program and part of the Vascular Biology Program on November 14th. Overall, there was high enthusiasm for this group of investigators. In their summary comments, the Board called out the need for succession planning, the role of senior investigators in organizational management, the importance of fully leveraging synergy between the MSI and BRI, and re-evaluation of promotions criteria.

At year's end, there were 45 trainees in the BRI. All four T32 Training Grant positions were filled in 2019: Tyce Kearl in Subra Malarkannan's lab, Lauren Pommert in Sid Rao's lab, Amy Siebert-McKenzie in Alan Mast's lab, and Jesse Sundlov in Peter Newman's lab. Yongwei Zheng in Demin Wang's lab was the 2019 Doolittle Fellow. Waseem Anani in Karin Hoffmeister's lab continued as the first Jacqueline and Arlen Fredrick Scholar. Theresa Bluemn in Nan Zhu's lab received the J. Evan Sadler Graduate Scholar Award, as voted by the BRI graduate students and postdoctoral fellows. Ten new graduate students selected the BRI as their place to train: Yongguang Zhang and Jianhui Wei from Fujian Normal University, and Lu Zhou and Ting Zhao from Shanghai Jiaotang University, all in Demin Wang's lab; and from Medical College of Wisconsin, Jian Shen and Moujtaba Kasmani in Weigo Cui's lab, and Ao Mei, Dandan Wang and Elaheh Hashemi in Subra Malarkannan's lab, and Hongyin Yu in Qizhen Shi's lab.

Gilbert C. White, II, MD

Interim Director, Blood Research Institute

Research By The Numbers – 2019

15
New NIH Grants

$116.6 Million
New Applications

33
Investigators

$15.5 Million
Research Revenues

4
Patents Granted

13
Core Labs

$469+ Thousand
Average Funding per Investigator

5
New Diagnostic Tests Developed

Transfusion Medicine

Transfusion Medicine research has a long history at Versiti, reflecting its basic mission to provide a safe and effective supply of blood products for patients who require transfusion.

Effective transfusion therapy requires knowledge of the biology and physiology of blood, satisfactory methods for collecting and storing blood cells with maximum preservation of function, immunologic aspects of blood transfusion, and an understanding of the many diseases in which transfusion of blood components can be beneficial.

Research conducted by the Transfusion Medicine group addresses each of these areas. Investigators in the Transfusion Medicine Program study basic biology and clinical implications of a range of transfusion-related issues.

Richard H. Aster, MD

CEO Emeritus and Senior Investigator, Blood Research Institute
Professor, Department of Medicine, Medical College of Wisconsin (MCW)
MD, University of Michigan 1957
Hematology, Harvard University 1965
Faculty, Harvard 1964-1970
Started at Versiti: 1970

Awards, Honors and Service

- Founder: GTI Diagnostics (subsidiary of VBRIF)
- Karl Landsteiner Award, American Association of Blood Banks (AABB)
- Henry Stratton Medal for translational research, American Society of Hematology
- Distinguished Service Award, Medical College of Wisconsin (MCW)
- Wallace Coulter Award for career achievement, American Society of Hematology, 2019
- Reviewer for the journals Blood, New England Journal of Medicine, Lancet, Nature, Science Translational Medicine and Transfusion
- Grant reviewer (Transfusion Medicine), Center for Scientific Review, National Institutes of Health

Funding

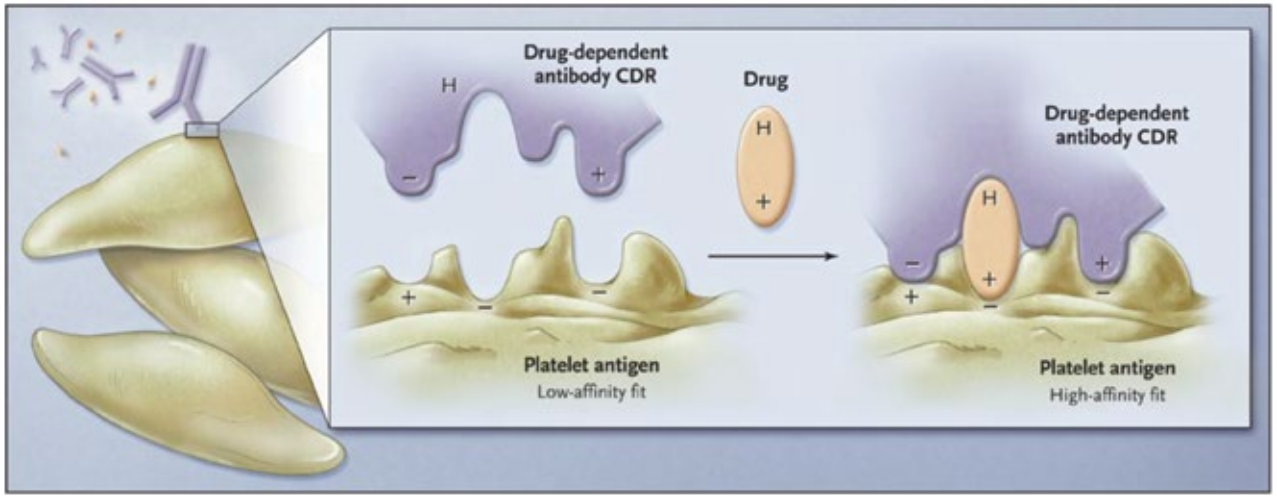
RO1-HL-13629-47 National Heart Lung and Blood Institute. Renewed June 1, 2017

Publications

1. Aster RH. Beta-lactam-induced severe neutropenia: a descriptive study. Fundam Clin Pharmacol. 2019 Apr;33(2):223-224. PMID: 30860628
2. Irani M, Siegal E, Jella A, Aster R, Padmanabhan A. Use of intravenous immunoglobulin G to treat spontaneous heparin-induced thrombocytopenia. Transfusion. 2019 Mar;59(3):931-934. PMID: 30556588
3. Vallatharasu Y, Hayashi-Tanner Y, Polewski PJ, Bottner WA, Rosenstein LJ, Uprety D, Bista A, Farnen JP, Aster R. Severe, prolonged thrombocytopenia in a patient sensitive to exenatide. Am J Hematol. 2019 Mar;94(3):E78-E80. doi: 10.1002/ajh.25381. PMID: 30575104

Research Interests

Immune destruction of red blood cells, white blood cells, and platelets is a major cause of morbidity and mortality. Dr. Aster's work is aimed at understanding the causes of blood cell destruction by autoantibodies, drug-induced antibodies, and antibodies triggered by blood transfusion or exposure to fetal blood cells during pregnancy. Recent studies in his laboratory have shown that metabolites generated in the body following exposure to various drugs can induce antibodies that cause platelet destruction and bleeding and provide new insights into the cause of "idiosyncratic" drug-sensitivity reactions. Findings made in these and related studies are defining new methods for antibody detection to improve diagnosis and treatment in patients with antibody-induced blood cell destruction and improved understanding of the molecular basis for these conditions.



Brian Curtis, PhD, D(ABMLI), MT(ASCP) SBB

Brian Curtis, PhD, D(ABMLI), MT(ASCP) SBB
Director, Platelet & Neutrophil Immunology Lab, Versiti
Senior Director, Diagnostic Hematology
Senior Investigator, Blood Research Institute, Versiti
Assistant Adjunct Professor, Clinical and Translational Science Institute, Medical College of Wisconsin
PhD, University of Wisconsin-Milwaukee
Started at Versiti: 1991

Research Interests

Antibodies specific for antigens carried on blood platelets and white blood cells (neutrophils) cause thrombocytopenia (low platelet count) and neutropenia (low neutrophil count) in various immune disorders and can be difficult to identify. Work in Dr. Curtis' laboratory has led to improved methods for detecting such antibodies and to new understanding of the blood disorders in which they are involved. Dr. Curtis serves as Director of the Platelet and Neutrophil Immunology Reference Laboratory of Versiti and applies his research findings to improve the effectiveness with which this laboratory enhances medical care for patients referred for diagnostic testing. One particular area of expertise for the lab is in diagnosis of Fetal and Neonatal Alloimmune Thrombocytopenia (FNAIT), a disorder in which a pregnant mother can make antibodies that destroy her child's platelets. Recent work in the Curtis lab has allowed for improved diagnosis and prevention of FNAIT.

Awards, Honors and Service

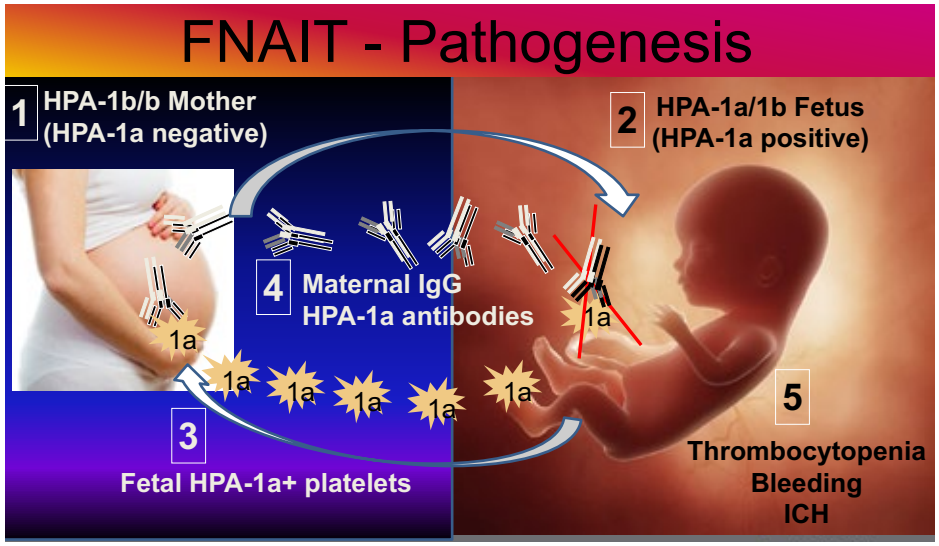
- Member, Editorial Board, Transfusion since 2011
- Member, ISBT Granulocyte Antigen Nomenclature Working Party, since 1997
- Member, ISBT International Granulocyte Immunobiology Steering Committee, since 2014
- Co-Chair, ISTH Platelet Immunology SSC, since 2015
- Member, Platelet Advisory Board, Ionis Pharmaceuticals, since 2016
- Co-chair, ISBT Platelet Immunobiology Working Party, Quality Subcommittee, since 2018

Funding

"Designer Blood Cells Using CRISPR-Cas 9 Gene Editing." Versiti Moonshot Fund, Versiti (PI).

Publications

1. Ameri AH, Curtis BR, Sykes DB. Immune neutropenia mediated by micafungin. Am J Hematol. 2019 Jul;94(7):830-832. PMID: 30945326
2. Mitta A, Curtis BR, Reese JA, George JN. Drug-Induced Thrombocytopenia: 2019 Update of Clinical and Laboratory Data. Am J Hematol. 2019 Mar;94(3):E76-E78. PMID: 30549322
3. Zhang N, Santoso S, Aster RH, Curtis BR, Newman PJ. Bioengineered iPSC-derived megakaryocytes for the detection of platelet-specific patient alloantibodies. Blood. 2019 Nov 28;134(22):e1-e8. PMID: 31697836
4. Jaleah Hawkins, Richard Aster, Brian R Curtis. Post-transfusion purpura: current perspectives. J Blood Med. 2019 Dec 9;10:405-415. doi: 10.2147/JBM.S189176
5. S.S. Khatri, B.R. Curtis, C. Yamada. A case of platelet transfusion refractoriness due to anti-CD36 and successful treatment outcome. Immunohematology 2019;35,4:139-144.



Gregory Denomme, PhD

Senior Investigator, Blood Research Institute, Versiti
Senior Director of Immunohematology and Innovation, Versiti
PhD, Microbiology and Immunology, University of Western Ontario, 1993
Started at Versiti: 2009

Research Interests

Dr. Denomme is Senior Director of Versiti's Immunohematology and Transfusion Service Laboratory, a division of Versiti Clinical Laboratories. He is an immunology and immunohematology-trained scientist with interests in the immune response to red cell antigens, the expression of blood groups, and bench-to-bedside studies in immunohematology. His work integrates immunogenetics with transfusion medicine to explore the genetic basis of blood group expression and the functional polymorphisms underlying the pathology of immune-mediated red cell hemolysis.

Awards, Honors and Service

- Petteway-Shepherd Award, North Carolina Association of Blood Bankers
- Working Party member, Red Blood Cell Immunogenetics and Blood Group Terminology
- International Society for Blood Transfusion
- Editorial Board Member, International Journal of Clinical Transfusion Medicine
- Editorial Board Member, Transfusion

Funding

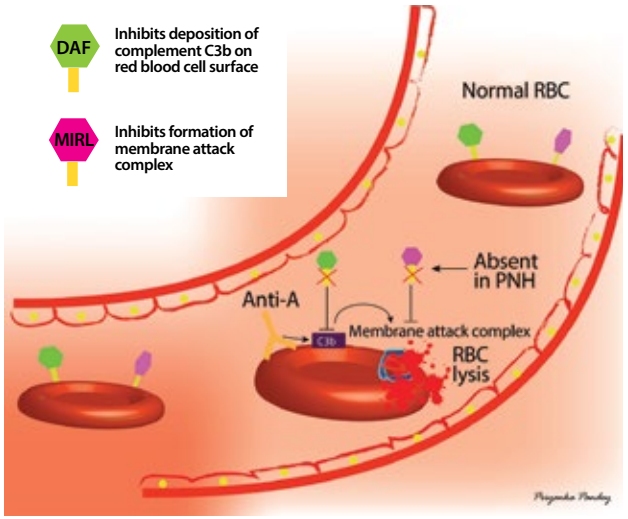
Designer blood cells: CRISPR/cas9 alteration of red blood cell and platelet antigens. G. Denomme (Principal Investigator) B. Curtis, P. Newman (Senior Investigator). Versiti Strategic Funding 2019.

Publications

1. Denomme GA, Anani WQ. Mass-scale red cell genotyping of blood donors: from data visualization to historical antigen labeling and donor recruitment. Transfusion. 2019 Sep;59(9):2768-2770. Review. PMID: 31246285
2. Sippert E, Volkova E, Denomme GA, Liu M, Liu Z, Rios M. New RHCE*ce variant allele in African descent holds 105C > T (silent) in cis to 48C in Exon 1 and 733G in Exon 5. Transfusion. 2019 Sep;59(9):3039-3040. PMID: 31002175
3. Storry JR, Clausen FB, Castilho L, Chen Q, Daniels G, Denomme G, Flegel WA, Gassner C, de Haas M, Hyland C, Yanli J, Keller M, Lomas-Francis C, Nogues N, Olsson ML, Peyrard T, van der Schoot E, Tani Y, Thornton N, Wagner F, Weinstock C, Wendel S, Westhoff C, Yahalom V. International Society of Blood Transfusion Working Party on Red Cell Immunogenetics and Blood Group Terminology: Report of the Dubai, Copenhagen and Toronto meetings. Vox Sang. 2019 Jan;114(1):95-102. PMID: 30421425
4. Volkova E, Sippert E, Liu M, Mercado T, Denomme GA, Illoh O, Liu Z, Rios M; Collaborative Study Group. Validated Reference Panel from Renewable Source of Genomic DNA Available for Standardization of Blood Group Genotyping. J Mol Diagn. 2019 May;21(3):525-537. PMID: 30872185

Antibody mediated lysis of PNH red blood cell clone

(Pandey et al. Blood Advances 2017)



Hervé Falet, PhD

Investigator, Blood Research Institute, Versiti
Assistant Professor, Medical College of Wisconsin
PhD, Paris Descartes University, 1997
Postdoctoral fellowship, Brigham and Women's Hospital and Harvard Medical School, 2001
Started at Versiti: 2016

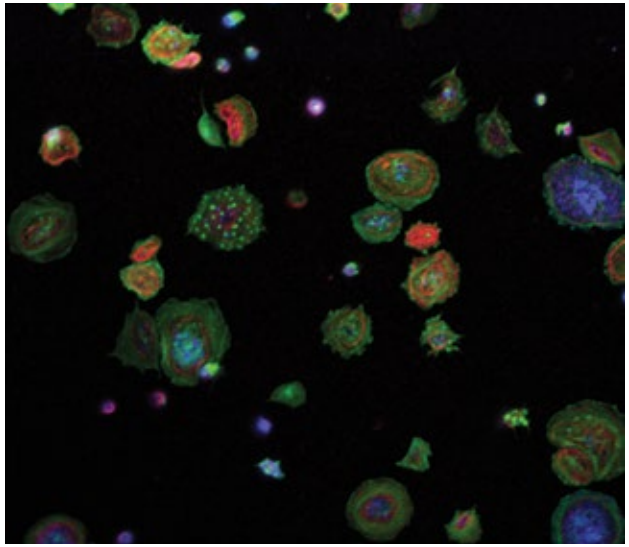
Research Interests

Dr. Hervé Falet received his master's and doctoral degree from Paris Descartes University and completed his postdoctoral fellowship at Brigham and Women's Hospital and Harvard Medical School. He joined the Blood Research Institute faculty in 2016. His primary research interests are associated with blood platelet production (thrombopoiesis) and function.

Platelets circulate in blood at a concentration of 150,000-450,000/µl that is maintained by a fine balance between production and clearance. At sites of vascular injury, platelets respond to external stimuli by rapidly changing shape and recruiting other platelets. Deficient platelet production, due to genetic causes, secondary to cancer therapy, or from unknown etiology, poses significant risks of mortality, mostly due to bleeding.

Blood platelets are produced in the bone marrow by megakaryocytes in a unique process that requires extensive intracellular membrane rearrangements. These include the formation of the demarcation membrane system, the surface-connected membrane extension that invaginates into the cell body and further develops to provide membranes for future platelets.

At the Blood Research Institute, Dr. Falet investigates the roles of novel membrane binding and deforming proteins in the formation and organization of the megakaryocyte demarcation membrane system. He anticipates that his studies will yield basic information related to megakaryocyte and platelet biology, and lead to the development of new approaches to reestablish thrombopoiesis in the setting of thrombocytopenia (low platelet count).



Awards, Honors and Service

- Member, Scientific Committee on Megakaryocytes and Platelets, American Society of Hematology 2019-2022

Funding

National Institutes of Health R01 HL126743, "Endocytosis in Platelet and Megakaryocyte Biology"

Publications

1. Giannini S, Falet H, Hoffmeister KM. Platelet glycobiology and the control of platelet function and lifespan. In: Platelets. Michelson AD, Cattaneo M, Frelinger AL, Newman PJ, Editors. Academic Press, Cambridge; 2019. pp.79-97
2. Eaton N, Drew C, Wieser J, Munday AD, Falet H. Dynamin 2 is required for GPVI signaling and platelet hemostatic function in mice. Haematologica. 2019 Jul 11. pii: haematol.2019.218644. doi: 10.3324/haematol.2019.218644. [Epub ahead of print] PMID: 31296575

"Platelet Fireworks", showing Dnm2-null platelets on a fibrinogen surface after GPVI activation. Winner, 2019 *Platelets* cover competition.

Joshua Field, MD, MS

Senior Medical Director, Versiti
Senior Investigator, Blood Research Institute, Versiti
Professor of Medicine, Medical College of Wisconsin
Medical Director, Adult Sickle Cell Disease Clinic, Froedtert Hospital
MD, Carver College of Medicine, University of Iowa, Iowa City, IA 2001
Started at Versiti: 2010

Research Interests

Dr. Field is concerned with clinical aspects and optimization of treatment for adults with sickle cell disease (SCD).

Awards, Honors and Service

- Member, American Society of Hematology Guidelines on Sickle Cell Disease Committee 2017 - present
- Chair, Acute Pain Taxonomy Project for American Pain Society/American Association for Pain Management 2017 - present
- American Society of Hematology: Committee on Quality, Washington, DC, 2016-present

Funding

"A Phase 1 Open-Label, Dose-Escalation/Dose-Expansion Safety and Tolerability Study of INCB059872 in Subjects with Sickle Cell Disease" Incyte Corporation 11/01/2017 – 10/31/2019

"C1701-202 A Randomized, Placebo-controlled, Phase 2 Study to Evaluate the Safety and Pharmacodynamics of Once-daily Oral IW-1701 in Patients with Stable Sickle Cell Disease" Ironwood Pharmaceuticals, Inc. 06/01-2018-present.

Publications

1. Field JJ, Ballas SK, Campbell CM, Crosby LE, Dampier C, Darbari DS, McClish DK, Smith WR, Zempsky WT Analgesic, Anesthetic, and Addiction Clinical Trial Translations, Innovations, Opportunities, and Networks-American Pain Society-American Academy of Pain Medicine Pain Taxonomy Diagnostic Criteria for Acute Sickle Cell Disease Pain. J Pain. 2019 Jul;20(7):746-759. PMID: 30578848
2. Karafin MS, Chen G, Wandersee NJ, Brandow AM, Hurley RW, Simpson P, Ward D, Li SJ, Field JJ. Chronic pain in adults with sickle cell disease is associated with alterations in functional connectivity of the brain. PLoS One. 2019 May 20;14(5):e0216994. PMID: 31107926
3. Karafin MS, Field JJ. The controversial role of red cell transfusions for sickle cell pain. Curr Opin Hematol. 2019 Nov;26(6):442-447. PMID: 31567433
4. Karafin MS, Mullins DE, Johnson ST, Nischik D, Feng M, Simpson P, Field JJ. Chronic pain persists in adults with sickle cell disease despite regular red cell transfusions. Transfus Apher Sci 2019 Aug;58(4):434-438. PMID: 31326289
5. Lindner JR, Belcik T, Widlansky M, Harmann LM, Karafin MS, Wandersee NJ, Puligandla M, Neuberg D, Linden J, Field JJ. Contrast-enhanced ultrasound detects changes in microvascular blood flow in adults with sickle cell disease. PLoS One. 2019 Jul 5;14(7):e0218783. doi: 10.1371/journal.pone.0218783. eCollection 2019. PMID: 31276520
6. Ruhl AP, Sadreameli SC, Allen JL, Bennett DP, Campbell AD, Coates TD, Diallo DA, Field JJ, Fiorino EK, Gladwin MT, Glassberg JA, Gordeuk VR, Graham LM, Greenough A, Howard J, Kato GJ, Knight-Madden J, Kopp BT, Koumbourlis AC, Lanzkron SM, Liem RI, Machado RF, Mehari A, Morris CR, Ogunlesi FO, Rosen CL, Smith-Whitley K, Tauber D, Terry N, Thein SL, Vichinsky E, Weir NA, Cohen RT, Klings ES. Identifying Clinical and Research Priorities in Sickle Cell Lung Disease. An Official American Thoracic Society Workshop Report. Ann Am Thorac Soc. 2019 Sep;16(9):e17-e32. PMID: 31469310
7. Wright N, Voshtina E, George G, Singavi A, Field J. Cryoglobulinemic vasculitis with interruption of ibrutinib therapy for chronic lymphocytic leukemia (CLL). Int J Hematol. 2019 Dec;110(6):751-755. PMID: 31494832
8. Clayton-Jones D, Matthie N, Treadwell M, Field JJ, Mager A, Sawdy R, George Dalmida S, Leonard C, Koch KL, Haglund K. Social and Psychological Factors Associated With Health Care Transition for Young Adults Living With Sickle Cell Disease. J Transcult Nurs. 2019 Dec 31:1043659619896837. doi: 10.1177/1043659619896837. [Epub ahead of print] PMID: 889479

Elodie

Elodie struggled with sickle cell disease while growing up. She would experience a crisis at least twice a month, forcing her to spend days at a time in the hospital. As an adult, Elodie receives blood transfusions every few weeks that help keep her strong and active. She has since obtained her master's degree and has experienced little to no sickle cell crises.

"The blood transfusions are making a really big difference. I have a very good quality of life. I exercise 3 days a week – and am able to swim every Friday."

Jerome Gottschall, MD

Senior Medical Director, Versiti
Senior Investigator, Blood Research Institute, Versiti
Professor of Pathology, Department of Pathology, Medical College of Wisconsin
MD, Ohio State University College of Medicine, 1974
Started at Versiti: 1979

Research Interests

Dr. Gottschall is a Co-Principal Investigator on the Recipient Epidemiology and Donor Evaluation Study-III (REDS III) sponsored by the National Heart Lung and Blood Institute in which Versiti is one of several participating organizations. REDS III includes studies on blood safety, blood availability, HIV transmission and other transfusion-related studies. REDS III utilizes large donor, component and recipient databases to help answer important transfusion-related questions. Among subjects to be studied are alloimmunization (immunization against transfused blood cells), impact of blood donation on donor iron levels, benefits of red cell transfusion in the elderly, and transfusion of various blood products in distinct clinical settings. Dr. Gottschall also is concerned with the clinical aspects of immune hemolytic anemia and in the status of iron levels in repeat blood donors. An avid athlete, Dr. Gottschall says sports have taught him three critical skills that he uses in his medical work and throughout his life: discipline; persistence; and setting goals.

Awards, Honors and Service

- Member, College of American Pathologists
- Member, American Association of Blood Banks (AABB)
- Member, American Society of Hematology

Publications

1. Bialkowski W, Blank RD, Zheng C, Gottschall JL, Papanek PE. Impact of frequent apheresis blood donation on bone density: A prospective, longitudinal, randomized, controlled trial. Bone Rep. 2018 Dec 12;10:100188. doi: 10.1016/j.bonr.2018.100188. eCollection 2019 Jun. PMID: 30581893
2. Bialkowski W, Tan S, Mast AE, Kiss JE, Kor D, Gottschall J, Wu Y, Roubinian N, Triulzi D, Kleinman S, Choi Y, Brambilla D, Zimrin A; NHLBI Recipient Epidemiology and Donor Evaluation (REDS)-III Study. Equivalent inpatient mortality among direct-acting oral anticoagulant and warfarin users presenting with major hemorrhage. Thromb Res. 2019 Nov 25;185:109-118. PMID: 31794885
3. Davis CS, Milia D, Gottschall JL, Weigelt JA. Massive transfusion associated with a hemolytic transfusion reaction: necessary precautions for prevention. Transfusion. 2019 Aug;59(8):2532-2535. PMID: 31241167
4. Jones AR, Patel RP, Marques MB, Donnelly JP, Griffin RL, Pittet JF, Kerby JD, Stephens SW, DeSantis SM, Hess JR, Wang HE...Gottschall JL; PROPPR Study Group. Older Blood Is Associated With Increased Mortality and Adverse Events in Massively Transfused Trauma Patients: Secondary Analysis of the PROPPR Trial. Ann Emerg Med. 2019 Jun;73(6):650-661. PMID: 30447946
5. Karafin MS, Tan S, Tormey CA, Spencer BR, Hauser RG, Norris PJ, Roubinian NH, Wu Y, Triulzi DJ, Kleinman S, Gottschall JL, Hendrickson JE. Prevalence and risk factors for RBC alloantibodies in blood donors in the Recipient Epidemiology and Donor Evaluation Study-III (REDS-III). Transfusion. 2019 Jan;59(1):217-225. PMID: 0427537

Matthew Karafin, MD

Medical Director, Medical Sciences Institute, Versiti
Associate Investigator, Blood Research Institute, Versiti
Associate Professor of Pathology, Medical College of Wisconsin
MD, Carver College of Medicine, University of Iowa, Iowa City, IA, 2007
Transfusion Medicine Fellowship, Johns Hopkins Hospital, 2011
Anatomical and Clinical Pathology Residency, Johns Hopkins Hospital, 2012
MS, Medical College of Wisconsin, Milwaukee, WI, 2015
Started at Versiti: 2012

Research Interests

Dr. Karafin's research interests include the use of red cell transfusion for patients with sickle cell disease, etiology and prevention of red cell alloimmunization, benefits and risks of red cell storage for patients with sickle cell disease, benefits and risks of red cell transfusions in the elderly, and the etiology and prevention of transfusion reactions.

Awards, Honors and Service

- Member, American Society for Apheresis (ASFA): Chair, Education Committee
- Member, American Association of Blood Banks (AABB): Chair, Molecular Testing Accreditation Program Unit Committee
- Member, College of American Pathologists (CAP)
- Member, American Society for Clinical Pathology (ASCP)
- Member, Alpha Omega Alpha (AOA)

Funding

HHSN268201100003I (Mast) 03/15/2011 – 03/14/2020 NIH/NHLBI $511,520 "Recipient Epidemiology and Donor Evaluation Study III (REDS III)" (Co-I)

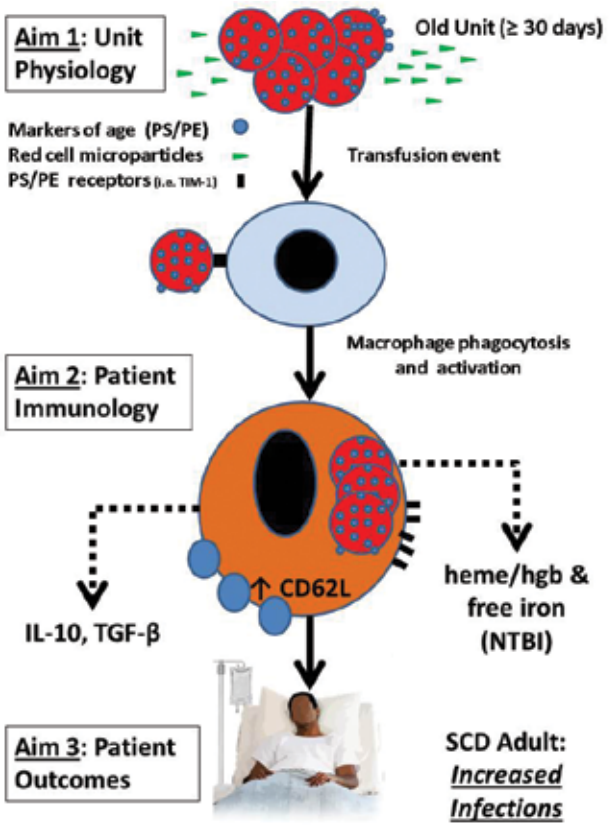
HHSN26819HB00003R (Mast) 04/01/2019 – 03/31/2026 NIH/NHLBI $1,000,038 "Recipient Epidemiology and Donor Evaluation Study-IV-Pediatric (REDS-IV-P)" (Co-I)

1R01HL148151-01 (MPI: Spitalnik, D'Alessandro, Karafin, Zimring) 09/25/2019 – 08/31/2023 NIH/NHLBI $173,014 (Versiti) "The Impact of Oxidative Stress on Erythocyte Biology" (Co-PD/PI)

1K23HL136787-01A1 (Karafin) 12/15/2017 – 12/15/2022 NIH/NHLBI $165,000 "The Effects of Older Red Cell Units in Adults with Sickle Cell Disease"(PI)

Publications

1. Karafin MS, Bruhn R, Roubinian NH, Chowdhury D, Qu L, Snyder EL, Murphy EL, Brambilla D, Cable RG, Hilton JF, St Lezin E; NHLBI Recipient Epidemiology and Donor Evaluation (REDS)-III Study. The impact of recipient factors on the lower-than-expected hemoglobin increment in transfused outpatients with hematologic diseases. Transfusion. Aug;59(8):2544-2550. PMID: 31270827
2. Karafin MS, Field JJ. The controversial role of red cell transfusions for sickle cell pain. Curr Opin Hematol. 2019 Nov;26(6):442-447. PMID: 31567433
3. Karafin MS, Francis RO. Impact of G6PD status on red cell storage and transfusion outcomes. Blood Transfus. 2019 Jul;17(4):289-295. Review. PMID: 31385801



Anand Padmanabhan, MD, PhD

Investigator, Blood Research Institute, Versiti
Medical Director, Therapeutic Services, Versiti
Associate Professor of Pathology, Medical College of Wisconsin
MD, Thanjavur Medical College, Thanjavur, TN, India, 2000
PhD, Brown University, 2006
Started at Versiti: 2010

Research Interests

Heparin is widely used to prevent and treat thrombosis, but some patients given this otherwise useful anticoagulant become immunized and produce antibodies that cause thrombocytopenia (heparin-induced thrombocytopenia, HIT). Many affected individuals experience thrombosis, which can be life threatening. Dr. Padmanabhan is engaged in studies to define the properties of heparin-induced antibodies that are most likely to cause thrombosis. Findings made are expected to advance the understanding of HIT and to improve laboratory diagnosis and treatment of this dangerous disorder.

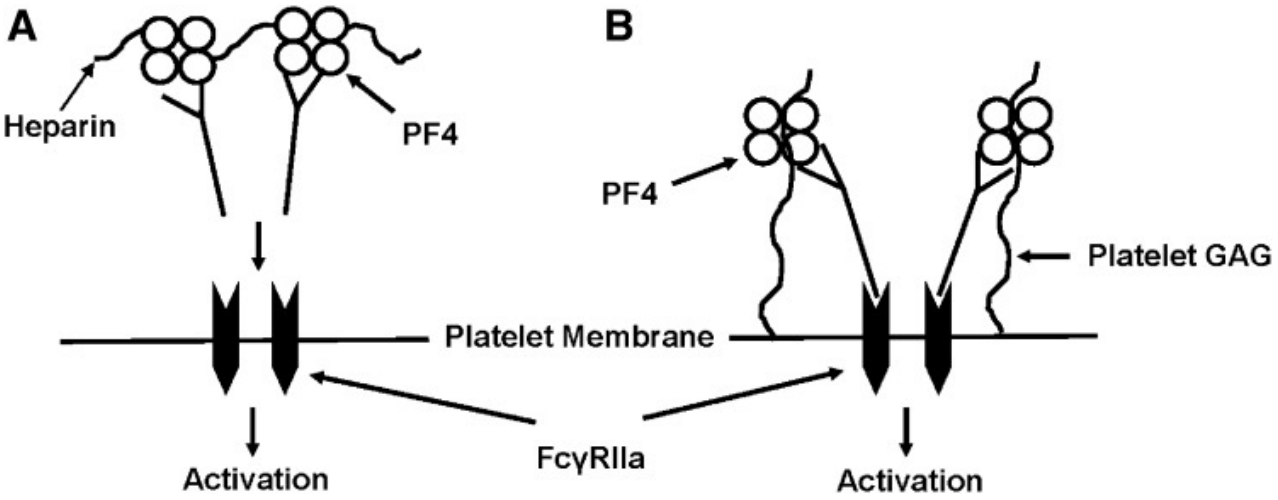
Awards, Honors and Service

- Editor, Journal of Clinical Apheresis (JCA), 08/2015-present
- Chair, JCA Writing Committee on the Use of Apheresis in Human Disease, (2016-present)
- Member, American Association of Blood Banks (AABB)
- Member, American Society for Apheresis (ASFA)
- Member, American Society of Hematology (ASH)

Publications:

1. Chawla D, Saad E, Khairi T, Padmanabhan A. Severe persistent heparin-induced thrombocytopenia in a renal transplant patient. Thromb Res. 2019 Oct 21;183:106-107. PMID: 31677588
2. Irani M, Siegal E, Jella A, Aster R, Padmanabhan A. Use of intravenous immunoglobulin G to treat spontaneous heparin-induced thrombocytopenia. Transfusion. 2019 Mar;59(3):931-934. PMID: 30556588
3. Padmanabhan A. New IDEaS for HIT treatment, anyone? Blood. 2019 May 30;133(22):2355-2356. PMID: 31147374
4. Padmanabhan A, Connelly-Smith L, Aqui N, Balogun RA, Klingel R, Meyer E, Pham HP, Schneiderman J, Witt V, Wu Y, Zantek ND, Dunbar NM, Schwartz GEJ. Guidelines on the Use of Therapeutic Apheresis in Clinical Practice - Evidence-Based Approach from the Writing Committee of the American Society for Apheresis: The Eighth Special Issue. J Clin Apher. 2019 Jun;34(3):171-354. PMID: 31180581

Figure: Model showing how we think HIT antibodies actually cause platelet activation in HIT (from Padmanabhan et al, Blood 2015)



Glycomics Center

The Glycomics Center, led by faculty member Dr. Karin Hoffmeister, opened its doors in 2016 for the Blood Research Institute. Analogous to Genomics and Proteomics, Glycomics focuses on defining the structures and functions of complex carbohydrates (sugars), as found in glycoproteins, glycolipids, and glycosaminoglycans.

Complex carbohydrates are important in many physiological processes and alterations in glycosylation are associated with vast numbers of blood related and unrelated diseases and disorders. The specific focus of the Center is to harness genomic with glycomic approaches with an emphasis on exploring transcriptional and epigenetic regulatory mechanisms of carbohydrate synthesis in health and disease. The data will help to understand and predict molecular mechanisms of carbohydrate expression and recognition by proteins important in human biology and disease. The Center will bring together scientists at Versiti and other institutions to understand the role that sugars play in biology.

Karin Hoffmeister, MD

Hauske Family Endowed Chair in Glycobiology
Senior Investigator, Blood Research Institute, Versiti
Professor of Biochemistry, Medical College of Wisconsin
Director of Translational Glycomics Center
MD, Technical University of Aachen, Aachen, Germany, 1993
Doctor of Medicine, Doctoral Research Program, Technical University of Aachen, 1995
Started at Versiti: 2016

Research Interests

Dr. Hoffmeister investigates how glycans regulate hematopoiesis and end-effector blood cells, including platelets. The general theme of Dr. Hoffmeister's research is to better understand the role of carbohydrates in hematopoietic stem cells, megakaryocytes and platelet function, survival, and interaction with other blood cells. Carbohydrate biosynthesis in nucleated cells is a highly regulated process involving several hundred glycosyltransferases. Correct glycan biosynthesis depends on the correct architecture and topology of the endoplasmic reticulum (ER) and Golgi apparatus. During maturation, differentiation and inflammation programmed remodeling of cell surface glycans takes place by the regulated expression of specific glycosyltransferases to regulate different biological functions. Dr. Hoffmeister's studies expand toward defining glycosyltransferases cell-specific transcriptional regulatory mechanisms during hematopoiesis to combine phenotypic surface carbohydrate expression with genomic and epigenetic data in hematopoietic cells.

Awards, Honors and Service

- Member, Hemostasis and Thrombosis Study Section
- Member, Transfusion Medicine Study Section, special panel
- Member, American Society of Hematology

Funding

RO1 HL089224-10 "Carbohydrate Mediated Platelet Clearance"

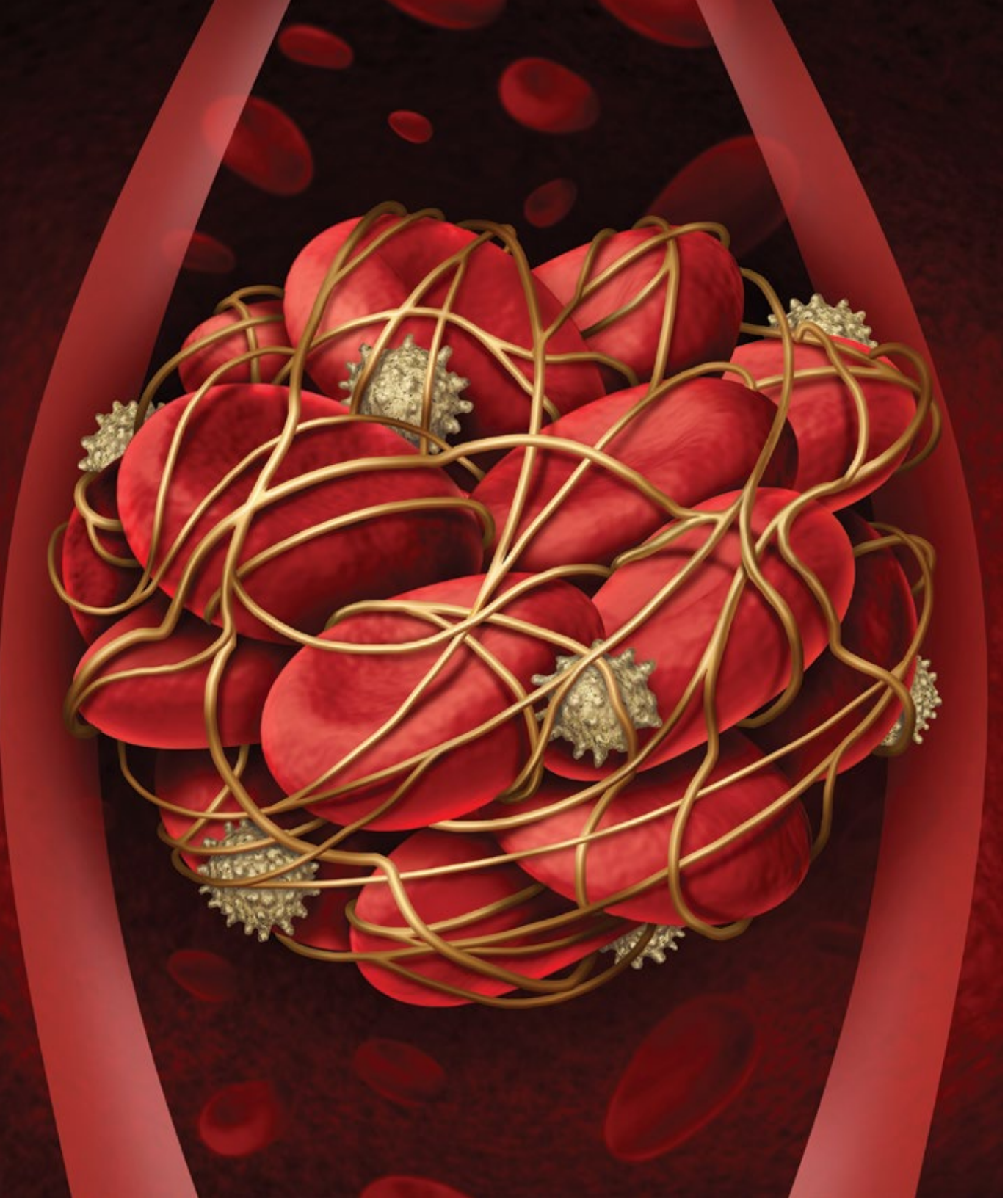
PO1 HL 107146-06 "Biosynthesis and Function of Lactosaminyl Glycans in Hematopoiesis"

R01 HL089224-04 "VWF- Mechanisms of Regulation"

K12 HL141954-01 "Glycans in Blood Hematopoiesis and Disease"

Publications

1. Irons EE, Lee-Sundlov MM, Zhu Y, Neelamegham S, Hoffmeister KM, Lau JT. B cells suppress medullary granulopoiesis by an extracellular glycosylation-dependent mechanism. Elife. 2019 Aug 13;8. pii: e47328. doi: 10.7554/eLife.47328. PMID: 31408003



Thrombosis, Hemostasis & Vascular Biology

The Thrombosis, Hemostasis and Vascular Biology Program is concerned with cellular and molecular mechanisms of normal blood clotting, pathological thrombosis, and events impacting the integrity of the blood vessels that transport blood throughout our body.

Studies conducted in the laboratories of the BRI range from basic scientific investigations of blood coagulation and platelet function, to the pathophysiology, treatment, and diagnosis of bleeding and clotting disorders.

Thomas C Abshire, MD

Executive Vice President, Medical Sciences Institute and Chief Medical Officer, Versiti
Senior Investigator, Blood Research Institute, Versiti
Professor of Pediatrics, Medicine and the CTSI, Medical College of Wisconsin
MD, Tulane University School of Medicine, 1979
Pediatrics, David Grant USAF Medical Center, Travis AFB, CA 1979-82
Pediatric Hematology/Oncology, University of Colorado Health Science Center, 1985-88
Started at Versiti: 2009

Other Significant Contributor/Key Personnel for the Administrative Core A (Clinical Acquisition Core) for Dr. Robert Montgomery's PPG; "Zimmerman Program on the Biology of VWD"

Awards, Honors, and Service

- Member, American Society of Hematology (ASH) and International Society of Thrombosis and Haemostasis (ISTH)
- Past President, Hemostasis and Thrombosis Research Society (HTRS)
- ASH Committee on Training
- Past Chair, American Thrombosis and Hemostasis Network (ATHN)
- Co-Chair, SSC Scientific Subcommittee on VWF, ISTH
- Best Doctors in America 2009-2018
- CTSI of SE WI Board of Directors

Funding

Past Funding

5R01HL112614-05 Montgomery/Abshire (Multiple PIs) 12/14/2013 – 11/30/2019 NIH/NHLBI "Comparative Effectiveness in the Diagnosis of VWD."

Current Grant

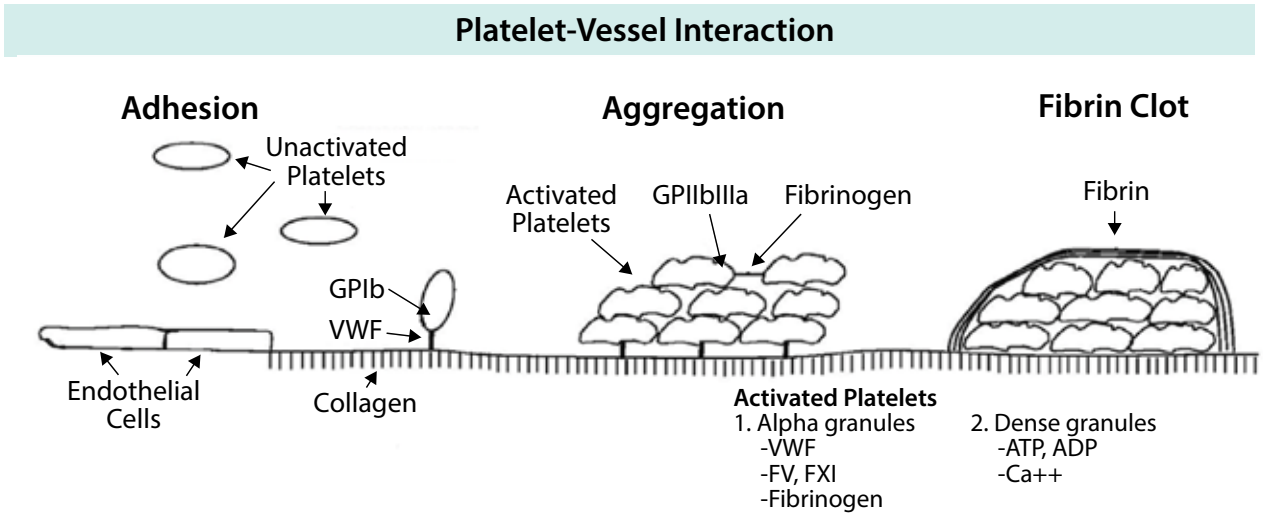
P01HL144457 (NIH/NHLBI) Other Significant Contributor 03/15/2019 – 02/29/2024 "Zimmerman Program on the Biology of VWD."

Publications

1. Warren BB, Jacobson L, Kempton C, Buchanan GR, Recht M, Brown D, Leissinger C, Shapiro AD, Abshire TC, Manco-Johnson MJ; "Joint Outcome Study Group Investigators. Factor VIII prophylaxis effects outweigh other hemostasis contributors in predicting severe haemophilia A joint outcomes". Haemophilia 2019 Sep;25(5):867-875.PMID: 31115111

Research Interests

One of Dr. Abshire's major research interests involves conducting clinical trials in patients with bleeding and thrombotic disorders with the aim of defining disease characteristics and evaluating new approaches to treatment. A recent focus is the evaluation of mild bleeding conditions in both children and adults, particularly those affected by von Willebrand Disease (VWD). With Robert Montgomery, MD, Dr. Abshire and a team of investigators from 12 centers across North America have just finished the sixth year (no cost extension) of a study funded by the NIH entitled "Comparative Effectiveness in the Diagnosis of VWD" which is focusing on new diagnoses of VWD and how to better define this bleeding disorder from a clinical, laboratory and molecular basis. Currently, he is



Lisa Baumann Kreuziger, MD, MS

Medical Director, Medical Sciences Institute; Associate Investigator, Blood Research Institute, Versiti
Associate Professor of Medicine, Division of Hematology/Oncology, Medical College of Wisconsin
MD, University of Wisconsin School of Medicine and Public Health, 2006
Started at Versiti: 2013

Research Interests

Venous thromboembolism occurs in more than a half million Americans every year. Anticoagulation after venous thromboembolism (VTE) can prevent recurrence but is associated with a risk of bleeding. Dr. Baumann-Kreuziger has developed a research network of >100 clinical researchers called the Venous thromboEmbolism Network US (VENUS). Versiti has supported the development of a coordinating center to allow clinical research in VTE to be performed faster, answer important questions, and help patients with VTE. Dr. Baumann Kreuziger also has an interest in how to prevent bleeding and thrombosis in patients requiring mechanical circulatory support devices for heart failure. Lastly, Dr. Baumann Kreuziger is involved with the Recipient Epidemiology and Donor Evaluation-III study.

Awards, Honors and Service

- American Society of Hematology Thrombophilia Guideline panel
- American College of Chest Physicians Antithrombotic Therapy for VTE Disease Guideline Panel Member

Funding

"Evaluating thrombi composition and persistent coagulation activation in the pathophysiology of left ventricular assist device (LVAD) thrombosis" NIH $80,000 (PI) 7/2015 – 6/2019

"Direct Oral Anticoagulants (DOACs) versus LMWH +/- warfarin for VTE in cancer: A Randomized Effectiveness Trial (CANVAS)" Alliance Foundation (PI) 05/27/2017 – present

"Post-Thrombotic Syndrome and Predictors of Recurrence in Catheter-Related Thrombosis"

Recipient Epidemiology and Donor Evaluation Study III (REDS III), Contract NIH/NHLBI: HHSN268201100003I (PI: Mast) 03/15/2011 – 03/14/2020.

Publications

1. Feih JT, Juul JJ, G Rinka JR, Baumann Kreuziger LM, Pagel PS, Tawil JN. Adequacy of hemostatic resuscitation improves therapeutic efficacy of recombinant activated factor VII and reduces reexploration rate for bleeding in postoperative cardiac surgery patients with refractory hemorrhage. Ann Card Anaesth. 2019 Oct-Dec;22(4):388-393. PMID: 31621674
2. Held N, Jung B, Sommervold L, Sing S, Kreuziger LB. Patient safety indicator-12 rarely identifies problems with quality of care in perioperative venous thromboembolism. Journal of Hospital Medicine, 2019;14, J Hosp Med. 2019 Nov 20;14:E1-E6.
3. Klein E and Baumann Kreuziger L (2019). Acquired von Willebrand Syndrome with Mechanical Circulatory Support. In D. Joyce and L. Joyce, Mechanical Circulatory Support: Principles and Applications (269-276). New York, NY, Oxford University Press.

Magdalena Chrzanowska, PhD, FAHA

Investigator, Blood Research Institute, Versiti
Associate Professor in Pharmacology and Toxicology, Medical College of Wisconsin
Research Member, Medical College of Wisconsin Cancer Center
Medical College of Wisconsin Cardiovascular Center
PhD, University of North Carolina at Chapel Hill, 1996
MSc, Jagiellonian University, Krakow, Poland, 1991
Started at Versiti: 2005

Research Interests

Endothelial cells (EC) cover the inner surface of blood vessels and perform many critical functions, such as preventing leakage of blood cells and plasma from the circulation, preventing inappropriate blood clotting, regulating selective transfer of cells and substances into and out of blood vessels, and maintaining the correct blood pressure.

Importantly, EC can adapt their functions to their environment, by sensing blood flow and the presence of inflammatory signals.

Dr. Chrzanowska studies how a protein, designated Rap1, regulates the response of EC to changes in blood flow and inflammation. Her work helps understand the processes causing hypertension and the narrowing and hardening of the blood vessel wall in atherosclerotic disease. In 2016, Dr. Chrzanowska succeeded in obtaining the renewal of grant support from the National Institutes of Health for her work.

The complications of cardiovascular disease remain major killers of the American population. The maintenance of normal cardiovascular function is critically dependent on vascular endothelium – cells that line blood vessels. Dr. Chrzanowska's research is focused on understanding molecular mechanisms underlying critical endothelial cell functions, such as preventing blood leakage, selective transfer of cells and substances to and from the blood stream, regulation of blood pressure, and restorative and pathogenic new blood vessel growth.

Dr. Chrzanowska's recent research revealed new mechanisms through which endothelial cells respond to the flow of blood and how defects in these responses contribute to atherosclerosis in an in vivo disease model. These are the first necessary steps in developing new strategies to restore endothelial function to prevent the progression of atherosclerosis. Furthermore, these studies provided novel insights into mechanisms through which endothelial cells maintain barrier under normal conditions. Importantly, Dr. Chrzanowska's studies identified potential novel therapeutic targets for pathological vascular hyper-permeability associated with early diabetes.

Awards, Honors and Service

- American Heart Association, ATVB Council – 2020 AHA Scientific Sessions Programming Committee 2019
- Affiliate Member, Clinical and Translational Science Institute of SE Wisconsin 2011-present
- Member, Medical College of Wisconsin Cardiovascular Center: Atherosclerosis, Thrombosis and Vascular Biology Signature Program 2011- present
- Member, Medical College of Wisconsin Cancer Center, Cancer Cell Biology Research Program - 2011 - Present
- NIH Vascular Cell and Molecular Biology Study Section – 2015-2019

Funding

NIH/NHLB R01 - HL111582-07 Chrzanowska-Wodnicka, M, (PI) 4/16/12 - 6/30/20 "Rap1 in endothelial homeostasis"

Publications

1. Ragunathrao VAB, Anwar M, Akhter MZ, Chavez A, Mao DY, Natarajan V, Lakshmikanthan S, Chrzanowska-Wodnicka M, Dudek AZ, Claesson-Welsh L, Kitajewski JK, Wary KK, Malik AB, Mehta D. Sphingosine-1-Phosphate Receptor 1 Activity Promotes Tumor Growth by Amplifying VEGF-VEGFR2 Angiogenic Signaling. Cell Rep. 2019 Dec 10; 29(11). PMID: 6927555.

Veronica H. Flood, MD

Associate Professor of Pediatrics, Division of Hematology/Oncology, Medical College of Wisconsin
Associate Medical Director, Comprehensive Center for Bleeding Disorders, Medical Sciences Institute
Associate Investigator, Blood Research Institute, Versiti
MD, Tufts University School of Medicine, 1999
Started at Versiti: 2016

Research Interests

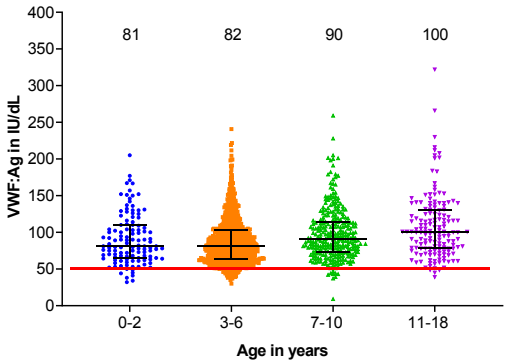
Dr. Flood is a pediatric hematologist and researcher at the Medical College of Wisconsin in Milwaukee. She received her medical degree from Tufts University School of Medicine and went on to complete a residency in pediatrics at Phoenix Children's Hospital and a fellowship in pediatric hematology/oncology at Oregon Health and Science University.

She began her research career as an undergraduate studying primate genetics in the Department of Anthropology at Harvard University. Her academic career in hemostasis research includes work on the biology of von Willebrand factor (VWF). Dr. Flood is interested in how VWF interacts with two of its main partners, platelet GPIb and collagen. Since collagen is exposed at sites of blood vessel injury, the VWF-collagen interaction is an important component of hemostasis. She also is interested in the genetics of von Willebrand disease (VWD). Through collaboration with Dr. Robert Montgomery and the Zimmerman Program for the Molecular and Clinical Biology of VWD, Dr. Flood has worked on characterizing genetic changes in VWD, with particular attention to variants that affect platelet and collagen binding. Dr. Flood has been funded by the National Heart Lung and Blood Institute since 2010, initially through a K08 award and subsequently transitioned to independent funding through an R01 grant.

Awards, Honors and Service

- Standing member, NIH Study Section, National Heart, Lung, and Blood Institute, Mentored Patient-Oriented Research review panel
- Vice-Chair, Mentored Research Award Committee, Hemostasis and Thrombosis Research Society

VWF levels lower in younger children.



- Co-Chair, Von Willebrand Disease Management Guideline Panel, ASH/ISTH/NHF/WFH
- Outstanding Medical Student Teacher, Medical College of Wisconsin, 2017-2018

Funding

R01 HL126810 "Mechanism of Type 4 Collagen Interactions with Von Willebrand Factor" (PI)

R01 HL112614 "Comparative Effectiveness in the Diagnosis of VWD" (Co-I)

Publications

1. Flood VH, Garcia J, Haberichter SL. The role of genetics in the pathogenesis and diagnosis of type 1 Von Willebrand disease. Curr Opin Hematol. 2019 Sep;26(5):331-335. PMID: 31261173
2. Slobodianuk TL, Kochelek C, Foeckler J, Kalloway S, Weiler H, Flood VH. Defective collagen binding and increased bleeding in a murine model of von Willebrand disease affecting collagen IV binding. J Thromb Haemost. 2019 Jan;17(1):63-71. PMID: 30565388
3. Weyand AC, Flood VH, Shavit JA, Pipe SW. Efficacy of emicizumab in a pediatric patient with type 3 von Willebrand disease and alloantibodies. Blood Adv. 2019 Sep 24;3(18):2748-2750. PMID: 31540901

* First author a trainee.

Kenneth Friedman, MD

Senior Medical Director Hemostasis Reference Lab, Diagnostic Labs, Versiti
Investigator, Blood Research Institute, Versiti
Professor of Internal Medicine and Pathology, Medical College of Wisconsin
MD, SUNY Upstate Medical University, Syracuse, NY 1980
Started at Versiti: 1997

Research Interests

Thrombotic microangiopathies are a collection of diseases characterized by formation of platelet/protein aggregates that obstruct the microcirculation, resulting in multi-organ dysfunction. Microthrombi in thrombotic thrombocytopenic purpura are rich in von Willebrand factor as a consequence of deficiency of the von Willebrand factor control enzyme ADAMTS13. Alternatively in atypical hemolytic uremic syndrome, the microthrombi are rich in fibrin as a consequence of disordered complement regulation and endothelial cell injury. The current focus of Dr. Friedman's research of thrombotic microangiopathies is the evaluation of patient plasma and genetic samples in order to identify patterns of disease, underlying risk factors and prognostic markers. Dr. Friedman's other area of interest relates to utilization of plasma and genetic markers to better understand the mechanisms underlying the bleeding that occurs in patients with defects of von Willebrand factor.

Awards, Honors and Service

- Co-Director, National Marrow Donor Program – Versiti Branch 2017
- Best Doctors in America 2018
- Medical Director of the Apheresis Center for the NMDP site in Milwaukee 2018
- Ad hoc Reviewer, Journal of Thrombosis and Hemostasis, Blood, and Transfusion and Transplantation 2019

Publications

1. Horn C, Négrier C, Kalina U, Seifert W, Friedman KD. Performance of a recombinant fusion protein linking coagulation factor IX with recombinant albumin in one-stage clotting assays. J Thromb Haemost. 2019 Jan;17(1):138-148. PMID: 30418692
2. Scully M, Cataland SR, Peyvandi F, Coppo P, Knöbl P, Kremer Hovinga JA, Metjian A, de la Rubia J, Pavenski K, Callewaert F, Biswas D, De Winter H, Zeldin RK; HERCULES Investigators...Friedman, K. Caplacizumab Treatment for Acquired Thrombotic Thrombocytopenic Purpura. N Engl J Med. 2019 Jan 24;380(4):335-346. PMID: 30625070
3. van Dorland HA, Mansouri Taleghani M, Sakai K, Friedman KD, George JN, Hrachovinova I, Knöbl PN, von Krogh AS, Schneppenheim R, Aebi-Huber I, Bütikofer L, Largiadèr CR, Cermakova Z, Kokame K, Miyata T, Yagi H, Terrell DR, Vesely SK, Matsumoto M, Lämmle B, Fujimura Y, Kremer Hovinga JA; Hereditary TTP Registry. The International Hereditary Thrombotic Thrombocytopenic Purpura Registry: Key findings at enrollment until 2017. Haematologica. 2019 Oct;104(10):2107-2115. PMID: 30792199

Alan

At 5 years old, Alan was diagnosed with hemophilia. Doctors did not expect him to live much longer than 40 years. He was eventually introduced to Versiti's Comprehensive Center for Bleeding Disorders (CCBD) and has been able to persevere past hemophilia. Now Alan is in his 80's, living a happy and healthy life with his wife Evelyn.

"I wouldn't be alive today without the CCBD."

Sandra Haberichter, PhD

Director of Hemostasis Reference Laboratory, Versiti
Senior Investigator, Blood Research Institute, Versiti
Associate Professor of Pediatrics, Medical College of Wisconsin
PhD, University of Wisconsin-Milwaukee, 1998
Fellowship, Blood Research Institute, Versiti, 1998-2003
Started at Versiti: 1998

Research Interests

The plasma protein von Willebrand factor (VWF) plays a critical role in enabling blood platelets to interact with damaged blood vessels and stop bleeding. Genetically determined abnormalities of VWF function and synthesis cause von Willebrand Disease (VWD), a source of abnormal bleeding that affects about one percent of the general population. Dr. Haberichter's work is aimed at characterizing various genetic defects that cause VWD and defining how these defects affect the structure and function of the large, highly complex VWF molecule. Recent findings have shown that low VWF levels in patients with a sub-type of VWD, designated Type 1C, decrease VWF levels by shortening the survival of VWF in the circulation and have led to a novel laboratory assay to diagnose this condition. Her current work is aimed at defining the molecular basis for accelerated clearance of VWF in patients with this form of VWD. Knowledge gained in these studies is expected to improve laboratory diagnosis and treatment of this common bleeding disorder.

Awards, Honors and Service

- Member, American Society of Hematology 2019
- Chair, ISTH SSC scientific committee on von Willebrand Factor 2019
- Member, International Society on Thrombosis and Haemostasis 2019

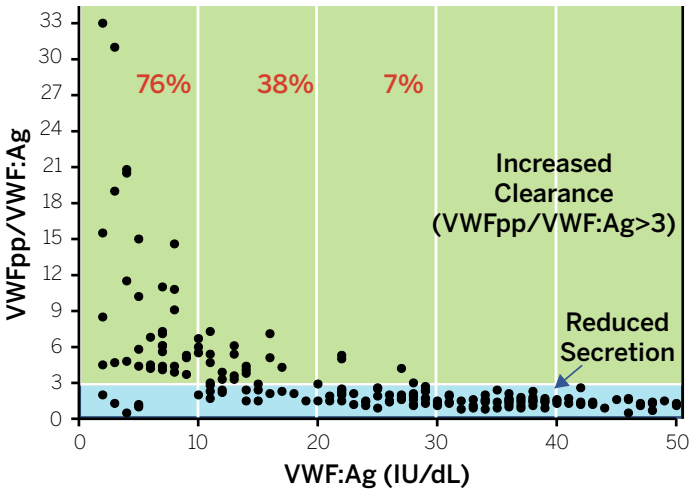
Funding

R01 HL136430 "VWF- Mechanisms of Regulation"

P01 HL144457 Project 2 – "Molecular Impact of Carbohydrates on VWF Biology"

Publications

1. Flood VH, Garcia J, Haberichter SL. The role of genetics in the pathogenesis and diagnosis of type 1 Von Willebrand disease. Curr Opin Hematol. 2019 Sep;26(5):331-335. PMID: 31261173
2. Hubbard AR, Haberichter SL; SSC subcommittee on von Willebrand factor of the ISTH. Establishment of an International Reference Reagent for standardization of von Willebrand factor binding to recombinant glycoprotein Ib (VWF:GPIbM and VWF:GPIbR): Official Communication of the SSC. J Thromb Haemost. Jun;17(6):1003-1005. PMID: 31102313
3. Husseinzadeh HD, Haberichter S. Evidence-Based Minireview: Perioperative management of the VWD patient at elevated thrombotic risk. Hematology Am Soc Hematol Educ Program. 2019 Dec 6;2019(1):601-603. PMID: 31808869
4. Sharma R, Haberichter SL. New advances in the diagnosis of von Willebrand disease. Hematology Am Soc Hematol Educ Program. 2019 Dec 6;2019(1):596-600. PMID: 31808831



Increased VWF clearance is prevalent in moderately severe type 1 VWD.

VWFpp/VWF:Ag < 3 predicts reduced synthesis/secretion phenotype (blue).

VWFpp/VWF:Ag > 3 predicts increased plasma VWF clearance (green).

76% of subjects with VWF:Ag ≤ 10 IU/dL and 38% of subjects with VWF:Ag = 11-20 IU/dL have an increased VWF clearance phenotype (type 1C).

Shawn Jobe, MD, PhD

Associate Investigator, Medical Sciences Institute/Blood Research Institute, Versiti
Associate Professor, Department of Pediatrics and Medicine, Medical College of Wisconsin
PhD, Medical College of Wisconsin, 1998
MD, Medical College of Wisconsin, 1999
Started at Versiti: 2013

Research Interests

Platelets are required to stop bleeding, but inappropriate platelet adhesion and activation results in thrombosis. Dr. Jobe's group is working to understand how platelet activation is regulated. They have identified a novel platelet mitochondrial mechanism that transforms the platelet's function from proaggregatory to procoagulant. Work in Dr. Jobe's lab currently is focused on understanding how platelet mitochondrially-mediated events are regulated and how they function to regulate hemostasis and thrombosis. Changes in mitochondrial metabolism are linked closely with many diseases associated with aging including diabetes, atherosclerosis and hypertension. Insights gained from these studies are expected to provide novel avenues for the treatment and prevention of thrombosis in aging-related diseases. Platelet procoagulant activity also is important in the prevention of bleeding. In other work, researchers in Dr. Jobe's lab are investigating how procoagulant platelets might work to prevent bleeding in patients with severe hemophilia.

Awards, Honors and Service

- Standing member American Heart Association Thrombosis/Hemostasis Study Section
- National Hemophilia Foundation Clinical Fellowship Advisory Board
- Member International Society on Thrombosis and Hemostasis

Publications

1. Kholmukhamedov A, Jobe S. Platelet respiration. Blood Adv. 2019 Feb 26;3(4):599-602. PMID: 30792189
2. Kholmukhamedov A, Jobe S. Procoagulant Platelets Get Squeezed to Define the Boundaries of the Hemostatic Plug. Arterioscler Thromb Vasc Biol. 2019 Jan;39(1):5-6. PMID: 30586335

Yan-Qing Ma, PhD

Investigator, Blood Research Institute, Versiti
PhD, Chinese Academy of Sciences, 2004
Started at Versiti: 2011

Research Interests

Integrins comprise an extensive family of cell membrane proteins that are essential for cell-cell communication and signaling. In blood platelets, the integrin allb/ß3 undergoes complex intracellular and extracellular structural changes that enable these cells to adhere to damaged blood vessels and to each other to control bleeding. This process must be carefully regulated to enable hemostasis to be achieved without causing a clot to be propagated inappropriately. Dr. Ma's current work is aimed at understanding intracellular signaling cascades in platelets that control structural changes in integrin allb/ß3 during platelet activation. A particular goal is to define how kindlin-3, a key integrin activator in platelets, coordinates with binding partners and creates a signaling network that regulates the platelet activation process. An important objective is to identify novel inhibitors of platelet function that can be useful for treatment and prevention of thrombosis.

Awards, Honors and Service

- Member, American Heart association
- Member, American Society of Hematology
- Member, International Society on Thrombosis and Hemostasis

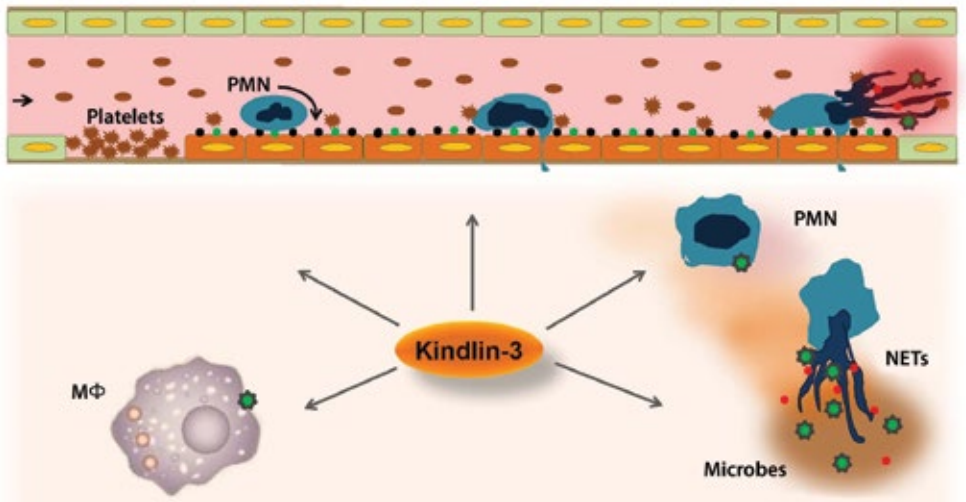
Funding

R01 HL131654 "Kindlin-3 Signaling in Blood Cells"

Publications

1. Yan Y, Yang H, Hu X, Zhang Z, Ge S, Xu Z, Gao J, Liu J, White GC, Ma YQ. Kindlin-3 in platelets and myeloid cells differentially regulates deep vein thrombosis in mice. Aging (Albany NY). 2019 Aug 31;11(17):6951-6959. PMID: 31477636
2. Gao J, Bao Y, Ge S, Sun P, Sun J, Liu J, Chen F, Han L, Cao Z, Qin J, White GC, Xu Z, Ma YQ. Sharpin suppresses ß1-integrin activation by complexing with the ß1 tail and kindlin-1. Cell Commun Signal. 2019 Aug 20;17(1):101. PMID: 31429758

Kindlin-3 signaling in platelets and neutrophils



Lynn Malec, MD, MSc

Medical Director, Comprehensive Center for Bleeding Disorders, Medical Sciences Institute
Associate Investigator, Blood Research Institute, Versiti
Assistant Professor of Medicine, Division of Hematology/Oncology, Medical College of Wisconsin
MD, UW Madison School of Medicine and Public Health, 2006
MSc, University of Pittsburgh, Institute for Clinical Research Education, 2014
Started at Versiti: 2016

Research Interests

Dr. Lynn Malec developed an interest in hemostasis during her Internal Medicine and Pediatrics residency at the University of Pittsburgh. This interest flourished during her fellowship in Pediatric Hematology/Oncology at Children's Hospital of Pittsburgh during which time she gained further expertise in the care of, and research involving, patients with congenital bleeding disorders across the age spectrum. During her fellowship, she pursued a Master's of Science in Clinical Research through the University of Pittsburgh Institute for Clinical Research Education. This rigorous training furthered her interest in clinical research involving patients with bleeding disorders.

Dr. Malec's current research interests involve the investigation of inhibitor prevention and eradication in patients with hemophilia and the impact of prophylactic use in this patient population. She currently is investigating the role that recombinant factor VIII Fc fusion protein (rFVIIIFc) has in immune tolerance induction and is conducting a multi-site observational study to evaluate the efficacy and safety of rFVIIIFc for ITI. Additionally, Dr. Malec was successful in competing for funding amongst a qualified pool of national junior investigators and received the DREAM Award through Hemostasis and Thrombosis Research Society (HTRS) to explore the impact of extended half-life products in preventing joint bleeds and joint damage in patients with hemophilia. Dr. Malec is engaged in the care of adult and pediatric patients with disorders of hemostasis and thrombosis, as well as other benign hematologic conditions.

Awards, Honors and Service

- Treasurer, Hemostasis and Thrombosis Research Society, 2018-present
- Working Group Member, National Heart, Lung, and Blood Institute (NHLBI) State of the Science Workshop Factor VIII Inhibitors: Generating a National Blueprint for Future Research, 2018.
- Learning Action Network Member, Foundation for Women and Girls with Bleeding Disorders 2014-present.

Funding

DREAM Award: Mentored Research Award sponsored by Hemostasis and Thrombosis Research Society (HTRS) and the American Thrombosis Hemostasis Network (ATHN) "Is Prophylaxis Putting Hemophilic Joints in the PINK: An ATHN-LINKED Observational Study into the Pink" 2017-2019 (Principal Investigator) $100,000

Children's Hospital of Wisconsin Hematology/Oncology/Transplant Pilot Project Funding Program "Pilot Study of Whole Genome Sequencing in Brother Cohorts with Severe Hemophilia A to identify Candidate Genes Implicated in Inhibitor Development 2018-2019 (Principal Investigator) $75,000

Bioverativ Investigator Initiated Funding Program "Hemophilia Inhibitor Response to Eloctate" 2016-2020 (Principal Investigator) $74,000

Publications

1. Croteau SE, Cheng D, Cohen AJ, Holmes CE, Malec LM, Silvey M, Thornburg CD, Wheeler AP, Kouides PA, Raffini LJ, Neufeld EJ. Regional variation and cost implications of prescribed extended half-life factor concentrates among U.S. Haemophilia Treatment Centres for patients with moderate and severe haemophilia. Haemophilia. 2019 Jul;25(4):668-675. PMID: 30993845

Alan Mast, MD, PhD

Senior Investigator, Blood Research Institute, Versiti
Medical Director, Medical Services, Versiti
Walter A. Schroeder Endowed Chair for Blood Research
Associate Professor, Department of Pathology, Medical College of Wisconsin
Associate Professor, Department of Cell Biology, Neurobiology and Anatomy, Medical College of Wisconsin
MD, Duke University, 1991
PhD, Duke University, 1991
Started at Versiti: 2003

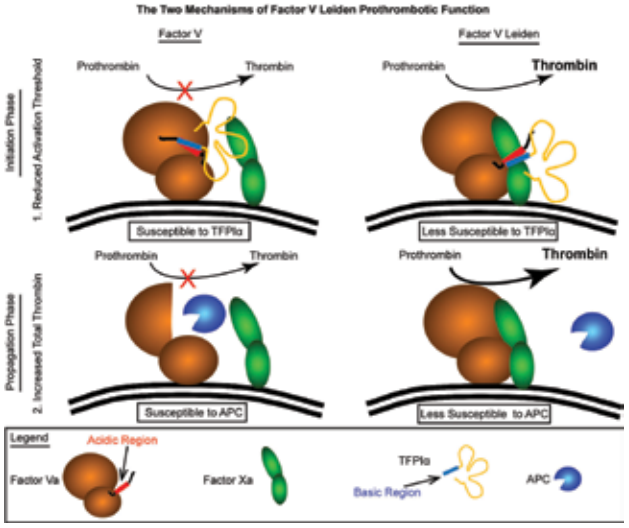
Research Interests

Blood donation removes a large amount of iron that is contained in red blood cells. Therefore, many regular blood donors become iron deficient. In his clinical research, Dr. Mast studies the effect of blood donations on iron metabolism and iron deficiency in the donor. His clinical research program has found that recovery of iron stores following blood donation takes more than six months, emphasizing the need for blood donors to take iron pills following each donation. A study found that taking 19 mg iron (the amount of iron in a typical multiple vitamin with iron) for 60 days following each donation is a simple and effective means for donors to replace iron lost during blood donation. A study of teenage blood donors found that teenagers are more susceptible to iron deficiency following blood donation than are adults, indicating that younger donors will benefit from additional safety measures to protect them from iron depletion.

In his basic research, Dr. Mast studies a protein designated "tissue factor pathway inhibitor (TFPI)." This protein plays a critical role in preventing blood from clotting inside blood vessels. His basic research program has made several important discoveries about the molecular interactions between TFPI and blood coagulation proteins. These have led to new ideas about how bleeding and clotting disorders occur. TFPI alters bleeding severity in hemophilia. Dr. Mast's laboratory is working to develop new pharmaceutical agents that block TFPI as a treatment for hemophilia.

Awards, Honors and Service

- Member, NIH Thrombosis and Hemostasis Study Section, 2019



- Member, American Society of Hematology Committee for Scientific Affairs, 2019
- Co-Chair, American Society of Hematology Working Group on Innovations in Clinical Trials, 2019
- Associate Editor, Journal of Thrombosis and Haemostasis, 2019

Funding

REDS-IV-P NHLBI
TFPI R01 NHLBI
Novo Nordisk Research Grant

Publications

1. Ellery PER, Hilden I, Thyregod P, Martinez ND, Maroney SA, Gill JC, Mast AE. Measurement of plasma and platelet tissue factor pathway inhibitor, factor V and Protein S in people with haemophilia. Haemophilia. 2019 Nov;25(6):1083-1091. PMID: 31608540
2. Kanias T, Stone M, Page GP, Guo Y, Endres-Dighe SM, Lanteri MC, Spencer BR, Cable RG, Triulzi DJ, Kiss JE, Murphy EL, Kleinman S, Gladwin MT, Busch MP, Mast AE; NHLBI Recipient Epidemiology Donor Evaluation Study (REDS)-III Program. Frequent blood donations alter susceptibility of red blood cells to storage- and stress-induced hemolysis. Transfusion. 2019 Jan; 59(1):67-78. PMID: 30474858
3. Spencer BR, Guo Y, Cable RG, Kiss JE, Busch MP, Page GP, Endres-Dighe SM, Kleinman S, Glynn SA, Mast AE; National Heart, Lung, and Blood Institute Recipient Epidemiology and Donor Evaluation Study-III (REDS-III). Iron status and risk factors for iron depletion in a racially/ethnically diverse blood donor population. Transfusion. 2019 Oct;59(10):3146-3156..PMID: 31318071

Robert R. Montgomery, MD

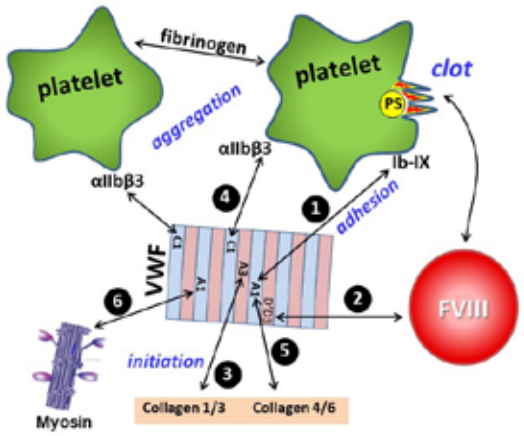
Senior Investigator, Blood Research Institute, Versiti
Attending Physician, Children's Hospital of Wisconsin
Professor of Pediatric Hematology & Population Health – Epidemiology, MCW
Professor of the Clinical and Translational Science Institute, Medical College of Wisconsin
Research Member, Hematologic Malignancy & Transplantation Research Program, MCW
MD, University of Pittsburgh Medical School, 1969
Started at Versiti: 1980

Research Interests

Hemophilia and von Willebrand Disease (VWD) are two major hereditary bleeding disorders that Dr. Montgomery's laboratory studies. The abnormal protein in hemophilia is Factor VIII (FVIII) and in VWD is von Willebrand factor (VWF). Although these are regulated by different genes, the two proteins bind together and help orchestrate the cessation of bleeding. In hemophilia Dr. Montgomery is exploring a unique form of gene therapy in which FVIII is induced to be synthesized and stored in platelets where it binds to VWF. This is not its normal site to be synthesized, but the platelet targets it to be released at the site where a blood vessel is damaged. This therapy can be effective even if the patient has begun to mount an immune response that normally blocks FVIII (this occurs in 30% of hemophilia patients). Gene therapy using this approach could be used as an alternative to using FVIII by-passing therapeutics that can sometimes run into more than $1M/yr. No one would have predicted that gene therapy could work in these individuals. Two other projects are directed at the molecular (DNA) causes of VWD. This is an international study that includes the USA, Canada, and Ireland that is supported by an NIH Program Project Grant.

Awards, Honors and Service

- Executive Secretary, Hemostasis and Thrombosis Research Society, 2019
- Member, Medical and Scientific Advisory Board National Hemophilia Foundation, 2019
- Chair, NHF Fellowship Review Committee, National Hemophilia Foundation, 2019
- Keynote Speaker, 2019 Earl W. Davie Symposium, University of British Columbia Centre of Blood Research, 2019



Binding Interactions of VWF

- Board Member, Great Lakes Hemophilia Foundation, 2019
- Keynote Speaker, National Outreach von Willebrand, 2019
- Keynote Speaker, VWDConnect National Meeting, 2019

Funding

RO1 HL139847 "Molecular Interactions of FVIII and VWF."

PO1 HL144457 "Zimmerman Program on the Biology of VWD."

RO1 HL112614 "Comparative Effectiveness in the Diagnosis of VWD."

Publications

1. Garcia J, Flood VH, Haberichter SL, Fahs SA, Mattson JG, Geurts AM, Zogg M, Weiler H, Shi Q, Montgomery RR. A Rat Model of Severe VWD by Elimination of the VWF Gene Using CRISPR/Cas9. Res Practice Thromb Haemost. 2019 Dec 29;4(1):64-71. eCollection 2020 Jan. PMID: 31989086
2. Chen J, Schroeder JA, Luo X, Montgomery RR, Shi Q. The impact of GPIbα on platelet-targeted FVIII gene therapy in hemophilia A mice with pre-existing anti-FVIII immunity. J Thromb Haemost. 2019 Mar;17(3):449-459. PMID: 30609275
3. Thornburg CD, Montgomery RR, Pipe SW. How we approach: Training pediatric coagulationists. Pediatr Blood Cancer. 2019 Dec;66(12):e27982.PMID: 31486588

Debra Newman, PhD

Senior Investigator, Blood Research Institute, Versiti
Professor, Department of Pharmacology & Toxicology/Department of Microbiology and Molecular Biology, Medical College of Wisconsin
PhD, Biology, Marquette 1989
Started at Versiti: 1989

Research Interests

Platelets are important in early wound healing, where they initially stick to damaged blood vessels and then aggregate with one another to form a platelet plug. Excessive bleeding occurs when platelet counts are low, or when platelets don't function well. Newborns who undergo heart surgery for congenital heart defects experience very severe bleeding. Dr. Newman's lab recently demonstrated that decreases in platelet count and function occur normally during heart surgery but can be corrected with platelet transfusion so that they will not complicate bleeding in newborn heart surgery patients. This research justifies administration of the right number of platelets at the right time to effectively control bleeding in this at-risk population. Deletion of segment 11.2 on the q arm of chromosome 22 gives rise to 22q11.2 Deletion Syndrome (22q11.2DS), which is commonly found in patients with congenital heart defects. One of the many genes that are deleted in 22q11.2DS encodes a component of an important platelet receptor (GPIBB). Dr. Newman's lab has recently demonstrated that loss of one copy of GPIBB is not associated with increased bleeding. This finding indicates that patients with 22q11.2DS who must undergo surgery for congenital heart defects are not at increased risk for severe bleeding because of loss of one copy of GPIBB.

A major focus of research in Dr. Newman's laboratory is Platelet Endothelial Cell Adhesion Molecule-1 (PECAM-1), which inhibits responses of many circulating blood cells, including platelets and T cells. T cells are immune cells that play an important role in clearing infections and eradicating tumors. Dr. Newman has discovered that PECAM-1 works with another potent T cell suppressor, Transforming Growth Factor ß (TFGß), to inhibit T cell anti-tumor responses. Her current work is dedicated to developing a better understanding of how PECAM-1 expression is regulated in T cells and of how PECAM-1 and TGFß work together to inhibit T cell responses. This research will help improve T cell-based therapies for treatment of cancer.

Awards, Honors and Service

- Member, Program Project Grant Review Parent Committee, National Heart, Lung & Blood Institute, National Institutes of Health 2017 – present
- Member, Planning Committee, International Society on Thrombosis & Haemostasis 2021 Annual Meeting 2019 – present
- Ad hoc Manuscript Reviewer: American Journal of Physiology – Cell Physiology; American Journal of Physiology - Heart and Circulatory Physiology; American Journal of Medical Genetics; The Anatomical Record; Arteriosclerosis, Thrombosis and Vascular Biology; Blood; BMC Immunology; Cell Adhesion and Migration; Circulation Research; Free Radical Biology and Medicine; Human Immunology; JCI Insight; Journal of Biological Chemistry; Journal of Cell Biology; Journal of Cell Science; Journal of Experimental Medicine; Journal of Histochemistry and Cytochemistry; Journal of Immunology; Journal of Thrombosis and Hemostasis; Molecular Biology of the Cell; Platelets; PLoS One; Proceedings of the National Academy of Sciences: USA; Science Reports; Science Signaling; Science Translational Medicine; Trends in Cardiovascular Medicine; Thrombosis and Haemostasis; Thrombosis Research; Transfusion

Funding

NIH R35- HL139937 (Co-Investigator)

Publications

1. Moroi AJ, Zwifelhofer NM, Riese MJ, Newman DK, Newman PJ. Diacylglycerol kinase ζ is a negative regulator of GPVI-mediated platelet activation. Blood Adv. 2019 Apr 9;3(7):1154-1166. doi: 10.1182/bloodadvances.2018026328. PMID: 30967391
2. Zwifelhofer NMJ, Bercovitz RS, Weik LA, Moroi A, LaRose S, Newman PJ, Newman DK. Hemizygosity for the Gene Encoding Glycoprotein Ib beta (GPIbα) Is Not Responsible for Macrothrombocytopenia and Bleeding in Patients with 22q11 Deletion Syndrome. J Thromb Haemost. 2019 Feb;17(2):295-305. PMID: 30549403

Peter Newman, PhD

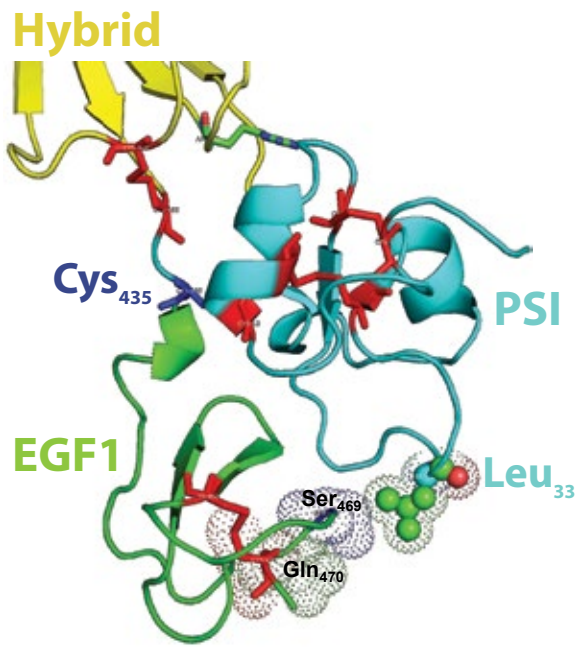
Jacquelyn Fredrick Endowed Chair for Foundational Research
Vice President for Research, Versiti
Associate Director Blood Research Institute, Versiti
Professor, Department of Pharmacology, Medical College of Wisconsin
Professor, Department of Cell Biology, Neurobiology and Anatomy, Medical College of Wisconsin
PhD, St. Louis University, 1983
Started at Versiti: 1983

Research Interests

Peter Newman's laboratory divides its attention between exploring the structure and function of the vascular cell adhesion and signaling receptor, PECAM-1, in platelets and endothelial cells, and the generation of antigenically-distinct megakaryocytes and platelets from induced pluripotent stem (iPS) cells - both funded by the Heart, Lung, and Blood Institute of the National Institutes of Health. Techniques range from CRISPR-mediated gene editing to protein crystallography to the development of animal models of platelet alloimmunity. Projects range from investigating the molecular basis of PECAM-1-mediated homophilic binding and the role of carbohydrate residues in this process to exploiting recent advances in CRISPR gene editing technology to generate megakaryocyte progenitor cells, megakaryocytes, and platelets from induced pluripotent stem cells to create platelet alloantigen-specific cell lines capable of long-term self-renewal, cryopreservation, and distribution.

Awards, Honors and Service

- R35 Outstanding Investigator Award, NIH National Heart, Lung, and Blood Institute 2018-25
- Distinguished Career Award, International Society of Thrombosis and Haemostasis 2013
- Chair, NIH Special Emphasis Panel, Consortium Linking Oncology with Thrombosis 2018
- Editor, Arteriosclerosis, Thrombosis and Vascular Biology (Journal of the American Heart Association) 2012-present



- Chair, BloodWorks Northwest Scientific Advisory Board 2012-present
- Editor, Arteriosclerosis, Thrombosis and Vascular Biology (Journal of the American Heart Association) 2012-present

Funding

2018-2025 - NIH Grant R35 HL139937 (Outstanding Investigator Award)

Publications

1. Zhang N and PJ Newman: Packaging functionally important plasma proteins into the α-granules of human induced pluripotent stem cell-derived megakaryocytes. J Tissue Eng Regen Med 13(2):244-252, 2019. PMCID: PMC6742440
2. Zwifelhofer NMJ, RS Bercovitz, LA Weik, A Moroi, S LaRose, PJ Newman, and DK Newman: Hemizygosity for the gene encoding glycoprotein Ibß is not responsible for macro-thrombocytopenia and bleeding in patients with 22q11 deletion syndrome. J Thromb and Haemostasis 17:295-305 2019. PMCID: PMC6410711
3. Moroi AJ, N Zwifelhofer, MJ Riese, DK Newman and PJ Newman: Diacylglycerol kinase zeta (DGKζ) is a negative regulator of GPVI-mediated platelet activation. Blood Advances 3:1154-1166, 2019. PMCID: PMC6457232
4. Zhang N, S Santoso, RH Aster, BR Curtis, and PJ Newman: Bioengineered iPSC-derived megakaryocytes for the detection of platelet-specific patient alloantibodies. Blood 134:e1-e8, 2019. PMCID PMC6887112. (with Inside Blood commentary 134:1887-1888)

Qizhen Shi, MD, PhD

Senior Investigator, Blood Research Institute, Versiti
Professor of Pediatric Hematology, Medical College of Wisconsin
MD, Fujian Medical University, Fuzhou, China, 1990
PhD, Fujian Medical University, Fuzhou, China, 1998
Hematology Fellowship, Medical College of Wisconsin 2000 – 2006
Started at Versiti: 2010

Research Interests

Development of inhibitory antibodies (inhibitors) against FVIII is a significant problem in the clinical care of patients with hemophilia A. One primary focus of Dr. Shi's research is to develop a gene therapy approach for the treatment of hemophilia A even with inhibitors. Dr. Shi's studies have shown that engineering blood stem cells to have FVIII made and stored in platelets can solve all the problems for hemophilia A. These studies show platelet-targeted gene therapy can efficiently correct the bleeding phenotype in hemophilia A mice even in the presence of inhibitors. Furthermore, platelet gene therapy can induce profound antigen-specific peripheral immune tolerance. Currently, Dr. Shi is exploring a safe non-genotoxic preconditioning for platelet gene therapy. In a separate line of research, Dr. Shi's team found that that FVIII's carrier protein, VWF, can modulate the antigenicity of FVIII, attenuating FVIII memory immune responses in hemophilia A mice. These studies provide important information about the impact of VWF on FVIII immune responses, which may aid the design of more effective protocols to prevent inhibitor development and to induce immune tolerance in patients with hemophilia A.

Awards, Honors and Service

- Ad Hoc Reviewer, Hemostasis/Thrombosis Study Section, NIH/NHLBI. 2/2019.
- Ad Hoc Reviewer, Small Business SEP, NIH/NHLBI, 11/2019.
- Co-Chair, the ISTH meeting. Melbourne, Australia. 7/2019.
- Editorial board member, Mol Ther – Meth & Clin Dev 2017-present

Funding

R01 HL102035 "Platelet Derived FVIII Gene Therapy of Hemophilia A". Role: PI

NHF Bridge Award "Investigation of VWF as an immunomodulator of the immunogenic response towards FVIII". Role: PI

1R01 HL142791A. "Phase I clinical trial testing Feasibility of hematopoietic stem cell gene therapy using platelet FVIII to safely improve hemostasis for severe hemophilia A with inhibitory antibodies to FVIII. Role: Co-I

Publications

1. Chen J, Schroeder JA, Luo X, Montgomery RR, and Shi Q. The impact of GPIbα on platelet-targeted FVIII gene therapy in hemophilia A with pre-existing anti-FVIII immunity. J Thromb Haemost. 2019 Mar;17(3):449-459.
2. Gao C, Schroeder JA, Xue F, Jing W, Cai Y, Subramaniam S, Rao S, Weiler H, Czechowicz A, and Shi Q. Immunotoxin-mediated non-genotoxic preconditioning for platelet gene therapy of hemophilia A mice. Blood Adv. 2019 Sep 24;3(18):2700-2711.
3. Jing W, Chen J. Cai Y, Chen Y, Schroeder JA, Cui W, Johnson BD, and Shi Q. Induction of activated T follicular helper cells is critical for anti-FVIII inhibitor development in hemophilia A mice. Blood Adv. 2019 Oct 22;3(20):3099-3110.

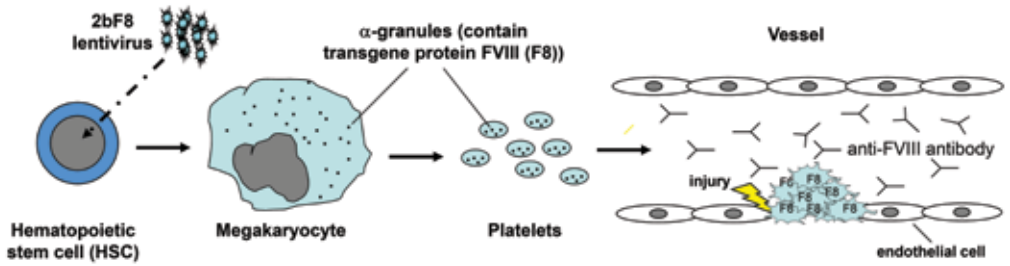


Fig. 1 Graphical summary of platelet-targeted gene therapy.

Roy Silverstein, MD

Senior Investigator, Blood Research Institute, Versiti
John and Linda Mellowes Professor and Chair
Department of Medicine, Medical College of Wisconsin
MD, Emory University School of Medicine 1979
Started at Versiti: 2011

Research Interests

Research by Dr. Silverstein's group concerns basic mechanisms underlying common vascular diseases, especially thrombosis and atherosclerosis, with particular emphasis on the role of a cellular receptor designated CD36 expressed on platelets, macrophages, fat cells and other tissues. CD36 enables the recognition by platelets and macrophages of danger signals generated in the body as the result of inflammation, oxidant stress, diabetes and cancer, and may play a role in the pro-thrombotic state associated with these conditions as well as in accumulation of cholesterol in blood vessel walls, leading to atherosclerosis. Recent work in the Silverstein lab has demonstrated that CD36 triggers very specific signaling pathways inside cells that promote these maladaptive responses. The remarkable diversity of CD36 functions suggests that further work will have implications for treatment and/or prevention of arterial disease, thrombosis and cancer.

Awards, Honors and Service

- President, American Society of Hematology 2019
- Chair, Medical College Physicians Board, 2018-2020
- Member, Clinical Executive Committee, Froedtert and Medical College of Wisconsin
- 26th William Maloney Lecture, Brigham and Women's Hospital Harvard Medical School
- Editorial Boards: J. Clinical Investigation and J. Experimental Medicine

Funding

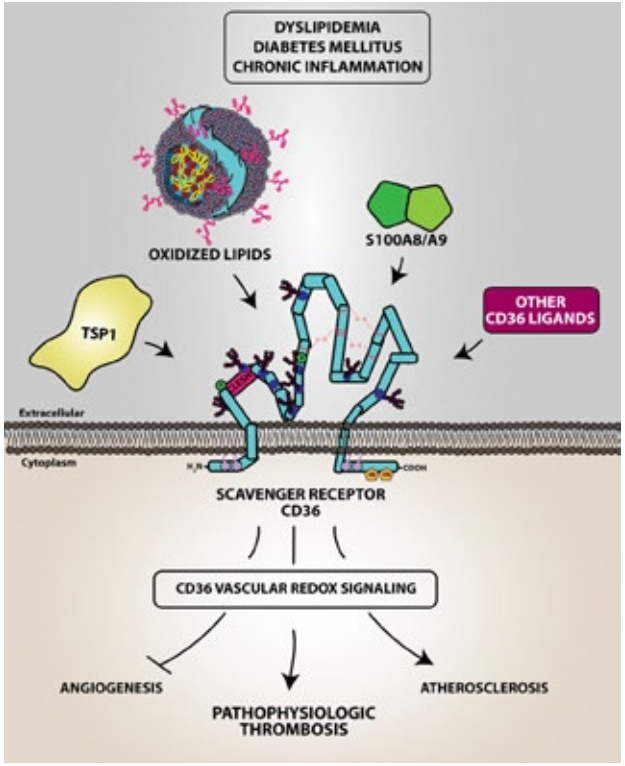
R01 HL142152: "ERK5 and CD36 link oxidative stress to platelet dysfunction and ischemic injury"

R01 HL126645: "MRP-14, CD36 and Thrombosis"

Advancing a Healthier Wisconsin Endowment Pre Program Project Pilot Program: Metabolic Control of Inflammation in Atherosclerosis by Macrophage Scavenger Receptors"

Publications

1. Chen Y, Yang M, Huang W, Chen W, Zhao Y, Schulte ML, Volberding PJ, Gerbec Z, Zimmermann MT, Zeighami A, Demos W, Zhang J, Knaack DA, Smith BC, Cui W, Malarkannan S, Sodhi K, Shapiro JI, Xie Z, Sahoo D, Silverstein RL. Mitochondrial Metabolic Reprogramming by CD36 Signaling Drives Macrophage Inflammatory Responses. Circ Res. 2019 Dec 6;125(12):1087-1102. PMID: 31625810
2. Yang M, Silverstein RL. CD36 signaling in vascular redox stress. Free Radic Biol Med. 2019 May 20;136:159-171. PMID: 30825500
3. Yue H, Febbraio M, Klenotic PA, Kennedy DJ, Wu Y, Chen S, Gohara AF, Li O, Belcher A, Kuang B, McIntyre TM, Silverstein RL, Li W. CD36 Enhances Vascular Smooth Muscle Cell Proliferation and Development of Neointimal Hyperplasia. Arterioscler Thromb Vasc Biol. 2019 Feb;39(2):263-275. PMID: 30567481.



Hartmut Weiler, PhD

Ziegler Family Chair for Research, Senior Investigator, Blood Research Institute, Versiti
Associate Professor, Department of Physiology, Medical College of Wisconsin
Director, Transgenic Core Facility, Medical College of Wisconsin / Blood Research Institute
Dr.rer.nat., Technische Hochschule Darmstadt Germany, 1989
Started at Versiti: 1997

Research Interests

Activation of the blood clotting system serves to stop bleeding when a blood vessel is injured, but it also is a natural part of the body's response to infections, inflammation, and cancer, and plays an important role in embryonic development. In 2019, the National Institutes of Health supported work in Dr. Weiler's laboratory to develop innovative therapeutic interventions targeting blood coagulation pathways in diseases such as severe sepsis, malaria, and bone marrow failure after exposure to lethal doses of radiation. Dr. Weiler holds the Ziegler Family Chair for Research, and also directs the joint Transgenic Core Facility of the Medical College of Wisconsin (MCW) and the Versiti Blood Research Institute. The facility provides a wide range of genome editing services facilitating the generation of genetically altered rodents serving as models for human disease.

Awards, Honors and Service

- NIH reviewer Special Emphasis Panel/Scientific Review Group (SBRI, STTR)
- Co-Chair: Coagulation pathways, ISTH Congress 2019, Melbourne, Australia
- 1st Esteemed Career Award of the International Society of Thrombosis and Hemostasis

Funding

"Zimmerman Program on the Biology of VWD" NHLBI -- 1P01HL144457

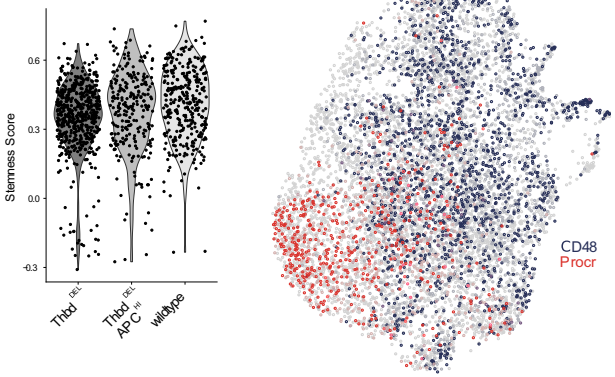
"Regulation of Innate Immunity by Coagulation Receptors" -- NHLBI -- 1RO1 HL133348

"Coagulation Factor Signaling in Malaria" -- NIH/NIAID -- R01HL130678

"Serpin Regulation of Coagulation Proteases" -- NHLBI -- R01HL062565

"Protein C Pathway Mitigation of Radiation-Induced Vascular Dysfunction" -- NIAID --U01AI133561

Transcriptional stemness of hematopoietic progenitors lacking Thrombomodulin

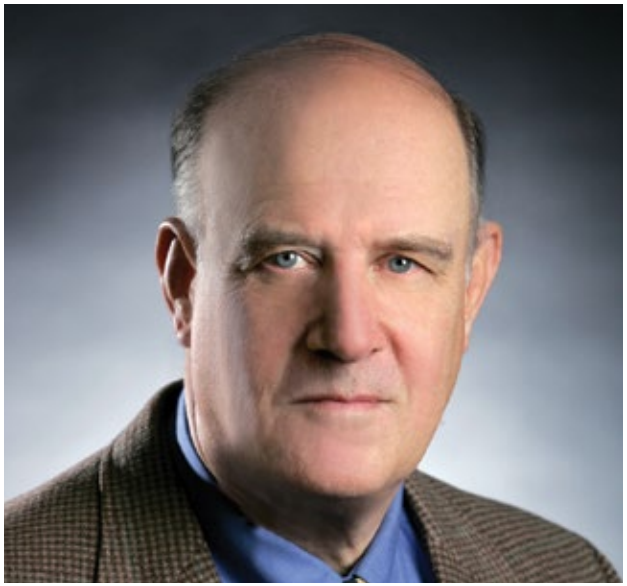


Publications

1. Gao C, Schroeder JA, Xue F, Jing W, Cai Y, Scheck A, Subramaniam S, Rao S, Weiler H, Czechowicz A, Shi Q. Nongenotoxic antibody-drug conjugate conditioning enables safe and effective platelet gene therapy of hemophilia A mice. Blood Adv. 2019 Sep 24;3(18):2700-2711. PMID: 31515232
2. Graf C, Wilgenbus P, Pagel S, Pott J, Marini F, Reyda S, Kitano M, Macher-Göppinger S, Weiler H, Ruf W. Myeloid cell-synthesized coagulation factor X dampens antitumor immunity. Sci Immunol. 2019 Sep 20;4(39). PMID: 31541031
3. Slobodianuk TL, Kochelek C, Foeckler J, Kalloway S, Weiler H, Flood VH. Defective collagen binding and increased bleeding in a murine model of von Willebrand disease affecting collagen IV binding. J Thromb Haemost. 2019 Jan;17(1):63-71. PMID: 30565388
4. Basu S, Liang HP, Hernandez I, Zogg M, Fields B, May J, Ogoti Y, Wyseure T, Mosnier LO, Burns RT, Carlson K, Weiler H. Role of Thrombomodulin expression on hematopoietic stem cells. J. Thromb. Haemost. 2019 (in press)

Gilbert White, II, MD

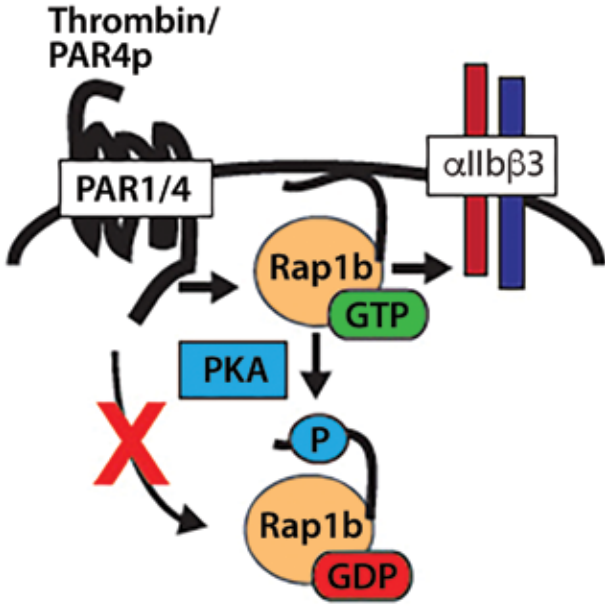
Senior Investigator/Director, Blood Research Institute, Versiti
Richard H. and Sara E. Aster Chair for Medical Research
Professor, Dept. of Medicine, Biochemistry, and Pharmacology & Toxicology, Medical College of Wisconsin
MD, University of North Carolina, Chapel Hill, 1971
Started at Versiti: 2004



Research Interests

Work by Dr. White's group is broadly aimed at understanding signaling pathways involved in the hemostatic responses by blood platelets. Recent focus has been on a small G protein, Rap 1b, which is present in high concentrations in platelets and is critical for platelet aggregation and the activation of integrins that are critical for the platelet-platelet interactions needed to form a hemostatic plug. Rap 1b also appears to be a critical target for cyclic AMP-dependent protein kinase (PKA) and phosphorylation of Rap 1b by PKA is involved in the inhibition of platelets by drugs that target the PKA pathway. Thus, Rap 1b may function as a unique and critical bi-directional modulator of platelet activation.

The ability of Rap 1b to bi-directionally modulate platelet function makes it a potentially interesting therapeutic target for anti-platelet therapy in the treatment of heart attacks and strokes.



Awards, Honors and Service

- Tibor J. Greenwalt Award, Versiti
- Board of Directors, National Hemophilia Foundation
- Board of Directors, Great Lakes Hemophilia Foundation
- Board of Directors, Versiti Blood Research Institute Foundation
- Chair, American Society of Hematology (ASH) Bridge Grant Review Program 2018
- Member, International Society of Thrombosis and Hemostasis (ISTH) Awards & Honors Committee 2018
- Editorial Board, Haemophilia
- Co-Editor, 6th edition of Thrombosis and Hemostasis

Funding

UL1 TR001435 Clinical and Translational Science Institute, NCATS

UG3 OD023190 All of US – Wisconsin, NIH Office of Director

T32 HL07209 "Research Training in Hematology and Transfusion Medicine", NHLBI

T32 GM080202 "Medical Scientist Training Program", NIGMS

Publications

1. Gao J, Bao Y, Ge S, Sun P, Sun J, Liu J, Chen F, Han L, Cao Z, Qin J, White GC, Xu Z, Ma YQ. Sharpin suppresses β1-integrin activation by complexing with the β1 tail and kindlin-1. Cell Commun Signal. 2019 Aug 20;17(1):101. PMID: 31429758
2. Yan Y, Yang H, Hu X, Zhang Z, Ge S, Xu Z, Gao J, Liu J, White GC, Ma YQ. Kindlin-3 in platelets and myeloid cells differentially regulates deep vein thrombosis in mice. Aging (Albany NY). 2019 Aug 31;11(17):6951-6959. PMID: 31477636

Jieqing Zhu, PhD

Investigator, Blood Research Institute, Versiti
Associate Professor, Department of Biochemistry, Medical College of Wisconsin
PhD, Institute of Microbiology, Chinese Academy of Sciences, Beijing 2003
Immune Disease Institute, Harvard Medical School, Boston 2009
Started at Versiti: 2011

Research Interests

Membrane protein complexes designated "integrins" function as cell surface receptors to regulate cell-cell and cell-matrix interactions critical for organ development, hemostasis, antigen recognition, cellular homing to specific body sites and inflammation. Dr. Zhu is using structural biology, biochemistry and cell biology techniques to investigate how particular structural domains of integrins function in integrin activation. These studies will guide the development of small molecule or antibody inhibitors of integrin function that can be useful in the treatment and prevention of thrombosis and a range of other conditions. Recent studies have revealed how integrin beta subunit becomes extended during activation and how the transmembrane domain contributes to integrin structural changes, which provide new information of developing next-generation integrin inhibitors that restrain integrin conformational activation.

Awards, Honors and Service

- Member, American Society of Hematology (ASH) 2019
- Member, American Society for Biochemistry and Molecular Biology (ASBMB) 2019
- Editorial Boards: Scientific Reports

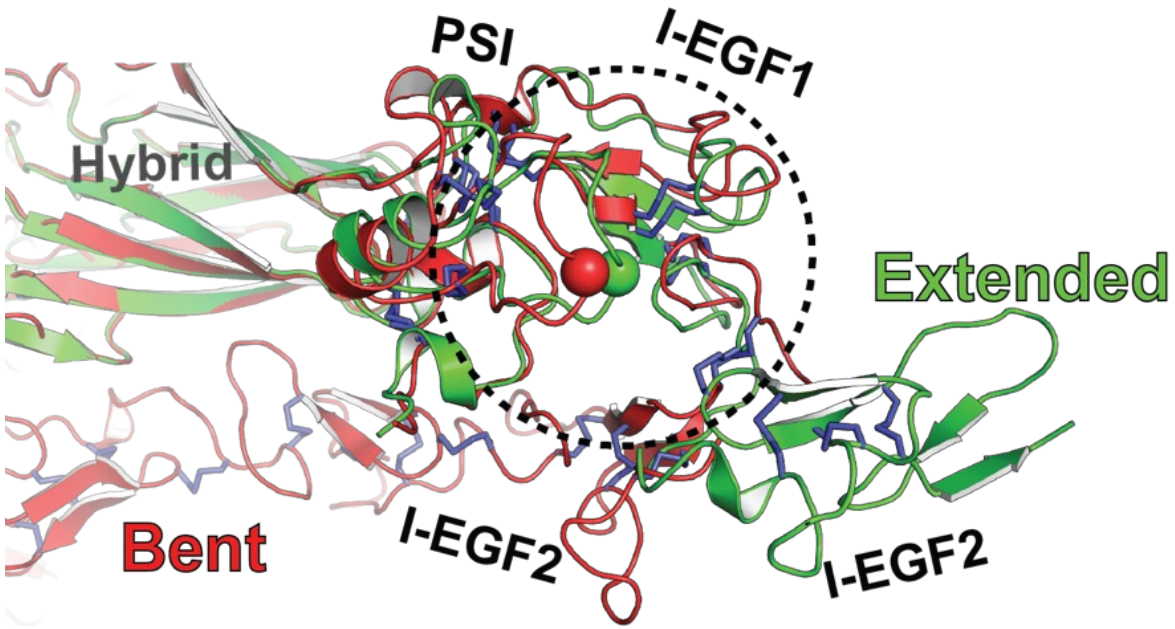
Funding

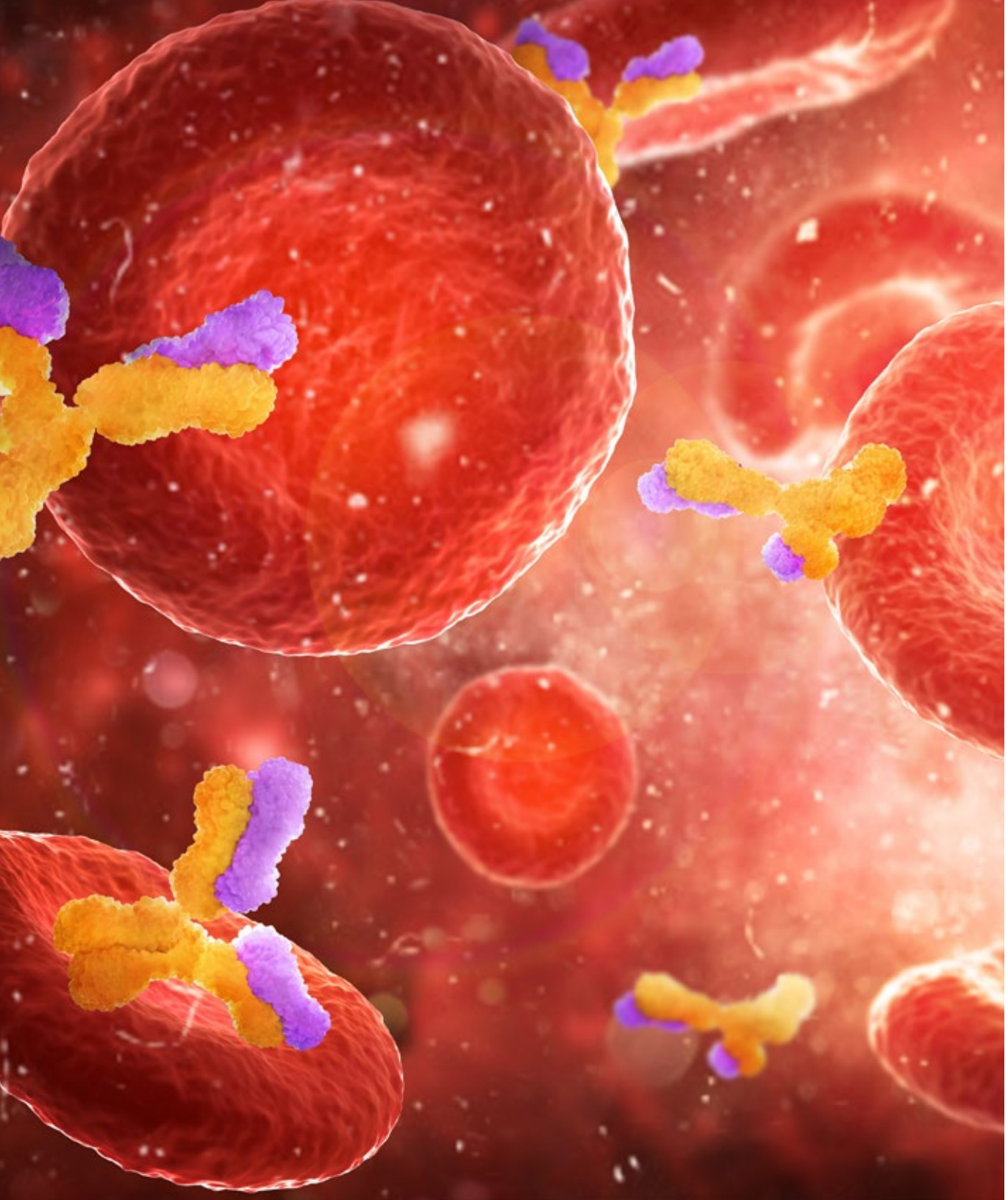
R01 HL131836 "Structural Transition of Cellular Integrins and Applications Thereof"

R35 HL139937 (PI: P. Newman) "Basic investigation and translational applications concerning the cell and molecular biology of blood and vascular cells"

Publications

1. Behnaz Bayat, Annalena Traum, Heike Berghöfer, Jieqing Zhu, Gregor Bein, Ulrich Sachs, and Sentot Santoso. Current anti-HPA-1a standard antibodies react with the β3 integrin subunit but not with αIIb β3 and αVβ3 complexes. Thrombosis and Haemostasis, 119 (11):1807-1815 (2019) PMID: 31587244





Immunology

Immunobiology has been a cornerstone of research at Versiti since 1947, going back to the early days of immunohematology. Studies by Versiti investigators led to the identification of some of the first antigen systems specific to red blood cells.

Versiti investigators facilitated the first bone marrow transplant from an unrelated donor to successfully treat bone marrow failure (aplastic anemia) and played key roles in creation of the National Marrow Donor Program.

Matthew Anderson, MD, PhD

Vice President and Medical Director, Diagnostic Laboratories
Associate Investigator, Blood Research Institute, Versiti
Assistant Professor, Pathology, Medical College of Wisconsin
MD, Medical College of Wisconsin, 2006
PhD, Medical College of Wisconsin, 2004
Started at Versiti: 2013

Research Interests

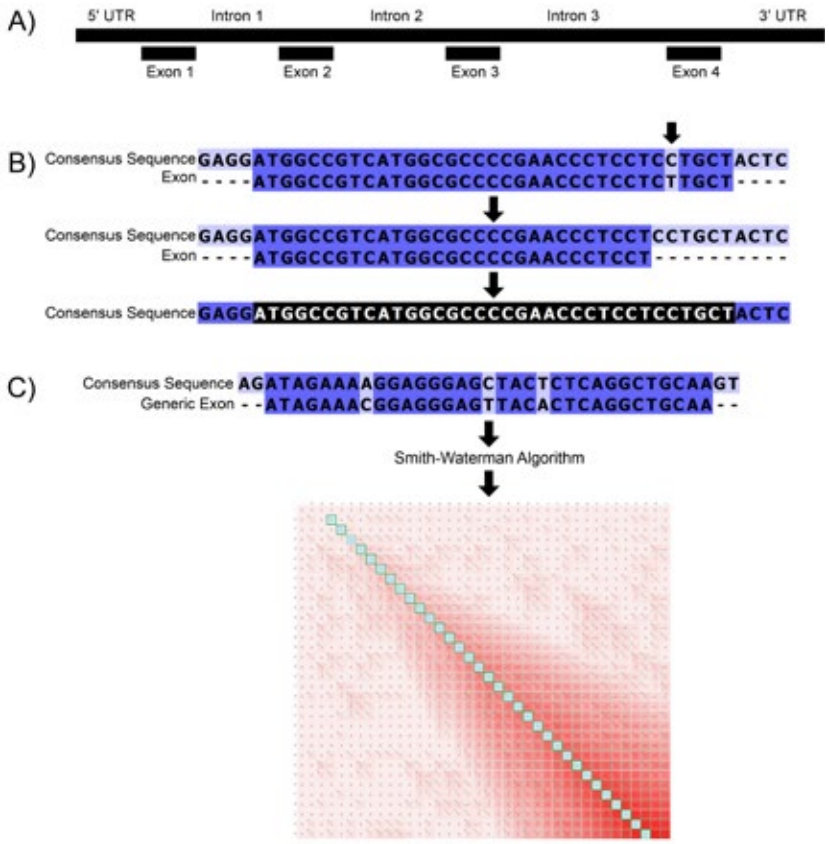
Dr. Anderson's research interests include the use of high-throughput sequencing technologies for clinical diagnostics and biomarker discovery, with a focus on transplantation. Human leukocyte antigens (HLA) are key molecular determinants of the adaptive immune response and also control the host immune response to hematopoietic and solid-organ transplants. Clinically, the success of a transplant critically depends on a high degree of similarity between the HLA molecules of the donor and recipient. Dr. Anderson's group has recently reported a novel bioinformatics approach (see figure) to directly compare HLA gene sequences from hematopoietic transplant donor and recipients, demonstrating that transplant pairs highly matched at a genetic level show improved transplant outcome. In the future, he plans to develop next-generation sequencing assays to analyze other genes important for the immune response to transplants and to monitor patients for rejection.

Awards, Honors and Service

- Member, American Society for Histocompatibility and Immunogenetics (ASHI)
- Member, Association for Molecular Pathology
- Fellow, College of American Pathologists

Publications

1. Compton CC, Robb JA, Anderson MW, Berry AB, Birdsong GG, Bloom KJ, Branton PA, Crothers JW, Cushman-Vokoun AM, Hicks DG, Khoury JD, Laser J, Marshall CB, Misialek MJ, Natale KE, Nowak JA, Olson D, Pfeifer JD, Schade A, Vance GH, Walk EE, Yohe SL. Preanalytics and Precision Pathology: Pathology Practices to Ensure Molecular Integrity of Cancer Patient Biospecimens for Precision Medicine. Arch Pathol Lab Med. Nov;143(11):1346-1363. PMID: 31329478
2. Vazirabad I, Chhabra S, Nytes J, Mehra V, Narra RK, Szabo A, Jerkins JH, Dhakal B, Hari P, Anderson MW. Direct HLA Genetic Comparisons Identify Highly Matched Unrelated Donor/Recipient Pairs with Improved Transplant Outcome. Biol Blood Marrow Transplant. 2019 May;25(5):921-931. PMID: 30537549
3. Anderson, M.W. Emerging next-generation sequencing technologies. Genomic Applications in Pathology (2nd edition). Netto, G. and Kaul, K.L.. (Eds). Springer-Verlag (2019) DOI: 10.1007/978-3-319-96830-8



Weiguo Cui, MD, PhD

Investigator, Blood Research Institute, Versiti
Associate Professor, Department of Microbiology and Immunology, Medical College of Wisconsin
MD/PhD, Tianjin Medical University, China, 2004
Dept. of Immunobiology, Yale University School of Medicine, 2012
Started at Versiti: 2012

Research Interests

T cell exhaustion is a crucial problem to fight off viral infection and cancer. Dr. Cui's studies are aimed at improving the understanding of how T cells become functionally exhausted in the face of viruses such as HIV and cancer. His current work is focused on the study of transcriptional and metabolic changes that take place in antigen-specific T cells during infection and cancer. An immediate goal is to identify specific factors that can reverse T cell exhaustion. Findings made will improve basic understanding of the immune response and have implications for the treatment and prevention of infectious diseases and cancer.

Awards, Honors and Service

- ACS Research Scholar Grant (RSG)
- Ad hoc review, IHD study section, NIH
- Method of manufacturing dual-specific t-cells for use in cancer immunotherapy. Weiguo Cui. US Patent (US 2018/0334651). Nov. 22, 2018.

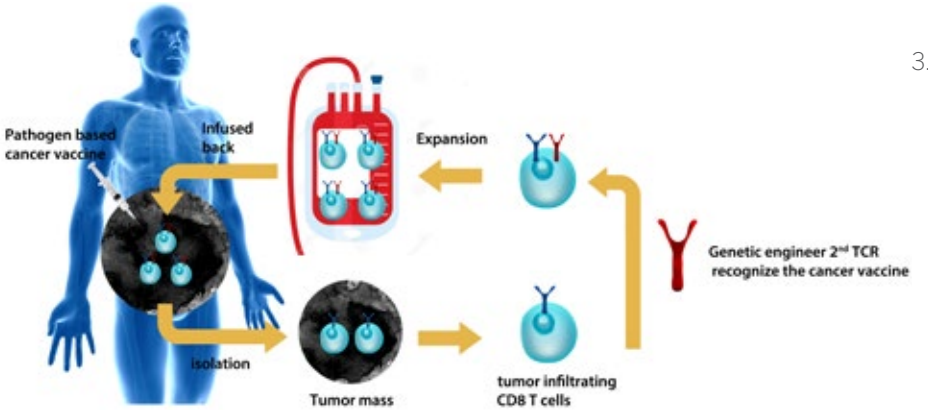
Funding

5R01AI125741-04 (PI) 05/16/2016-04/30/2021 NIH/NIAID "The cellular and transcriptional control of CD8-T cell functional adaptation to chronic viruses."

HDTRA11710052 (Co-I) 01/01/2018-08/20/2020 DTRA via ioGenetics "Comparative Computational Modeling of Immune Responses to Vaccines"

RSG-17-186-01 (PI) 01/01/18–12/31/21 American Cancer Society "Harnessing BATF-boosted Anti-tumor CD8 T cells in Cancer Immunotherapy"

MCW Cancer Center (MPI) "BCG boosted Adoptive Cell Transfer Immunotherapy to Treat Bladder Cancer"



TORFP E02 (Co-I) 08/04/19–02/03/22 NIH/NCI "Chemoprevention with Aerosolized Let-7 micro RNA in Mouse Models of Non-small Cell Lung Cancer"

1R01DK121747-01 (Co-I) 08/22/19–05/31/23 NIH/NIDDK "Shaping Diabetogenic T Cells by IL-27 in Type I Diabetes"

1R01AI148403 (PI) 09/01/19–08/31/24 NIH/NIAID "Phenotypic, Functional and Transcriptional Heterogeneity in T Cell Exhaustion"

Publications

1. Xin G, Zander R, Schauder DM, Chen Y, Weinstein JS, Drobyski WR, Tarakanova V, Craft J, Cui W. Single-cell RNA sequencing unveils an IL-10-producing helper subset that sustains humoral immunity during persistent infection. Nat Commun. 2018 Nov 28;9(1):5037.
2. Newman DK, Fu G, McOlash L, Schauder D, Newman PJ, Cui W, Rao S, Johnson BD, Gershan JA, Riese MJ. Frontline Science: PECAM-1 (CD31) expression in naïve and memory, but not acutely activated, CD8+ T cells. J Leukoc Biol. 2018 Nov;104(5):883-893.
3. Wesley EM, Xin G, McAllister D, Malarkannan S, Newman DK, Dwinell MB, Cui W, Johnson BD, Riese MJ. Diacylglycerol kinase ζ (DGKζ) and Casitas b-lineage proto-oncogene b-deficient mice have similar functional outcomes in T cells but DGKζ-deficient mice have increased T cell activation and tumor clearance. Immunohorizons. 2018 Apr 1;2(4):107-118.

Bonnie Dittel, PhD

Senior Investigator, Blood Research Institute, Versiti
Professor, Department of Microbiology and Immunology, Medical College of Wisconsin
PhD, University of Minnesota
Postdoctoral Training, Yale University
Started at Versiti: 2000

Research Interests

Autoimmunity occurs when the immune system mounts an inappropriate attack on one's own body tissues. Dr. Dittel's laboratory is concerned with immune regulation that occurs during multiple sclerosis (MS), the most prevalent autoimmune disorder affecting the central nervous system (CNS). For this work, she is utilizing a mouse model of MS designated experimental autoimmune encephalomyelitis (EAE). Current studies are aimed at understanding how key cells of the immune system (T and B lymphocytes) interact to influence the autoimmune process that causes damage to CNS tissue. In recent studies we have identified a new subset of B lymphocytes (BDL) that induce the proliferation of a critical subset of T lymphocytes designated T regulatory cells (Treg) via GITRL that are essential for controlling autoimmunity. Dr. Dittel also is investigating how immune cells propagate EAE by studying the mechanisms whereby they open the blood-brain barrier and induce neuronal damage. Findings made are expected to suggest new approaches for treating MS and other immune disorders affecting the nervous system.

Awards, Honors and Service

- Journal of Neuroimmunology, Editorial Board
- Brain, Behavior, and Immunity, Editorial Board
- NIAID, Investigator Program Project Application (P01), Chair
- NINDS, Clinical Neuroimmunology and Brain Tumors
- NIH, Small Business: Neuroscience Assay, Diagnostics and Animal Model Development
- NIAID, Cellular and Molecular Immunology B
- National Multiple Sclerosis Society, Fellowship Review Committee
- Research Partner of the Year, National Multiple Sclerosis Society

Funding

Advancing Healthier Wisconsin, Neuroscience Research Center, Medical College of Wisconsin Targeting myeloperoxidase in neuroinflammation – Principle Investigator

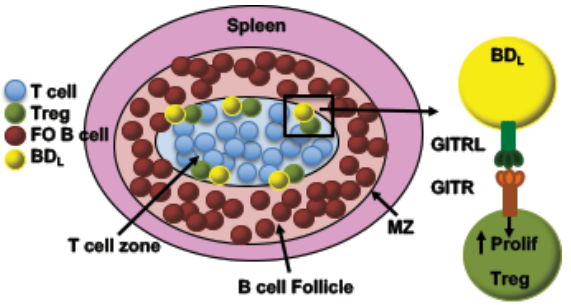
R21AI145323, NIAID A novel human regulatory B cell subset – Principal Investigator

R21NS106451, NHLBI Trifunctional peptides in the Ischemic Cascade – Co-investigator

National Multiple Sclerosis Society - B Cell Regulation in EAE/MS – Principle Investigator

Publications

1. Cowie AM, Dittel BN, Stucky CL. A Novel Sex-Dependent Target for the Treatment of Postoperative Pain: The NLRP3 Inflammasome. Front Neurol. 2019 Jun 12;10:622. doi: 10.3389/fneur.2019.00622. eCollection 2019. Review. PMID: 31244767
2. Dittel LJ, Dittel BN, Brod SA. Ingested ACTH blocks Th17 production by inhibiting GALT IL-6. J Neurol Sci. 2019 Nov 27;409:116602. PMID: 31812846
3. Johnson KE, Lange PT, Jondle CN, Volberding PJ, Lorenz UM, Cui W, Dittel BN, Tarakanova VL. B cell-intrinsic SHP1 expression promotes gammaherpesvirus-driven germinal center response and the establishment of chronic infection. J Virol. 2019 Dec 12;94(1). PMID: 31597758
4. Ray A, Khalil MI, Pulakanti KL, Burns RT, Gurski CJ, Basu S, Wang D, Rao S, Dittel BN. Mature IgDlow/- B cells maintain tolerance by promoting regulatory T cell homeostasis. Nat Commun. 2019 Jan 14;10(1):190. PMID: 30643147



Jack Gorski, PhD

Senior Investigator, Blood Research Institute, Versiti
Assistant Professor, Microbiology and Immunology, Medical College of Wisconsin
PhD, University of Cincinnati, 1976
Started at Versiti: 1986

Research Interests

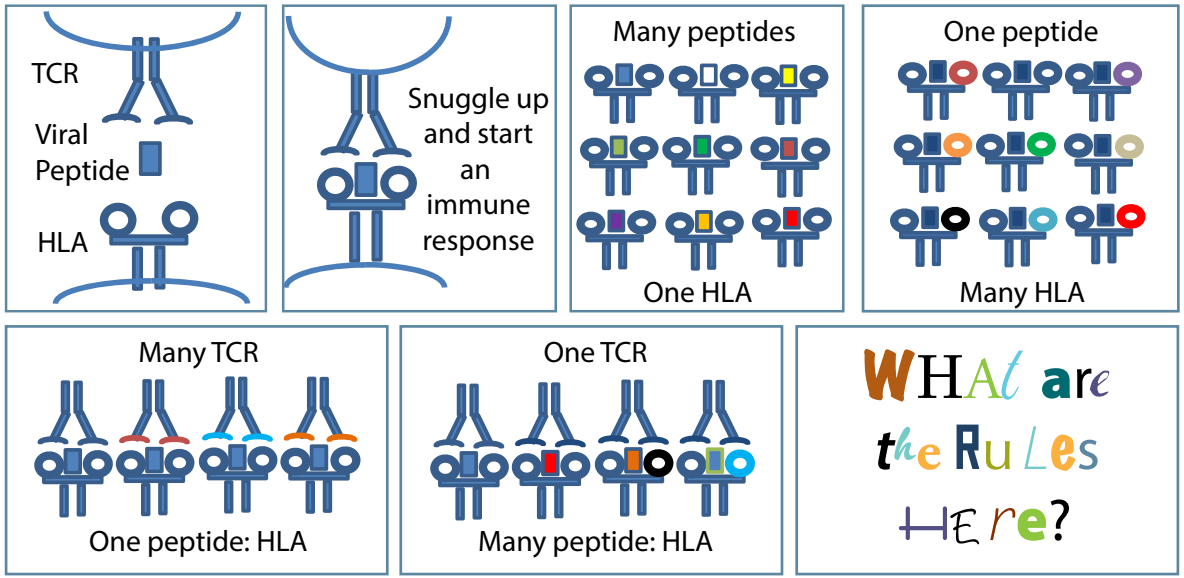
The immune response is a complicated process involving direct and indirect communication between many specialized types of cells. Dr. Gorski studies this process at a molecular level. Recent studies have provided new insights into how the immune system recognizes and generates a response against protein fragments (peptides) from germs or viruses. He is the inventor of innovative methods to characterize genetic differences between individuals that determine which protein fragments can be recognized, and how to measure the range of unique immune cells that recognize these protein fragments. Dr. Gorski currently studies how immune responses are affected by aging, how T cell responses differ between healthy children and children with an autoimmune disease, and how the spread of influenza among older persons in the US can be tracked utilizing the tools of molecular biology. Recently he has been investigating the use of blood cells as a cellular network to identify differences in serum samples that can indicate the immune state of the person providing the serum. Finally, he is very interested in how specificity for a pathogen arises from a system whose components are not very specific. These studies will advance the basic understanding of the human response and its relation to autoimmunity, tissue transplantation and infectious disease.

Awards, Honors, and Service

- Director, Center for Human Immunology, Blood Research Institute

Publications

1. Kumar P, Rajasekaran K, Nanbakhsh A, Gorski J, Thakar MS, Malarkannan S. IL-27 promotes NK cell effector functions via Maf-Nrf2 pathway during influenza infection. Sci Rep. 2019 Mar 21;9(1):4984. PMID: 30899058
2. Naumova EN, Yassai MB, Demos W, Reed E, Unruh M, Haribhai D, Williams CB, Naumov YN, Gorski J. Age-Based Dynamics of a Stable Circulating Cd8 T Cell Repertoire Component. Front Immunol. 2019 Aug 6;10:1717. PMID: 31447830
3. Simpson RB, Alarcon Falconi TM, Venkat A, Chui KHH, Navidad J, Naumov YN, Gorski J, Bhattacharyya S, Naumova EN. Incorporating calendar effects to predict influenza seasonality in Milwaukee, Wisconsin. Epidemiol Infect. 2019 Sep 11;147:e268. PMID: 31506136



Subramaniam Malarkannan, PhD

Senior Investigator and Gardetto Chair for Immunology and Immunotherapy, Blood Research Institute
Professor of Medicine, Microbiology & Immunology, and Pediatrics, Medical College of Wisconsin
PhD Madurai Kamaraj University, Madurai, TN, India, 1991
Started at Versiti: 2000

Research Interests

Natural Killer (NK) cells are a type of white blood cell that specializes in killing virus-infected and malignant cells. Due to this specialty, there is a great deal of interest in using NK cells for therapeutic purposes. Dr. Malarkannan's group studies basic, translational, and clinical aspects of NK cells. Using single-cell sequencing, his group has determined the developmental heterogeneity of human NK cells in healthy individuals and patients with rare inherited diseases. The group's studies have identified pathways that influence target cell killing and associated inflammatory changes. This work may show how these pathways can be manipulated to maximize the killing effect and minimize the adverse effects of NK cell therapy. This constitutes a new form of transfusion therapy for treatment of malignant conditions.

Awards, Honors and Service

- 2019 Adhoc Reviewer, NIAID, Transplantation, Tolerance, and Tumor Immunology (TTT) Study Section, NIH
- 2019 External Member, Grant Review Committee, University of Lyon, France

Funding

R38 HL143561, Stimulating access to Research in Residency (StARR).

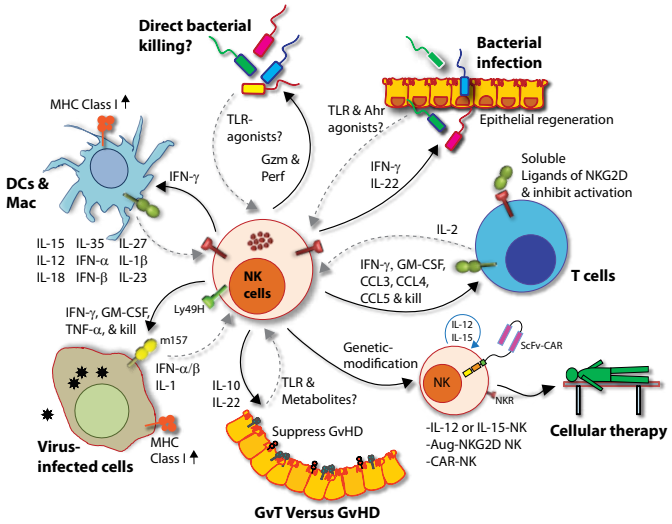
MACC Fund, "Targeting Pediatric Cancer with 'Next-Gen' CARs

"AHW, Honing inflammatory responses in cancer treatment with designer CAR-T cells.

Ann's Hope Foundation, Requirement of metabolic reprogramming in NK cells during the clearance of melanoma.

Publications

1. Kumar P, Rajasekaran K, Nanbakhsh A, Gorski J, Thakar MS, Malarkannan S. IL-27 promotes NK cell effector functions via Maf-Nrf2 pathway during influenza infection. Sci Rep. 2019 Mar 21;9(1):4984. PMID: 30899058
2. Nanbakhsh A, Srinivasamani A, Holzhauer S, Riese MJ, Zheng Y, Wang D, Burns R, Reimer MH, Rao S, Lemke A, Tsaih SW, Flister MJ, Lao S, Dahl R, Thakar MS, Malarkannan S. Mirc11 Disrupts Inflammatory but Not Cytotoxic Responses of NK Cells. Cancer Immunol Res. 2019 Oct;7(10):1647-1662. PMID: 31515257
3. Schloemer NJ, Abel AM, Thakar MS, Malarkannan S. In Vivo Assessment of NK Cell-Mediated Cytotoxicity by Adoptively Transferred Splenocyte Rejection. (2020) Methods Mol Biol. 2097:115-123. doi: 10.1007/978-1-0716-0203-4-8. (PMID:31776923).
4. Yang C, Siebert JR, Burns R, Gerbec ZJ, Bonacci B, Rymaszewski A, Rau M, Riese MJ, Rao S, Carlson KS, Routes JM, Verbsky JW, Thakar MS, Malarkannan S. Heterogeneity of human bone marrow and blood natural killer cells defined by single-cell transcriptome. Nat Commun. 2019 Sep 2;10(1):3931. PMID: 31477722



Nykia

Nykia Julian knew something was wrong with her heart. She was struggling to maintain a normal life and could not walk up stairs without getting winded. Eventually, doctors told her she would not have much longer to live without a new heart. Thanks to the kindness of a donor, Nykia was able to get the transplant she needed to live a healthy life.

“I’m able to do a lot that I wasn’t able to do before. I’m able to travel, I’m able to walk upstairs, I’m able to walk further.”

Matthew Riese, MD, PhD

Associate Professor, Departments of Medicine (Division of Heme/Onc), Microbiology and Immunology, and Surgery, Medical College of Wisconsin
Associate Investigator, Blood Research Institute, Versiti
PhD, Medical College of Wisconsin, 2002
MD, Medical College of Wisconsin, 2004
Started at Versiti: 2012

Research Interests

Immunotherapies for the treatment of malignancy have recently begun to demonstrate impressive success in achieving long-term disease control and eradication; however, the therapies work in a minority of patients. The Riese lab is investigating ways to improve upon existing cancer immunotherapies by targeting "off" switches inside T cells, the cells responsible for killing cancer cells. His studies have incorporated both oncology and chemistry and have allowed him to blend medicine and research throughout his career.

Awards, Honors and Service

- Member, American Society of Clinical Oncology 2008-current
- Member, American Association for Cancer Research 2015-current
- Member, Society for Leukocyte Biology 2015-current

  Member, Society for Immunotherapy of Cancer, 2018-current

Funding

GI Innovations. 8/1/2019-7/31/2020. Evaluation of GI-101 on DGK-deficient T cells in conjunction with STING agonists (PI-Riese).

Bristol-Myers Squibb. 9/1/2016-6/1/2019. "Studies of novel compounds in mice models." (PI-Riese)

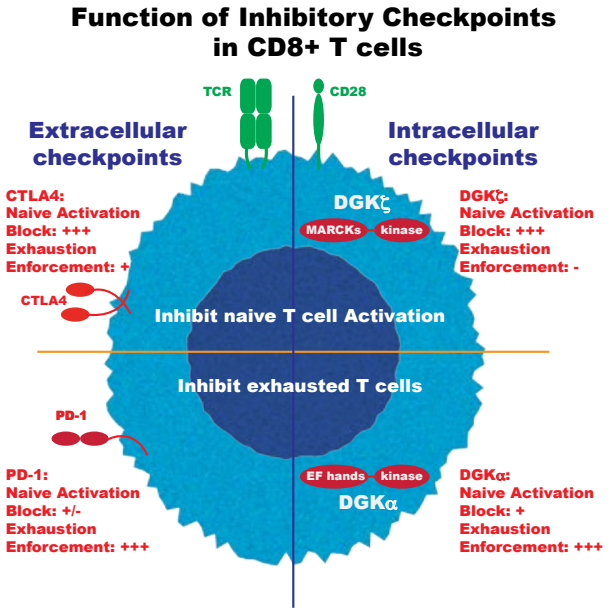
MCW Cancer Center. 11/1/2017-10/31/2019. "Optimizing Cancer Adoptive Cellular Therapies with Epigenetic Strategies." (PI-Riese, co-PI Rao).

Advancing a Healthier Wisconsin. 1/1/2018-12/31/2019. "Engineering Inflammation-free CAR Therapy." (PI-Malarkannan, co-PI Riese).

MCW Center of Immunology. 12/1/2018-11/30/2019. "Unbiased Genomic Analysis of the Pancreatic Cancer Immune Microenvironment." (PI-Riese).

Publications

1. Jing W, McAllister D, Vonderhaar EP, Palen K, Riese M, Gershan J, Johnson BD, Dwinell MB. "STING agonist inflames the pancreatic cancer immune microenvironment and reduces tumor burden in mouse models." Journal of Immunotherapy of Cancer. 2019 Apr 29;7(1):115.
2. Yang C, Siebert JR, Burns R, Gerbec ZJ, Bonacci B, Rymaszewski A, Rau M, Riese MJ, Rao S, Carlson KS, Routes JM, Verbsky JW, Thakar MS, Malarkannan S. "Heterogeneity of human bone marrow and blood natural killer cells defined by single-cell transcriptome." Nature Communications. 2019 Sep 2;10(1):3931.
3. Sitaram P, Uyemora B, Malarkannan S, Riese MJ. "Beyond the cell surface: targeting intracellular negative regulators to enhance T cell anti-tumor activity." International Journal of Molecular Science 2019 Nov 20;20(23).



Demin Wang, PhD

Senior Investigator, and John B. and Judith A. Gardetto Chair for Cancer Research
Blood Research Institute, Versiti
Adjunct Faculty, Department of Microbiology and Immunology, Medical College of Wisconsin
PhD, University of Tennessee, 1995
Started at Versiti: 2000

Research Interests

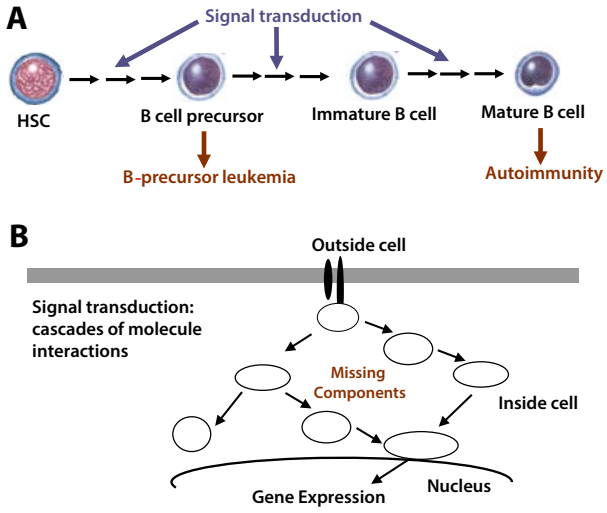
Dr. Wang is concerned with self-renewal and differentiation of cells (hematopoietic stem cells, HSCs) that give rise to blood cells and to the subset of white blood cells (B lymphocytes) that produce antibodies. His studies are designed to identify and functionally characterize signaling molecules and pathways that are critical to HSC and B cell biology. Recent studies have identified a novel and important CXCR5+PD-1+ follicular helper CD8 T cells that control B cell tolerance. These findings further our understanding of B cell tolerance regulation. Furthermore, recent studies have identified new mechanisms that limit the production of autoantibodies causing heparin-induced thrombocytopenia and thrombosis (HIT). Work in these fields is expected to provide an improved understanding of autoantibody formation in human disease and suggest new approaches to prevention and treatment of autoimmunity.

Awards, Honors, and Service

- Editorial Board, Blood, 2017-present
- Host R. Douglas Ziegler Innovation Lecture, 2019
- Member, ZRG1 IMM-J03, Special Emphasis Panel, NHLBI, NIH, 2019
- Member, ZRG1 F07 U 20 L, Fellowship Study Section, NHAID NIH, 2019

Funding

R01 AI079087 "PLCγs in B cell biology and autoimmunity"



R01 HL130724 "B cell responses in heparin-induced thrombocytopenia"

NIH SBIR "Development of a New Carbohydrate-based Anticoagulant Drug"

Publications

1. Chen Y, Yu M, Zheng Y, Fu G, Xin G, Zhu W, Luo L, Burns R, Li QZ, Dent AL, Zhu N, Cui W, Malherbe L, Wen R, Wang D. CXCR5+PD-1+ follicular helper CD8 T cells control B cell tolerance. Nat Commun. 2019 Sep 27;10(1):4415. PMID: 31562329
2. Wen R, Wang D. PTPRJ: a novel inherited thrombocytopenia gene. Blood. 2019 Mar 21;133(12):1272-1274. PMID: 30898775
3. Zheng Y, Zhu W, Haribhai D, Williams CB, Aster RH, Wen R, Wang D. Regulatory T Cells Control PF4/Heparin Antibody Production in Mice. J Immunol. 2019 Oct 1;203(7):1786-1792 PMID: 31471526

Renren Wen, PhD

Investigator, Blood Research Institute, Versiti
PhD, University of Tennessee Medical School, Memphis 1996
St. Jude Children's Research Hospital, Memphis 1996-2000
Started at Versiti: 2000

Research Interests

T and B lymphocytes are two important cell types in our adaptive immune system. Whereas B cells secrete antibodies that are essential for protection against extracellular pathogens, T cells are critical for the control of infection by intracellular pathogens, and for enabling B lymphocytes to efficiently produce antibodies. Dr. Wen's work is aimed at more fully understanding the signaling pathways that govern proper B and T cell activation and communications.

Dr. Wen's lab studies two signaling molecules that play roles in both B and T cells: phospholipase gamma, mutations of which have been associated with human autoinflammatory diseases and T cell lymphomas, and Bcl10, mutations which have been associated with human B cell lymphomas. Dr. Wen's lab also studies heparin-induced thrombocytopenia (HIT), a disease that is caused by clinical administration of heparin, which in some patients can result in limb- and life-threatening thrombosis. Dr. Wen's lab has cloned a group of antibodies that can activate platelets and contribute to the development of HIT. Her current work is focused on identifying the underlying molecular mechanisms that control the development and activation of the B cells that generate these platelet-activating antibodies, and her work will potentially lead to improved diagnosis and HIT treatment.

Awards, Honors and Service

- Member, American Society of Hematology
- Member, International Society on Thrombosis and Hemostasis

Funding

R01 1R01HL148120-01 Wen (PI) 06/01/19-05/31/2023 NIH/NHLBI "Molecular Basis of the Humoral Immune Response in Heparin-Induced Thrombocytopenia" Role: Principal Investigator

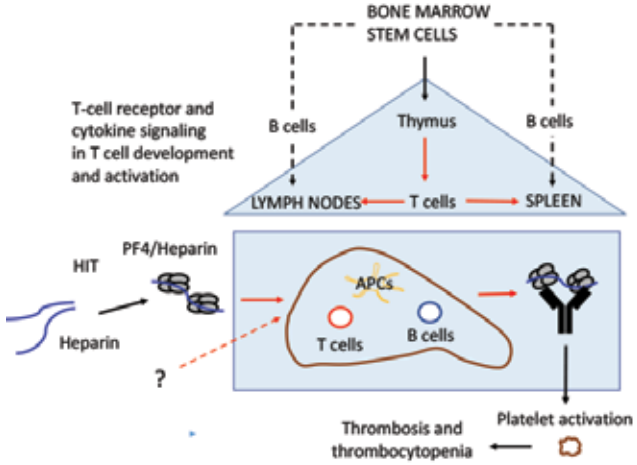
5R01 AI079087-08 Wang (PI) 06/15/2008 – 08/31/2019 NIH/NIAID "PLCγs in B Cell Biology and Autoimmunity" Role: Co-Investigator

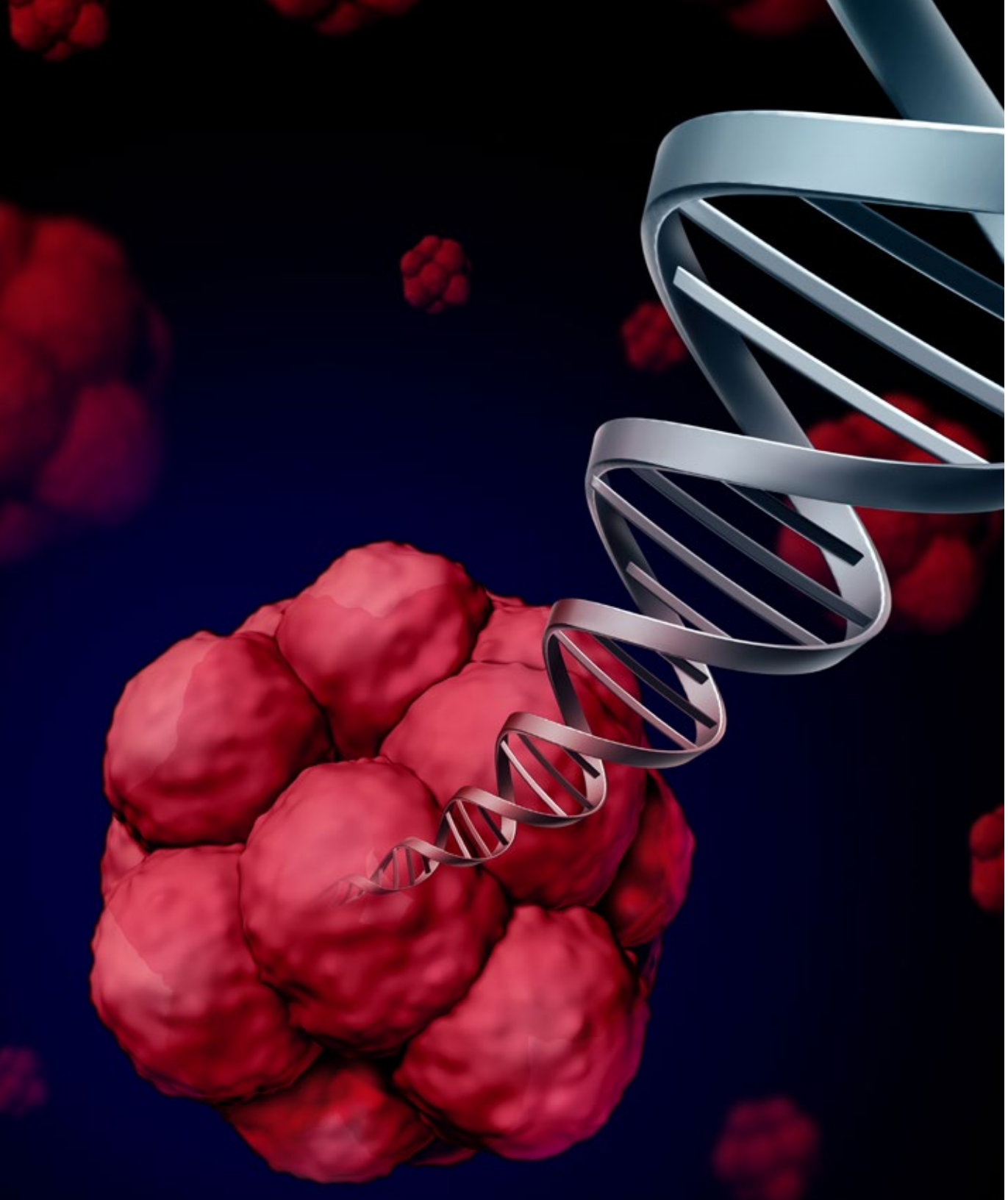
R01 HL130724 Wang (PI) 12/01/2016-11/30/2020 "B cell responses in heparin-induced thrombocytopenia" Role: Co-Investigator

R01 AI083636-06 Salomon (PI) 05/08/2017-04/30/2022 "Phosphoproteomic Analysis of Feedback Networks in T Cell Signaling" Role: Co-investigator

Publications

1. Chen Y, Yu M, Zheng Y, Fu G, Xin G, Zhu W, Luo L, Burns R, Li QZ, Dent AL, Zhu N, Cui W, Malherbe L, Wen R, Wang D. CXCR5+PD-1+ follicular helper CD8 T cells control B cell tolerance. Nat Commun. 2019 Sep 27;10(1):4415. PMID: 31562329
2. Wang Z, Vaughan TY, Zhu W, Chen Y, Fu G, Medrzycki M, Nishio H, Bunting ST, Hankey-Giblin PA, Nusrat A, Parkos CA, Wang D, Wen R, Bunting KD. Gab2 and Gab3 Redundantly Suppress Colitis by Modulating Macrophage and CD8+ T-Cell Activation. Front Immunol. 2019 Mar 18;10:486. PMID: 30936879
3. Wen R, Wang D. PTPRJ: a novel inherited thrombocytopenia gene. Blood. 2019 Mar 21;133(12):1272-1274. PMID: 30898775





Stem Cells

Research in Stem Cell Biology and Hematopoiesis is aimed at understanding the many factors that regulate the process of how normal blood cells are formed (hematopoiesis), as well as understanding disease mechanisms that lead to abnormal hematopoiesis, which either could lead to a failure of healthy blood cell production or cause leukemia.

Studies in this area are bringing Versiti into the fields of regenerative medicine, and cancer biology. These studies reflect an ongoing commitment to expanding foundational research into areas that will fundamentally improve the understanding and treatment of currently incurable blood diseases.

Stem Cell Biology investigators are using cutting-edge technology to characterize molecular mechanisms involved in regulation of hematopoietic stem cells and their maturation into mature red cells, white cells and platelets.

Karen Carlson, MD, PhD

Assistant Professor, Department of Internal Medicine, Division of Hematology and Oncology,
Medical College of Wisconsin
Assistant Investigator, Blood Research Institute, Versiti
MD/PhD University of Wisconsin-Madison, 2004
Hematology/Oncology Fellowship: New York Presbyterian, Weill-Cornell Medical Center, 2011
Started at Versiti: 2016

Research Interests

Dr. Karen-Sue Carlson is a board certified clinical hematologist. She joined the faculty at the Medical College of Wisconsin as an Assistant Professor of Medicine in the Division of Hematology and Oncology in 2013, and was appointed Assistant Investigator at the Blood Research Institute in 2016.

She maintains an active clinical focus on diseases of disordered hematopoiesis including aplastic anemia, acute and chronic leukemias, and myelodysplastic and myeloproliferative syndromes at the Medical College of Wisconsin and Froedtert Hospital. At the Blood Research Institute, her research focuses on the bone marrow microenvironment.

Dr. Carlson studies how extracellular matrix dynamics control communication between the peripheral nervous system and the bone marrow. This matrix-nerve-bone marrow axis is an important regulator of blood cell production.

Dr. Carlson's long-term goal is to apply what she learns about the biology of the bone marrow microenvironment to develop targeted therapies that will help her patients with hematopoietic diseases.

Awards, Honors and Service

- Best Doctors designation 2018, 2019
- Faculty Development Award, Department of Medicine, Medical College of Wisconsin, 2019
- Top Patient Experience Award, Froedtert and the Medical College of Wisconsin, 2019

Funding

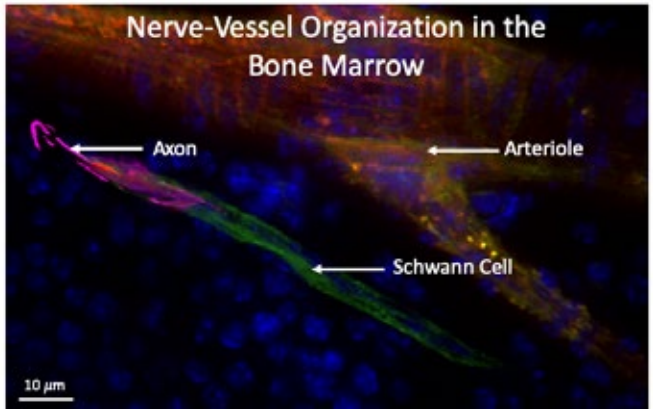
"Bone Marrow Failure in Mice Deficient for the Extracellular Matrix Component, Laminin-gamma1" (1K08HL127187-03) NHLBI (NIH) PI 4/1/ 15 – 3/31/20

"Neural Regulation of AML", Froedtert Hospital and the Scott Garrett Leukemia Research Foundation - 12/10/ 19 – 12/31/20

Paulette Kroll Leukemia Research Fund, MCW -6/13/19 Ongoing

Publications

1. Fields B, DeLaForest A, Zogg M, May J, Hagen C, Komnick K, Wieser J, Lundberg A, Weiler H, Battle M, Carlson K*. "The Adult Murine Intestine is Dependent on Constitutive Laminin-γ1 Synthesis". Scientific Reports. 2019 Dec 17;9(1):19303 . PMID: 31628891.
2. Tang J, Zhu N, Rao S, Carlson K*. Stem Cell Damage During Chemotherapy—Can we do better?. Best Practices & Research Clinical Haematology. 2019 Mar;32(1):31-39. PMID: 30927973
3. Basu S, Liang HP, Hernandez H, Zogg M, Fields B, May J, Ogoti Y, Wyseure T, Mosnier LO, Burns RT, Carlson K, Weiler H. Role of Thrombomodulin expression on hematopoietic stem cells. Journal of Thrombosis and Hemostasis. 2019 Oct 19. PMID: 31628891
4. Yang C, Siebert JR, Burns R, Gerbec ZJ, Bonacci B, Rymaszewski A, Rau M, Riese M, Rao S, Carlson KS, Routes JM, Verbsky JM, Thakar MS, Malarkannan S. Heterogeneity of Human Bone Marrow and Blood Natural Killer Cells Defined by Single-cell Transcriptome. Nature Communications. 2019 Sep 2;10(1):3931. PMID: 31477722.
5. Izaguirre-Carbonell J, Christiansen L, Burns R, Schmitz J, Li C, Mokry RL, Blumen T, Zheng Y, Shen J, Carlson KS, Rao S, Wang D, Zhu N. Critical role of jumonji domain of JMJD1C in MLL-rearranged leukemia. Blood Advances. 2019 May 14;3(9):1499-1511. PMID: 31076406.



John Pulikkan, PhD

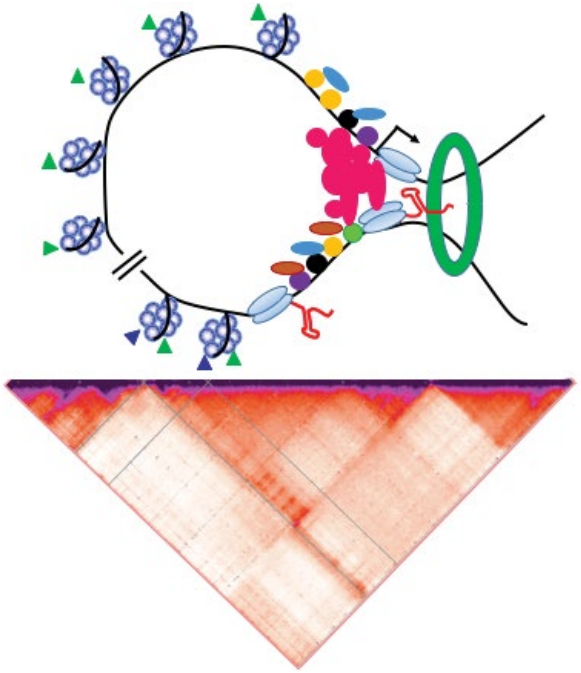
Associate Investigator, Blood Research Institute, Versiti
Assistant Professor, Cell Biology, Neurobiology and Anatomy, Medical College of Wisconsin
PhD, Ludwig Maximilians University of Munich, Munich, Germany, 2008
Postdoctoral Associate, University of Massachusetts Medical School, Worcester, MA, 2010-2018
Started at Versiti: 2018

Research Interests

The past decade of leukemia research has indicated in a comprehensive understanding of the role of genetic and epigenetic changes in leukemogenesis. However, only recently has the three-dimensional genome architecture been implicated in leukemogenesis. While much is known about transcription factor deregulation in AML, our understanding of chromatin structure and how transcription factors regulate higher-order genome architecture is limited. Our lab is interested in understanding the interplay between transcription factors (RUNX1 and C/EBPα) and chromatin dynamics in myeloid differentiation and how this is altered in AML. Our goal is to identify and characterize novel therapeutic targets and translate them to the clinic with academic and pharmaceutical collaborations.

Awards, Honors and Service

- Young Investigator Grant, Alex's Lemonade Stand Foundation for Childhood Cancer 2014
- Discovery Grant, Lauri Strauss Leukemia Foundation 2014
- Scholar Award in Basic Research, American Society of Hematology (ASH) 2013
- Member, American Association for Cancer Research (AACR) 2018
- Member, International Society for Stem Cell Research (ISSCR) 2018
- Member, American Society for Hematology 2018



Sridhar Rao, MD, PhD

Associate Investigator, Blood Research Institute, Versiti
Associate Professor of Pediatrics, Division of Hematology/Oncology, Medical College of Wisconsin
MD, University of Chicago, Pritzker School of Medicine, 2001
PhD, University of Chicago, 1999
Started at Versiti: 2010

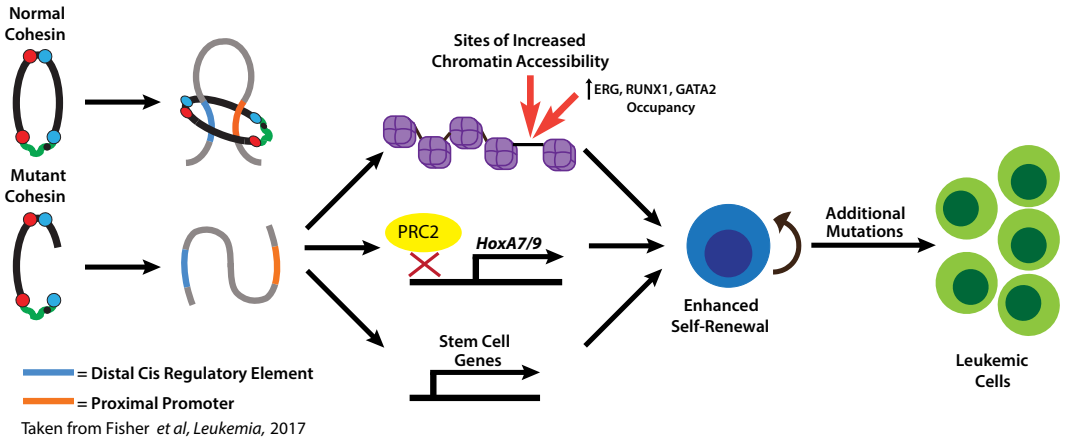
Research Interests

Acute Myelogenous Leukemia (AML) is a common malignancy, but despite modern chemotherapy, the majority of patients relapse. Dr. Rao's laboratory focuses on how altered gene expression causes diseases such as cancer. His long-term goal is to understand how gene expression derangements can be targeted to develop less toxic, more effective chemotherapies to treat blood-derived cancer.

Dr. Rao's laboratory focuses on Acute Myelogenous Leukemia (AML) because it represents a significant clinical challenge, with up to 50% of patients relapsing. Recent data has indicated a large number of genes (>200) can be mutated in AML, making it difficult to understand how specific, targeted therapies can be developed. In Dr. Rao's lab, they have focused on how mutations in a specific group of genes, termed the cohesin complex, cause AML. Recent work indicates that mutations in the cohesin complex promote bone marrow cells to divide abnormally, and this predisposes them to acquire additional mutations which ultimately cause leukemia. Because the cohesin mutation likely occurs early in the process, targeting these mutations could lead to new therapies. Dr. Rao is currently working on different targeted therapy agents already in clinical trial to determine if they could be used to treat patients with cohesin-mutated AML. He has also expanded his research interest into Ewing's Sarcoma, in which cohesin mutations have also recently been identified.

Awards, Honors and Service

- Member, American Society of Hematology 2019
- Member, American Association for Cancer Research 2019
- Permanent Member, NIH CSR Standing Study Section (DEV2) 2019



Taken from Fisher *et al, Leukemia,* 2017

Funding

National Cancer Institute- R01 "Cohesin Mutations in AML"

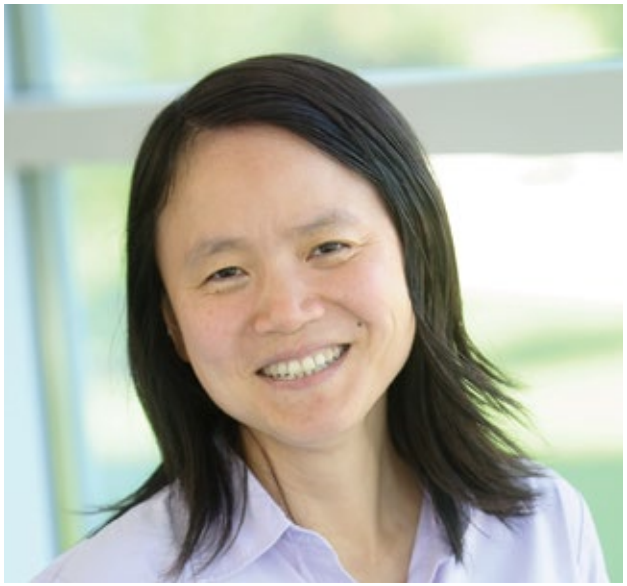
Midwest Athletes Against Childhood Cancer (MACC Fund)

Publications

1. Rao S. Closing the loop on cohesin in hematopoiesis. Blood. 2019 Dec 12;134(24):2123-2125. PMID: 31830276
2. Reimer M Jr, Pulakanti K, Shi L, Abel A, Liang M, Malarkannan S, Rao S. Deletion of Tet proteins results in quantitative disparities during ESC differentiation partially attributable to alterations in gene expression. BMC Dev Biol. 2019 Jul 8;19(1):16. PMID: 31286885
3. Tang J, Zhu N, Rao S, Carlson KS. Stem cell damage after chemotherapy- can we do better? Best Pract Res Clin Haematol. 2019 Mar;32(1):31-39 Review. PMID: 30927973

Nan Zhu, PhD

Associate Investigator, Blood Research Institute, Versiti
Assistant Professor, Cell Biology, Neurobiology and Anatomy, Medical College of Wisconsin
PhD, Boston University, Boston, MA, 2007
Started at Versiti: 2015



Research Interests

Research in Dr. Zhu's laboratory focuses on understanding epigenetic regulation in normal and malignant hematopoiesis with emphasis on the role of such regulation in hematopoietic stem cells (HSC) as well as leukemia stem cells (LSC). Epigenetic regulation refers to changes in gene activities that are independent of the underlying gene sequences. Epigenetic regulators play an important role in normal development and differentiation. More recently, they emerged as important players in the development of cancer as evident by recurrent mutations across a spectrum of cancers. Dr. Zhu's lab has previously screened and identified several epigenetic regulators as important for the maintenance of acute myeloid leukemia (AML). Currently, Dr. Zhu is working on elucidating their role in normal and malignant stem cell function and understanding the precise underlying molecular mechanism. Dr. Zhu has shown that one of the epigenetic regulators, JMJD1C, is important for LSC function but dispensable for HSC function, thus a potential therapeutic target. Dr. Zhu's lab is extending this finding using pre-clinical models of human AML as well as studying the molecular mechanism of how JMJD1C functions in AML. The ultimate goal of our research is to identify therapeutic targets and developed targeted therapy in AML based on knowledge gained from our research.

Funding

- NIH(NCI) R37CA229751 "The histone demethylase JMJD1C in human Acute Myeloid Leukemia"
- ASH Scholar Award "Understanding Molecular Mechanism of JMJD1C Function in AML"
- R. Douglas Ziegler Innovation Award "CRISPR/Cas9 Screen to Identify Driver Non-coding Variants in AML Leukemogenesis"

Publications

1. Chen Y, Yu M, Zheng Y, Fu G, Xin G, Zhu W, Luo L, Burns R, Li QZ, Dent AL, Zhu N, Cui W, Malherbe L, Wen R, Wang D. CXCR5+PD-1+ follicular helper CD8 T cells control B cell tolerance. Nat Commun. 2019 Sep 27;10(1):4415. PMID: 31562329
2. Deshpande A, Chen BR, Zhao L, Saddoris K, Kerr M, Zhu N, Mali P, Deshpande AJ. Investigation of Genetic Dependencies Using CRISPR-Cas9-based Competition Assays. J Vis Exp. 2019 Jan 7;(143). PMID: 30663717
3. Izaguirre-Carbonell J, Christiansen L, Burns R, Schmitz J, Li C, Mokry RL, Bluemn T, Zheng Y, Shen J, Carlson KS, Rao S, Wang D, Zhu N. Critical role of Jumonji domain of JMJD1C in MLL-rearranged leukemia. Blood Adv. 2019 May 14;3(9):1499-1511. PMID: 31076406
4. Tang J, Zhu N, Rao S, Carlson KS. Stem cell damage after chemotherapy- can we do better? Best Pract Res Clin Haematol. 2019 Mar;32(1):31-39 Review. PMID: 30927973

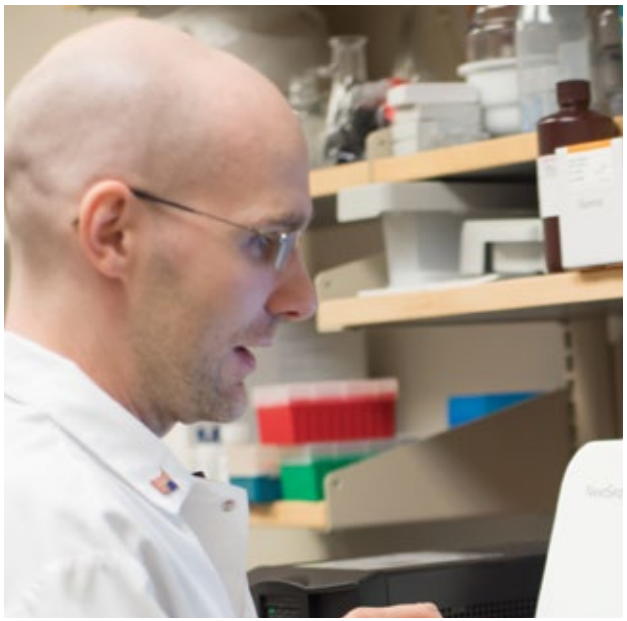
Leukemia Cells

Isaac

When he was 2, Isaac was diagnosed with leukemia. During two years of treatment, he received numerous blood transfusions as his cancer went into remission. However, in 2015, it came back and Isaac underwent a successful bone marrow transplant that saved his life. Now happy and healthy, Isaac wants to grow up and be a cancer researcher and give back to other patients just like him.

“If it wasn’t for someone donating bone marrow, my son... I don’t know where he’d be right now.” - Isaac’s mom, Fawnda

Core Laboratories



Modern biomedical research requires access to a wide range of specialized technologies. The Versiti Blood Research Institute maintains cutting-edge technology platforms that give researchers from the BRI, and its affiliates on the Medical College of Wisconsin (MCW) campus, access to state-of-the-art equipment and expertise. These centralized core laboratories are a shared resource and are staffed by technical specialists that support individual research projects. Currently, the BRI is home to 13 different core laboratories.

The **Molecular Cell Biology Core** offers DNA sequencing using both capillary-based and next-generation platforms and quantitative assays for DNA and RNA utilizing several different instrument platforms, such as a QuantStudio 6 Flex Real-time PCR system for rapid measurement of gene activity. Both 10X Genomics and Fluidigm systems are available for molecular analysis of single cells.

The **Protein Chemistry Core** synthesizes peptides using a microwave-enhanced Liberty 1 synthesizer and offers peptide purification and a variety of post synthesis peptide modifications. The Core aids investigators with protein purifications using AKTA and Agilent chromatography systems.

The **Hybridoma Core** produces murine and rat monoclonal antibodies for research and diagnostic purposes.

The **Flow Cytometry Core** utilizes two Becton Dickinson LSR II multicolor cytometers, one BD FACS Celesta, one BD Accuri cytometer, a BD FACSAria high-speed cell sorter, and a BD FACS Melody cell sorter.

The **Microscopic Imaging Core** includes the confocal/multiphoton laboratory featuring an Olympus FV1000-MPEconfocal, multiphoton microscope as well as an inverted Nikon TE200, a Nikon Eclipse Ti2 inverted fluorescence microscope, a Zeiss Axioscope and a Zeiss Lumar V12 stereo microscope with fluorescence capabilities. A PhD Imaging Specialist manages the Microscopic Imaging Core.

The **Biophysics Core** is equipped with a BIAcore 3000 Plasmon Resonance Spectrometer and an Octet Red 96 from forte BIO that enables scientists to study protein-protein interactions in real time.

The **Viral Vector Core** is shared between the BRI and MCW and specializes in vectors based on lentivirus, adenovirus and adeno-associated virus needed for research in the field of gene therapy and other experimental applications.

The **Thrombosis Core** maintains a spinning disk confocal microscope system for in vivo studies on thrombosis. This core also features an in vitro flow system designed to recapitulate the in vivo conditions of flowing blood in the vasculature (VenaFlux system from Cellix Ltd; Zeiss inverted microscope with phase contrast, fluorescence and incubation capabilities).

The **Histology Core** specializes in tissue preparation, cutting of fixed and frozen sections and various staining techniques. This core is staffed by a histology technician with 30 years of experience in experimental and clinical histology.

The **Gene Editing Core** is available as a resource for researchers that want to make targeted mutations in cells using recently developed CRISPR technology.

The **Computational Biology Core** is led by a PhD scientist who collaborates with BRI investigators to analyze and interpret their next generation sequencing data. The Core provides not only data analysis but also integration and development of new computational methods to analyze and integrate genome wide data sets.

The joint **BRI/MCW Transgenic Core** aids in the generation of genetically altered animal models for the study of human disease.

The **Clinical Trials Research Office (CTRO)** supports the work of our Clinical Investigators, interfacing with the other research support services at the BRI. Services provided by the CTRO include but are not limited to clinical trial design and activation, study coordination and management, data collection, adverse event reporting, regulatory support and compliance, budgeting and contract negotiation, and financial management.

Versiti Blood Research Institute Foundation

The Versiti Blood Research Institute Foundation raised a record amount of funds in 2019, thanks to many of you, who believe in and support the researchers highlighted in this report.

We kicked off our **Campaign for a Center for Stem Cell and Cell Therapy Research**, a $10.4M campaign that will help the Versiti Blood Research Institute grow in this cutting-edge field. Blood is the original cellular therapy, so it makes sense that Versiti takes the lead in developing these new treatments for a host of diseases, including cancer, hemophilia, and stroke, to name a few.

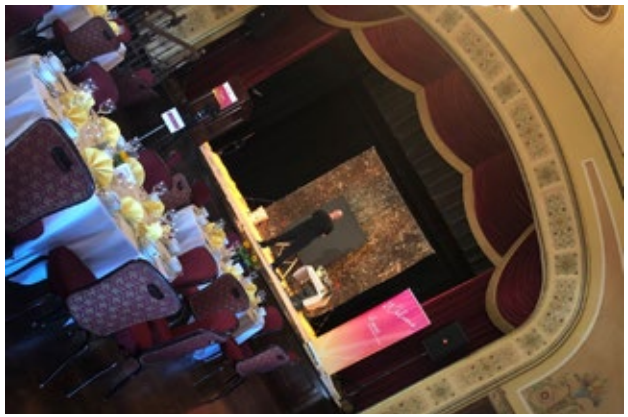
Two events that highlighted the important research taking place at the Versiti Blood Research Institute included:

Our **Research and Roses** event welcomed nearly 100 people to the BRI to hear from Drs. Peter Newman and Tom Abshire who talked about the connection between basic and clinical research – two fields that merge here at Versiti to benefit patients.

The **2019 Imagine Gala** was a fun and moving event held at the Wisconsin Club. We welcomed 220 guests who heard from Jack Zbiegien and Christine Roehling, both of whom have experienced, firsthand, the life-changing, patient-focused care that Versiti provides to our communities.

As the Foundation works to expand awareness of the Versiti Blood Research Institute in our community and beyond, we appreciate all that you do for us. You are our best advocates – sharing with your networks the important, life-changing work that happens at the BRI.

If you'd like to learn more about how you can help, please contact the Foundation office at 414-937-6799.



Intellectual Property

Novel Approaches to Help Patients

Basic and applied biomedical research studies are aimed primarily at understanding normal and abnormal biology. This aids disease diagnosis, treatment and prevention. Research findings impact patients and patient care when companies develop products and services from new discoveries. Intellectual property and patents help to differentiate and protect these new markets. Federal guidelines encourage protection of grant-supported discoveries through patents and other mechanisms that have the potential to transform research findings into products and services that benefit the health of the public.

The Technology Transfer Office of Versiti helps to identify, protect, and commercially partner discoveries to serve patient needs. Net revenues generated support further research. In 2019, four new patents were filed, and licensing income totaled $414,752. Versiti has inked more than 50 license agreements with industry partners. The number of new invention disclosures per research dollar increased 60% over 2018.

Mission Statement

The Technology Transfer Office supports Versiti's organizational mission of bringing life-saving solutions to the patient through a departmental focus on placing innovations into the hands of customers and colleagues.

Background

Inventor Tibor Greenwalt and colleagues discovered a white cell filtration method for blood in the 1960s. Patent activity increased in the 1980s with the discovery of the human platelet antigen system. Currently, the Technology Transfer Office provides intellectual property, contract management, and business management administrative services for the organization. A cross functional team called the Technology Transfer Review Group provides executive oversight for this function.

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