3. Xu R, Chan YP, Bradel-Tretheway B, Akyol-Ataman Z, [illegible] Skiniotis G, Lee B, Zhou ZH, **Broder CC**, [illegible] fusion Nipah Virus Fusion Glycoprotein Reveals a Novel Hexamer for Trimer Assembly. PLoS Pathog 8;11(12):e1005322. doi: 10.1371/journal.ppat.1005322. PMID: 26646856.
4. Mire CE, Satterfield BA, Geisbert JB, Agans KN, Borisevich V, [illegible] Chan YP, Cross RW, Fenton KA, **Broder CC**, Geisbert TW (2016). Pathogenic Differences between Nipah Virus Bangladesh and Malaysia Strains in Primates: Implications for Antibody Therapy. Sci Rep. 3;6:30916. doi: 10.1038/srep30916.

B. Positions and Honors

Positions and Employment

1990 -92 National Research Council, [illegible] Associate, Laboratory of Infectious Diseases, National Institutes of Health, Bethesda, Maryland.

1993 -96 IRTA Fellow, LVD, NIAID, NIH, Bethesda, Maryland.

Assistant Professor, Department of Microbiology and Immunology, Joint appointment, Molecular and Cell Biology Graduate Program, Uniformed Services University, Bethesda, Maryland.

2000 -05 Associate Professor, Department of Microbiology and Immunology, Joint appointment, Emerging Infectious Diseases Graduate Program, USUHS, Bethesda, Maryland.

2005 - [illegible] appointment, Emerging Infectious Diseases [illegible]

2006 -18 Director, Emerging Infectious Diseases Graduate Program, USU, Bethesda, Maryland.

2018 - Chair, Department of Microbiology and Immunology, USUHS, Bethesda, Maryland.

Other Experience and Professional Affiliations

2009 Member, National Veterinary Stockpile Nipah Virus [illegible] Australia

2011 Member, Discontools Nipah Virus Infection Panel Expert Group: Gap analysis. International Federation for Animal Health Europe, Brussels, Belgium

2011 Invited expert, National Academies, Washington, DC: Evaluation of site-specific risk assessment for the National Bio- and Agro-Defense Facility (NBAF), Manhattan, Kansas

Editorial Board of *J. of Virology* [illegible]

Honor and Awards

1996 The Fellows Award for Research Excellence, Office of Science Education, NIH

1996 American Association for the Advancement of Science, Science Magazine, Newcomb Cleveland Prize

1996 Outstanding Instructor in Virology, USUHS, School of Medicine

2005 The Henry W. Award for Excellence in Basic Science Research

2013 The 3rd Sidney Pestka Lecture: 22nd Annual PhD Philosophic Inquiry

2013 The 2013 Federal Laboratory Consortium (FLC) Award for Excellence in Technology Transfer

2013 Second Finalist for the [illegible] Prize

2013 The CSIRO Chairman's Medal, The Commonwealth Scientific and Industrial Research Organisation (CSIRO); Australia's national [illegible]

2014 The Cinda Helke Award for Excellence in Graduate Student Advocacy

2016 The James J. Leonard Award for Excellence in Translational/Clinical Research

2019 The 2019 Federal Laboratory Consortium (FLC) Award for Excellence in Technology Transfer

2019 USU Outstanding Biomedical Graduate Educator Award

C. Contributions to Science

1. **My Ph.D. thesis** [illegible] **discovery and characterization of a specific receptor for human plasmin on Group A Streptococci during a rotation p**[illegible] revealed that certain group A streptococci elaborated surface receptors [illegible] fibrinolytic enzyme, plasmin, [illegible] towards the zymogen precursor plasminogen. [illegible]

other serine proteases. T[illegible] bacterium-bound p[illegible] retained enzymatically ac[illegible] and its ability to hydrolyze a fibrin c[illegible] inhibitor. Since the [illegible] organism, is produced s[illegible] producing a[illegible] active enzyme tha[illegible] destruction of the extracellular matrix environment fi[illegible] the "flesh-eating streptococci".

a. Lottenberg R, **Broder CC**, Boyle MDP (1987). Identification of a Specific Receptor for Plasmin on a Group A Streptococcus. Infection and Immunity 55(6):1[illegible]-1[illegible].
b. **Broder CC**, Lottenberg R, Boyle MDP (1988). Mapp[illegible] Recognized by its Unique Group A Sp[illegible] 26[illegible]5.
c. **Broder CC**, Lotte[illegible] R, von Mering GO, Johns[illegible] eukaryotic plasmin rece[illegible] produced by the same microorganism. **J. Biol. Chem.** 266:4922-28.
d. Lottenberg R, **Broder CC**, Boyle MDP, Kain SJ, Schroeder BL, Curtis[illegible] Cloning, Sequence Analysis, and Expression in *Escherichia coli* of Strept[illegible] Plasmin Receptor. **J. Bacteriology** 174:5264-5270.

2. **My independent postdoctoral relationship to [illegible] on the early stages of HIV-1 envelope glycoprotein mediated membrane fusion**

vir[illegible] fusion and generated the first [illegible] of T-cell [illegible] and Macrophage-tropic HIV-1 e[illegible] type glycoprotein (Env) encoding recombinant vaccinia virus vectors and used these tools [illegible] cellular tropism [illegible] HIV-1 could be explained by specific [illegible] fusion factors required for the different classe[illegible] of HIV-1 Envs. I also developed the [illegible] st soluble [illegible] HIV-1 [illegible] glycoprotein and explored the impo[illegible] oligomeric structure in terms of its presentation conformational and virus-neutralizing epitopes through the development and characterization of more than 100 murine monoclonal antibodies.

a. **Broder CC**, Dimitrov DS, Blumenthal R, Berger EA (1993). The block to HIV-1 envelope glycoprotein-mediated membrane fusion in animal cells expressing human CD4 c[illegible] by a human cell component(s). **Virology** 193:483-491.
b. Nussbaum O, **Broder CC**, Berger E[illegible] Envelope Glycoprotein/CD4 Mediated Cell Fusion: A Novel Recombinant Vaccinia Virus-Based Assay Measuring Activation of a Reporter Gene by Bacterio-phage T7 RNA Polymerase Selectively in Fused Cells. **J. V[illegible]**
c. **Broder CC**, Earl PL, Long D, Moss B, Doms RW (1994). Antigenic implications of HIV-1 envelope glycoprotein quaternary structure: oligomer specific and sensitive mAbs. **PNAS** 91:11699-11703.
d. **Broder CC**, Berger EA (1995). Fusogenic Selectivity of the Envelope Glycoprotein Is a Major Determinant of HIV-1 Tropism for CD4+ T-Cell Lines vs. Macrophages. **PNAS** 92:9779-9783.

3. **My early studies on the cellular and viral membrane fusion tropism of HIV-1 and the development of a sensitive and specific reporter gene assay of cell-cell membrane fusion facilitated the discovery of the first [illegible]**

hypothesized existed, and this [illegible] macrophage-tropic Envs (CCR5) the HIV-1 coreceptor. These findings were significant beyond the work in HIV research leading to numerous new directions in understanding HIV pathogenesis as well as many therapeutic strategies.

a. Feng Y, **Broder CC**, Kennedy PE, Berger EA (1996). HIV-1 Entry Cofactor: Functional cDNA Cloning of a Seven-Transmembrane, G Protein-Coupled Receptor. **Science** 272:872-877.
b. Alkhatib G, Combadiere C, **Broder CC**, Feng Y, Kennedy PE, Murphy PM, Berger EA (1996). CC CKR5: a RANTES, MIP-1α, MIP-1β Receptor as a Fusion Cofactor for Macrophage-Tropic HIV-1. **Science** 272:1955-1958.

c. Rucker J, Samson M, Doranz BJ, Libert F, Berson JF, Yi Y, Collman RG, **Broder CC**, Vassart G, Doms RW, Parmentier M (1996). Regions in beta-chemokine receptors CC CKR5 and CC CKR2b Determine HIV-1 Coreceptor Specificity. Cell 87:1-[illegible]

d. Edinger AL, Amedee A, Miller K, Doranz BJ, Endres M, Sharron M, Samson M, Lu Z-H, Clements JE, Murphey-Corb M, Peiper SC, Parmentier M, **Broder CC** (1997). Differential utilization of CCR5 by macrophage and T cell tropic simian immunodeficiency virus strains. PNAS USA, 94:4005-4010.

4. **My initial work on HIV-1 entry led to further independent studies which investigations characterizing the roles of the HIV-1 coreceptors in the virus entry process.** These studies revealed the interplay between the HIV-1 entry coreceptors, pointed important determinants of the coreceptors involved in HIV-1 Env interaction, and also revealed possible avenues of how an HIV-1 Env might engage and differentially use the CXCR4 and CCR5 coreceptors, [illegible] collaborative follow-up studies exploring the utility of soluble oligomeric HIV-1 envelope glycoprotein gp140 subunit vaccine immunogens (gp140) which initiated at NIH while a postdoctoral fellow, with the unusual R3 HIV-1 Env isolate and led to the first NIAID project grant funded at USU.

a. Chabot DJ, Zhang PF, Quinnan GV, **Broder CC** (1999). Mutagenesis of CXCR4 Identifies Important Domains for HIV-1 X4 Isolate-Envelope Glycoprotein Mediated Membrane Fusion and Virus Entry and Reveals Cryptic Coreceptor Activity for R5 Isolates. J. Virol. 73:[illegible]

b. Xiao X, Wu L, Stamatatos L, Peng H, Vogel R, Chen H, Chabot DJ, **Broder CC**, Sattentau QJ, Dimitrov DS (1999). Conformational and functional surface association between CD4 and CCR5. PNAS 96:7496-7501.

c. Chabot DJ, Chen H, Dimitrov DS, **Broder CC** (2000). N-linked Glycosylation in CXCR4 Masks Coreceptor Function for CCR5-Dependent HIV-1 Isolates. J. Virol. 74:4404.

d. Zhang PF, Chen H, Dong M, Choudhary A, Bouma P, Zhang P, Dimitrov DS, Chabot DJ, Robinson J, Lewis GK, Zolla-Pazner S, Nussenzweig MC, Zhang L, Cherpelis S, Gorny MK, Parren PWHI, Burton DR, Sodroski J, Moore JP, Sattentau QJ, Wyatt R, Sanzari J, Cao Y, Chen Z, Katinger H, Montefiori DC, Ho DD, Wrin T, Petropoulos CJ, Ruprecht RM, Lewis MG, Spenlehauer C, Marx PA, Narayan O, Pauza CD, Clements JE, Duliege AM, Hendry RM, Tang S, Lu S, Pantophlet R, Burton DR, Doms RW, Moore JP, Stamatatos L, Barnett SW, Anderson DE, Pugach P, Martin-Garcia J, Cunningham P, Kleanthous H, Balachandran H, Shaw GM, Stamatatos L, Bailes E, Gao F, Bibollet-Ruche F, Hahn BH, Wei X, Chen W, Chen W, Zhou T, Zhu P, Byrum R, Yoder A, Staprans SI, Malenbaum S, Duliege AM, Cao J, Zhang P, Ba L, Fang G, Kotler DP, Jin C, Duliege AM, Cloyd MW, Kayman SC, Ruprecht RM, Choudhagra R, Baba TW, Isacson R, Ye SY, Voss G, **Broder CC**, Quinnan GV Jr (2007). Extensively cross-reactive anti-HIV-1 neutralizing antibodies induced by gp140 immunization. PNAS USA. 104(24):10193-8.

5. **My most recent research has been on emerging viruses that impact human and domestic livestock populations, including Australian bat lyssaviruses (rabies-like viruses), filoviruses (Ebola and Marburg) and the henipaviruses (Hendra and Nipah).** My lab was the first to publish on Hendra in Australia. I obtained the first NIAID funded project providing monetary support to an overseas laboratory (2[illegible] years, c[illegible] from structural biochemistry, *in vitro* [illegible] the development and testing [illegible] fusion inhibitors, subunit vaccine and neutralizing human monoclonal antibodies (mAb), and supported the development of the feline and ferret models of Hendra and Nipah infection and pathogenesis in Australia and the development of the first henipavirus p[illegible] the *in vivo* efficacy of the Hendra/Nipah vaccine (HeV-sG) and an anti-HeV/NiV G-specific neutralizing human mAb. One human mAb (m102.4) [illegible] used by compassionate use on emergency protocol in 15 people in Australia and one individ[illegible] United States, [illegible] 2016. The henipavirus subunit vaccine, HeV-sG, was launched; called Equivac® HeV (Zoetis, Inc.) and is the [illegible] of the henipavirus entry receptors (ephrin-B2/B3) and produced soluble versions of the G and [illegible] facilitating their structural solutions.

a. Bossart KN, Zhu Z, Middleton D, Klippel J, Crameri G, Bingham J, McEachern JA, Green D, Hancock TJ, Chan YP, Hickey AC, Dimitrov DS, Wang LF, **Broder CC** (2009). A neutralizing human monoclonal antibody protects against lethal disease in a new ferret model of acute Nipah virus infection. PLoS Pathogens 5:[illegible].

b. Bossart KN, Geisbert TW, Feldmann H, Zhu Z, Feldmann F, Geisbert JB, Mire CE, Donaldson EF, Hashiguchi T, Razuvajevski ES, Grolla A, Wong G, Engelhardt JA, Yan L, Feldmann F, Arnantz C, Yang Y, Dimitrov AS, Callison J, Chan Y-P, Hickey AC, Dimitrov DS, **Broder CC**, Collin N, Barrett PN, Marzi A, Fouchier RAM, Feldmann H (2011). Model of Ebola virus pathogenesis in aerosol exposed guinea pigs. PLoS ONE 6:[illegible].

2005 [illegible] Medical Institute, student intern, [illegible]
National Institute of Neurolog[illegible]
2005-06 [illegible] graduate resea[illegible]
Maryland, College Park, MD.
2007 -08 Undergraduate [illegible] research assistant, Biology Department, Honors research, Department of Biology, University of Maryland, College Park, MD.
2008 -09 Research assistant, Department of Pharmacology, Uniformed Services University, Bethesda, MD.
2010 Research assistant, Department of Microbiology, Uniformed Services University, Bethesda, MD.
2010 -16 Graduate research student, Department of Microbiology, Uniformed Services University, Bethesda, MD.
2016 -17 Postdoctoral fellow, Henry M. Jackson Foundation, Department of Microbiology, Uniformed Services University, Bethesda, MD.
2017 -18 Scientist, Henry M. Jackson Foundation, Department of Microbiology, Uniformed Services University, Bethesda, MD.
2019 - Research Assistant Professor, Uniformed Services University, Department of Microbiology and Immunology, Uniformed Services University, Bethesda, MD.

Other Experience and Professional Membership

2009 Mentor, A[illegible] student mentoring, Bethesda-Chevy Chase High School, Bethesda, MD.
2009-10 Mentor, [illegible], Washington, D.C.
2013 Mentor, high school, undergraduate, and graduate students, Uniformed Services University, Bethesda MD.
2014 Participant, American Society of Microbiology, Kavli Institute
2014 -15 Volunteer, AAAS/Senior Scientists and Engineers STEM Volunteer Program
2014 -17 Member, American Society of Tropical Medicine and Hygiene
2014 -19 Member, American Society of Microbiology
2015 -16 Member, USU Global Health Interest Group

Honors

2[illegible]
2005 -07 Semester [illegible] Honors
2006 College Park Life Sciences Scholars Program, [illegible]
2008 High Honors, Biology Departmental Honors Program
2015 USU Research Days Gr[illegible]
2015 NSF East Asia and Pacific S[illegible]
2015 -16 Val G. Hemming Fellowship, Henry M. Jackson Foundation

C. C[illegible]

1. **Virus-host interactions:** My Ph.D. thesis research was focused on virus-host interactions; understanding bats as hosts of zoonotic viruses and Australian bat lyssavirus (ABLV) cellular entry. [illegible] exploring the antiviral mechanisms that enable cellular persistence of viruses in bats, particularly, authophagy. Findings revealed that the autophagy pathway is induced upon infection with Australian bat lyssavirus (ABLV), a rabies virus-related virus carried by Australian flying fox bats. The combined pharmacological and genetic studies of the autophagy pathway in the [illegible] indicated that autophagy functions as an antiviral defense. The study also demonstrated that bat-derived cell lines have elevated levels of basal autophagy, [illegible] contribute to the ability of bats to act as host to these viruses. An additional finding from these studies was that activation of autophagy may have therapeutic benefits during [illegible] virus infection.

Wellcome Trust: Tropical Medicine Interest Group, Member
Member
Gates Foundation: Founding Board, Global Alliance to Improve Nutrition; Grand Challenges in Global Health, Scientific Advisory Board; Scientific Advisory Committee, Member
One Health Commission: Advisors
Consortium of Universities for Global Health
National Center for Synthetic Engineering and Biotechnology, National Science and Technology Development
Institute for Healthcare Improvement: Scientific
Council on Health Research for Development
Nevin Scrimshaw International Nutrition Foundation
American Federation for Clinical Research: Member
American Society for Microbiology: Member and Fellow
American Association for the Advancement of Science: Member
American Society for Clinical Investigation
Association of American Physicians: Member
New York Academy of Sciences: Member

Honors

1972 - American Board of Internal Medicine, Diplomate, Internal Medicine and Infectious Disease
1973 -76 Career Scientist Award, Health Research Council of the City of New York
1974 -79 Research Career Development Award, NIAID
1981 Oswald Avery Award, Infectious Diseases Society of America
1991 Heath Clark Lecturing Professor, University
1997 Maxwell Finland Lectureship, Infectious Diseases Society of America
2000 Edward R. Barsky Award, Physicians Forum/Physicians for Social Responsibility
2002 Alexander Fleming Award, Infectious Diseases Society of America
2002 National Academy of Medicine, Elected Member
2005 Rockefeller Fellowship, Rockefeller Foundation, Bellagio
2013 Distinguished Leadership, [illegible]

Contributions to Science

1. **I rediscovered Shiga toxin during my fellowship, while participating in a training program at the Institute of Nutrition for Central America and Panama in 1**[illegible] I have focused on Shiga toxin and its role in pathogenesis. The major recognized virulence factor of *Shigella* was its ability to invade gut epithelial cells. The previously described Shiga toxin was thought until I showed that a protein produced by *Shigella dysenteriae* [illegible] damage of the bowel and body inflammatory exudates similar to dysentery in a rabbit model. [illegible] proved the two toxin activities were due to the same protein. My work [illegible] (Stx), sequenced the binding subunit, identified its mammalian cell receptor, described its translocation to the cell cytoplasm via receptor [illegible] and identified [illegible] receptor and [illegible]. This work paved the way to understand the pathogenesis of *E. coli* O157 and other serotypes associated with hemorrhagic colitis and hemolytic-uremic syndrome [illegible]. My [illegible] essential for a rapid commercial diagnostic test for all Stx producing bacteria. I was principal investigator [illegible] all of these studies.

   a. **Keusch GT**, Grady GF, Mata L, McIver JM (1972). The pathogenesis of *Shigella dysenteriae* 1 (Shiga) Enteritis: [illegible]

b. D[illegible] Shigella diarrhea. IX. Simplified high yield purification of Shiga toxin and characterization of subunit composition, [illegible] by the use of [illegible] monoclonal antibodies. **J. E[illegible] Med**. 1[illegible]

c. Jacewicz M, Clausen H, Nudelman E, Donohue-Rolfe A, Keusch GT (1986). Pathogenesis of Shigella diarrhea. XI. Isolation of a shigella toxin binding glycolipid from rabbit jejunum and HeLa cells and its identification as globotriaosylceramide. **J. Exp. Med.** 163:1391-1404.

d. Kandel G, Donohue-Rolfe A, Donowitz M, Keusch GT (1989). Pathogenesis of Shigella diarrhea. XVI. Selective targetting of Shiga toxin to villus cells of rabbit jejunum explains the effect of the toxin on intestinal [illegible]

2. **In the [illegible]980's I began work** on the mo[illegible] parasite to identify a trypsin activated mannose-6-P lectin mediating binding to mammalian cell surfaces. These properties were consistent with [illegible] with activation in upper small bowel [illegible] We also discovered a Cryptosporidium parvum lectin which mediated binding to mammalian cells, and showed it is a member of a family of mucin-like glycoproteins containing N-acetylgalactosamine. Together these pioneering studies de[illegible] the pathogenesis of intestinal protozoal infections. I was primary or co-investigator in all of these studies.

a. Lev B, Ward H, **Keusch GT**, Pereira MEA (1988). Trypsin activation in Giardia lamblia by host proteases: A novel host parasite interaction. **Science** 239:71-73.

b. Ward HD, Alroy J, Lev BI, **Keusch GT**, Pereira ME[illegible] (1988). Analysis of [illegible] carbohydrates of Giardia lamblia. Detection of N-acetyl-D-glucosamine as the only saccharide moiety and identification of two distinct subsets of trophozoites by lectin binding. **J. Exp. Med.** [illegible]

c. Hamer DH, Ward H, Tzipori S, Pereira MEA, Alroy JP, **Keusch GT** (1994). Attachment of Cryptosporidium parvum sporozoites to MDCK cells in vitro. **Infect Immun** 62:2208-13.

d. Ortega-Barría E, Ward HD, **Keusch GT**, Pereira MEA (1994). Growth inhibition of the intestinal parasite Giardia lamblia by a glycan binding [illegible] 2288.

3. **I have made multiple contributions to the understanding of nutrition-infection interaction in laboratory and field research.** I developed a rat model of [illegible] malnutrition and demonstrated macrophage functional abnormalities, including chemotaxis, phagocytosis, and intracellular bacterial activity. Together with colleagues in Guatemala [illegible] malnourished children and [illegible] decreased s[illegible] Type [illegible] activity, complement deficiency, [illegible] deficits, and their reversal with nutrition interventions. Subsequent studies in Zaire (now DRC) in AIDS [illegible] elevated pro-inflammatory cytokine levels that could drive metabolic shifts underlying cachexia. HIV-infected but clinically stable non-wasted subjects also had high pro-inflammatory cytokine levels; however this was countered by elevated levels of the antagonist cytokines IL-1RA and TNF α-soluble receptor p55 at a molar ratio known to block inflammatory effects of IL-1β and TNF α in vitro. This was the first biologic plausible mechanism to explain the observation of weight and body composition in long-term clinical non-progressors.

a. **Keusch GT**, Douglas SD, Hammer G, Braden K (1978). Macrophage [illegible] experimental protein calorie malnutrition. II. Cellular and humoral factors for chemotaxis, phagocytosis and intracellular bacterial activity. [illegible]

1982 -94 Associate Professor of Immu[illegible]
1994 - Professor and Chair, Department [illegible] University School of Medicine
2007 -14 Associate Director, National Emerging Infectious Diseases Laboratories Institute, Boston University
2009 -14 Associate Provost for Research, Boston University Medical Campus
2014 - Director, National Emerging Infectious Diseases Laboratories Institute, Boston University

Other Experience and Professional Membership

1989 -92 Immunobiology Study Section, NIH
1996 -00 Immunobiology Study Section, NIH
2004 Immunobiology Study Section, NIH
1978 - American Association of Immunologists
1991 - American Society for Microbiology
1992 -93 [illegible]
1997 -98 Chair, Cell Biology [illegible]
2000 Chair, Cell Biology and Immunology Predoctoral Committee, HHMI/NRC
2001 -03 Member, Research Training Fellowships for Medical Students Committee, HHMI
2002 Member, Research Training Fellowships for Medical Students Committee, HHMI
2002 Advisory Panel [illegible]
2005 -06 Chair, "Med int[illegible] Review Committee, HHMI
2007 Member, NIH Review Panels on "B Cell Immunology and [illegible] Vaccines"
2009, 10 Member, NIH Review Panels on "Basic HIV Discovery Research"
2011 -13 Member, *ad hoc* Review Panel, "Immune Mechanisms of Virus Control" [illegible] NIH/NIAID
2011 -14 SmithGroup JJR Science & Technology Advisory Board
2016, 17 Reviewer, National Research Foundation, Competitive Research Program, Singapore
Ongoing: Security Risk Assessment (SRA) [illegible] CDC Program in Biological Select Agents and Toxins (BSAT)
Ongoing: BSL-4 suit-trained and certified, Boston University

Honors

1979 [illegible]
2015 Fellow of [illegible]

C. Contributions to Sci[illegible]

1. **Innate role of B lymph[illegible]** [illegible] secreted IgM antibodies had unique functions in concentrating pathogens and antigen into secondary lymphoid organs, and prevented dissemination into vital organs. We sought [illegible] responsible for these activities, and to understand the consequences for the immune system. We demonstrated that IgM immune complexes became co[illegible] transported these com[illegible] innate role for this subset of B lymphocytes in the early steps of initiation of primary immune responses. A role for orchestrated transport of antigen and immune complexes [illegible] widely accepted as early events in immune responses.

   a. Ferguson AR, **Corley RB** (2005). Accumulation of marginal zone B cells and accelerated loss of follicular dendritic cells in NF-κB p50-deficient mice. **BMC Immunology** 6:8.
   b. Ferguson AR, Youd ME, **Corley RB** (2001). Marginal zone B cells [illegible] immune complexes o[illegible] follicular dendrit[illegible] month").
   c. Youd ME, Ferguson AR, **Corley RB** (2002). Synergistic roles of IgM and complement in antigen transport and follicular localization. **Eur. J. Immunol.** 32: 2[illegible]-2337.

2. **The [illegible] of the [illegible] forms of IgM antibodies in immune responses.** Data from my laboratory and others had [illegible] indicated that IgM antibodies were not always secreted as pentameric molecules with J chain, but [illegible] evidence existed to indicate [illegible] shared functions with pentameric IgM, or if they had unique functions. [illegible] monomers, had discrete activities. [illegible] could be deleterious [illegible] autoimmune diseases. [illegible] monomers did not fix complement, [illegible] ability to function in antigen trapping, and could also accelerate disease manifestations in autoimmune prone mice. These data supported the important role for strict quality control standards in the assembly and secretion of IgM antibodies for maintenance of proper homeostasis in the immune system.

a. Youd ME, Luus L, **Corley RB** (2007). IgM monomers accelerate disease manifestations in autoimmune prone *fas*-deficient mice. **J. Autoimmunity** [illegible]: 333-343.
b. Hughey CT, Brewer JW, C[illegible] normal and autoimmune B cells: Implications for the physiologic role of IgM hexameric IgM. **J. Immunol.** 161: 4091-4097.
c. Brewer JW, **Corley RB** (1997). Late events in assembly regulate the polym[illegible] activity of secretory IgM. **Mol. Immunol.** 34: 33[illegible]
d. Brewer JW, Randall TD, Parkhouse RME, **Corley RB** (1994). IgM hexamers? **Immunol. Today** 15: 165-168.

3. **Quality control in modulating the assembly and [illegible]** [illegible] whether the addition of J chain was responsible for catalyzing assembly of IgM into [illegible] assembly occurred was controversial. We demonstrated, however, that IgM assembly is regulated in the endoplasmic reticulum by a process involving thiol regulation. Further, J chain plays a role in mediated IgM assembly, and its addition is a terminally late step in the production of polymeric IgM. To complete these studies, we made use of various biochemical assays including pulse chase experiments. We also cloned a [illegible] J chain to demonstrate its role in modulating polymer assembly, and this work remains the definitive description of IgM assembly.

a. Reddy PS, **Corley RB** (1999). The [illegible] secretory IgM. **Immunology Today** 20: 582-588.
b. Brewer JW, **Co[illegible]** [illegible] the redox state of proteins in the endoplasmic reticulum [illegible]
c. Brewer JW, Randall TD, Parkhouse RME, **Corley RB** (1994). Mechanism and subcellular location of secretory IgM polymer assembly. **J. Biol. Chem.** 269: 17338-17348.
d. Randall TD, Brewer JW, **Corley RB** (1992). Direct evidence that J chain regulates the polymeric structure of IgM in antibody secreting B cells. **J. Biol. Chem.** 267: 16606-16607.

4. **Defining mouse [illegible] mammary tumor virus as an endogenous [illegible] of B lymphocytes in the MMTV life cycle.** Prior to these studies there was evidence for the existence of that endogenous superantigens which played important roles in shaping [illegible] cell repertoires in mice, but the identity [illegible] process, we identified [illegible] mammary tumor virus, Mtv-9, as [illegible] activation of B cells, and [illegible] cycle.

a. Sharma S, King LB, **C[illegible]** of endogenous mouse mammary tumor proviral *env* transcripts following B cell stimulation. **J. Immunol.** 141: 2510-2518.

1;110(40):16157-62.

4. Menachery VD, Yount BL, **Sims A**, Agnihothram S, Gralinski LE, Plante JA, Graham RL, Scobey Royal S, Pickles RJ, Randell SH, Lanzavecchia A, [illegible] WIV1-CoV poised for human emergence. **PNAS** 15;113(11):3048-53.

B. Positions and Honors

Positions and Employ[illegible]

1993 American [illegible]
1994 Albert Einstein College of Medicine Summer Student Award
1996 -01 Graduate Student, Laboratory of Mark Denison, Vanderbilt University, Nashville, TN
1999 Dissertation Enhancement Award, Vanderbilt University
2001-02 Postdoctoral Fellow, Duke University, [illegible]
2002 -04 Infectious Disease Pathogenesis Training Grant Fellow, [illegible]
2002 -05 Postdoctoral Fellow, University of North Carolina at Chapel Hill
2005 -17 Research Assistant Professor, Department of Epidemiology, [illegible]
2017- [illegible]

C. Contr[illegible]

1. In vitro m[illegible] [illegible] newly identified or emerged hu[illegible] human conducting airway can be cultured at an air liquid interface and following maturation recapitulate the morphology of the airway epithelium. These cultures provide a unique in vitro model and for one human coronavirus, HKU1, provide the only in vitro model for studying this virus.

a. **Sims A**, Pyrc K, Dijkman R, Jebbink M, Long C, Deming D, Donaldson E, Vabret A, Baric R, van der Hoek L, Pickles R (2010). Culturing the unculturable: human coronavirus HKU1 infects, replicates, and produces progeny virions in human ciliated airway epithelial cell cultures. **J Virol**. 84(21):11255-63.

b. **Sims A**, Baric RS, Yount B, Burkett SE, Collins PL, Pickles RJ (2008). SARS-CoV infection of human ciliated airway epithelium: the role of the ciliated cell in viral spread in the conducting airways of the lung. **J Virol**. 79(24):15511-15524.

c. Huang Y, Dong W, Milewska A, Golda A, Qi Y, Zhu Q, Marasco W, Baric R, **Sims A**, Pyrc K*, Li W, Sui J* (2015). HCoV-HKU1 Spike protein uses O-acetylated sialic acid as a receptor determinant and employs HE protein as a receptor-destroying enzyme. **J Virol**. 89(14):7202-13. *indicates co-senior author.

2. Development of coronavirus infectious clones. The generation of coronavirus infectious clones has drastically increased the understanding of how specific genes or open reading frames affect replication and pathogenesis as well as identifying sets of mutations that can make coronavirus genome [illegible] proof live [illegible] vaccine candidates.

a. Thornbrough JM, Jha BK, Yount B, Goldstein [illegible] Weiss SR (2016). Middle East Respiratory Syndrome Coronavirus NS4b Protein Inhibits Host RNase L Activation. **MBio** 29;7(2).

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the virus [illegible] nd cell inte[illegible] ct over a 7[illegible]
Role: [illegible] ect [illegible]