3. Xu K, 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-of-Trimers [illegible] 8;11(12):e1005322. doi: 10.1371/journal.ppat.1005322. PMID: [illegible]
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 [illegible]

1990 -92 National Research Council [illegible] Associate, Lab[illegible] Allergy and Infectious Diseases, National Institute 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, [illegible] 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 [illegible]

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, [illegible] 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 [illegible]

1996 American Association for the Advancement [illegible] Magazine, Newcomb Cleveland Prize

1996 Outstanding Instructor in Virology, USUHS, School of Medicine

2005 The Henry Wu Award for Excellence in Basic Science Research

2013 [illegible] 3rd Sidney Pestka Lecture; 22nd Annual [illegible]

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

2013 Second Finalist for the [illegible] Infectious Diseases Research Centre Eureka Prize

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

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 ([illegible]) Award for Excellence in T[illegible]

2019 USU Outstanding Biomedical Graduate Educator Award

C. Contributions to Science

1. **My Ph.D. thesis [illegible] centered on the discovery and characterization of a specific receptor for human plasmin on Group A Streptococci during a rotation [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, fin[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(8): 1915-1918.

b. **Broder CC**, Lottenberg R, Boyle MDP (1988). Mapping [illegible] Recognized by [illegible] Unique [illegible] 26[illegible]5.

c. **Broder CC**, Lotten[illegible] R, von Mering GO, Johns[illegible] [illegible] eukaryotic plasmin rece[illegible] [illegible] produced by the same microorganism. **J. Biol. Chem.** 266:4922-28.

d. Lottenberg R, **Broder CC**, Boyle MDP, Kain SJ, Schroeder BL, Curtiss [illegible] Cloning, Sequence Analysis, and Expression in *Escherichia coli* of a Streptococcal Plasmin Receptor. **J Bacteriology** 174:5285-5293.

2. **My independent postdoctoral relationship [illegible] on the early stages of HIV-1 envelope glycoprotein-mediated** virus [illegible] fusion and generated the first panel of T-cell [illegible] and Macrophage-tropic HIV-1 envelope glycoprotein (Env) encoding recombinant vaccinia virus vectors and used these tools [illegible] cellular tropism of HIV-1 could be explained by specific [illegible] fusion factors required for the different classes of HIV-1 Envs. I also developed the first soluble [illegible] glycoprotein and explored the [illegible] oligomeric structure in terms of its presentation of 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 can be overcome by a human cell component(s). **Virology** 193:483-491.

b. Nussbaum O, **Broder CC**, Berger EA (1994). Fusogenic Mechanisms of Enveloped-Cell Fusion 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. **JV** 68:5411-5422.

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** 92:9004-9008.

3. **My early studies on the cellular and viral mechanisms of 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 breakthroughs in HIV research leading to numerous new directions in understanding HIV pathogenesis as well as new therapeutic strategies.

a. Feng Y, **Broder CC**, Kennedy PE, Berger EA (1995). 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. R[illegible]cker J, Samson M, Doranz BJ, Libert F, Berson [illegible] Y, Collman RG, **Broder CC**, [illegible]sart G, Doms R[illegible] ([illegible]96). Regions in [illegible] Chemokine R[illegible] Determine HIV-1 Cof[illegible] Specificity. **Cell** 87:1[illegible]
d. Edin[illegible] AL, Amedee A, Miller K, Dor[illegible] [illegible]J, Endres M, Shar[illegible]n M, Samson M, Lu Z-h, Clements JE, Murphey-Corb M, Peiper SC, Parmentier M, **Br**[illegible] (1997). Differential utilization of CCR5 by macrophage and T cell tropic simian immunodeficiency virus strains. **PN**[illegible] USA. 94:4005-4010.

4. **My initi[illegible] work on HIV-1 entry led to further independent studies whi[illegible] investigations characte[illegible] the roles of the HIV-1 coreceptors in the virus entry process.** These studies revealed the interplay between the HIV-1 entry [illegible]ceptors, mapped important [illegible] of the coreceptors involved in HIV-1 Env interaction, and also revealed possible avenues of how an HIV-1 Env might engage an[illegible] differently utilize the CXCR4 and CCR5 corec[illegible] en[illegible] collaborative follow-up studies exploring the utility of soluble oligomeric HIV-1 envelope glycopro[illegible] subunit vaccine immunogens (gp140) which I initiated at NIH while [illegible] postdoctoral [illegible]ellow, with the unus[illegible] R2 HIV-1 Env isolate and led t[illegible] first NIAID pr[illegible] project grant funded at USU.

a. Chabot DJ, Zhang PF, Quinnan GV, **Broder CC** (1999). Mutagenesis of CXCR4 [illegible] Importa[illegible] Domains for HIV-1 X4 Isolate Envelope-Mediated Membrane Fusio[illegible] entry and Reveals Cryptic Corecep[illegible] Activity for R5 isolates. **J Vir**[illegible] 73:[illegible]
b. Xiao X, Wu L, St[illegible] [illegible], **Broder CC**, [illegible] Dimitrov DS (1999). [illegible] surface association between CD4 a[illegible]CCR5. **PNAS** 96:7496-7501.
c. Chabot DJ, Chen H, Di[illegible] **Broder CC** (2000). N-linked Gly[illegible]ylation in CXCR4 Masks Coreceptor Function for CCR5-Dependent HIV-1 Isolates. **J Virol** 74:44[illegible]
d. Zhang PF, Cham F, Dong M, Choudhary A, Bouma P, Zha[illegible] N, 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 popula[illegible] including Australian bat lyssavirus ([illegible]-like virus), filoviruses (Ebola and Marburg) and the he[illegible]viruses (Hendra and Nipah).** My lab was the first to [illegible]blish on Hendra [illegible] Australia. I obtained the first NIAID funded project providing monetary sup[illegible] an overseas laboratory (2[illegible] years, c[illegible] from structural biochemistry, in vivo [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 developm[illegible] of the first nonhuman 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 (m1[illegible] [illegible] used by compassionate ther[illegible] protocol in 13 people in Australia and one indivi[illegible] United State[illegible] 2016. The henipavirus subunit vaccine, HeV-sG, was launched; called Equivac® HeV (Zoetis, Inc.) and is the f[illegible] of the henipavirus entry receptors (ephrin-B2/B3) and produced solub[illegible]e versions of the G an[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 Y-P, Hickey AC, Dimitrov DS, Wang L-F, **Bro**[illegible] (2009). A neutralizing human monoclonal antibody protects against lethal disease in a new ferret model of acute Nipah virus infection. **PLoS Pathogens** [illegible]. PMCID: PMC2753220.
b. Bossart KN, Geisbert [illegible] Feldmann H, Zh[illegible] D, Scott [illegible] Yang Y, Dimitrov AS, Callison J, Chan Y-P, Hickey AC, Dimitrov DS, **Broder** [illegible]

2005 -06 Undergraduate student intern, Medical Institute, student intern, C
National Institute of Neurolog
2005 -08 Undergraduate resea Pass
Maryla College Park, MD.
2007 -08 Undergraduate h assistant, Biology Department Honors research, Departme
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 Po doctoral Fellow, Henry M. Jackson Foundation, Department of Microbiology, Unifo
Services Univ , Bethesda, MD.
2017 -18 Sc Henry Mo Foundation, Department of Microbiology, Unifo
University, Bethesda, MD.
2019 - Research Assistant Professor, Uniformed Services Univ Department of Microbiology and Immunology, Uniformed Services University, Bethesda, MD.

Other Experience and Professional Membership

2009 Mentor, A
20 -10 Me
2013 Mentor, high school, undergraduate, and graduate students, Uniformed Services University, Bethesda MD.
2014 Partici American Society of Mic
2014 -15 Vo 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 Glob Health Interest

Honors

2
2005 -07 Se Honors
2006 College Park Life Sciences Scholars Pr
2008 High Honors, Biolo Departmental Honors Program
2015 USU Research Days Gr
2015 NSF East Asia and Pacific S
2015 -16 Val G. Hemming Fellowship, Henry M. Jackson Founda

C. C

1. **Virus-host inter ctions:** My Ph.D. thesis research was focused on virus-host interactions; understanding bats as hosts of zoono ses and Australian bat lyssavirus (ABLV) cellular entry, exploring the antiviral me chanisms that enable cellular persi authophagy. Findings revealed that the autophagy pathway is induced upon in ection with Australian bat lyssa virus (ABLV), a rabies virus-related virus carried by Australian flying fox bats. The studies pharmacological and genetic studies of the autophagy pathway in the indicated that autophagy functions as an antiviral defense. The study also dem onstrated that bat-derived cell lines have elev ated levels of basal autophagy, which contribute to the ability of bats to act as host to these viruses. An additional finding from these studies was that activation of autoph may have therapeutic benefits d infection.

Wellcome Trust: Tropical Medicine Interest Group, Member [illegible]
Member
Gates Foundation: Founding Board, Global Alliance [illegible] Improve Nutrition; Grand Challenges in Global Health, Scientific Advisory Board [illegible] sory Committee, M [illegible]
One Health C [illegible] visors
Consortium of Universiti [illegible]
National Center for Genetic Engineering and Biotechnology, National Science and Technology Development [illegible]
Institute for Healthcare Improvement: Scientific [illegible]
Council on Health R [illegible]
Nevin Scrimshaw International Nutrition Found [illegible]
American Federation for Clinical Research: Me [illegible]
American Society fo [illegible]
American Association for the Advancement of Science: Member
American Society [illegible]
Association of American P hysicians: Member
New York Academy of Sciences: Member

Honors

1972 - American Board of Internal Medicine, Diplomate, Internal Medici [illegible]
1973 -76 Career Scientist Award, Health R [illegible]
1974 -79 Research Career Development Award, NIAID
1981 Oswald Avery Award, Infectious Diseases Society of America
1991 Heath [illegible] ng Professor, University [illegible]
1997 Maxwell Finland Lectureship, Infectious Diseases Society of America
2000 Edward R. Barsky Award, Physicians Forum/Physicians for Social Re [illegible]
2002 Alexander Fleming Award, Infectious Diseases Society of America
2002 National Academy of Medicine, Elected Member
[illegible]
2013 Disting [illegible] ed Lead [illegible]

C. Contributions to Science

1. **I rediscovered Shiga toxin during my fellowship, while participating in a training [illegible] Institute of Nutrition for Central America and Panama in** [illegible] has focused on Sh [illegible] and its role in pathogenesis. The major recognized virulence factor of Shigella was its ability to invade gut epithelial cells; the previously described Shiga "neurotoxin" was long [illegible] until I showed that a protein pro [illegible] *Shigella dysenteriae* [illegible] damage of the bowel a [illegible] body inflammatory exudat [illegible] ar to dysentery in a rabbit model [illegible] proved the two toxin activities were due to the same protein. My wo [illegible] of Shiga (Stx), sequenced the binding subunit, identified its mammalian cell receptor, described its translocation to the cell cytoplasm via recepto [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] essential for a rapid commercial diagnostic test for all Stx-producing bacteria. I was principal in [illegible] all of these st [illegible]

   a. **Keusch GT**, Grady GF, Mata L, McIver JM (1972). The pathogenesis of Shigella [illegible] Ente [illegible] 51:1212-1218.

b. D[illegible]
Sh[illegible]la diarrhea. IX. Simplified high yield purification of S[illegible] toxin and charac[illegible]tion of subunit composition [illegible] by the use of s[illegible] monoclonal antibodies. **J. E[illegible] Med** [illegible]
c. Jacewicz M, Clause[illegible]H, Nudelm[illegible] Donohue-Rolfe A, Keusch GT (19[illegible]). Patho[illegible] 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** (19[illegible]). Pathogenesis of Shigella d[illegible] Selective targetting of Shiga toxin to villus cells of rabbit jejunum explains the eff[illegible] of the toxin [illegible] intestin[illegible]

2. **In th[illegible]980's I began work on the m[illegible]**
fi[illegible] parasi[illegible] These properties were cons[illegible] with activation in u[illegible] We also discovered a Cryptosporidium parvum le[illegible] which mediated binding to mammalian cells, and showed it is a member of a family of mucin-like glyc[illegible]teins containing [illegible]-acetylgalactosamine [illegible] Together these p[illegible] the pathoge[illegible] [illegible]ese studies.

a. Le[illegible]B, Ward H, **Keusch GT**, Pereira MEA (19[illegible]) [illegible]ctin activation in G[illegible] by host protea[illegible] A novel host parasite i[illegible]

b. Ward HD, Alroy J, Lev BI, **Keu[illegible]** (1988). Ana[illegible] Giar[illegible] [illegible] two distinct subs[illegible] of trophozoites by lectin b[illegible] **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-Barr[illegible] E, Ward HD, **Keusch GT**, Pereira MEA (1994). Growth inhibition of the in[illegible]stinal parasite Giardia lambli[illegible] by a dietary [illegible] **J. Clin. I**[illegible] 2288.

3. **I have made multiple contributions to the understanding of nutrition-infecti[illegible] inter[illegible] laboratory and field research.** I developed a rat model of [illegible]gy malnutrition and demonstrated macrophage functional abnorm[illegible]ities, including chemotaxis, phagocytosis, and intracellular bacter[illegible] activity. Together w[illegible] colleagues in Guate[illegible] malnourish[illegible] children [illegible], decreased s[illegible] comple[illegible]nt deficiency [illegible] T-c[illegible] deficits, and the[illegible] reversal with [illegible]entions. Subseque[illegible] st[illegible] Zaire (now DR[illegible]) [illegible] elevat[illegible] pro-inflammatory cytokine levels that could drive metabolic shifts underlying [illegible]chexia. HIV-infe[illegible] stable non-was[illegible]bjects also had high pro-inflammatory cytokine levels; however this was countered by elevated levels of the antagonist cytokines IL-1RA and TNFα-soluble receptor p55 at a molar ratio known to block inflammatory effects of IL-1β and TNFα in vitro. This was the first biologic[illegible] plausible [illegible] to explain the preservation of weight and body composition in long term clinical non-progressors.
a. **Keusch GT**, Douglas SD, Hammer G, Braden K (1978). Macroph[illegible] experimental protein calorie malnutrition. II. Cellular and humoral factors for chemotaxis, phagoc[illegible] and intracellular ba[illegible]ericidal acti[illegible]

1982 -94 Associate Professor of Immunology, [illegible]
1994 - Professor and Chair, Department of [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 Memberships

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
2000 Chair, Cell Biology and Immunology Predoctoral Committee, HHMI/NRC
2001 -03 Member, Research Training Fellowships for Medical Students Committee, HHMI
2001 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 Protective HIV-1 Vaccines"
2009, 10 Member, NIH Review Panels on "Basic HIV Discovery Research"
2011 -13 Member, *ad hoc* Review Panel, "Immune Mechanisms of Virus Control" Program, 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] for CDC/US Department of Health and Human Services Biological Select Agents and Toxins (BSAT)
Ongoing: BSL-4 suit-trained and certified, Boston University

Honors

1979 [illegible]
2015 Fellow of [illegible]

C. Co[illegible]

1. **Innate role of B lymph[illegible]**
secreted IgM antibodies had unique functions in concentrating pathogens and antigen into secondary lymphoid organs, and prevented dissemination into vital organs. We sought 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 widely accepted as early events in immune responses.

   a. Ferguson AR, **Corley RB** (2006). Accumulation of marginal zone B cells and associated loss of follicular dendritic cells in NF-κB p50 deficient mice. **BMC Immunology** 6:8.
   b. Ferguson AR, Youd ME, **Corley RB** (2004). Marginal zone B cells [illegible] immune complexes on follicular dendritic cells [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]337.

and others had indicated that IgM antibodies were not always secreted as pentameric molecules with J chain, but little evidence existed to indicate if they had shared functions with pentameric IgM, or if they had unique functions. We found that monomeric IgM was more efficient than pentameric IgM, could be deleterious in autoimmune diseases, and that monomers did not fix complement. Monomers did not have the 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 hexamers accelerate disease manifestations in autoimmune *gld/gld fas*-deficient mice. **J. Autoimmunity** 29: 333-343.
b. Hughey CT, Brewer JW, Colley DG, Parkhouse RME, **Corley RB** (1998). Differential expression of hexameric and pentameric IgM in normal and autoimmune B cells: Implications for the physiologic role of hexameric IgM. **J. Immunol.** 161: 4091-4097.
c. Brewer JW, **Corley RB** (1997). Late events in assembly regulate the polymeric 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] control in the field [illegible] whether the addition was responsible for catalyzing assembly of IgM into [illegible] assembly occurred controversial. We demonstrated, however, that IgM assembly is regulated in the endoplasmic reticulum by a process involving thiol regulation. Further, J chain [illegible] 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 and sequenced 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, **Corley RB** (1[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 localization 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: 18661-18667.

4. **Defining mouse** [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 the B cell repertoire in mice, but the identity of these superantigens was unknown. During different investigations into this process, we identified mouse mammary tumor virus, MTV-9, as [illegible] activation of B cells, and later linked this to superantigen [illegible] cycle.

a. Sharma S, King LB, **Corley RB** (1989). Transient activation 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, La Vecchia A, WIV1-CoV poised for human emergence. **PNAS** 15:113(11):3048-53.

B. Personal and Professional

Positions and Employ

1993 Ameri
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,
2002 -04 Infectious Disease Pathogenesis Training Grant Fellow
2002 -05 Postdoctoral Fellow, University of North Carolina at Chapel Hill
2005 -17 Research Assistant Professor, Department of Epidemiology
2017- P

C. Contri

1. In vitro mo

newly identified or emerged hum
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 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 uncultivable: 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 L, 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**, Potempa K*, Li W, Sui J* (2015). HCoV-HKU1 Spike protein uses O-acetylated sialic acid as an attachment receptor determinant and employs HE protein as a receptor-destroying enzyme. **J Virol**. 89(14):7202-13. *indicates co-senior au

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 genomes less fit/attenuated proof live vaccine candidates.

a. Thornbrough JM, Jha BK, Yount B, Goldstein S, Li Y, Elliott R, Sims AC, Baric RS, Silverman RH, Weiss SR (2016). Middle East Respiratory Syndrome Coronavirus NS4b Protein Inhibits Host RNase L Activation. **MBio** 29;7(2).

b. Menachery VD, Gralinski LE, Mitchell HD, Dinnon KH 3rd, Leist SR, Yount BL Jr, Graham RL, McAnarney ET, Stratton KG, Cockrell AS, Debbink K, **Sims A**, Waters KM, Baric RS (2017). Middle East Respiratory Syndrome Coronavirus Nonstructural Protein 16 Is Necessary for Interferon Resistance and Viral Pathogenesis. **mSphere** 2(6). e00346-17.

determine the in
the virus
Role: