IBC Meeting Minutes

March 19, 2026 (Thursday) at 11:00 A.M.
via Zoom Conference Bridge

IBC members present:

Tom Greenough (Chair)	X	Shaoguang Li		Carol Schrader	X	Edward Jaskolski (alt)	X
Lisa Cavacini	X	Philip Tai	X	Mohan Somasundaran		Timothy Kowalik (alt)	
Colleen Driskill	X	Robert Klugman*	X	Eric Rouse	X	Regino Mercado-Lubo (alt)	X
Kris Giaya	X	Amelia Houghton		Sharone Green (alt)		Casey Moran (alt)	X
Hardy Kornfeld		Melissa Scannell	X	Jennifer Wang (alt)		Richard Ellison III (alt)	

Non-members present: Patrice Rando (IACUC/IBC Office)
*RK- left at 12:10

I. Introductory Remarks

1) The Chair brought to the attention of the Committee the meeting minutes from the previous IBC meeting. **Meeting Decision: Vote to approve February 12, 2026, Meeting Minutes**

II. Report on incidents/accidents from Employee Health Services (EHS)

Past incidents that remain open:

1) 07/08/2025: Needlestick in BSL-3. Potential TB exposure. 6 month follow-up 04/10/2026
2) 10/24/2025: Cut working with HVI using defective gloves. 6 month serology 04/23/2026
3) 11/26/2025: BSL-3. Wet sleeve noted; skin intact. Repeat IGRA screening planned?
4) 12/08/2025: Cut noted after cleaning BSC. Low likelihood HIV exposure; PEP; serology planned 06/08/26.

Recent incidents:

1) 03/17/2026: Needlestick. No rsNA or infectious agent. Carbidopa.
2) 3 mouse bites. No rsNA or infectious agent.

III. Protocols Reviewed Administratively

1) Investigator: Haran, J
Title: Department of Emergency Medicine Research Labs
IBC Registration: I-654-26, Renewal
Reviewer: Mercado-Lubo
Training Verification: Acceptable pending completion of PI training
Brief Summary: The Department of Emergency Medicine Research Labs are used for clinical research. They are utilized by various Investigators for processing and manipulation of human samples obtained in a number of different clinical protocols. Typically, samples are processed in the lab, stored there, and then packaged and shipped to another lab that is designated by the protocol. Samples may be frozen and shipped later (in batches). Some projects involve on-site testing of human samples using new POC instruments in development, although currently no such projects exist.
BSL/ABSL: BSL-2
NIH Guidelines: N/A

IV. Protocols to Discuss

1) Investigator: Brown, R
Title: Gene silencing therapeutics for ALS-FUS
IBC Registration: I-552-23, Amendment
Training Verification: Acceptable pending completion of PI training
Brief Summary: ALS is a devastating, progressive motor neuron (MN) degenerative disorder that starts focally and spreads to denervate most skeletal muscles, leading to death from respiratory failure, typically in 4 or 5 years. In this project, we will test the ability of novel gene silencing approaches to delay the disease progression in mouse models of FUS-ALS with the goal of developing therapeutics with improved efficacy and tolerability for ALS patients with mutations in the FUS gene.

- Aim 1. Characterize a mouse FUS-ALS model that is homozygous for human FUS transgene (hFUS tg/tg) and homozygous for a knockin mutation that replaces most of the mouse transferrin receptor gene with the equivalent human sequence (hTFRC ki/ki).
  Aim 2. Optimize suppression of FUS in mouse motor neurons by adeno-associated viruses (AAVs) expressing artificial microRNAs (amiRs).
  Aim 3. Evaluate the suppression of neuropathology and disease progression as a function of age of AAV-miR dosing in the FUS-ALS model.

Brief Summary and Review by Primary Reviewer

Overview and Objectives: Amyotrophic lateral sclerosis (ALS) is a neurodegenerative condition with no known cure and a 100% fatality rate. The objective of this amendment is to test the ability of novel gene silencing approaches to delay disease progression in mouse models of FUS-ALS with the goal of developing therapeutics with improved efficacy and tolerability for ALS patients with mutations in the FUS gene.
Aim 1. Characterize a mouse FUS-ALS model that is homozygous for human FUS transgene (hFUS tg/tg) and homozygous for a knockin mutation that replaces most of the mouse transferrin receptor gene with the equivalent human sequence (hTFRC ki/ki).
Aim 2. Optimize suppression of FUS in mouse motor neurons by adeno-associated viruses (AAVs) expressing artificial microRNAs (amiRs).
Aim 3. Evaluate the suppression of neuropathology and disease progression as a function of age of AAV-miR dosing in the FUS-ALS model.

Experimental Approach: ALS-FUS mice will be injected with recombinant AAV (rAAV) that express artificial miRNAs to suppress expression of the human FUS transgene. Motor function, disease progression, weight and serum biomarkers will be monitored for improvement after treatment. rAAV is be prepared by the UMass Viral Vector core using standard protocols to prevent viral replication. Animals will be injection with various viral doses up to 10e14 vector genomes/kg. Intravenous injections will be performed using standard procedures: facial vein injection in P1 neonatal animals or tail vein injection in adult animals at ages P28 and older. Animals will be monitored until euthanasia at either a pre-defined age or reaching a humane endpoint. Blood and various tissues will be processed to examine FUS expression and neuropathology.
The activity of some constructs may be tested by transfection of cell lines in vitro.

IBC Discussion and Vote

Discussion:	The reviewer discussed that the lab uses transgenic animals but hasn't described these particular transgenic animals. The reviewer had some questions about the role of transferrin receptors being used. The reviewer discussed that there are some inconsistencies between the registration and vector checklist regarding AAV being replication deficient which needs to be rectified.
Meeting Decision:	Vote to approve upon completion of action items.
BSL/ABSL:	**BSL-2; ABSL-1 with Special Precautions; Administration to Animals using BSL-2 Precaution/ Sharps Safety**
NIH Guidelines:	III-D, III-E, III-F

2) Investigator: Fitzgerald, K
Title: Innate immunity to bacteria, viruses, and fungi
IBC Registration: **I-284-25, Amendment**
Training Verification: Acceptable
Brief Summary: Amendment request to change SARS-CoV-2 work from BSL-3 to BSL-2. The CDC laboratory Biosafety Guidelines for working with SARS-CoV-2 mentions: "At a minimum, BSL-2 facilities, practices, and procedures are recommended for diagnostic research, anatomic pathology, environmental testing, and virus propagation utilizing SARS-CoV-2. At a minimum, ABSL-2 is recommended for work with these viruses in animal models." Centers for Disease Control and Prevention. (2025, September 5). *Laboratory biosafety guidelines for working with SARS-CoV-2*. https://www.cdc.gov/covid/php/lab/index.html

Brief Summary and Review by Primary Reviewer

Overview and Objectives: The long-term goal of our research is to obtain a comprehensive understanding of how the innate immune response is activated and regulated following infection with viral, bacterial and fungal pathogens, as well as to understand the factors that initiate autoimmune diseases. The main objective of these studies is to examine basic principles of the immune response to infection both in vitro and by manipulating the mouse model.
Amendment to reduce SARS-CoV-2 biocontainment from BSL-3/ABSL-3 to BSL-2 with enhanced precautions and ABSl-2

Experimental Approach: A combination of biochemical, molecular and genetic approaches to identify key components (receptors, signaling molecules, cytokines etc.) in the innate immune response. A combination of shRNA/siRNA approaches in human cell lines and mouse cell lines as well as the use of cells from mice with targeted deletions in these key components will be used. Live or UV/heat inactivated bacteria, viruses, fungi, and pathogen-derived products like lipopolysaccharide (LPS) will be used to treat cells in vitro. Activation of signal transduction pathways in vitro will be evaluated. Additionally, disease models will be used to monitor the role of innate receptors/signaling molecules in vivo. Pathogens include: Listeria monocytogenes, Salmonella typhimurium, Candida albicans, Sendai Virus (will not be used in vivo), Vaccinia Virus, mouse cytomegalovirus, Herpes Simples Virus-1, Influenza Virus, and Human Coronavirus SARS-CoV-2.

IBC Discussion and Vote

Discussion: This is the first protocol that is looking to decrease the Biosafety level from BSL-3 to BSL-2 working with SARS-CoV2. The lab is adding 2 strains of SARS-CoV2.

Meeting Decision: Vote to approve upon completion of action items. The committee discussed the use of surgical mask at all times and that N-95 should be used if aerosols are possible. For decontamination; the lab should use Bleach, not Vesphene. DURC/PEPP questions were reviewed. An update to the SOP's is required with most recent contact information and EHS hours.

BSL/ABSL: **BSL-2+; ABSL-2**

NIH Guidelines: III-D

3) Investigator: Galván-Peña, S
Title: Systemic impact of gut microbiome
IBC Registration: **I-936-26, New**
Reviewer: Schrader
Training Verification: Acceptable pending completion of PI training
Brief Summary: The gut carries a huge collection of naturally occurring microbes (the microbiome) that regulate and maintain the immune system even beyond the intestines. We do not yet fully understand how the microbiome impacts the immune system beyond the gut.
Our main goal is to understand how intestinal immune cells integrate signals from the microbiome and communicate with other cells in and outside the gut.

Brief Summary and Review by Primary Reviewer

Overview and Objectives: The main goal is to understand how intestinal immune cells integrate signals from the microbiome and communicate with other cells in and outside the gut.

Experimental Approach: In vivo Rainbow-CRISPR approach, achieved by bone marrow reconstitution of irradiated mice with stem cells (HSC/LSK cells) from Cas9-expressing mice transduced with lentiviral vectors expressing gRNAs.10 weeks later, to allow for reconstitution and differentiation, cells are isolated from different organs and stained for flow cytometry analysis. Findings from this model will be confirmed using human cells and tissues, and mice may be lavaged with human fecal material to alter their microbiome.

IBC Discussion and Vote

Discussion: The reviewer discussed this is a new protocol from a newer UMass Chan Principal Investigator. The reviewer discussed that the lab may be sorting a mouse line, but it is not described in the registration. The reviewer discussed that they did a great job filling out the forms and discussed minor action items.

Meeting Decision: Vote to approve upon completion of action items.

BSL/ABSL: **BSL-2; ABSL-2**

NIH Guidelines: III-D, III-F

4) Investigator: Henninger, N
Title: Sarm knockout to block ischemic and traumatic axonal injury in the mouse
IBC Registration: **I-657-26, Renewal**
Reviewer: Tai
Training Verification: Acceptable

Brief Summary: *Sarm1* is an endogenous gene that promotes axonal degeneration under normal conditions. *Sarm1* knockout (KO) has been shown to prevent axonal degeneration in fly and rodent models of neurological disease.
We propose to use both germline and conditional *Sarm1* knockout mice to test the view that Sarm1-knockout not only increases axonal structural integrity but also preserves axonal function after traumatic brain injury (TBI). These experiments will help to bridge the current gap between previously observed axonal preservation and improved functional outcomes after TBI in knockout mice.

Brief Summary and Review by Primary Reviewer

Overview and Objectives: The research team proposes to use both germline and conditional Sarm1 knockout mice to test the hypothesis that Sarm1-knockout, not only increases axonal structural integrity, but also preserves axonal function after traumatic brain injury (TBI). The study will help to bridge the current gap between previously observed axonal preservation and improved functional outcomes after TBI in knockout mice.

Experimental Approach: The research team previously showed that Sarm1 germline knockout mice are protected from TBI. These mice have been previously described in other publications. The team hypothesizes that Sarm1 knockout in post-mitotic neurons in the adult mouse brain will neuronautonomously protect the brain after TBI.
They aim to produce conditional knockouts by leveraging Cre-loxP technology. Spatial control (tissue specificity) will be achieved by using mice that express Cre driven by a promotor that is specific to the tissue of interest. By using tamoxifen-inducible Cre-systems (Cre-ERT), temporal control of target gene expression will be achieved.
Conditional knockout mice will be generated through two approaches:
Sarm1fl/fl transgenic mice were previously generated using CRISPR technology by inserting two loxP sites into the Sarm1 gene flanking essential exons via microinjection of linearized DNA (long ssDNA) into fertilized C57BL/6-strain inbred mouse embryos.
Transgenic animals will be crossed with the appropriate transgenic mouse lines expressing the inducible Cre (all available through the Jackson labs, Bar Harbor, ME). For example, for conditional Sarm1 disruption in neurons, microglia, astroglia, and oligodendroglia, Wfs1CreERT2 (stock #009614), CX3CR1CreER (stock #021160), hGFAPCreERT2 (stock #012849), or Plp1CreERT (stock #005975) mouse strains will be used, respectively.
Cre-ERT activity will be induced via intraperitoneal injection of tamoxifen (75-200 mg/kg) at a concentration of 20 mg/mL once a day for 5 consecutive days in mice ~2 months of age per IACUC Policy 3.02 - Use of Non-Pharmaceutical Grade Tamoxifen in Mice. To validate the recombination properties of the Cre lines and tamoxifen treatment regimens, mice will be crossed to a global double-fluorescent Cre reporter line Gt(ROSA)26Sor (Jackson labs; e.g., stock #007676). Homogenous cell-type specific Cre recombination 1 week after tamoxifen (but not vehicle) injection in Cre-transgenic mice has been shown for the proposed Cre lines.
To determine the cell types in which Sarm1 disruption leads to axon protection following TBI, histological evaluations 48 hours after TBI, which require the use of paraformaldehyde perfusion in some mice, will be performed.

IBC Discussion and Vote

Discussion: Reviewer discussed if the lab plans to continue to use Cas9 for generating transgenic mice, then they should revise the registration form. Reviewer discussed action items.
Meeting Decision: Vote to approve upon completion of action items.
BSL/ABSL: **BSL-1; ABSL-1**
NIH Guidelines: III-E

5) Investigator: Lawrence, J
Title: Translational Epigenetics by Genomic Editing
IBC Registration: I-660-26, Renewal
Reviewer: Cavacini
Training Verification: Acceptable pending completion of PI training
Brief Summary: To modify the epigenome to study regulation by XIST RNA and its derivatives (e.g. A-repeat segment) in human and mouse iPS cells and their differentiated products, in vitro.
To generate mouse models of Down Syndrome carrying transgenes of XIST to test silencing of a trisomic chromosome and potential phenotypic correction, and to also test in vivo delivery of the transgene into mouse tissues.
To examine the effects on cell pathologies of silencing chr21 or specific genes on chr21 in existing iPS cells initially derived from Down Syndrome patient fibroblasts.
To investigate protein factors or repetitive DNA sequences that contribute to the epigenetic regulation of the genome by XIST RNA or other nuclear RNAs or proteins, such as SAF-A.
To investigate in cells in culture the role of the APP gene and factors involved in cell aging to the development of Alzheimer Disease, particularly in Down Syndrome.

Brief Summary and Review by Primary Reviewer

Overview and Objectives: To modify the epigenome to study regulation by XIST RNA and its derivatives (e.g. A-repeat segment) in human and mouse iPS cells and their differentiated products, in vitro. To generate mouse models of Down Syndrome carrying transgenes of XIST to test silencing of a trisomic chromosome and potential phenotypic correction, and to also test in vivo delivery of the transgene into mouse tissues. To examine the effects on cell pathologies of silencing chr21 or specific genes on chr21 in existing iPS cells initially derived from Down Syndrome patient fibroblasts. To investigate protein factors or repetitive DNA sequences that contribute to the epigenetic regulation of the genome by XIST RNA or other nuclear RNAs or proteins, such as SAF-A. To investigate in cells in culture the role of the APP gene and factors involved in cell aging to the development of Alzheimer Disease, particularly in Down Syndrome.

Experimental Approach: Much of this work will utilize cells carrying an XIST transgene, or mice carrying an Xist transgene, which we have already made in recent years under the prior approved IBC protocol. Recombinant plasmids were introduced into human/mouse cell lines by transfection reagents (Lipofectamine 2000) or electroporation. To generate chromosome silencing mouse models, Down syndrome (trisomic) mouse embryonic stem cell lines or fertilized oocytes were prepared using standard culture techniques. New experiments will utilize these same protocols to introduce modifications of the XIST transgenes into human iPS cells or mouse ES cells. In addition, the XIST transgene, in some cases a selection gene (e.g. puromycin or neomycin) or a reporter gene (GFP), will be inserted into a specific locus of mouse chromosome 16 using genome editing technology (ZFNs or CRISPR/Cas9). In some cases, the transgenes will be introduced into cultured cells or fertilized embryos using recombinant adeno-associated viruses, as has been implemented in the laboratory of Dr. Jaime Rivera. The AAV are replication deficient, non-pathogenic and are routinely used by the Transgenic Animal Core at UMASS. To study in vivo delivery of chromosome silencing transgenes (XIST gene), recombinant plasmids will be packaged into recombinant AAV viruses and delivered into mouse organs. The recombinant AAV viruses will be provided by the Viral Vector Core of the Gene Therapy Center at UMASS.
Another component of our work using neural cells or endothelial cells differentiated from existing human iPS cells which carry the XIST transgene and were generated a few years ago. In some cases, we will modify that XIST transgene to make it smaller, using the same procedures as described above. We are also deriving cerebral organoids in culture from these cells. All of our work uses established protocols or minor modifications thereof.

A substantial part of our research involves extracting RNA from the cells and examining the effects of transgene expression on the transcriptome of the cells. This type of analysis is done both for the Down Syndrome related studies using XIST transgenes, and for more basic biological studies in which no XIST transgene is involved. A substantial part of the work also involves molecular cytological analysis of cells, in which we use fluorescence microscopy to visualize certain genes, RNAs, and proteins in cells.

IBC Discussion and Vote

Discussion:	The reviewer discussed that there were no changes in the registration since the previous IBC submission. The reviewer mentioned it's unclear from the registration if human cells are being used.
Meeting Decision:	Vote to approve upon completion of action items.
BSL/ABSL:	**BSL-2; BSL-2 Enhanced Flow Sorting; ABSL-1 with Special Precautions; Administration to Animals using BSL-2 Precautions/ Sharps Safety**
NIH Guidelines:	III-D, III-E, III-F

6) Investigator: Wang, Y
Title: Isolation of fetal cells from circulating maternal blood in mice
IBC Registration: I-937-26, New
Reviewer: Giaya
Training Verification: Acceptable pending completion of PI training
Brief Summary: The overarching goal of this research is to develop and validate a highly sensitive and reliable method for isolating circulating fetal cells (CFCs) from maternal blood, thereby enabling noninvasive, genome-wide prenatal genetic diagnosis.

Specifically, this study seeks to:

- Aim 1. Establish and optimize methodologies for the efficient isolation of circulating fetal cells (CFCs) from maternal blood.

We will refine our existing techniques for CFC enrichment and sorting using pregnant mouse models. By combining physical and molecular capture methods, including fluorescence-activated cell sorting (FACS), magnetic affinity cell separation (MACS), and molecular tagging, we aim to maximize the yield and purity of CFCs recovered from maternal circulation.

- Aim 2. Identify and validate fetal-specific biomarkers for CFC detection and capture.

Using both wild-type and transgenic (EGFP-expressing) mouse models, we will characterize the molecular and cellular features of isolated CFCs to identify robust fetal-specific RNA and protein markers that distinguish them from maternal cells. Antibody and oligonucleotide affinity reagents targeting these biomarkers will be tested for their ability to selectively recognize and isolate fetal cells from maternal blood.

- Aim 3. Characterize fetal cell subtypes and genomic composition to enable single-cell prenatal genetic analysis.

We will perform single-cell RNA sequencing and whole-genome amplification of isolated CFCs to define their transcriptional profiles, confirm paternal allele inheritance, and evaluate their suitability for noninvasive genetic testing. Comparative analyses between different gestational ages and mouse strains will further refine the molecular catalog of fetal cell types present in maternal circulation.

Brief Summary and Review by Primary Reviewer

Overview and Objectives: Develop and validate a highly sensitive and reliable method for isolating circulating fetal cells (CFCs) from maternal blood, thereby enabling noninvasive, genome-wide prenatal genetic diagnosis.

Specifically, this study seeks to:

- Aim 1. Establish and optimize methodologies for the efficient isolation of circulating fetal cells (CFCs) from maternal blood.Refine existing techniques for CFC enrichment and sorting using pregnant mouse models. By combining physical and molecular capture methods, including fluorescence-activated cell sorting (FACS), magnetic affinity cell separation (MACS), and molecular tagging, aiming to maximize the yield and purity of CFCs recovered from maternal circulation.
- Aim 2. Identify and validate fetal-specific biomarkers for CFC detection and capture.

Using both wild-type and transgenic (EGFP-expressing) mouse models, they will characterize the molecular and cellular features of isolated CFCs to identify robust fetal-specific RNA and protein markers that distinguish them from maternal cells. Antibody and oligonucleotide affinity reagents targeting these biomarkers will be tested for their ability to selectively recognize and isolate fetal cells from maternal blood.

- Aim 3. Characterize fetal cell subtypes and genomic composition to enable single-cell prenatal genetic analysis.

They will perform single-cell RNA sequencing and whole-genome amplification of isolated CFCs to define their transcriptional profiles, confirm paternal allele inheritance, and evaluate their suitability for noninvasive genetic testing. Comparative analyses between different gestational ages and mouse strains will further refine the molecular catalog of fetal cell types present in maternal circulation.

Experimental Approach: This study will use inbred C57BL/6J mouse male mice lacking fluorescent protein transgenes to evaluate CFC-capturing reagents using non-fluorescent wild-type fetal cells. EGFP transgenic mice on a C57BL/6 background will serve as controls to generate EGFP-expressing CFCs. Female breeders will be wild-type CD1 mouse (outbred) or FVB/NJ mouse (inbred).

For each gestation group, one male will be housed with 1–2 females for 2–3 days and then separated. Males will be individually housed and used for mating up to once per week. Up to two gestation groups may be established per week, each using up to 40 males and 28–40 females. Females will be maintained for 8–17 days after mating (11–19 days total gestation) to allow sampling across developmental stages.

At the designated gestational time point, up to 2.0 mL of maternal blood will be collected by cardiac puncture under isoflurane anesthesia. Dams will then be euthanized, and selected fetuses and placentas collected for analysis. Up to 2.0 mL of blood may also be collected from up to two non-pregnant females for optimization studies. Occasional tail blood collection may be performed from males and females as needed.

Maximum animal use will not exceed 2,080 female mice per year (6,240 over 3 years) and 320 male mice per year (960 over 3 years combined for C57BL/6J and EGFP males). Based on prior observations, approximately 8,008 fetuses per year may be generated (2,080 females × 0.35 pregnancy rate × ~11 fetuses per dam). Pregnant mice will be euthanized prior to parturition, and offspring will not be maintained. Fetuses will be euthanized at the designated gestational stage, and any live-born pups will be euthanized.

The study will establish 0–2 gestation groups per week (up to 52 per year; 156 over 3 years).

IBC Discussion and Vote

Discussion:	The reviewer discussed that although it's clear that they are breeding mice, much of this protocol is unclear as to what work is being done where. The lab mentions use of another facility at Harvard but there aren't enough details to determine what is being done at what location. Clarification regarding what work is being done in the lab at UMass is required.
Meeting Decision:	Vote to approve upon completion of action items.
BSL/ABSL:	**BSL-1; BSL-2 Flow Sorting; ABSL-1**
NIH Guidelines:	III-E

V. Report on incidents/accidents/issues involving BSL-3 & ABSL-3 Facilities

1) Changes to SAT manual; distributed
2) ABSL-3 scrubs (response to CDC observation); dedicated scrubs under PPE required. Architect and facilities working on a change room to use for scrubs. SOP drafted to use locker rooms pending changing room renovations.
3) BSL-3 new lab space design meetings.
4) Collaborators from other institutions using BSL-3/ABSL-3 facilities.

VI. Information from the field (Senior Biosafety Officer)

1) New Director for chemical and lab safety unit (CELS).
2) Audit of labs; chemical storage/use/etc.
3) RFP for biohazard waste disposal (Stericycle vs other).

VII. Other Business

Acknowledgement Items:

1) Luzuriaga 631-25, Hafer 443-24 Update-new study collecting, processing, storing and shipping human samples (CRC and Biorepository)
   Pfizer clinical trial (PI Jennifer Wang): Shionogi RSV Protocol v2.0 (Amendment 1) "A phase 2b, multicenter, randomized, double-blind, placebo controlled, parallel-group study to evaluate the safety, tolerability, and efficacy of S-337395 in symptomatic nonhospitalized adults with respiratory syncytial virus who are at high risk of progression to severe disease"

2) Hayward 917-25 Update- EPI-321-02_Protocol_v7.0 "A Phase 1/2, Open-label, Dose-escalation Study to Evaluate the Safety, Tolerability, and Biological Activity of EPI-321, an AAVrh74-delivered Epigenetic Editing Therapy in Adult FSHD Patients" Amendment 111925: changes in safety lab monitoring, number of participants, new time points for biopsies and immunology. "Data from a 30OCT2025 data snapshot showed non-serious, log-grade (CTCAE Grade 1) laboratory abnormalities emerge in the 4-6 week window in initial participants administered the 2×10^{13} vg/kg IV dose level; these abnormalities could in theory increase in severity or seriousness at the higher dose level (4×10^{13} vg/kg IV)." Increased staggering time frame to monitor for adverse effects at higher doses. Added rituximab and sirolimus to immune suppression for higher doses.

Adjourned at 1:23pm