

IBC Meeting Minutes
July 16, 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		Mohan Somasundaran		Timothy Kowalik (alt)	
Colleen Driskill	X	Robert Klugman		Eric Rouse	X	Regino Mercado-Lubo (alt)	X
Kris Giaya	X	Amelia Houghton	X	Sharone Green (alt)		Casey Moran (alt)	X
Hardy Kornfeld		Melissa Scannell		Jennifer Wang (alt)		Richard Ellison III (alt)	

Non-members present: Patrice Rando (IACUC/IBC Office)

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 June 18, 2026 Meeting Minutes**

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

Past incidents that remain open:

None.

Recent incidents:

- 1) 06/27/2026: Pipette laceration. Human stem cells; certified negative for BBP. CLOSED
- 2) 07/13/2026: Mouse bite. No infectious agent or rsNA. Swelling prompted attempt at ED visit. Follow-up in EHS - improved.

III. Protocols Reviewed Administratively

- 1) Transfer of PI: Wang I-631 (CRC lab) transferring from Luzuriaga to Wang
Administrative approval at BSL-2 as before recommended

IV. Protocols to Discuss

- 1) Investigator: Aronin, N
Title: Transgenic Models of Huntington's Disease: siRNA Delivery in AAV and Lentivirus
IBC Registration: I-243-26, Renewal
Training Verification: **Acceptable pending completion of PI training**
Brief Summary: The goal of this project is to determine how gene silencing can be applied to the treatment of Huntington's Disease.
 1. We will use AAV to deliver RNA silencing genes to transgenic mice carrying the human Huntington gene.
 2. We will use AAV to deliver allele specific siRNA to transgenic mice carrying the human Huntington gene.
 3. We will use human brain tissue to evaluate alternative polyadenylation of huntingtin mRNA in diseased versus control human brain tissue.
 4. We will use siRNAs to silence genes associated with Huntington's Disease.

Brief Summary and Review by Primary Reviewer

Overview and Objectives: The goal of this project is to determine how gene silencing can be applied to the treatment of Huntington's Disease.

Experimental Approach: 1) AAV will be used to deliver RNA silencing genes to transgenic mice carrying the human Huntington gene. 2) Human brain tissue will be evaluated for alternative polyadenylation of huntingtin mRNA in diseased versus control human brain tissue. 3) siRNAs will be used to silence genes associated with Huntington's Disease.

IBC Discussion and Vote

Discussion:	The reviewer discussed this is a 5-year renewal and details the experimental approach, including the use of AAV to deliver RNA silencing agents in transgenic mice and the use of human brain tissue for alternative polyadenylation studies. The reviewer identified action items, including the need to clarify the use of lentivirus (if any) and to complete various sections of the protocol. The reviewer also highlighted the need for accurate and complete responses in the protocol, including the replication defective status of vectors.
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 Precautions/ Sharps Safety
NIH Guidelines:	III-D, III-E, III-F

- 2) Investigator: Clayton, K
Title: Clayton Lab: Characterization of HIV-infected macrophage interactions with immune cells
- IBC Registration:** **I-804-26, Renewal**
Training Verification: **Acceptable**
Brief Summary: 06/23/26: Updates include work with de-identified HIV+ HEAL cohort patient samples (obtained from a collaboration at Brigham Women's Hospital – classified as not human subjects research), THX humanized mouse samples (obtained from a collaboration at Massachusetts General Hospital), new strains of Mtb, HHV-6A/6B/7-infected CD4+ T cell sample runs on the Pathology Department 10X Genomics Instrument, use of lentiviral constructs to deliver shRNAs to CD4+ T cells, CRISPR/Cas9 ribonucleoprotein-based editing of CD4+ T cells and macrophages to make them more susceptible to perforin and NK cell-mediated killing, and mRNA and lentiviral-based overexpression of HIV genes in uninfected human CD4+ T cells and macrophages to make them more susceptible to perforin and NK cell-mediated killing.
- The overall goal of the Clayton Lab is to characterize the interactions of pathogen-infected macrophages and CD4+ T cells with cytolytic cells of the immune system (CD8+ T cells and NK cells) to identify mechanisms of resistance to killing. This includes several complementary specific objectives:
- 1) Identify differential mechanisms of NK cell recognition of HIV/SHIV/SIV-infected CD4+ T cells, monocyte-derived macrophages (MDMs) and alveolar macrophages (AMs).
 - 2) Characterize HIV/SHIV/SIV-infected CD4+ T cells, MDMs, and AMs that survive interactions with cytolytic CD8+ T cells (CTL) and NK cells, including cells derived from human PBMCs, human bronchial alveolar lavage (BAL), and non-human primate tissues.
 - 3) Assess the responses above to human macrophages (MDMs and AMs) infected *in vitro* with *Mycobacterium tuberculosis* (Mtb), human cytomegalovirus (HCMV), and Ebola virus (through our

- Boston University collaboration). In addition, assess the responses above to human CD4⁺ T cells infected with human herpes virus 6A, 6B, and 7 (HHV-6A, HHV-6B, and HHV-7)
- 4) Discover pathways intrinsic to macrophages that antagonize perforin-mediated cell death. Ultimately, knowledge of common survival signatures of pathogen-infected macrophages (MDMs and tissue-resident macrophages), which will be compared to pathogen-infected CD4⁺ T cells, will guide the development of therapeutics to enhance the sensitivity of these infected cells to killing by CTLs and NK cells. In addition, we are proposing to assess immune interactions with macrophages infected with *Mtb* and (separately) HCMV, with or without HIV co-infection, and CD4⁺ T cells infected with HHV-6A/6B/7, with or without HIV, to determine if multiple pathogens employ common mechanisms in macrophages and CD4⁺ T cells to escape cytolytic immune responses.

Brief Summary and Review by Primary Reviewer

Overview and Objectives: The overall goal is to characterize the interactions of pathogen-infected macrophages and CD4⁺ T cells with cytolytic cells of the immune system (CD8⁺ T cells and NK cells) to identify mechanisms of resistance to killing. Several complementary objectives have been described in details in the original protocol. Updates to the protocol include work with de-identified HIV⁺ HEAL (HIV Eradication And Latency) cohort patient samples (obtained from a collaboration at Brigham Women's Hospital – classified as not human subjects research), THX humanized mouse samples (obtained from a collaboration at Massachusetts General Hospital), new strains of *Mtb*, HHV-6A/6B/7-infected CD4⁺ T cell sample runs on the Pathology Department 10X Genomics Instrument, use of lentiviral constructs to deliver shRNAs to CD4⁺ T cells, CRISPR/Cas9 ribonucleoprotein-based editing of CD4⁺ T cells and macrophages to make them more susceptible to perforin and NK cell-mediated killing, and mRNA and lentiviral-based overexpression of HIV genes in uninfected human CD4⁺ T cells and macrophages to make them more susceptible to perforin and NK cell-mediated killing.

Experimental Approach: De-identified human PBMCs derived from the HIV⁺ HEAL (HIV Eradication And Latency) cohort patients (a collaboration with Dr. Athe Tsibris at BWH) – designated as not human subjects research by the UMass IRB (06/22/26). Fresh and fixed tissues (Blood, GI tract, lymph nodes, feces, saliva, BAL, lung, and brain) from uninfected and HIV-infected THX humanized mice (obtained via overnight FedEx shipments from our collaborator, Dr. Alejandro Balazs, at MGH)

- Human PBMCs from the HIV⁺ HEAL cohort will be depleted of NK cells and CD8⁺ T cells using magnetic isolation kits, followed by stimulation with cytokines and TCR agonists for 24 hours. The cells will then be lysed for qPCR analysis of HIV transcripts. The conditioned media from these samples will be stored for cell-free HIV RNA (qPCR) and Gag p24 measurements (ELISA).
- Healthy donor CD4⁺ T cells infected with HHV-6A, HHV-6B, or HHV-7 will be resuspended to high concentrations in media and loaded onto Pathology Department 10X Genomics instrument (in the BSL-2 space), which will be used for RNA sequencing.
- Healthy donor CD4⁺ T cells will be transduced with lentiviruses expressing shRNA to target viral RNAs of interest, followed by infection with either HIV or HHV-6A/6B/7. This will be used to assess the effects of specific viral genes on the host phenotype without altering the viral genomes.
- Healthy donor uninfected CD4⁺ T cells and macrophages will be electroporated/transfected with mRNA or transduced with lentiviruses to express viral proteins (HIV Nef, Vpu, Vpr, and Vif) to assess the effects on the cell phenotype and susceptibility to NK cell-mediated killing.
- CRISPR/Cas9 ribonucleoproteins will be used to edit CD4⁺ T cells and macrophages to make them more susceptible to perforin and NK cell-mediated killing. CRISPR/Cas9 ribonucleoproteins (RNPs) targeting host pathways predicted to affect perforin activity will be used to edit human monocytes, which will then be matured into macrophages. These cells will then be subjected to recombinant perforin (commercial source), followed by flow cytometry analysis of fixed cells to determine viability and phenotypic changes. Edited cells treated +/- perforin will also be sorted using the FACS Aria II.

(BSL2 containment – Flow Cytometry Core) and deep sequenced to assess for signatures enriched in cells that survive treatment with these cytolytic proteins.

IBC Discussion and Vote

Discussion: The reviewer discussed this is a 5-year renewal on characterizing HIV-infected macrophage interactions with immune cells. The reviewer detailed the updates to the protocol, including the use of de-identified HIV and HIV eradication study samples, humanized mouse samples, and new strains of MTB and human herpes virus six as well as the addition of recombinant human HIV clones and recombinant MTB strains, including those expressing luciferase or fluorescent reporter proteins. The reviewer discussed action items including updating contact information, completing SOPs, and ensuring proper training for lab members.

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

BSL/ABSL: **BSL-3; BSL-3 Flow Sorting**

NIH Guidelines: III-D, III-E, III-F

- 3) Investigator: Kim, J
Title: Mouse Metabolism in Pathogen-Challenged Mice (UMass Mouse Metabolic Phenotyping Center / Kim)
- IBC Registration:** I-693-22, Amendment
- Training Verification: **Acceptable**
- Brief Summary: 1. Diphtheria toxin (DT) is needed to induce alpha cell loss in alpha cell-specific diphtheria toxin receptor transgenic mice for the study of glucagon metabolism in the context of obesity.
2. To avoid pleiotropic effects of IL-12 induced by systemic administration, we will use AAV-constitutively expressing IL-12 (AAV8-IL12) or luciferase (AAV8-Luc; control) under the transcriptional control of a hepatocyte-specific promoter (a1-antitrypsin promoter fused to the albumin enhancer) to selectively target the liver.
3. To achieve siRNA-mediated liver macrophage silencing of IL-12, we will use fluorescently-labeled GeRPs for targeted delivery of siRNA to potentially silence the IL-12 gene in liver macrophages in vivo.

Brief Summary and Review by Primary Reviewer

Overview and Objectives: This amendment introduces the use of diphtheria toxin, AAV8-IL-12 and AAV8-Luc vectors, and siRNA-loaded GeRPs into the registration.

Experimental Approach: AAV-constitutively expressing IL-12 (AAV8-IL12) or luciferase (AAV8-Luc) will be injected via tail vein. Following treatment mice will be used for in vivo metabolic phenotyping, and livers and brains will be used for molecular analysis.

Fluorescently-labeled GeRPs containing IL-12 targeting siRNA or non-specific siRNA (control) will be given via tail vein (8 doses) over a 2-week period. GeRPs-treated mice will be used for in vivo metabolic and behavioral phenotyping, and livers and brains will be used for molecular analysis.

IBC Discussion and Vote

Discussion: The reviewer discussed this was an amendment submitted for the UMass Mouse Metabolic Phenotyping Center, and the overall objective of the study is to perform quantitative analysis of cytokines, hormones, and lipid levels in tissues

collected from mice infected from influenza, as well as coxsackie virus. The lab is looking to add three things. The first is diphtheria toxin, and they're using it to induce alpha cell ablation in alpha cell-specific diphtheria toxin receptor transgenic mice for studies investigating glucagon metabolism in the context of obesity. The second is AAV8 expressing IL 12 and AAV8 vector expressing luciferase to minimize pleiotropic effects associated with systemic IL-12 administration. Third, they are adding fluorescently labeled glucan particles loaded with siRNA to silence IL 12. The reviewer discussed action items.

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 Precautions/ Sharps Safety

NIH Guidelines:

III-D, III-E, III-F

4) Investigator:

Wang, J

Title:

In vitro Cap-snatching of Host RNAs by Hantaviruses

IBC Registration: **I-616-25, Amendment**

Training Verification: **Acceptable**

Brief Summary:

To use protocols such as CapSeq (see Reference 1) or siRNA silencing combined with deep-sequencing to examine the host response of cells to different hantavirus strains (Andes, Hantaan, Puumala, and Sin Nombre) in infected human cultured cells.

Brief Summary and Review by Primary Reviewer

Overview and Objectives: To examine the host response of cells to different hantavirus strains in infected human cultured cells, this amendment will add siRNA silencing in vitro, human precision cut lung slices (PCLS), and use of the Andes hantavirus strain.

Experimental Approach: They will infect a human bronchoepithelial cell line, A549, or human precision cut lung slices (PCLS) with different strains of hantavirus in the BSL-3 suite using appropriate precautions and following the hantavirus standard operating procedures (see enclosed document). In some cases, A549 cells will be pre-treated with siRNAs targeting pro-viral host factors. RNA and proteins from infected A549 cells will be harvested using TRIzol at specific time points following infection. TRIzol samples will be removed from the BSL-3 suite and used for transcriptional analysis and western blots in the Wang lab. Infected PCLS will be fixed with formalin then transferred to the Wang lab for image analysis with RNAscope or immunohistochemical staining. Different hantaviruses strains will be used to determine if host responses are strain-specific. Viral stocks will be expanded as needed in Vero cells in the BL-3 suite but only in small volumes (≤20mls).

IBC Discussion and Vote

Discussion:

The reviewer discussed that this is an amendment to examine host responses to different hantavirus strains in human cultured cells and precision-cut lung slices. The reviewer detailed the new strains being added and, in some cases, the cells will be pretreated with the siRNAs that they're adding to target proviral host factors however, no animal work proposed. The reviewer discussed the experimental approach, including the use of siRNA for gene silencing and the need for updated agent-specific awareness slides. A member of the committee emphasized the importance of updating the hantavirus SOP, developing a medical SOP for the Andes virus and distributing the updated agent awareness slides to BSLB-3/ABSL-3 Users in real-time. The IBC gave approval to receive the Andes virus strains with the understanding that they are kept in freezer storage

	until a Medical SOP is approved by the IBC, and other action items are adequately addressed
Meeting Decision:	Vote to approve upon completion of action items.
BSL/ABSL:	BSL-3
NIH Guidelines:	III-F

5) Investigator:	Wang, J
Title:	Countering Coronavirus Disease 2019 (COVID-19)
IBC Registration:	I-755-25, Amendment
Training Verification:	Acceptable
Brief Summary:	Change from BSL-3 to BSL-2 per the latest recommendations from NIH

Brief Summary and Review by Primary Reviewer

Overview and Objectives: To characterize SARS-CoV-2 infection as well as that of other human coronaviruses in order to (1) discover compounds with antiviral activity and (2) cellular components that are crucial for cell entry and/or viral replication, utilizing different human cell lines for capability of productive infections.

Experimental Approach: This amendment is to request approval to reduce biocontainment requirements from BSL-3/ABSL-3 to BSL-2/ABSL-2 in alignment with new guidelines for SARS-CoV-2 research. There are no new experiments proposed. As before:

Antiviral screening assays. Screens will be performed in collaboration with the Khvorova, Watts, and additional labs. Screening plates will be set-up by the collaborating labs (i.e. Khvorova, Watts, additional labs) and then transferred to the Wang lab for the SARS-CoV-2 infection work.

SARS-CoV-2 will be propagated in vitro, including ancestral and circulating variants (e.g. omicron), mouse-adapted strains MA10), and engineered strains (plasmid). Human coronavirus OC43 will be used as a control virus as needed for experiments.

Various human cells (A549-ACE2, Huh7, CaCo2, HEK293T-ACE2), primary tissues (human islets, precision cut lung slices) and NHP cells (Vero) will be used for propagating viruses in vitro to determine antiviral effects of various compounds and to identify host factors that impact viral infection and replication. Mouse-adapted SARS-CoV-2 (BEI NR-55329) will be used to infect mice to test immunologic responses and antiviral and antihost siRNAs. Various SARS-CoV-2 strains may be used for hamster and K18 hACE2 transgenic mice infections to test antiviral agents or evaluate immunologic responses to infection.

It is now proposed that SARS-CoV-2 work will be performed under BSL-2/ABSL-2 conditions.

Work on antiviral siRNAs and antisense oligonucleotides (ASO) will continue; to identify host and virus targeting oligos that reduce/prevent SARS-CoV-2 infection. This will occur in vitro:

- Preparation of the antiviral screen multi-well plates

Multi-well plates for the antiviral screen will be set up under the approved protocol from the collaborating lab (i.e., Khvorova lab for chemically modified siRNAs). Briefly, compounds are diluted in media to the appropriate final concentration in a dilution plate then transferred to the assay plate. Cells to be used in the screen are washed with PBS, trypsinized, resuspended in growth media, counted, and then diluted to the appropriate concentration of cells for the screen. Diluted cells are then added to each well of the assay plate containing the diluted compounds.

- SARS-CoV-2 infection of antiviral screen multi-well plates and monitoring viral loads

Antiviral screening plates transferred from the collaborating lab will be infected with SARS-CoV-2. Cells will be monitored for cytopathic effect and infectious viral load will be assayed by plaque or other approved assays. At appropriate time points, cells will be lysed and the RNA extracted using the approved TRIzol-based extraction method for virus inactivation. Extracted RNA will be used in RT-qPCR or bDNA assays to confirm reduction of viral RNA or knockdown of transcript.

Lead candidates will be tested in vivo in murine models with intranasal delivery of both virus and oligonucleotides. The effects of commensal bacterial on SARS-CoV-2 infection will be evaluated. Mice will be inoculated with *Prevotella salivae* or *Veillonella infantium* intranasally 48 hours prior to infection with SARS-CoV-2. For the animal models of infection, animals will be monitored for weight loss, survival, and lung tissue harvested and processed for viral load and inflammatory markers (e.g. cytokine expression). Hamsters also serve as a model for SARS-CoV-2 infection, but there are no immediate plans to perform oligonucleotide therapeutic experiments or commensal bacteria experiments (i.e., these deliveries are not linked to our IACUC protocol for hamsters).

IBC Discussion and Vote

Discussion: The reviewer discussed this is a protocol amendment requesting approval to reduce biocontainment requirements from BSL-3 to BSL-2. The reviewer details the experimental approach, including antiviral screening assays, the use of different SARS-CoV-2 variants, and the inclusion of human and non-human primate cells. The reviewer recommended approval at BSL-2 with enhancements, including the development of a BSL-2 SOP and the use of a designated procedure room after minor action items are addressed.

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

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

NIH Guidelines: III-D, III-F

6) Investigator: Wu, J
 Title: Detection of Increase or Decrease of DNA/Histone Methylation in Newborns from Obese and Non-Obese Mothers with or without Metabolic Syndromes

IBC Registration: **I-684-26, Renewal**

Training Verification: **Acceptable**

Brief Summary: My laboratory focuses on identifying novel epigenetic genes in childhood obesity and pediatric non-alcoholic fatty liver. Our overall goal is to develop effective therapies for childhood obesity related metabolic disorders.

Brief Summary and Review by Primary Reviewer

Overview and Objectives: Identifying novel epigenetic genes in childhood obesity and pediatric non-alcoholic fatty liver. Overall goal is to develop effective therapies for childhood obesity related metabolic disorders

Experimental Approach: Overexpress or Knock down candidate genes in mice by tail-vein injecting rAAV
 Overexpressing or knocking down the candidate genes: rAAV will be produced by Viral Core (Jun Xie). IVIS imaging of mice at the Animal Imaging Core at time points 6, 10, 14, 18, and 22 weeks post-injection. Light-microscope cell-growth checks. Flow core sorting unfixed mouse and human tumor cells.

GOI: Protein arginine methyltransferase; mitochondrial fitness (mouse PRMT5)

From 2021 description:

"To characterize novel molecular pathways regulated by PRMT5, PRMT5 small molecule inhibitors will be tested as potential therapeutic agents using cell culture and diet-induced obesity models. The specific experimental procedures include:

1. Isolate white blood cells from cord blood
2. Analyze gene expression by RNA-seq
3. Analyze epigenetic landscape by ATAC-seq

4. Overexpress candidate genes in mice by rAAV8
5. Knockdown candidate genes in mice by rAAV8"

IBC Discussion and Vote

Discussion: The reviewer discussed this is a 5-year renewal for a protocol focusing on epigenetic markers and novel therapeutic targets. Details are needed for full risk assessment, however information can be gleaned from responses to various sections of the registration and from prior renewal in 2021. A flow sorting addendum is needed.

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 Precautions/ Sharps Safety**

NIH Guidelines: III-D, III-E, III-F

7) **Investigator:** Wu, J
Title: Epigenetic Markers in Neuroblastoma
IBC Registration: **I-812-26, Renewal**
Training Verification: **Acceptable**
Brief Summary: My laboratory studies the epigenetic mechanisms mediating cancer cell proliferation, metabolism, drug resistance, and metastasis. One of the foci for my laboratory is identifying novel therapeutic targets in pediatric cancers. We will characterize novel molecular pathways in tumor subpopulations including cancer stem cells. microRNAs, lncRNA and classical genes (e.g. transcription factors) are evaluated as potential therapeutic targets using cell culture and xenograft tumor models. Overall goal is to develop improved therapies for neuroblastoma and other pediatric cancers to reduce treatment-related toxicity and improve overall cure rates.

Brief Summary and Review by Primary Reviewer

Overview and Objectives: The research team studies the epigenetic mechanisms mediating cancer cell proliferation, metabolism, drug resistance, and metastasis. Specifically, they seek to identify novel therapeutic targets in pediatric cancers. They will characterize novel molecular pathways in tumor subpopulations including cancer stem cells. Various genes will be evaluated as potential therapeutic targets using cell culture and xenograft tumor models. The overall goal is to develop improved therapies for neuroblastoma and other pediatric cancers to reduce treatment-related toxicity and improve overall cure rates.

Experimental Approach: The studies will involve cloning human cDNAs and expressing them in tissue culture. The team will also use CRISPR/Cas and siRNAs to downregulate and upregulate endogenous oncogenes and immunomodulators. Some experiments will use exogenous expression of human oncogenes and other proteins involved in cell cycle, proliferation, and metastasis. Gene products are naturally occurring proteins in normal cells. No toxic genes will be used. Cloning vectors include pCDNA3.1 (and variants) and a 3rd generation lentiviral packaging system. Recombinant proteins will be expressed in normal and malignant tumor cell lines (neuroblastoma lines in particular). Tumor growth will be evaluated in cell culture and in mouse xenografts. Transgenic animal models of neuroblastoma will also be used to test genetic interactions and possible therapeutic pathways. The Viral Vector and RNAi Cores will be used to produce shRNAs to knockdown candidate genes using replication-defective lentiviral vectors. Lentivirus transductions will be performed under BSL-2, animal housing under ABSL-1+BBP with special precautions. The Mutagenesis Core will be used to generate TALEN/CRISPR constructs targeting genes of interest. The Animal Imaging Core will be used to monitor tumor growth in live animals (IVIS system). The Flow Core will be used to sort cells expressing GFP/RFP at BSL-2 enhanced containment.

IBC Discussion and Vote

Discussion:	The reviewer discussed this is a 5-year renewal for a protocol studying epigenetic mechanisms mediating cancer cell proliferation, metabolism, drug resistance, and metastasis. The reviewer detailed the experimental approach, including the use of CRISPR-Cas and siRNAs, and the need for updated SOPs and flow sorting addendum and identified major and minor action items, including distinguishing viral vectors from bacterial plasmids, updating the vector checklist, and clarifying the use of transgenic mice. The reviewer emphasized the importance of accurate and complete registration details for risk assessment and approval. The overall goal is to develop improved therapies for neuroblastoma and other pediatric cancers to reduce treatment-related toxicities and improve overall cure rates. Chemotherapeutic agents are not under IBC purview (chemical safety oversight).
Meeting Decision:	Vote to approve upon completion of action items.
BSL/ABSL:	BSL-2+; BSL-2 Enhanced Flow Sorting; ABSL-1 + BBP with Special Precautions; Administration to Animals using BSL-2 Precautions/ Sharps Safety
NIH Guidelines:	III-D, III-E, III-F

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

- 1) Shutdown on BSL-3/ ABSL-3: for facility reverification at the end of August 2026
- 2) Fire alarm issue resolved

VI. Information from the field (Senior Biosafety Officer)

N/A

VII. Other Business

Updated forms and templates: Registration, Animal Addendum, Medical Alert Card template, Viral Vector Facts Sheet (new version)

[IBC Animal Addendum_062526.docx](#)

[Medical Alert Card Template_062526.docx](#)

[IBC Registration Form Example_062526.docx](#)

[Viral Vector Fact Sheet V6.pdf](#)

[Viral Vector Fact Sheet V7.pdf](#)

Acknowledgement Items:

N/A

Adjourned at 1:11pm