SAFETY Meeting Minutes
UAMS IBC

MEETING TIME RECORDS

Meeting start time: 6/6/2025 12:48 PM
Meeting end time: 6/6/2025 01:26 PM
Meeting type: Virtual

Name of Regular/Alternate Member	Status (Member or Alternate)	Present by Teleconference?
Ha-Neui Kim	Member	Yes
Matthew Jorgenson	Member	Yes
Robert Hunter	Member	No
RC Ragsdale	Member	No – Voted via e-mail
Kimberly Murphy	Member	Yes
Lindsey Clark	Member	Yes
James Douglas	Member	Yes
Amanda Holloway	De Novo	No
James Bishop	Member	Yes
Youssef Aachoui	Member	Yes
Jia Liu	Member	Yes
Yuet-Kin Leung	Member	Yes
Melaney Gee	Member	Yes
Mark Manzano	Member	No
Christine Simecka Morgan	Member	No
Antino Allen	Member	Yes
KyoungHyun Kim	Member	Yes
James Townsend	De Novo	No
Shengyu Mu	Member	Yes
Kikumi Ono-Moore	De Novo	Yes
Zhiqiang Qin	Member	No

QUORUM INFORMATION

Number of SAFETY members on the roster: 18

Number required for quorum: 9
Quorum: Yes

All members present via teleconference received all pertinent material before the meeting and were able to actively and equally participate in all discussions.

ATTENDANCE STATUS AND VOTING KEY	
ABSTAIN:	Present for the vote but not voting "For" or "Against."
ABSENT:	Absent for discussion and voting for reasons other than a conflict of interest.
RECUSED:	Absent from the meeting during discussion and voting because of a conflict of interest.
SUBSTITUTION:	When regular members and their alternate(s) are listed in the ATTENDANCE table above and an alternate member serves as a substitute for the regular member this identifies the name of the alternate to indicate which individual is serving as the voting member for this vote. May be deleted if there are no substitutions.

GUEST NAMES
Elisabeth Skaggs

Previous Meeting minutes approved: Yes

REVIEW OF SUBMISSIONS

The review and discussion of the protocols listed below included the following elements: the agents involved and their characteristics; types of manipulations planned; the source(s) and nature of the nucleic acid sequences; the host organism(s) and vector(s) to be utilized; whether expression of a foreign gene is intended and, if so, the specific protein(s) to be produced; the containment conditions to be applied, including biosafety level and any special provisions; and the relevant sections of the NIH Guidelines.

All IBC members present were reminded to identify any conflicts of interest as each registration was reviewed.

For each protocol reviewed, it was confirmed that the Principal Investigator (PI) and laboratory personnel have received appropriate training in the safe conduct of research.

De Novo Review

1. Review of SPROTO202500000036

Title:	Biomarkers of Retinal Ischemia
Investigator:	Abdelrahman Fouda
Submission ID:	SPROTO202500000036
Description:	The retina is the most metabolically active tissue in the human body, even more than the brain. Its oxygen dependency makes it highly susceptible to vascular injuries that might affect blood supply. Retinal vessels form for two capillary plexuses that anastomose but the main retinal arteries are terminal arteries without anastomosis. Hence, any occlusion can lead to the ischemia of the entire inner retinal layer. Retinal ischemia is a common cause of vision loss since it is the main contributor in the pathogenesis of many retinal vascular diseases including diabetic retinopathy (DR) and retinal vascular occlusions. These conditions are associated to the development of neovascularization through mediators such as vascular endothelial growth factor (VEGF) and other chemokines. VEGF is the most well-known mediator and marker found in retinal ischemia and has led to the development of therapies for multiple retinal vascular diseases. Understanding the retinal metabolic response to circulating high glucose levels in diabetic patients is critical for development of new therapeutic strategies to treat DR. Furthermore, in the setting of tissue ischemia, cellular metabolism is shifted from oxidative phosphorylation in the mitochondria to glycolysis in the cytosol. The retinal glycolytic function and glycolytic enzymes expression have never been investigated under diabetic or ischemic conditions. Our ongoing preclinical studies in mouse models of diabetic and ischemic retinopathy show reduced retinal glycolytic response under diabetes which is associated with decreased expression of key glycolytic enzymes in the diabetic retina. Examining the glycolytic response in the human diabetic and ischemic retinal disease will provide a better understanding of what regulates angiogenesis and might lead to the development of new diagnostic and therapeutic options in the future. This study aims to evaluate potential biomarkers and pathological mediators of retinal ischemia in different body fluids in subjects with diseases typically associated with retinal hypoperfusion and ischemia and in healthy subjects as control. Samples will be collected in the clinic then transferred to the research lab for aliquoting and further processing. The samples will be either processed for protein estimation or sent to

	Proteomics core for analysis. The handling of all the samples will be done inside the BSC and with the required PPE. Our findings from the human samples will be further validated on the bench side using different types of primary cells, in vitro and in vivo animal models. For the animal studies, we will further determine the underlaying mechanisms of our findings using the ischemia reperfusion, optic nerve crush and the oxygen induced retinopathy mice models. All of our animal models are established in our lab and approved by the IACUC. for in vitro studies, we will use different primary cell and cell lines including bovine retinal endothelial cells, human retinal endothelial cells, primary isolated macrophages and R28 mixed neuronal cell line. We will use the recombinant DNA products (siRNA or plasmids) to knockdown or overexpress HDAC3 in human or bovine retinal endothelial cells. Our in vitro studies include different insults such us oxygen glucose deprivation, hypoxia, hyperoxia or high glucose treatment. All experiments will be conducted as previously described by us and others while maintaining the required biosafety measures stated in our biosafety protocol. We have other ongoing project studying the CD47/SIRP neutralization as a treatment for ischemic stroke. The summary of research of this project is included below. 1) Relevance: Ischemic stroke is the fifth leading cause of death in the US and a leading cause of morbidity and long-term disability. Current treatments are only available for selected patients who present to a comprehensive stroke center within a certain time window. Here we propose to target CD47, a ‘do not eat me’ signal expressed on healthy cells and can be upregulated on dying cells under pathological conditions. Neutralization of CD47 with antibodies have been shown to enhance apoptotic (dying) cells clearance by phagocytic cells in a process called efferocytosis (phagocytosis of dying cells). 2) Overall Goals: We propose anti-CD47 antibody as a promising therapy for ischemic stroke to enhance clearance of dying cells and reduce inflammation. This proposal will investigate the potential use of anti-CD47 antibody as a treatment for ischemic stroke by quantifying the efferocytosis index and stroke outcomes in a murine stroke model. We will also investigate the effect of HDAC3 deletion or diabetes on stroke outcomes.

	3) Specific Aims: The central hypothesis is: Administration of CD47 neutralizing antibody will enhance efferocytosis of apoptotic neurons after stroke and lead to better disease outcomes. This will be tested by two specific aims: Aim 1: To determine the effect of CD47 neutralizing antibody on microglial/macrophage activation/infiltration and apoptotic cell clearance after stroke. Aim 2: To determine the effect of CD47 neutralizing antibody on long-term ischemic stroke outcome. 4) Experimental Approach: To test our approach, we will use multiple levels of analyses including cellular, molecular, immunohistochemical, and behavioral techniques. To induce stroke, mice will be subjected to temporary middle cerebral artery occlusion (tMCAO). To examine aim 1,anti-CD47 will be administered intraperitoneally at 3 hours and at day 2 post-tMCAO, which is representative of a clinically useful time frame to determine the effect of CD47 neutralization on the phagocytic cell response and apoptotic cell clearance post-stroke. Animals will be euthanized at 2 and 4 days post-stroke by transcardial perfusion under anesthesia. High resolution imaging will be utilized to examine macrophage/microglia interaction with apoptotic neurons in the brain after stroke. To examine aim 2, we will examine the effect of anti-CD47 treatment on stroke functional recovery. Injured animals will be evaluated longitudinally by neurobehavioral tests that specifically assess cognitive, motor and balance functions. Animals will be euthanized by transcardial perfusion under anesthesia 30 days post-stroke. Histological/immunohistochemical analyses will be used after euthanization to evaluate apoptosis, neuronal degeneration, and neuroinflammation. Infarct size analysis will be conducted using magnetic resonance imaging (MRI) in vivo. Collectively, these experiments will further our understanding of the mechanisms and functional benefits provided by anti-CD47 treatment or HDAC3 deletion and diabetes on stroke outcomes.
Agent Containment:	Biological Containment Levels: • Primary Human Tissue: BSL-2 • Animal Tissue: BSL-2 • Animal Blood: BSL-2 • Human Blood: BSL-2 • Human retinal endothelial cells : BSL-2 • Bovine retinal endothelial cells : BSL-2

Applicable NIH Guidelines:	• Section III-F • Section III-F-1 • Section III-D • Section III-D-3-a

a. **Determination:** Modifications Required

b. **Required modifications:** Minor revisions necessary. Please see comments and respond to all queries.

c. **Votes:**

For:	14
Against:	0
Recused:	0
Absent:	4
Abstained:	0

De Novo Review

2. Review of SPROTO202500000032

Title:	Epigenetic mechanisms in acute myeloid leukemia (BP339)
Investigator:	Samrat Roy Choudhury
Submission ID:	SPROTO202500000032
Description:	Acute Myeloid Leukemia is the third most prevalent cancer among the hematological malignancies in the United States and in the state of Arkansas. AML is presented with diverse epigenetic alterations, including recurrent mutations in the epigenetic modifiers (DNMT3a, TET2, and IDH1/2) or cytolological abnormalities resulting in the epigenetic gene fusions (KMT2A fusions or AML1-ETO9). Another fascinating feature of the AML is the shift in epigenetic driver mutations across the age groups. However, exceptional transcriptional competence was observed due to the over-expression of Retionoic Acid Receptor alpha (RARα)-PML fusion in a cohort of AML patients, irrespective of the age difference. Despite distinct epigenetic modifiers have been identified in the pediatric and adult course of the disease, similar phenotypic changes favoring the aggressive leukemogenesis have been manifested in many cases. Therefore, there remains a gap in our understanding of the epigenetic switching mechanisms of leukemic development and differentiation between different age groups. Our research focus is aimed at investigating the epigenetic regulatory mechanisms at the enhancers and promoters of the critical oncogenes and tumor suppressors that drive malignant proliferation and invasion during leukemogenesis in the AML

	patients, characterized with BET (BRD4), SET (KMT2A, EZH2, and NSD1) or runt (CBFA2T3) over-expressed domains. Using the multi-omics platform, we intend to identify the alterations in DNA methylation, histone modifications and transcription factor assemblies in and outside the topologically assorted domains (TAD) in AML. To address our objectives, we aim to perform the following routine experiments: # Next-Generation Epigenetic Sequencing techniques: We will perform enhanced reduced representation bisulfite sequencing (eRRBS) and TAB sequencing in a panel of human pediatric and adult AML cell lines (as mentioned in section-C) to determine DNA methylation and hydroxymethylation respectively. Gene expression will be determined using RNA-Seq. Genomic DNA and RNA will be extracted using the QIAGEN all prep kit (RLT buffer in the kit will be handled per chemical waste disposal regulations. We will be using commercial primary antibodies for the chromatin pull down experiments for the ChIP sequencing reactions. Libraries will be prepared with the commercially available kit from the Illumina Inc. and New England Biolabs (kits are non-biohazards). #Experimental validation of the epigenomics data: We will examine the effects of target gene over expression and knockdown to see what additional genes/proteins are affected, if AML aggressiveness is altered, and if the response to immunotherapy or small molecule inhibitors are altered. We will primarily use a 3rd generation, 4-plasmid, lentivirus system will be used and will involve 5 plasmids, two packaging plasmids (pRSV-Rev, pMDLg/pRRE), one envelope plasmid (pMD2.G), and three transfer plasmids (pLKO.1puro, pLJM1-EGFP, and pLenti-puro). The plasmids will be amplified using one of two E. coli cell lines (NEB stable, TOP10). Gibson assembly will be performed using PCR on an open bench for cloning the target genes into the transfer plasmid pLJM1-EGFP and for cloning CRISPR and sgRNA into the transfer plasmid pLKO.1puro. The samples to be amplified and ligated will only contain the transfer plasmids which do not contain any lentivirus structural genes and human transcription factor genes will be prepared in a BSL-2 hood in a 96-well plate with an adhesive cover. The plate will remain sealed and will not be opened outside of the BSL-2 hood. We will generate the lentivirus/ retrovirus in HEK 293T cells. The viruses will then be collected from the medium and applied to the AML cell lines.

	#Targeted epigenetic editing using CRISPR tools: We will fuse epigenetic modifiers such as the catalytic domain of Ten-Eleven Translocation Enzyme (TET1CD) and DNA methyltransferase 3A (DNMT3ACD), histone acetylases (HAT), histone deacetylases (HDAC) to the dCas9 enzyme in the lentiviral plasmid having the features, as mentioned in the previous section. Fusion proteins will have additional fluorescence markers such as mCherry or eGFP. Selection of transduced cells will be performed with 1 µg/mL Puromycin hydrochloride (Fisher Scientific) for 14 days. Induction of the fusion protein expression will be achieved with 1 µg/mL Doxycycline (Fisher) for 72 h and effectiveness of AML chimeric proteins on the downstream proteins will be confirmed by RT-qPCR and immunoblotting. All the cell culture, molecular biology, and Next-Generation Sequencing experiments will be carried out in ACRI, laboratory R4035 or R4036 # Animal research: A. Bioluminescence assay Project 1: Leukemic burdens measured with bioluminescence will be compared pairwise between mice administered with MED12/12L modified cells vs mice administered with vehicle (PBS), to identify differences among the groups within each measurement day using repeated-measures-ANOVA post-hoc comparisons and no adjustment will be made for multiple comparisons. Project 2: Leukemic burdens measured with bioluminescence will be compared pairwise between mice administered with DNMT1 KO, DNMT3B KO and both DNMT1 KO and DNMT3B KO construct modified cells vs mice administered with vehicle (PBS), to identify differences among the groups within each measurement day using repeated-measures-ANOVA post-hoc comparisons and no adjustment will be made for multiple comparisons. Leukemic burdens measured with bioluminescence will be compared pairwise between mice administered with DNMT3B KO-1, DNMT3B KO-2 modified cells vs mice administered

	with vehicle (PBS), to identify differences among the groups within each measurement day using repeated-measures-ANOVA post-hoc comparisons and no adjustment will be made for multiple comparisons. Project 3: Leukemic burdens measured with bioluminescence will be compared pairwise between mice administered with MOLM-13 or WSU-AML + vehicle vs MOLM-13 or WSU-AML + 8p, to identify differences among the groups within each measurement day using repeated measures ANOVA post-hoc comparisons and no adjustment will be made for multiple comparisons. B. Chemo-sensitivity assay Briefly, n=18 mice from each group × sex combination will be randomized at 6 mice/treatment into one of 3 daunorubicin treatments starting at week 4 of the injection. Daunorubicin in 100 µL of PBS (or PBS alone) will be administered intravenously 3 times at once per week. Leukemic growth will be monitored and analyzed every 2 weeks by bioluminescent imaging for 6 months. Tumor burdens determined over time from the bioluminescent imaging will be subjected to mixed-effect ANOVA for purposes of estimation. Briefly, n=24 mice, two groups of treatment combinations 6 mice/sex (n=24; control group=12, treatment group= 12). Mice from group-1 (wild-type AML cells with no genetic modification) and group-2 (DNMT1/3B modified AML cells) from week 2 of injection will be orally administered with Venetoclax at 25 mg/kg. Both the group will receive 15 mg Venetoclax in 15% Cremophor EL + 85% Saline (oral suspension of 200 microliter (µL)). Drugs will be orally administered 6 times per week. Anti-leukemic effects of the drugs will be monitored every 7 days using IVIS imaging for 3 months. The progression of leukemic growth will be monitored by phenotypic changes in addition to bioluminescence (IVIS imaging). All the proposed animal-related work will be carried out at the UAMS, WPRCI animal vivarium facility. Dr. Ryan Porter will be assisting us with training for the Imaging Center.

Agent Containment:	Biological Containment Levels: • Lentivirus: BSL-2 • E. coli: BSL-1 • E. coli: BSL-2 • U-937 (Human Myeloid Leukemia Cell Line): BSL-2 • ME-1 (Human Acute Myeloid Leukemia Cell Line): BSL-2 • Kasumi-1 (Acute Myeloid Leukemia Cell Line): BSL-2 • TF-1 Human Cell Line: BSL-2 • HEK293T Human Cell Line: BSL-2 • MV4-11 (Human Acute Myeloid Leukemia Cell Line): BSL-2 • KG-1a (Human Acute Myelogenous Leukemia Cell Line): BSL-2 • HL-60 (Human Leukemia Cell Line): BSL-2 • M-07e Cell Line: BSL-2 • MOLM-13 (Human Acute Myeloid Leukemia Cell Line): BSL-2 • WSU-AML: BSL-2 • MEGAL Cell Line: BSL-2
Applicable NIH Guidelines:	• Section III-D-1-a • Section III-D-4 • Section III-D-1

a. **Determination:** Modifications Required

b. **Required modifications:** Minor revisions necessary. Please see comments and respond to all queries.

c. **Votes:**

For:	14
Against:	0
Recused:	0
Absent:	4
Abstained:	0

De Novo Review

3. Review of SPROTO202500000033

Title:	Scavenger Receptors in disease
Investigator:	Steven Post
Submission ID:	SPROTO202500000033
Description:	A critical component of inflammatory diseases is the interaction between the various tissue cells and inflammatory cells. We are interested in the interactions between various

	stromal cells, extracellular matrix, and macrophages; and in particular the role of specific proteins in mediating these interactions. The protein of interest is the macrophage scavenger receptor class A (SR-A) and its interactions with modified extracellular proteins and tumor cells. To study these interactions, we will examine SR-A expression and importance in physiology/pathophysiology using mouse models that do or do not express SR-A. Tissues will be harvested from these animals and used for routine cell biological and biochemical assays. Cell lines in which protein expression has been altered will also be used to study the impact on cell function and interaction.
Agent Containment:	Biological Containment Levels: • Animal Tissue: BSL-2 • Human Embryonic Kidney 293 (HEK293): BSL-2 • THP-1: BSL-2 • Raw-278: BSL-2 • MCF7 Human Cell Line: BSL-2 • J774: BSL-2 • Animal Cells: BSL-2 • Mouse Bone Marrow Macrophages: BSL-2 • Chinese Hamster Ovary (CHO) Cells: BSL-1 • Animal Cells: BSL-2 • 4T1 Murine Cell Line: BSL-2 • BT20 Human Cell Line: BSL-2 • E0771 Murine Cell Line: BSL-2
Applicable NIH Guidelines:	• Section III-D-1 • Section III-D

a. **Determination:** Modifications Required

b. **Required modifications:** Minor revisions necessary. Please see comments and respond to all queries.

c. **Votes:**

For:	14
Against:	0
Recused:	0
Absent:	4
Abstained:	0

De Novo Review

4. Review of SPROTO202500000034

Title:	The Role of Endothelial TRPC3 Channels in Neurovascular Coupling (BP266)
Investigator:	Fang Zheng
Submission ID:	SPROTO202500000034
Description:	This study uses the mouse brain as a model to study the role of a family of newly discovered membrane proteins in the regulation of cerebral blood flow. These proteins are called TRPC channels (canonical transient receptor potential channels), and they form openings (channels) in the outer cell membrane and gate the flow of calcium ions from the extracellular space into neurons. New findings from this study will provide the foundation for designing new therapy for seizure, stroke and other brain diseases. This proposal seeks to generate an inducible and brain-specific endothelial TRPC3 knockout line and to use this novel mouse line to elucidate the role of TRPC3 channels expressed in cerebral vasculature endothelial cells in seizure-induced reduction of cerebral blood flow. A toxic chemical, tamoxifen, will be used to induce the expression of cre, a protein that will modify TRPC3 gene in brain endothelial cells in mice. Tamoxifen powder (Sigma) will be dissolved in corn oil in a chemical hood in Room 513-2 in Biomed II. Tamoxifen solution will be transported to DLAM facility in double container to be administered to mice in the hood inside the animal room in DLAM or Rm 513-2. For Genotyping, tail samples will be collected in DLAM facility using DLAM required PPE. Genomic DNA isolation and PCR will be performed in Rm 513-2 of Biomed II, with standard laboratory PPE. Tamoxifen will be prepared in a Chemical Fume Hood in Biomed II room 513-2.
Agent Containment:	
Applicable NIH Guidelines:	

a. **Determination:** Modifications Required

b. **Required modifications:** Minor revisions necessary. Please see comments and respond to all queries. Agent containment and applicable NIH guidelines need to be indicated.

c. **Votes:**

For: 14
Against: 0
Recused: 0

Absent: 4
Abstained: 0

Initial Protocol

5. Review of SPROTO202500000028

Title:	Mechanisms of DNA Damage Repair
Investigator:	Kirk West
Submission ID:	SPROTO202500000028
Description:	1. Determine the mechanisms of proteins within the DNA damage response to better understand the development of chemoresistance. This project will elucidate how proteins directly impact DNA damage repair choice and the process of repairing damaged DNA. This project will involve the use of human cell lines that are transiently transfected with plasmids containing antibiotic resistance genes to ampicillin, kanamycin, blasticidin, zeocin, hygromycin, or puromycin. No known pathogenic genes will be contained in these plasmids. Plasmids will contain open reading frames for endogenously coded DNA damage repair proteins with or without loss of function mutations to identify domain specific functions. Common siRNA techniques involving the direct transfection of siRNA into human cell culture will be carried out for gene Knockdown experiments. The CRISPR cas9 system will be used to generate endogenous mutations within the human cell line genomes to overcome the potential negative effects of transient transfection. Cells will primarily be used for three types of experiments, Western blotting/Proteomics, confocal microscopy, or flow cytometry. To induce DNA damage cells will be treated with DNA damaging drugs such as hydroxyurea, camptothecin, etoposide, mitomycin C, cisplatin, olaparib, or ultraviolet light, or ionizing radiation by using the fee for service XRAD in the Radiation Oncology/Radiation Health department. 2. Determine the role of oligomerization of DNA damage proteins on their function. This project will involve the purification of proteins to study their biochemical mechanisms in an In vitro setting. We will utilize E. coli expression by transforming them with a plasmid encoding the gene of interest for purification. No known pathogenic genes will be introduced. Plasmids containing antibiotic resistance genes for ampicillin, kanamycin, or spectinomycin will be used. For

	some recombinant proteins an insect cell expression system will be used. In this system pFastBac vectors containing the opening reading frame of the gene of interest will be recombined into a bacmid by transformation into Gibco MAXEFFICIENCY DH10Bac E. coli. These bacmids will then be transfected into SF9 insect cells from Gibco to produce baculovirus. Baculovirus will then be used to infect SF9 cells for recombinant protein production.
Agent Containment:	Biological Containment Levels: • E. coli: BSL-2 • 293T: BSL-2 • Baculovirus: BSL-2 • E. coli: BSL-2 • E. coli: BSL-2 • RPE-1 Human Cell Line: BSL-2 • HeLa cells: BSL-2 • E. coli: BSL-2 • E. coli: BSL-2 • U2OS: BSL-2
Applicable NIH Guidelines:	• Section III-D-1-a • Section III-D-4-a • Section III-D

a. **Determination:** Modifications Required

b. **Required modifications:** Minor revisions necessary. Please see comments and respond to all queries.

c. **Votes:**

For:	14
Against:	0
Recused:	0
Absent:	4
Abstained:	0

De Novo Review

6. Review of SPROTO202500000037

Title:	HDAC11 in diabetic cardiomyopathy
Investigator:	Rushita Bagchi
Submission ID:	SPROTO202500000037
Description:	This research investigates the role of epigenetic regulators such as histone deacetylase 11 (HDAC11) in diabetes. HDACs are largely known to influence chromatin and gene expression, but also can cause changes in protein modifications. Our

	previous work has established the role of HDAC11 in obesity. Diabetes and obesity are known to increase the risk of heart failure in humans. Deficiency of HDAC11 in mice has been shown to be protective against diet-induced obesity. Here we are extending our studies and exploring the mechanism(s) that may lead to heart failure in the setting of diabetes. This study used mouse and cell culture models with genetic or pharmacological targeting of HDAC11 to test the hypothesis that HDAC11 promotes diabetic cardiomyopathy through effects on genes involved in lipid metabolism, and affects cardiomyocyte homeostasis. We will use a mixed model of pharmacologic and diet intervention to cause diabetic heart disease in mice- a single injection of streptozotocin will be sufficient. Recombinant DNA approaches (plasmids, adenovirus, lentivirus) will be used to alter gene expression in cell culture models. Recombinant adeno associated virus may be used in some cases to induce Cre recombinase expression in mouse hearts which do not express the cre transgene. In mice that already have the Cre transgene, tamoxifen injection will be used to activate and drive its translocation to the nucleus to cause gene deletion. Doxorubicin will be used to study cardiotoxicity mechanisms in cell culture systems. Human cells will not be used in animals. All animal work and cell culture with be done at UAMS. Two BSCs are available for cell culture in the UAMS lab in Biomed 1 B218.
Agent Containment:	Biological Containment Levels: • Human Serum: BSL-2 • Primary Human Tissue: BSL-2 • Animal Tissue: BSL-2 • Animal Tissue: BSL-1 • Adenoassociated virus AAV: BSL-2 • Lentivirus: BSL-2 • Adenovirus: BSL-2 • E. coli: BSL-2 • Valvular Cells: BSL-2 • Cardiomyocytes: BSL-2 • Human Vascular Endothelial Cells: BSL-2

	• Mouse Bone Marrow Macrophages: BSL-2 • Human iPS Cells: BSL-2 • Human Epithelial Cells: BSL-2 • Mouse Primary Endothelial Cells: BSL-2 • HEK293: BSL-2 • Human Mesenchymal Stem Cells: BSL-2 • Aortic Smooth Muscle Cells: BSL-2 • Adipocytes: BSL-2 • Human Vascular Smooth Muscle Cells: BSL-2 • Human Valvular Cells: BSL-2 • Fibroblasts: BSL-2 • RAW 264.7 Macrophage Cell Line: BSL-2
Applicable NIH Guidelines:	• Section III-D-4 • Section III-F • Section III-F-1 • Section III-D

a. **Determination:** Modifications Required

b. **Required modifications:** Minor revisions necessary. Please see comments and respond to all queries.

c. **Votes:**

For:	14
Against:	0
Recused:	0
Absent:	4
Abstained:	0

REVIEW OF OTHER AGENDA ITEMS