

SAFETY Meeting Minutes
UAMS IBC

MEETING TIME RECORDS

Meeting start time: 5/1/2026 12:01 PM

Meeting end time: 5/1/2026 1:20 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 – voted by e-mail
Kimberly Murphy	Member	Yes
Lindsey Clark	Member	Yes
James Douglas	Member	Yes
James Bishop	Member	No
Youssef Aachoui	Member	No
Jia Liu	Member	Yes
Yuet-Kin Leung	Member	Yes
Melaney Gee	Member	Yes
Mark Manzano	Member	Yes
Christine Simecka Morgan	Member	No
Antino Allen	Member	No
KyoungHyun Kim	Member	No
James Townsend	Ex Officio	Yes
Shengyu Mu	Member	Yes
Kikumi Ono-Moore	Ex Officio	Yes
Zhiqiang Qin	Member	No

QUORUM INFORMATION

Number of SAFETY members on the roster: 17

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
Alyse Briggs, AJ Ewll, Cole Birmingham, Riya Patel

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.

Initial Protocol

1. Review of SPROTO202600000015

Title:	Lab Safety Protocol for Schuller de Almeida
Investigator:	Maria Schuller De Almeida
Submission ID:	SPROTO202600000015
Description:	<p>NIH Guidelines, Sections: III-D-4-c-(1), III-D-4-c- (2), III-E-3-a, III-F-8-C-II, III-F-8-C-VII, and III-F-8-C-VIII</p> <p>To study osteoporosis pathophysiology, genetically modified mice using cre-lox technology are used to achieve conditional gene deletion or overexpression at a specific developmental time point and in a specific tissue constitutively. Gene and protein expressions are compared to normal and non-target tissues and cell culture models by western blotting and RNA gene expression assays. Skeletal and bone phenotype comparisons are made between both young and old, and both intact and sex steroid-depleted conditions.</p> <p>Additional investigational laboratory techniques used include DNA extraction for genotyping, dual-energy x-ray absorptiometry to visualize and quantify changes in skeletal density, x-ray microtomography to visualize and quantify bone architectural changes, and histomorphometry to visualize and quantify bone cell location, number and distribution in the bone microenvironment.</p> <p>Bone marrow is collected and cultured to form either the bone cells that create bone matrix, osteoblasts (Ob), or the bone cells that destroy bone matrix, osteoclasts (Oc). Blood is collected and processed to extract plasma to test for circulating markers in enzyme-linked immunosorbent assays (EIA) for osteoblastic bone formation or osteoclastic bone resorption. Dysregulation or imbalance of these processes affect the strength and structural integrity of the skeleton that leads to osteoporosis disease.</p> <p>NIH Guidelines, Sections III-D-1-a and III-F-1</p> <p>Under BSL-2, gene targeting lentiviruses allow gene silencing in-vitro by RNA interference. Non-replicating RNAi molecules are used to downregulate the expression of individual target genes in primary cells or cell lines to explore individual gene and protein functions as well as their relationships to other genes and proteins.</p> <p>NIH Guidelines, Sections III-D-1-a and III-F-1</p> <p>Under BSL-2, using a replication-defective, cre-recombinase-</p>

	<p>targeting adenovirus expressing green fluorescent protein, the targeted gene modification expression in the genetically modified mouse strains we possess can be confirmed and tested in vitro using harvested primary cells prior to scaling up for large-scale in vivo experiments.</p>
<p>Agent Containment:</p>	<p>Biological Containment Levels:</p> <ul style="list-style-type: none"> • Animal Tissue: BSL-1 • Animal Serum: BSL-1 • Animal Blood: BSL-1 • MEF (Mouse Embryo Fibroblasts): BSL-2 • MLO-Y4: BSL-2 • C2C12 Murine Myoblast Cell Line: BSL-2 • NIH3T3: BSL-2 • Murine Bone Marrow Stem Cells: BSL-2 • Mouse Bone Marrow Macrophages: BSL-2 • Chinese Hamster Ovary (CHO) Cells: BSL-2 • UAMS-32: BSL-2 • Lentivirus: BSL-2 • Muscle Tissue: BSL-1 • Adenovirus: BSL-2 • HeLa cells: BSL-2 • PBMC: BSL-2 • LNCaP: BSL-2 • MCF7 Human Cell Line: BSL-2 • OB-6 osteoblastic cells: BSL-2 • E. coli: BSL-1 • E. coli: BSL-1 • ST2: BSL-2
<p>Applicable NIH Guidelines:</p>	<ul style="list-style-type: none"> • Section III-D-1-a • Section III-F-2 • Section III-E-3 • Section III-E-3-a • Section III-D-4 • Section III-D-4-c-(1) • Section III-F • Section III-E • Section III-F-8-C-II • Section III-F-1 • Section III-D-1 • Section III-F-8-C-VII • Section III-D-4-c-(2) • Section III-D • Section III-D-4-c • Section III-F-8-C-VIII

- a. **Determination:** Modifications Required
- b. **Required modifications:**

Committee Determination: Modifications Required.

Please review and respond to all comments throughout submission.

Please contact BSO J.C. Douglas (jdouglas@uams.edu) or Lindsey Clark (Lclark4@uams.edu) with any questions/concerns. Thank you!

c. Votes:

For: 10
Against: 0
Recused: 0
Absent: 7
Abstained: 0

De Novo Review

2. Review of SPROTO202600000026

Title:	Studying pathogenic Borrelia spirochetes (BP101)
Investigator:	Jon Blevins
Submission ID:	SPROTO202600000026
Description:	<p>The goal of this work is to identify genes that contribute to pathogenesis of spirochetes belonging to the genus Borrelia. Specifically, we are studying the bacteria that cause Lyme disease (LD) and relapsing fever (RF). Borrelia burgdorferi, Borrelia turicatae, and Borrelia duttonii represent the current/active focus of our lab, but we have three additional RF Borrelia (e.g., Borrelia hermsii, Borrelia crocidurae, and Borrelia recurrentis) that we cultivate and may include in our future genetic and infection studies.</p> <p>1) To assess the contributions of specific genes during infection, we disrupt genes of interest in Borrelia using allelic exchange and plasmid-based suicide vectors. The suicide vector is generated with an antibiotic resistance marker interrupting the gene being targeted for mutagenesis. The mutational vector is constructed in an E. coli cloning strain. The mutational vector is electroporated into Borrelia and the target gene is rendered non-functional upon integration of the marker into the target gene via homologous recombination.</p> <p>2) Phenotypic analyses are performed on Borrelia mutant clones and differences are confirmed by genetic complementation (e.g., reintroduction of an intact copy of the gene of interest). Complementation requires cloning the intact</p>

	<p>gene and adjacent regulatory regions into either a borrelial shuttle vector or a site-specific integration vector. The latter approach inserts the gene cassette into the bacterial genome. The resulting constructs are transformed into a <i>Borrelia</i> mutant clone and phenotypic analyses are repeated.</p> <p>3) Gene expression studies employing transcriptional reporters require the cloning of promoter regions of <i>Borrelia</i> genes of interest upstream of either a promoterless green fluorescent protein (GFP), red fluorescent protein (TagRFP or mCherry), or promoterless luciferase genes (firefly or Renilla). Reporter constructs are then transformed into <i>Borrelia</i>.</p> <p>4) Inducible expression studies are facilitated by cloning the <i>Borrelia</i> gene of interest under the control of a lac/IPTG-inducible promoter. Constructs are generated in <i>E. coli</i> and transformed into <i>Borrelia</i> on a borrelial shuttle vector or site-specific integration construct that inserts the gene cassette into the genome of the bacterium.</p> <p>5) Recombinant protein expression requires cloning the <i>Borrelia</i> gene of interest into an expression construct. The resulting construct is transformed into one of several <i>E. coli</i> expression strains, and recombinant proteins are then purified from <i>E. coli</i>. These recombinant proteins are used as antigen for generation of antisera or for in vitro functional assays.</p>
<p>Agent Containment:</p>	<p>Biological Containment Levels:</p> <ul style="list-style-type: none"> • Animal Serum: BSL-2 • Human Serum: BSL-2 • Human Blood: BSL-2 • Animal Tissue: BSL-2 • <i>Borrelia recurrentis</i>: BSL-2 • <i>Staphylococcus aureus</i>: BSL-2 • <i>Borrelia duttonii</i>: BSL-2 • <i>E. coli</i>: BSL-2 • <i>Borrelia turicatae</i>: BSL-2 • <i>Borrelia burgdorferi</i>: BSL-2 • <i>Salmonella enterica</i>: BSL-2 • <i>Pseudomonas aeruginosa</i>: BSL-2 • <i>Borrelia hermsii</i>: BSL-2 • <i>Borrelia crocidurae</i>: BSL-2 • <i>Bacillus subtilis</i>: BSL-2
<p>Applicable NIH Guidelines:</p>	<ul style="list-style-type: none"> • Section III-D-1-a • Section III-D-2-a • Section III-D-1 • Section III-D-2

	• Section III-D
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- a. **Determination:** Modifications Required
- b. **Required modifications:**
 Committee Determination: Modifications Required.
 Please review and respond to all comments throughout submission.
 Please contact BSO J.C. Douglas (jdouglas@uams.edu) or Lindsey Clark (Lclark4@uams.edu) with any questions/concerns. Thank you!
- c. **Votes:**
 - For:** 10
 - Against:** 0
 - Recused:** 0
 - Absent:** 7
 - Abstained:** 0

De Novo Review

3. Review of SPROTO202600000031

Title:	Role of Micro-RNA in Vascular Malformations
Investigator:	Graham Strub
Submission ID:	SPROTO202600000031
Description:	Vascular anomalies are a heterogeneous group of congenital blood vessel disorders more typically referred to as birthmarks, which are subcategorized into vascular tumors and vascular malformations. Lymphatic (LM) are congenital lesions characterized by the formation of dilated lymphatic channels that can infiltrate adjacent structures and cause significant morbidity due to mass effect, infection, cosmetic deformity, and recurrence after treatment. These lesions vary in size and location and can result in significant or life-threatening morbidity due to infiltration and compression of critical structures. Somatic gain-of-function mutations in PIK3CA have been identified in the majority of these lesions, yet the precise mechanisms underlying their initiation and progression remain unclear. MicroRNAs (miRNAs) act as post-transcriptional regulators of gene expression and are necessary for vascular and lymphatic endothelial cell growth and development; however, their functions in vascular malformations are unknown. microRNA-21 (miR-21), a known oncogenic microRNA, is a driver of endothelial cell proliferation.

	<p>In this proposed work, our long-term goal is to identify suitable miRNA targets for the development of deliverable, miRNA-based therapeutics. identify miRNA targets for potential therapeutic development. This proposal aims to profile differentially expressed miRNAs in vascular malformations, particularly LM versus normal tissues, identify their mRNA and protein targets, and assess their functional impact. Tissue samples will be collected from consented patients and stored in a coded biorepository. Endothelial cells will be isolated and subjected to comprehensive molecular analyses, including RT-PCR, sequencing, proteomics, and functional assays. This work aims to uncover miRNA-driven mechanisms in vascular malformations, particularly LMs and identify novel targets for therapeutic intervention.</p>
<p>Agent Containment:</p>	<p>Biological Containment Levels:</p> <ul style="list-style-type: none"> • Human Serum: BSL-2 • Primary Human Tissue: BSL-2 • Human Blood: BSL-2 • Lentivirus: BSL-2 • Human Dermal Lymphatic Endothelial cells (HDLECs) : BSL-2 • Human Primary Endothelial Cells: BSL-2
<p>Applicable NIH Guidelines:</p>	<ul style="list-style-type: none"> • Section III-D-1-a • Section III-F • Section III-F-1 • Section III-D-1 • Section III-D

a. **Determination:** Modifications Required

b. **Required modifications:**

Committee Determination: Modifications Required.

Please review and respond to all comments throughout submission.

Please contact BSO J.C. Douglas (jdouglas@uams.edu) or Lindsey Clark (Lclark4@uams.edu) with any questions/concerns. Thank you!

c. **Votes:**

For:	10
Against:	0
Recused:	0
Absent:	7
Abstained:	0

REVIEW OF OTHER AGENDA ITEMS