

FY-2022 ANNUAL REPORT



Boston University National Emerging
Infectious Diseases Laboratories



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Picture on front cover: SARS-CoV-2 neurodissemination in the transgenic humanized K18hACE2 mouse brain at the level of the hippocampus: SARS-CoV-2 Spike (orange); neurons-NeuN (teal); microglia-Iba1 (magenta); astrocytes-GFAP (green); blood vessels-CD31 (red); DAPI-grey. Douam and Crossland labs.. Students: Anna Tseng and Devin Kenney

Picture on back cover: Post-primary tuberculosis in the immunocompetent but susceptible C3H.B6-sst1 mouse lung: pan-macrophage-Iba1 (blue), M1 macrophages-iNOS (teal), M2 macrophages-Arg1 (yellow); T cells-CD3epsilon; B cells-CD19-green; DAPI-grey. Kramnik and Crossland labs. Student: Anna Tseng. Post-doc: Shivraj Yabaji

September 1, 2022

Letter from the Director

We are in the latter half of the third year of the ongoing COVID-19 pandemic. Last year, as I wrote this letter, we were in the midst of the emergence of the SARS-CoV-2 “delta” variant, and now we face yet another variant evolution, the “omicron” variant(s). Each variant that emerges becomes dominant because it is more infectious than those that appeared before. While hospitalizations and death from COVID-19 continue, vaccines (or prior COVID infections) have largely blunted the high incidence of severe disease evident in the first waves of the pandemic. COVID exhaustion is evidenced by a largely unmasked population which has tried to return to pre-COVID ways of life. Yet, almost 6.5 million lives have been lost, with over 1 million of those in the US, evidence of the tragedy that has unfolded during the pandemic. The number of people who have been infected continues to increase. How long immunity to infection will last is unknown, nor is it known how many more variants this virus can evolve, but we will likely be contending with the SARS-CoV-2 virus for the rest of our lives. We are fortunate to have one of our accomplished physicians, Nahid Bhadelia, NEIDL investigator and the founding director of CEID (the Center for Emerging Infectious Disease Research and Policy), serving in the White House as a senior policy advisor for global COVID response.

COVID-19 is not the only emerging infectious disease we have to contend with. Monkeypox, a disease largely unknown outside of 7 countries in central and western Africa where it is endemic has now spread widely outside of Africa. Infections have increased in Africa over the past several years and there have been warnings about its ability to emerge elsewhere that have been largely ignored. It has now spread to more than 80 countries with over 41,000 cases, including over 14,100 in the US. We have new generation vaccines to smallpox which are highly likely to be effective against monkeypox, but the available doses are not sufficient for this unexpectedly large outbreak. Polio outbreaks are also feared to be on the horizon in New York and in London. While both monkeypox and polio can spread “under the radar” and take hold rapidly, we do have the tools to diagnose, vaccinate high risk members of the population, and contain the outbreaks. Nevertheless, these latest outbreaks emphasize how important it is to invest in our public health systems, and pay attention to warning signs that have not been heeded.

We are fortunate to have a cadre of investigators with diverse pathogen expertise in the NEIDL, capable of studying these pathogens, including those with experience with the poxviruses (Connor) and Picornaviruses, of which polio is a member (Saeed). However, there remain many pathogens we know exist for which we have few tools to combat, leaving those of us in the research space plenty to do. We are also reminded often that there remain many pathogens capable of infecting humans still undiscovered in nature. Just this month, a newly described virus, named Langya Henipavirus, and likely of animal origin, has been associated with the infection of several dozen humans, causing febrile disease as well as more severe symptoms. The Henipavirus genus includes Nipah virus, a virus that causes severe disease in humans with a very high case fatality rate. Nipah virus is a high priority virus for study and is considered one that has high pandemic potential (the virus outbreak in the 2011 movie “Contagion” was modeled after Nipah virus. Several investigators in the NEIDL have the expertise to study this virus, have been planning and seeking funding to study it, and are now working on various aspects of this virus, from understanding its replication and testing potential vaccines and therapeutics (Griffiths, Fearn, Mühlberger and colleagues). These investigators will bring many of the lessons learned from studies initiated during the COVID-19 pandemic to accelerate research on this virus.

In addition to these viruses, NEIDL researchers continue to study many other pathogens of importance to human health. These include a number of filoviruses, including Ebola virus, Sudan virus, Marburg virus, and the more recently discovered Lloviu virus isolated from bats in Europe (Mühlberger, Griffiths, Davey, Hume). Of course, we continue to study SARS-CoV-2, not only to understand the complexity of the virus, but to develop and test therapeutics and next generation vaccine candidates. Our faculty also have been developing animal models to study the disease caused by the virus (Griffiths, Davey, Honko, and Douam) as well as study “long COVID”, the plethora of symptoms that can persist for months or longer in patients who have recovered from COVID-19. These studies have benefited from our accomplished expert in the use of telemetry to interrogate disease processes (Honko) as well as our Veterinary Pathologist (Crossland) and his team. We also continue to study diseases that largely impact the very young, like

Respiratory Syncytial Virus (Fearn) and Enterovirus D68 (Saeed), as well as one of the most “successful” pathogens on earth, Mycobacterium tuberculosis (Kramnik). We are also fortunate to have been selected for the BU Provost’s Basic Life Sciences joint hiring initiative, resulting in the recruitment of a new NEIDL investigator who will join September 1, 2022, Dr. Adriana Tomic. Dr. Tomic will join Boston University as an Assistant Professor of Microbiology and Biomedical Engineering. She is an accomplished immunologist who uses machine learning/artificial intelligence to interrogate human responses to pathogens and vaccines, using influenza virus vaccines as one model system.

As I have noted before, we cannot do the science without our entire team of professionals who are dedicated to our overall success. Everyone has to work together – not only the scientific staff and trainees, but also our biosecurity team, facility engineers and mechanics, environmental health and safety professionals, and our information technology staff. None of the science could be done without this group of talented staff. Activities carried out by these groups are highlighted in this report. At the same time, we have continued to keep the community informed of the work that we are doing through the efforts of our community relations team, led by Valeda Britton. This team also spearheaded a number of activities to educate the young professionals of the future, and some of these have been highlighted as well.



Ronald B. Corley, Ph.D.
Professor of Microbiology
Director, National Emerging Infectious Diseases Laboratories

Mission Statement

The Boston University National Emerging Infectious Diseases Laboratories (NEIDL) mission is to generate and translate fundamental knowledge on high priority emerging infectious diseases for the benefit of the public health, locally, nationally, and globally.

Emerging infectious diseases are defined as those that have newly appeared and been recognized in the population or have existed but are rapidly increasing in incidence or in geographic range. To meet our missions the NEIDL will:

1. Perform innovative basic, translational, and clinical research on emerging infectious diseases, especially those NIH/NIAID identifies as high Priority Category A, B, and C pathogens, in order to develop diagnostic tests, treatments and vaccines to promote the public's health.
2. Provide education and training in these areas of research, in order to develop the next generation of scientists in this field, and to support a national response in the event of a biodefense emergency.
3. Establish a research facility with the highest attention to community and laboratory safety and security.

Strategic Goals

To successfully fulfill its mission, NEIDL has developed and is implementing a strategic plan to:

- i. Partner with academic departments across the university to recruit a cadre of investigators, as well as to develop research staff with expertise in the scientific disciplines required to investigate the pathogenesis of emerging infectious diseases caused by category A, B and C agents. We encourage and support the development of national and international research collaborations in order to carry out our mission.
- ii. Develop physiologically relevant models for the comparative study of these pathogens, mimicking as closely as possible the human disease process. Not only does this require that we recruit faculty with expertise in animal modeling and veterinarian pathology, but also develop the needed services to support these investigations.
- iii. Move promising basic research as rapidly as possible to translational, preclinical, and clinical research in animals and humans in partnership with appropriate collaborators.
- iv. Create and establish the methodologies needed to advance the development and testing of vaccines, therapeutics, and diagnostics for these agents.
- v. Train scientists and related support personnel in the requirements to perform maximum containment research in a safe and secure environment.
- vi. Maintain the flexibility needed to support a national response in the event of a biodefense emergency.
- vii. Ensure a "safety first" environment for the conduct of all activities in the NEIDL

NEIDL by the Numbers

Total amount funded
\$38 Million

114 Total Publications
33 co-authored by multiple
NEIDL faculty

73 Funded Sponsored
Projects

50 by Core Faculty
23 by Affiliate Faculty

14 Core Faculty
7 Affiliate Faculty

86 trained staff at BSL-3
16 trained in FY22

63 trained staff at BSL-4
18 trained in FY22

14 Postdocs
22 Graduate Students
6 Undergraduate Students
4 Visiting Scientists
3 Research Scientists
18 Research Lab Support Staff
19 Animal Science support Staff
55 Operations staff
6 Administrative staff

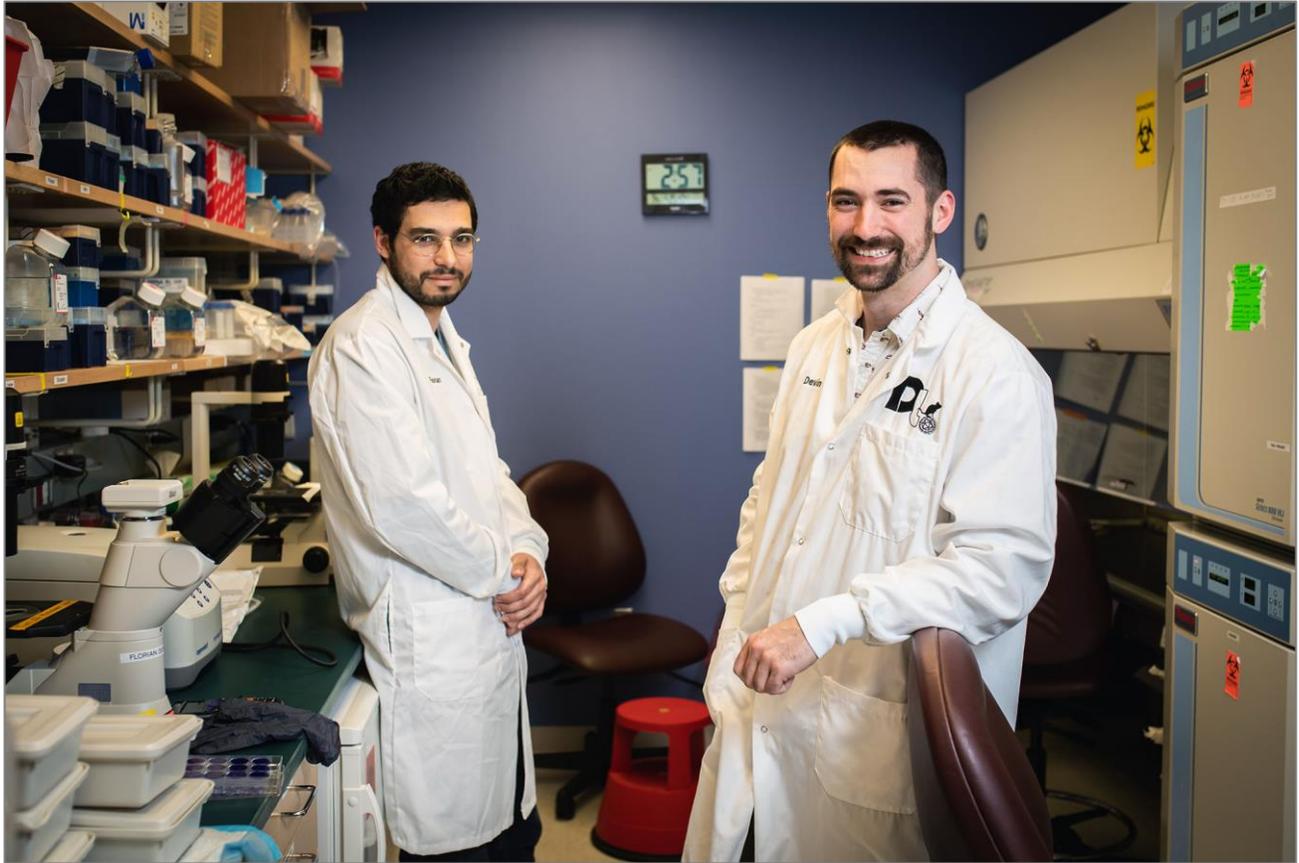
Featured Research

COVID-19

Why Do Some People Get Sicker than Others from COVID?

New study of virus in the lung suggests 11 “protection-defining genes” may help many avoid worst symptoms

Original article from The Brink by Andrew Thurston & Jackie Ricciardi. April 29, 2022



Florian Douam (left), a BU School of Medicine assistant professor of microbiology, and Devin Kenney, a PhD student in Douam’s lab, say their findings could propel the development of drugs that better prime immune systems for taking on the coronavirus.

COVID-19 vaccines have saved at least a million lives in the United States alone, but for many people, a lingering fear remains: if—or when—they get hit by the coronavirus, just how bad will it be? Will they breeze through with little more than a sore throat—or will it saddle them with long-term complications, perhaps even push them to the brink of death?

Since SARS-CoV-2 first began storming around the world in early 2020, COVID-19 has claimed six million lives and counting, according to the World Health Organization. And yet, the vast majority of people who’ve contracted COVID—some 99 percent of the more than 500 million confirmed cases—have survived their brush with the disease.

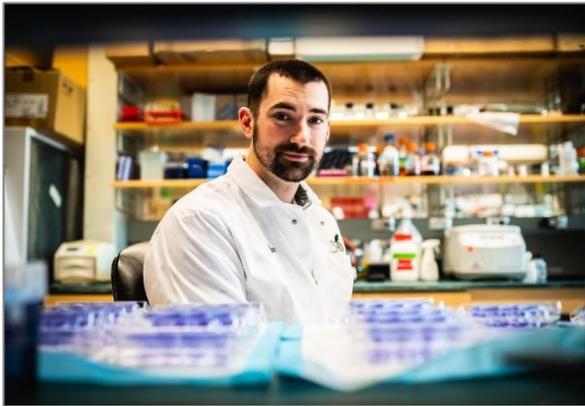
So, why is it that some people are so badly affected by COVID when many are barely scratched by it? Age and other health conditions increase the risk of getting really sick, but a new study suggests that those who escape the worst symptoms might also have the right balance of a type of immune cells called macrophages.

White blood cells found in every tissue, macrophages—part of a group of cells called myeloid cells, the guards of the immune system—are healers. They’re crucial in wound repair, streaming to an injury to help the body patch itself up. They also take on invaders, gobbling up and digesting anything that looks like it doesn’t belong in the body, from dead cells to harmful bacteria. That attack mode helps keep us healthy, but it also seems to be a factor in severe COVID-19 cases. Evidence has been growing that many COVID deaths are caused by a hyper-immune response: rampaging

macrophages attacking not just the virus, but also our bodies, causing excessive inflammation and damaging heart and lung tissue.

In a study published in *Cell Reports*, a team of researchers at Boston University’s National Emerging Infectious Diseases Laboratories (NEIDL) and Princeton University looked at why that was happening, examining COVID’s impact on those who get dangerously sick—and those who don’t. By studying lungs that seem to easily deflect SARS-CoV-2 or quickly bounce back from infection, they found a set of genes that determine whether immune cells mount a solid defense—or turn rogue and land someone on a ventilator. The findings could help efforts to develop new drugs that better prime immune systems for taking on the virus.

“If you can understand why most people are protected against COVID and how their body protects them, then you could potentially harness this knowledge to develop therapeutics and other advances,” says Florian Douam, a BU School of Medicine assistant professor of microbiology who co-led the study.



Douam (right) and Kenney (left) studied lungs that seem to easily deflect or recover from SARS-CoV-2 in order to better understand why most people survive COVID. Douam says the work they are “doing here is really upstream” and could lay the foundation for future immunotherapy strategies.

Why Are Some Lungs Protected against COVID?

After two years of sickness and swabbing, a lot of scientists know about how SARS-CoV-2 is transmitted and how our bodies react when we get it—but there’s also a lot they don’t understand. Take the lungs: We know COVID-19 can leave lungs full of liquid and inflamed, sometimes scarred by sepsis. But most of what’s known about COVID in the lungs is driven by samples taken from those who died from the disease—not those who lived through it.

“You can only access the lung when the patient dies,” says Douam, who’s based at NEIDL. “You cannot obviously get someone who had a mild disease and tell them, ‘Oh, give me your lung.’ In contrast to lung autopsy samples from diseased patients, the lungs from milder or asymptomatic patients are just much harder to access. When you have the diseased lung, you get a snapshot of the end-stage disease.”

To get around this challenge, Douam and the research team developed a new model—a mouse engrafted with human lung tissue and bolstered with a human immune system derived from stem cells—for monitoring the different stages of SARS-CoV-2 infection and COVID-19 disease. Douam says that mice with human lung tissue, but without the human immune system, don’t react well to infection—the lung tissues are damaged in a similar way to people with a severe case of the disease. But when they studied mice that also had a humanized immune system, it was different. “We were barely seeing any virus in the lungs,” he says. “The lung was protected. Then we asked the question, ‘Why is the lung protected?’ And this is where we found the macrophages.”

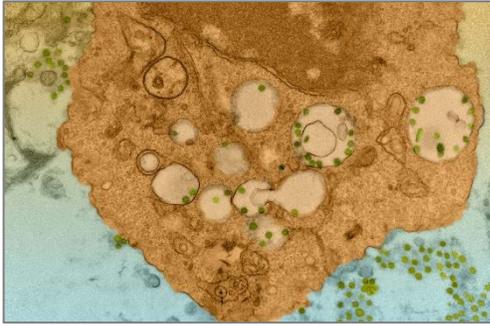
“Protection-Defining Genes”

According to Devin Kenney, a PhD student in Douam’s lab and lead author on the latest paper, one signature of lungs that were more severely impacted by COVID was a lack of macrophage diversity. They were dominated by a pro-inflammatory macrophage—the cells that usually respond to viruses and bacteria—called M1.

“It seems they drive this hyper-inflammatory response,” says Kenney (MED’27), “and it leads to a more severe disease state.”

By contrast, those immune systems that mixed in more of the cells that typically help in wound repair—M2 or regulatory macrophages—fared better.

“If you have a more diverse macrophage population that has both regulatory and inflammatory macrophages, you can more effectively regulate the signals driving antiviral responses, shutting them off when appropriate,” he says. “Then, the immune system can clear the virus really rapidly, protect the tissue.”



SARS-CoV-2 particles can be seen in a macrophage in the human lung tissue-engrafted model. Picture courtesy of Douam and Kenney, taken in collaboration with the Harvard Medical School Electron Microscopy Facility

The researchers tied this positive antiviral response to a set of 11 genes they called “protection-defining genes.” In cases of effective resistance, these genes were working harder, or what’s known as upregulated.

“We now know not only that macrophages can promote protection in the lung tissue,” says Douam. “We also know the key set of genes that these macrophages need to express to protect the lung.”

What they don’t know yet is why some people can put a diverse mix of macrophages to work while others can’t. That’s a target for future studies.

“What we’re doing here is really upstream,” says Douam. “If you can generate knowledge and better understand the molecular processes driving lung protection from COVID-19, then once you get this really good comprehensive picture of what’s happening, you can start designing potential immunotherapy strategies.”

And that’s the end goal of this work. Knowing that some genes are critical in the COVID fight gives potential fresh targets for drugs. With new coronavirus variants springing up and taking root at a rapid rate, says Douam, it’s important that scientists find alternatives to medications that target the virus itself.

“The virus, over time, can start escaping these types of drugs,” he says. “It’s not the virus itself that makes you critically ill, it’s an overreaction of the immune system.”

Finding drugs that help patients have a more balanced immune response could “complement the antiviral strategy.”

This work was mainly supported by a Boston University start-up fund, a Peter Paul Career Development Professorship, and the National Institutes of Health. Other researchers involved in leading the study include John H. Connor, a MED associate professor of microbiology, Nicholas Crossland, a MED assistant professor of pathology and laboratory medicine, and Andrew Emili, a College of Arts & Sciences professor of biology and biochemistry, as well as Alexander Ploss, an associate professor of molecular biology at Princeton University.

TWIV 891: “LLOV” in the time of Ebola; interview with Elke Mühlberger and Adam Hume

Elke, Adam, and Gabor join TWiV to discuss their work on Lloviu virus, a filovirus, including recovery of infectious virus from a DNA copy of the genome and from Schreiber’s bats in Hungary.

Original interview from This Week in Virology (TWiV) by Vincent Racaniello, Rich Condit, Kathy Spindler, and Brianne Barker. April 21, 2022

NEIDL faculty members **Elke Mühlberger** and **Adam Hume** joined the cast of This Week in Virology (TWiV) to discuss their work on Lloviu virus, a newly discovered member of the filovirus family which includes highly pathogenic viruses such as Ebola and Marburg virus. They discuss their research generating recombinant Lloviu virus as a tool to study this European filovirus which had not been previously cultured. They were also joined by Gábor Kemenesi, a collaborator from the University of Pécs (Hungary), who was able to use innovative field techniques to sequence and recently isolate Lloviu virus from Schreiber’s bats in Hungary. Together, they talk about the discovery of this virus as well as explore its ecology (informed by Gábor’s work) and describe the work of Drs. Mühlberger and Hume examining factors that may be involved in potential virus spillover to humans as well as what pathogenic threat the virus may pose to humans.



Sources:

Characterization of recombinant Llovio virus: <https://pubmed.ncbi.nlm.nih.gov/35120176/>

Isolation of Llovio virus from bats: <https://pubmed.ncbi.nlm.nih.gov/35361761/>

Inside the Insectary: How BU scientists study diseases from mosquitoes – without getting bitten

Original article from The Brink by Devin Hahn & Andrew Thurston. April 4, 2022

It's an unwanted ritual of summer: vainly splatting at mosquitoes as they nibble your exposed legs and arms, then enduring days of irritated itching from inflamed bites. For most Americans, it's just an annoyance, but for many around the world, those bites can be deadly. Globally, 400,000 people die from malaria every year, another 40,000 from dengue—both diseases are primarily transmitted by mosquitoes.

At Boston University's National Emerging Infectious Diseases Laboratories (NEIDL), researchers are looking at ways to halt mosquito-borne viruses—known as arboviruses—by studying the role of mosquito saliva proteins in facilitating disease transmission.

They're especially interested in *Aedes aegypti* mosquitoes—sometimes called the yellow fever mosquito—which is typically found in tropical and subtropical climates. This disease-carrying insect is now finding a home across much of the southern United States, and is expected to fly even farther north as the climate warms, potentially becoming a regular nuisance in New England and Canada by 2100.

At NEIDL, up to 2,000 mosquitoes are held in an Arthropod Containment Level 3 (ACL-3) insectary, with researchers having to make their way through a series of containment barriers to get to their lab to study the insects. Infected mosquitoes are locked in double cages, while mesh and plastic cover every lab entrance—researchers also keep bug zappers on hand as an added precaution. The biosafety measures allow the researchers—suited in Tyvek lab coats and N95 respirators—to study viruses commonly transmitted by *Aedes aegypti*, like chikungunya, dengue, West Nile, yellow fever, and Zika.

“Without specific medication to treat arbovirus infection—and the only effective arboviral vaccine is the yellow fever vaccine developed in 1937—the prevention of arboviral transmission is limited,” says Fabiana Feitosa-Suntheimer, a NEIDL senior research scientist and ACL-3 insectary manager. If we can better understand “the mechanism in which mosquito saliva interacts with arbovirus during transmission to the host,” she says, it may give antivirals and vaccines something new to target: take down the saliva, stop the spread. “I hope that finding new mosquito saliva proteins with prophylactic potential will contribute tremendously to the control of arbovirus diseases in humans.”



Watch the [video](#) inside the insectary to see how the researchers are learning more about mosquitoes and the diseases they carry.

This research is supported by the National Institutes of Health and NEIDL. Feitosa-Suntheimer is the lead researcher on the project; Florian Douam, a BU School of Medicine assistant professor of microbiology, is a coinvestigator.

Study explores molecular mechanisms underlying SARS-CoV-2 infection of macrophages and the induction of pro-inflammatory cytokines

Original article from Medical Science News, by Neha Mathur, April 1, 2022

In a recent study posted to the bioRxiv* pre-print server, researchers examined the role of sialic acid-binding immunoglobulin-type lectins-1 (CD169), a myeloid cell-specific receptor, in facilitating severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection of angiotensin-converting enzyme 2 (ACE2)-deficient macrophages and its effect on pro-inflammatory cytokine expression.

Background

The monocytes and macrophages, including tissue-resident alveolar macrophages, have not been directly associated with productive SARS-CoV-2 infection as they do not express ACE2; however, studies have reported the presence of SARS-CoV-2 ribonucleic acid (RNA) and antigen in these cells.

The aberrant activation of these cells in severe coronavirus disease 2019 (COVID-19) robustly induces interferon-stimulated gene (ISG) expression and high levels of pro-inflammatory cytokines, including interleukin 6 (IL-6), tumor necrosis factor α (TNF α), and IL-1 β , all potential drivers of acute respiratory distress syndrome (ARDS).

Studies on post-mortem tissues from deceased COVID-19 patients have shown tissue-resident alveolar macrophages and inflammatory myeloid cell populations enriched with SARS-CoV-2 RNA. However, studies have barely investigated whether SARS-CoV-2 can establish a productive infection in monocytes and macrophages and the molecular mechanisms contributing to the myeloid cell-intrinsic hyperinflammatory phenotype.

Some recent reports demonstrated that dendritic cell (DC)-mediated SARS-CoV-2 trans infection of ACE2+ epithelial cells was facilitated by CD169 as it binds to highly sialylated SARS-CoV-2 spike (S) protein.

About the study

In the present study, researchers showed that CD169 binds to the SARS-CoV-2 S and mediates SARS-CoV-2 entry into macrophages even in the absence of ACE2, leading to restricted cytosolic expression of viral genomic and sub-genomic (sg) RNA.

They used two different human macrophage models, phorbol 12-myristate 13-acetate (PMA)-differentiated human leukemia monocytic cell line (THP1) cells (THP1/PMA), and monocyte-derived macrophages (MDMs).

They cultured CD14+ monocytes in the presence of human AB-serum and macrophage colony-stimulating factor (M-CSF) for six days to obtain differentiated primary MDMs, with ACE2 expression under the detection limit. They infected these cells with a SARS-CoV-2 S-pseudotyped lentivirus. Further, they incubated THP1 cells stably expressing wildtype (wt) CD169, mutant CD169 (R116A), or both wt CD169 and ACE2 with recombinant SARS-CoV-2 S protein and used flow cytometry (FC) to observe the relative S binding.

Study findings

The FC analysis showed a significantly reduced S binding in THP1/CD169-R116A cells, indicating that CD169 recognizes the sialylated S protein. Co-expression of the CD169 and ACE2 in THP1 cells enhanced S-pseudotyped lentiviral infection by greater than 11-fold and three-fold compared to cells only expressing CD169 or ACE2, respectively, suggesting a cooperative binding of CD169 and ACE2 to SARS-CoV-2 S. Furthermore, these results reinforced that the CD169-S interaction alone is enough to promote SARS-CoV-2 entry and fusion in macrophages in an ACE2-independent manner.

The authors observed a marked attenuation in the S-pseudotyped lentiviral infection in CD169+ macrophages upon treatment with the N-terminal domain (NTD)-targeting antibodies, suggesting that the CD169-S interaction facilitates SARS-CoV-2 entry in a manner distinct from ACE2. Accordingly, the results have implications for antibody treatment for COVID-19 patients, as current therapies primarily focus on the receptor-binding domain (RBD)-targeting antibodies. However, the NTD-targeting neutralizing antibodies blocking ACE2-dependent and ACE2-independent entry might also potentially suppress myeloid/macrophage-intrinsic inflammatory signature.

In THP1/PMA macrophages, the authors observed that the CD169-mediated SARS-CoV-2 entry was as effective as that mediated by ACE2. Consequently, there were no significant differences in the guide RNA (gRNA) levels at early times (up to six hours post-infection (hpi)) between CD169+, ACE2+ or CD169+/ACE2+ THP1/PMA macrophages, suggesting the absence of any restrictions to the initial steps of SARS-CoV-2 replication, such as attachment and fusion, in CD169+ THP1/PMA macrophages.

SARS-CoV-2 RNA synthesis occurs within the endoplasmic reticulum (ER)-derived double-membrane vesicles (DMVs). The authors observed fewer double-stranded RNA (dsRNA) in the SARS-CoV-2 S-pseudotyped lentivirus-infected CD169+ macrophages, likely reflecting the selective impairment of the formation of DMVs and in turn SARS-CoV-2 RNA. However, upon restoring the ACE2 expression in the THP-1/PMA and primary MDMs, they started infectious SARS-CoV-2 production, suggesting that the macrophages were permissive to viral replication in an ACE2-dependent manner.

Interestingly, clinical treatment with remdesivir (RDV) blocked de novo SARS-CoV-2 RNA expression and significantly reduced the pro-inflammatory cytokine expression in non-productively infected CD169+ macrophages. This finding suggested that the newly synthesized SARS-CoV-2 RNAs viz., negative sense gRNA, dsRNA, and sgRNAs are key drivers of innate immune activations in the macrophages, not the S protein interaction with ACE2. This discovery presents a novel therapeutic strategy for combating hyper inflammation in COVID-19 patients.

Conclusions

Although CD169 facilitated SARS-CoV-2 entry in the macrophages and monocytes in an ACE-2 independent manner and enhanced the kinetics of SARS-CoV-2 replication and magnitude of infection even during the ACE2-mediated entry, its molecular mechanism remains unclear. Nevertheless, the study highlighted a post-entry role of ACE2 in the SARS-CoV-2 life cycle in the macrophages.

Future studies should further explore the ACE2-independent CD169-mediated mechanism of SARS-CoV-2 entry in the macrophages resulting in the hyperactivated immune response during SARS-CoV-2 infection for providing insights into the molecular-level post-entry restrictions on viral replication and the viral determinants that trigger the innate immune activation.

Important notice

bioRxiv publishes preliminary scientific reports that are not peer-reviewed and, therefore, should not be regarded as conclusive, guide clinical practice/health-related behavior, or treated as established information.

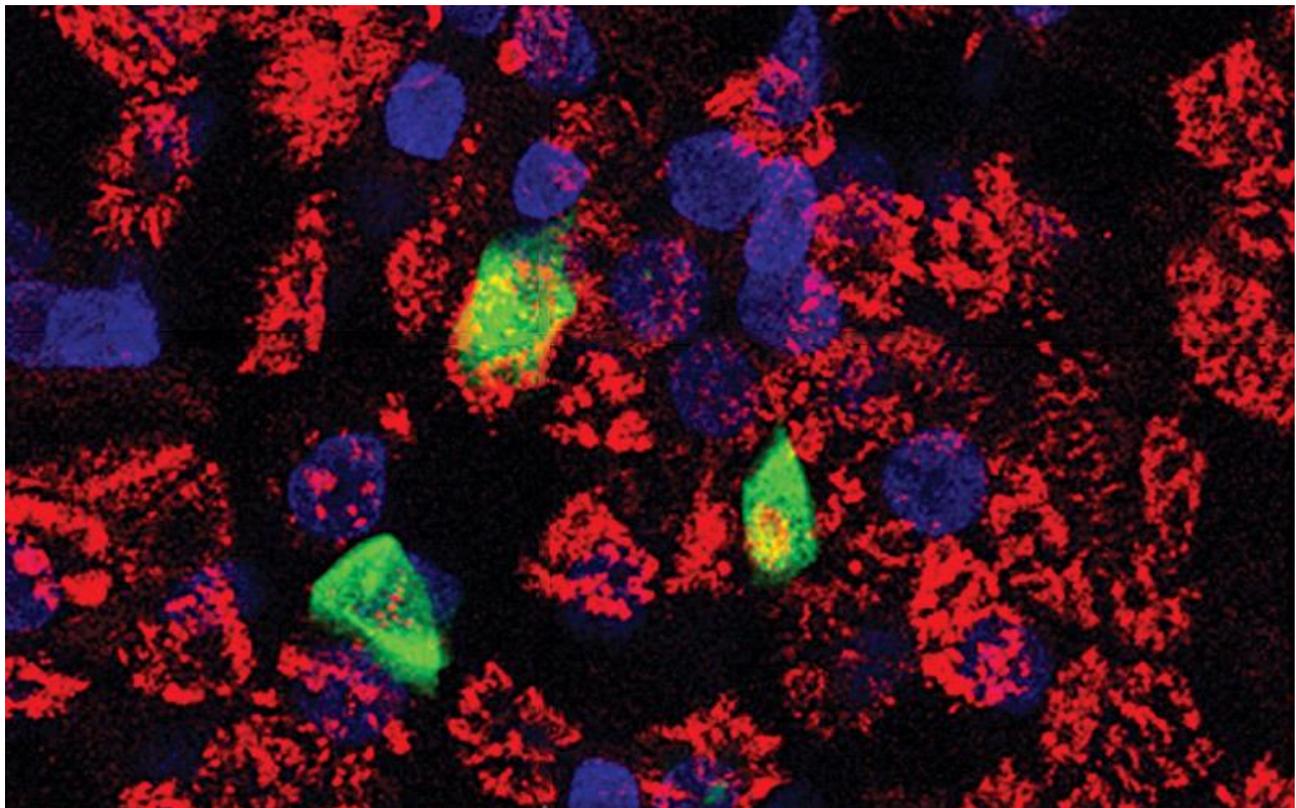
Journal reference:

Sallieu Jalloh, Judith Olejnik, Jacob Berrigan, Annuurun Nisa, Ellen L Suder, Hisashi Akiyama, Maohua Lei, Sanjay Tyagi, Yuri Bushkin, **Elke Mühlberger**, Suryaram Gummuluru. (2022). CD169-mediated restrictive SARS-CoV-2 infection of macrophages induces pro-inflammatory responses. *bioRxiv*. doi: <https://doi.org/10.1101/2022.03.29.486190>
<https://www.biorxiv.org/content/10.1101/2022.03.29.486190v2>

A respiratory model of COVID-19, made from patients' own cells

Original article posted on February 8, 2022 by Nancy Fliesler | Basic/Translational, Research
<https://answers.childrenshospital.org/model-airway-covid-19/>

Under a microscope, a number of individual cells are seen. Those infected by SARS-CoV-2 stand out by appearing much brighter.



Derived from patients' cells, a new airway model containing ciliated respiratory endothelial cells recapitulates SARS-CoV-2 infection and can be used to test drugs. Three ciliated cells in this model are infected with the virus. Image: Chantelle Simone Roach, Boston Children's, and Jake La Suer, Boston University

What happens in our respiratory tract once COVID-19 invades? A three-dimensional airway model, made from patient-derived stem cells, could provide answers about the initial stages of infection. The model not only replicates the infection process, but can be used to test potential antiviral drugs.

Ruby Wang, MD, attending physician in Boston Children's Division of Pulmonary Medicine, led the research in collaboration with the Center for Regenerative Medicine of Boston University (BU) and Boston Medical Center and BU's National Emerging Infectious Disease Laboratories (NEIDL).

"Human primary bronchial cells are the gold standard for studying lower respiratory infections," says Wang. "But the value of our model is that it doesn't require getting tissue from patients' airways, an invasive procedure. Our platform provides an inexhaustible supply of cells for modeling purposes and produces the relevant airway cell types."

From blood cells to a model airway

To build the model, Wang and BU colleagues Finn Hawkins, MB, BCh, and Darrell Kotton, MD, obtained blood cells from two individuals. They first genetically “reprogrammed” the cells back to a stem cell state, together with Thorsten Schlaeger, PhD, and George Daley, MD, PhD, from Boston Children’s Stem Cell Program. They then added factors to get the stem cells to form all the major types of epithelial cells that line the trachea and bronchi. These included multi-ciliated, secretory, and basal cells. The model airway carried the hallmark cell receptor ACE2 as well as TMPRSS2, a key enzyme that aids in viral entry.

Using live SARS-CoV-2 in BU’s Biosafety Level 3 facility, Wang’s group and NEIDL researchers [Adam Hume, PhD](#), and [Elke Muhlberger, PhD](#), successfully infected the model airway. They showed that multi-ciliated airway cells are the initial point of viral entry.

“That’s important, because ciliated cells help propel airway mucus and entrapped pathogens up away from the lungs,” says Wang. “If the ciliated cells are injured, the virus can more easily get down to the lower lung.”

Modeling SARS-CoV-2 infection, testing treatments

Once infected, the model airway mounted a robust antiviral response, producing type 1 and type 3 interferons. The team also saw a marked inflammatory response. They observed increased production of inflammatory signaling molecules and increased expression of genes stimulated by interferon (natural antivirals that rally the immune response), and genes involved in activating immune cells.

Because the model airway genetically matches the patient from whom it’s derived, it enables testing of how specific underlying conditions may affect susceptibility to COVID-19.

“We can also use CRISPR gene editing to see the effects of different genetic mutations,” says Wang. She, Stuart Rollins, MD, and other members of her lab are currently using stem cells from cystic fibrosis (CF) patients to study how a CF airway responds to SARS-CoV-2 and other pathogens.

Perhaps most importantly, scientists can use the model to test potential treatments. The team tested the antiviral remdesivir and found a decrease in viral replication. They found the same when they tested camostat mesylate, which inhibits TMPRSS2, confirming that the virus requires TMPRSS2 to infect airway cells.

The team’s findings appear in the journal *American Journal of Physiology – Lung Cellular and Molecular Physiology*. In the future, Wang plans to use the airway model to study Omicron, Delta, and other new variants and their response to treatments in both healthy patients and those with airway diseases such as CF.

RESEARCH THAT MATTERS – NEIDL: RIGHT PLACE, RIGHT TIME

Microbiologist [Robert Davey’s](#) ongoing novel coronavirus research was boosted with \$400,000 through Harvard from the Massachusetts Consortium on Pathogen Readiness, part of \$1.6 million in funding that BU researchers received.

Original article from BU Annual Report. January 31, 2022

When news broke that an unknown and dangerous virus had reached US shores and was rapidly spreading, researchers at BU didn’t blink. By mid-March 2020, they were already working with live samples of the novel coronavirus at the National Emerging Infectious Diseases Laboratories (NEIDL).

“NEIDL was built to be able to study emerging infectious diseases and respond to national emergencies,” says Director Ronald Corley, chair and professor of microbiology at the School of Medicine. “This pandemic has demonstrated the value of having a facility like NEIDL and all the expertise that we’ve brought into it.”

Built in 2008, NEIDL is a state-of-the-art research facility that supports the work of investigators who focus on infectious diseases that are—or have the potential to become—major public health concerns. It is one of only a handful of facilities in the United States that houses biosafety level 2 (BSL-2), BSL-3, and BSL-4 laboratories, which are designed to permit investigators to work safely with these emerging pathogens. NEIDL is owned and operated by Boston University but is also one component of a national network of secure facilities that studies emerging infectious diseases.

Since the COVID-19 outbreak, NEIDL scientists dropped nearly every other research project to focus on understanding and combating the novel coronavirus.

BU researchers quickly received \$1.6 million in new funding through Harvard from the Massachusetts Consortium on Pathogen Readiness to further advance coronavirus research, from modeling infections in lung organoids to developing better and faster testing to screening thousands of drug compounds in search of new treatments.

During the pandemic, global vaccine development focused heavily on activating immune cells—B cells—which are responsible for creating antibodies. But with the mutating variants, NEIDL researchers are now looking at other ways human cells activate the immune system in response to SARS-CoV-2 infection, specifically T cells, considered the body's killers as they are sent out to destroy infected cells.

Along with colleagues from the Broad Institute of MIT and Harvard, BU researchers have been studying the types of "red flags" the human body uses to enlist the help of T cells. Their findings, published in *Cell* in June 2021, could open the door to even more effective and powerful vaccines against the coronavirus and its rapidly emerging variants.

"We swung into full action right away because my laboratory had already generated human cell lines that could be readily infected with SARS-CoV-2,"

says **Mohsan Saeed**, a NEIDL virologist and a co-corresponding author of the paper. "This virus wants to go undetected by the immune system for as long as possible. Once it's noticed by the immune system, it's going to be eliminated, and it doesn't want that."



Microbiologist ROBERT DAVEY's ongoing novel coronavirus research was boosted with \$400,000 through Harvard from the Massachusetts Consortium on Pathogen Readiness, part of \$1.6 million in funding that BU researchers received.

Researchers identify a new protein that enables SARS-CoV-2 access into cells

Findings may lead to the development of new antiviral therapeutics against COVID-19

Original Article from Science Daily published on January 27, 2022

Summary:

Researchers have identified extracellular vimentin as an attachment factor that facilitates SARS-CoV-2 entry into human cells. Vimentin is a structural protein that is widely expressed in the cells of mesenchymal origin such as endothelial cells and a potential novel target against SARS-CoV-2, which could block the infection of the SARS-CoV-2.

The entry of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) into human cells is an essential step for virus transmission and development of COVID 19. Although the lung epithelial cells are its initial target, SARS-CoV-2 also can infect endothelial cells. Endothelial cells are the major constituents of the vascular system and cardiovascular complication is a hallmark of severe COVID-19. Angiotensin-converting enzyme 2 (ACE2) is the entry receptor for SARS-CoV-2. However, the possible involvement of other cellular components in the viral entry is not fully understood.

A team of researchers from the Boston University School of Medicine (BUSM) has identified extracellular vimentin as an attachment factor that facilitates SARS-CoV-2 entry into human cells. Vimentin is a structural protein that is widely expressed in the cells of mesenchymal origin such as endothelial cells and a potential novel target against SARS-CoV-2, which could block the infection of the SARS-CoV-2.

"Severe endothelial injury, vascular thrombosis, and obstruction of alveolar capillaries (tiny air sacs scattered throughout the lungs) are common features of severe COVID-19. Identification of vimentin as a host attachment factor for SARS-CoV-2 can provide new insight into the mechanism of SARS-CoV-2 infection of the vascular system and can lead to the development of novel treatment strategies," said corresponding author Nader Rahimi, PhD, associate professor of pathology & laboratory medicine at BUSM.

The researchers used liquid chromatography-tandem mass spectrometry (LC-MS/MS) and identified vimentin as a protein that binds to the SARS-CoV-2 spike (S) protein and facilitates SARS-CoV-2 infection. They also found that depletion of vimentin significantly reduces SARS-CoV-2 infection of human endothelial cells. In contrast, over-expression of vimentin with ACE2 significantly increased the infection rate. "More importantly, we saw that the CR3022 antibody inhibited the binding of vimentin with CoV-2-S-protein, and neutralized SARS-CoV-2 entry into human cells," explained Rahimi.

Collaborators from BUSM included [Elke Mühlberger, PhD](#), Vipul Chitalia, MD, PhD and Catherine E. Costello, PhD.

These findings appear online in the Proceedings of the National Academy of Sciences.

This research was supported in part through a grant from BUSM Genome Science Institute and ARC-COVID-19 (NR and CEC); NIH grants R24 GM134210 and S10 OD021728 (to CEC), funding from BUSM COVID-19 ARC (to CEC and NR) and BUSM Clinical & Translational Science Institute awards from NIH NCATS grant UL1TR001430 (to EM and CEC). Fast Grants (EM), the Evergrande COVID-19 Response Fund Award from the Massachusetts Consortium on Pathogen Readiness (EM and AGS). NIH R01 AI146779 (AGS) and NIGMS T32 GM007753 (AGS).

Journal Reference:

Extracellular vimentin is an attachment factor that facilitates SARS-CoV-2 entry into human endothelial cells

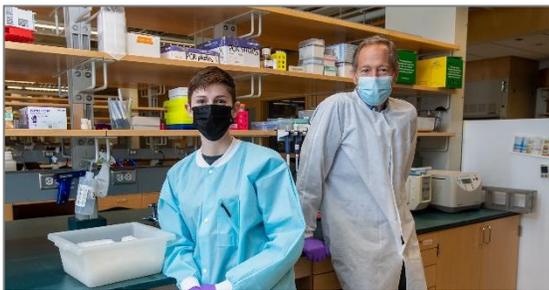
Razie Amraei, Chaoshuang Xia, Judith Olejnik, Mitchell R. White, Marc A. Napoleon, Saran Lotfollahzadeh, Blake M. Hauser, Aaron G. Schmidt, Vipul Chitalia, Elke Mühlberger, Catherine E. Costello, Nader Rahimi. . *Proceedings of the National Academy of Sciences*, 2022; 119 (6): e2113874119 DOI: 10.1073/pnas.2113874119

COVID ON CAMPUS

BU Scientists Are Prepared to Detect Omicron—and Other Variants

BU's National Emerging Infectious Diseases Laboratories (NEIDL) has been monitoring COVID-19 variants from BU and Boston Medical Center tests since February

Original article from The Brink By Kat J. McAlpine. December 8, 2021



NEIDL scientists Jackie Turcinovic (left) and John Connor analyze the genomic sequences of all positive COVID-19 tests that come from BU faculty, staff, and students. Photo by Cydney Scott

When will the coronavirus variant known as Omicron arrive at BU? Although it's hard to predict, it seems like a foregone conclusion that it's only a matter of time given how quickly Omicron has spread around the world—already infecting people in more than 30 countries across 6 continents, and at least 16 US states. On December 4, the first known case of Omicron in Massachusetts was detected in a woman in her 20s who lives in Middlesex County.

One thing's for certain—if and when Omicron infects a member of BU's community, its presence will be quickly detected.

The University's testing program—which screens a pool of all students, faculty, and staff that come to BU's campuses—hinges upon its Clinical Testing Lab, which was rapidly outfitted with machinery and custom software to screen nasal swabs for

coronavirus over the summer of 2020. Fully operational for the last year and a half, it has screened over two million swabs and is capable of processing up to 6,000 coronavirus tests per day.

And for every positive test result that diagnoses a new case of COVID-19, that swab is then couriered over to BU's National Emerging Infectious Diseases Laboratories (NEIDL), where scientists perform genomic sequencing to determine the strain of the virus—mostly the Delta variant, since July 2021—and to detect whether transmission occurred from sources outside of the BU community or from within. The NEIDL also performs variant sequencing for Boston Medical Center (BMC), BU's primary teaching hospital and the safety-net hospital for the city of Boston.

The NEIDL's variant sequencing efforts took off in February 2021, after NEIDL virologist [John Connor](#) teamed up with BMC infectious disease clinician Karen Jacobson and other clinical staff to propose a plan for sequencing anonymized positive test results from BU's Clinical Testing Lab, as well as patient samples from BMC's COVID Biorepository and samples from BMC clinicians and staff.

Jacobson says the NEIDL’s variant sequencing effort is a real example of science that would not be possible without a big team of collaborative scientists, including infectious disease specialist Tara Bouton of BMC and BU School of Medicine (MED), Cassandra Pierre of BMC and MED, and Harvard University epidemiologist William Hanage. “This is not something that just a few investigators could pull off,” she says.

When Delta arrived on the scene at the end of June, “all of sudden, within the span of two weeks, there was a rapid shift,” says Jackie Turcinovic, a third year PhD student and one of the primary NEIDL scientists who performs variant sequencing. Positive results started coming in for sequencing much more rapidly, and case reports indicated Delta was causing more symptomatic than asymptomatic cases. The amount of viral material in each positive sample was also typically much higher than the lab had seen with other coronavirus variants.

“There was this shift from trying to squeeze out [enough viral material from samples to allow for sequencing] to suddenly seeing samples with tons of virus—it was very odd, almost a little creepy,” Turcinovic says. “The change in presentation and urgency seemed really different.”

According to Judy Platt, BU’s chief health officer and executive director of Student Health Services, viral materials from positive test results are analyzed by NEIDL scientists to reveal the unique genetic sequence of the virus, and to match those sequences up with databases cataloging all currently known COVID variants from around the world. There are several steps involved in being able to sequence the viral genome, a process which takes several days.

Variant sequencing is not considered a clinical diagnostic for individuals. Instead, it’s used to study how COVID-19 is spreading and evolving at the population level. For regulatory reasons, BU is not permitted to tell individuals if they have a variant form of COVID-19—NEIDL scientists are not even aware of which person the sequenced samples came from; they just know the virus sample was collected from someone at BU.

“The most direct thing we’ve been able to do is see what the pattern of virus spread is,” says Connor, a BU School of Medicine associate professor of microbiology. “From the genomes we can get a pretty good idea of whether positives at BU are coming from person-to-person spread versus transmission from cases outside of the BU system. If we see two virus genomes that are identical, we know two people were in the same room together.”

That has helped the team understand where most cases at BU are coming from and corroborate the findings of BU Healthway’s contact tracing team.

The contact tracing team is on call 12 hours a day, 7 days a week, addressing positive COVID-19 cases as soon as they come up. On a slow week, the contact tracing team might investigate between 50 and 60 cases, a number that can grow to more than 100 during periods after long weekends or holiday breaks, when more positive cases tend to surface. A contact tracer’s day involves just what you would expect: a lot of phone calls and a lot of hard conversations.



Third year PhD student Jackie Turcinovic in the Connor Lab. Photo by Cydney Scott

If we see two virus genomes that are identical, we know two people were in the same room together.

John Connor

To date, BU’s contact tracing team has discovered that the majority of spread within BU’s community actually occurred off campus, from external sources, rather than from in-class or in-office interactions. That’s largely due to the power of vaccines—more than 95 percent of BU faculty, students, and staff are fully vaccinated against COVID—and BU’s community health protocols, which require people, regardless of vaccination status, to wear masks in common spaces shared with others.

It’s those unmasked interactions—within households, at social gatherings, during travel, or in other settings—that are largely contributing to the vast majority of cases at BU.

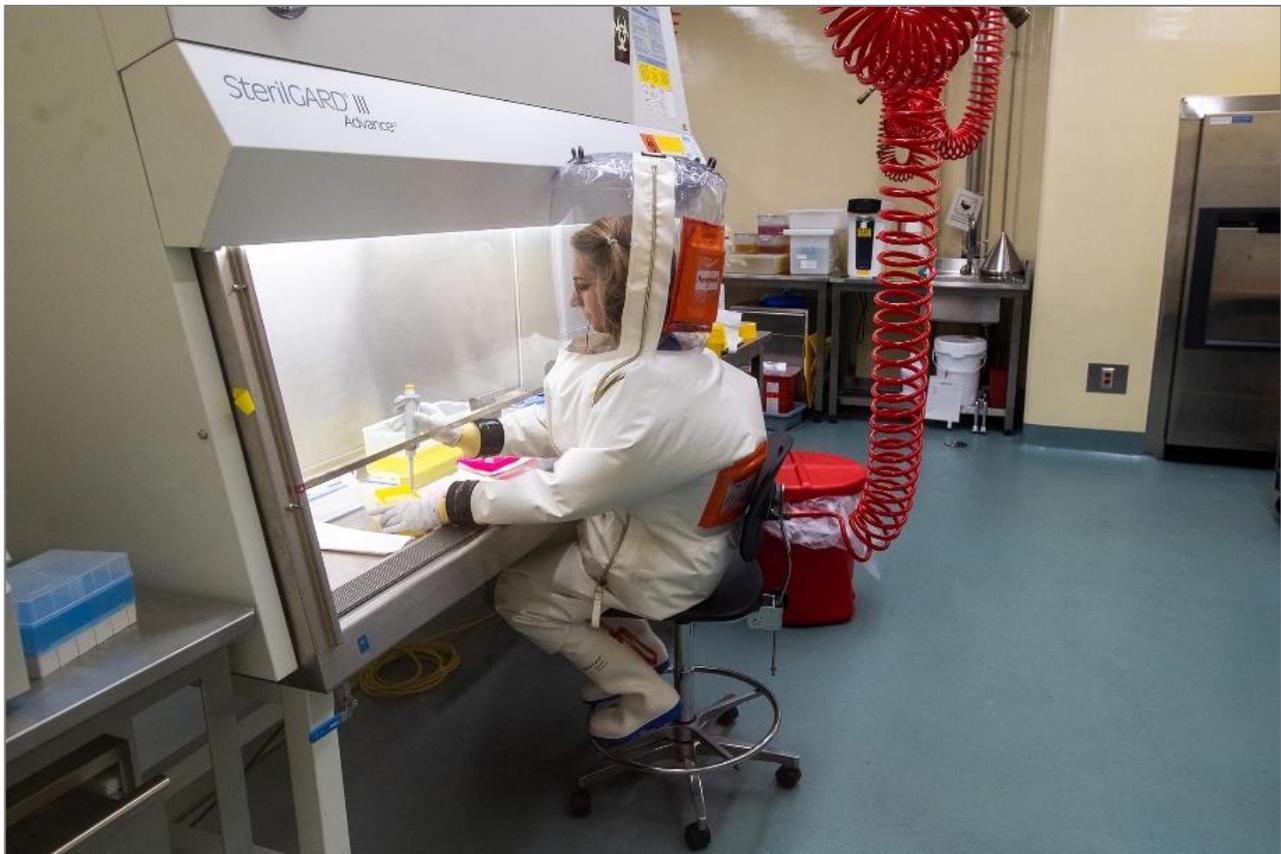
“Most true breakthrough cases are linked to clubs, bars, and socializing in crowded, indoor spaces,” says Hannah Emily Landsberg (Sargent’12, SPH’13), associate director of Student Health Services. “It does put in perspective your level of risk—if you go to a 10-person gathering, that’s less risky than going to several bars or big parties.”

“Unmasked, prolonged social events can lead to COVID—whether you’re vaccinated or not,” Platt says.

With Omicron spreading around the world, scientists have been racing to understand if the new variant, which has the most mutations ever seen on a coronavirus variant, will be able to evade immunity from vaccination. Preliminary indications are showing that Omicron may cause milder infections than earlier variants, although scientists caution that it’s too soon to be sure.

The emergence of the Delta and now Omicron variants likely foreshadows that the world will continue living amongst the SARS-CoV-2 virus for a long time.

“In Massachusetts, we’ve been very fortunate that we’ve had some of the highest rates of vaccine acceptance,” says Jacobson, who in addition to treating patients at BMC is also a BU School of Medicine associate professor of medicine in the section of infectious diseases. “But the virus has mutated in ways that [make it] hard to put the genie back in the



Pulmonary fellow Ellen Suder (MED '26, '26) working in the high containment lab, BSL-4, at BU's National Emerging Infectious Diseases Laboratories (NEIDL). Photo by Cydney Scott

bottle—the virus is figuring out ways to [mutate] over time. A lot of arrows point toward SARS-CoV-2 becoming an endemic virus [like influenza], one that is much milder and comes around seasonally.”

SCHOOL OF MEDICINE

MED’s Biology of the Lung Funded through Its 50th—Yes, 50th—Year *BU’s longest running federally funded training program began in 1975*

Excerpt from original article BU Today By Joel Brown. September 23, 2021

When the School of Medicine training program called Biology of the Lung: A Multidisciplinary Program began, on July 1, 1975, Gerald Ford was president, all four Beatles were alive, and gas cost about 50 cents a gallon.

“I was in elementary school when this started, so I wasn’t spending a lot of time thinking about lung biology or pulmonary disease or anything else,” **Joseph Mizgerd**, now program co-principal investigator, says with a smile.

The National Institutes of Health (NIH) has funded Biology of the Lung from the beginning and has just renewed it for five more years, taking it through years 46 to 50 of training predoctoral and postdoctoral scientists, both PhDs and MDs, in lung biology and pulmonary disease. The \$4.1 million renewal will fund stipends and other expenses for a dozen trainees a year until 2026.

While Biology of the Lung appears to be the longest running grant-funded training program at the University, the scientists who run it would much rather talk about the innovative idea at its core. “There are certain well-worn, traditional paths in training a doctor or a researcher, and this program is not one of those. It’s very different,” says Darrell Kotton, who joined Mizgerd as a co–principal investigator on the grant this summer.

Mizgerd, the Jerome S. Brody, MD, Professor of Pulmonary Medicine, is a MED professor of medicine, microbiology, and biochemistry and BU Pulmonary Center director. Kotton, the David C. Seldin Professor of Medicine, is a MED professor of medicine and pathology and founding director of the Center for Regenerative Medicine (CReM).

Dr. Mizgerd is a NEIDL faculty member, and both Drs. Mizgerd and Kotton have productive collaborations in the NEIDL.

Training doctors and researchers

Some fellows have found themselves on the front lines in the pandemic.

“My dissertation research actually focuses on filoviruses—that’s Ebola and Marburg,” says Ellen Suder (MED’26’26), a PhD candidate in microbiology and a program trainee who has been doing a lot of COVID-related work at BU’s National Emerging Infectious Diseases Laboratories (NEIDL) high-containment labs. “Over the past year and a half, I’ve picked up a lot of research on SARS-CoV-2. It’s been a really amazing experience to be involved with all that.”



Ellen Suder preparing to work in the NEIDL high containment lab. Photo by Cydney Scott

The NEIDL lab where she works offered virology help to scientists around the Boston area last year as soon as the seriousness and duration of the outbreak became clear. “We ended up accruing 20-plus collaborators at a bunch of labs in the Boston area—a lot of BU labs, a lot of other universities, some companies,” Suder says. “It was very much an all-hands-on-deck situation. I just started volunteering to take on samples that needed to be generated and things like that.

“The biggest project that our lab did last year was this collaboration with Darryl Kotton’s lab at the CReM. We came up with all of these potential drug targets and curated a list of drugs that were either approved for something else entirely or were in clinical trials, and tested if they would be viable in preventing this infection. What we found out that was really cool was in the cell-type model we normally use they weren’t terribly effective at preventing infection, but in the actual stem cell–derived lung cells, they worked a lot better.

“Now I’m really interested to see what I can do with these lung cell models,” she says, “studying the viruses we normally study.”

Research Publications

The NEIDL faculty continue to publish innovative studies on a diversity of human pathogens in high impact journals, illustrative of their diverse experience, expertise, and research interests. Many of the papers reflect the important collaborative nature of modern biomedical science, demonstrating widespread collaboration among NEIDL faculty and other faculty at Boston University and other institutions in the US as well as internationally. Collaborations with industry are also reflected in the publications.

Publications marked with a **red asterisk *** are coauthored by 2 or more **NEIDL core faculty (blue font)** or **Associate Faculty (black font)**.

Global COVID-19 vaccine inequity: The scope, the impact, and the challenges.

Asundi A, O'Leary C, **Bhadelia N**. *Cell Host Microbe*. 2021 Jul 14;29(7):1036-1039. doi: 10.1016/j.chom.2021.067. PMID: PMC8279498

Interruption of tuberculosis detection and care during the Ebola virus disease epidemic (2014-2015) in Liberia: time-series analyses for 2013-2017.

Gray KL, Walker NF, Martineau F, **Bhadelia N**, Harmon-Gray WM, Skrip LA, DeMarco J, Konwloh P, Dunbar N. *Int J Infect Dis*. 2021 Nov;112:13-20. doi: 10.1016/j.ijid.2021.08.041. Epub 2021 Aug 22. PMID: 34433096

Optimizing Highly Infectious Disease Isolation Unit Management: Experiences From the Infectious Diseases Isolation and Research Unit, Fort Portal, Uganda.

Alum S, Asimwe M, Kanyomozi G, Nalikka J, Okwaro P, Migisha I, Muhindo B, Wailagala A, Okello S, Blair P, Waitt P, **Bhadelia N**, Ayebare R, Kwicien A, Saunders D, Lamorde M, Kibuuka H, Clark D. *Disaster Med Public Health Prep*. 2021 Nov 25:1-5. doi: 10.1017/dmp.2021.339. Online ahead of print. PMID: PMC8886060

Institutional policies and readiness in management of critical illness among patients with viral hemorrhagic fever.

DiLorenzo MA, Baker CA, Herstein JJ, Evans L, Lowe JJ, Gibbs SG, **Bhadelia N**. *Infect Control Hosp Epidemiol*. 2021 Nov;42(11):1307-1312. doi: 10.1017/ice.2020.1416. Epub 2021 Feb 15. PMID: 33583468

Pandemic Response Requires Research Samples: A U.S. Safety-Net Hospital's Experience and Call for National Action.

Ragan EJ, McCallum C, Marathe J, Cole M, Hofman M, Henderson AJ, Flack T, Miller NS, Burks EJ, Zhao GQ, Denis R, Lin NH, Jacobson KR, Andry CD, Pelton SI, Duffy ER, **Bhadelia N**. *Ann Intern Med*. 2021 Dec;174(12):1727-1732. doi: 10.7326/M21-2857. Epub 2021 Nov 2. PMID: 34724402

Coronavirus Disease-2019 and Heart Failure: A Scientific Statement From the Heart Failure Society of America.

Bhatt AS, Adler ED, Albert NM, Anyanwu A, **Bhadelia N**, Cooper LT, Correa A, Defilippis EM, Joyce E, Sauer AJ, Solomon SD, Vardeny O, Yancy C, Lala A. *J Card Fail*. 2022 Jan;28(1):93-112. doi: 10.1016/j.cardfail.2021.08.013. Epub 2021 Sep 1. PMID: PMC8408888

The MAVS Immune Recognition Pathway in Viral Infection and Sepsis.

Sharma A, Kontodimas K, **Bosmann M**. *Antioxid Redox Signal*. 2021 Dec;35(16):1376-1392. doi: 10.1089/ars.2021.0167. Epub 2021 Sep 28. PMID: PMC8817698

GLP-1 Analog Liraglutide Improves Vascular Function in Polymicrobial Sepsis by Reduction of Oxidative Stress and Inflammation.

Helmstädter J, Keppeler K, Aust F, Küster L, Frenis K, Filippou K, Vujacic-Mirski K, Tsohataridis S, Kalinovic S, Kröller-Schön S, Oelze M, **Bosmann M**, Münzel T, Daiber A, Steven S. *Antioxidants (Basel)*. 2021 Jul 23;10(8):1175. doi: 10.3390/antiox10081175. PMID: PMC8388926

The MAVS immune recognition pathway in viral infection and sepsis.

Sharma A, Kontodimas K, **Bosmann M**. *Antioxid Redox Signal*. 2021 Dec 5. doi: 10.1089/ars.2021.0167. PMID: PMC8817698 (available on 2022-12-01)

*Neutrophil Extracellular Traps as an Exacerbating Factor in Bacterial Pneumonia. **

Sanders NL, Martin IMC, Sharma A, Jones MR, Quinton LJ, **Bosmann M**, **Mizgerd JP**. *Infect Immun*. 2022 Mar 17;90(3):e0049121. doi: 10.1128/IAI491-21. Epub 2022 Feb 7. PMID: PMC8929384

Advocacy of targeting protease-activated receptors in severe coronavirus disease 2019.

Subramaniam S, Ruf W, **Bosmann M**. *Br J Pharmacol*. 2022 May;179(10):2086-2099. doi: 10.1111/bph.15587. Epub 2021 Jul 7. PMID: PMC8794588 (available on 2023-05-01)

A Single-Cell Lung Atlas of Complement Genes Identifies the Mesothelium and Epithelium as Prominent Sources of Extrahepatic Complement Proteins.

N, Jayaraman A, Reinhardt C, Campbell JD, **Bosmann M**. *Mucosal Immunology*, 2022 Jun 7. PMID: PMC9173662

*Humanized mice reveal a macrophage-enriched gene signature defining human lung tissue protection during SARS-CoV-2 infection. **

Kenney DJ, O'Connell AK, Turcinovic J, Montanaro P, Hekman RM, Tamura T, Berneshawi AR, Cafiero TR, Al Abdullatif S, Blum B, Goldstein SI, Heller BL, Gertje HP, Bullitt E, Trachtenberg AJ, Chavez E, Nono ET, Morrison C, Tseng AE, Sheikh A, Kurnick S, Grosz K, **Bosmann M**, Ericsson M, Huber BR, **Saeed M**, Balazs AB, Francis KP, Klose A, Paragas N, Campbell JD, **Connor JH**, Emili A, **Crossland NA**, Ploss A, **Douam F**. Cell Rep. 2022 Apr 19;39(3):110714. doi: 10.1016/j.celrep.2022.110714. Epub 2022 Apr 4. PMID:PMC8977517

Fatal neuroinvasion and SARS-CoV-2 tropism in K18-hACE2 mice is partially independent on hACE2 expression. *
Carossino M, Montanaro P, O'Connell A, Kenney D, Gertje H, Grosz K, Ericsson M, Huber BR, Subramaniam S, Kirkland TA, Walker JR, Francis KP, Klose AD, Paragas N, Kurnick S, **Bosmann M**, **Saeed M**, Balasuriya U, **Douam F**, **Crossland NA**. bioRxiv. 2021 Sep 23:2021.01.13.425144. doi: 10.1101/2021.01.13.425144. Preprint. PMID: PMC7814818 **Updated**.

Forebrain Neural Precursor Cells Are Differentially Vulnerable to Zika Virus Infection.
Shelton SM, Soucy AR, Kurzion R, Zeldich E, **Connor JH**, Haydar TF. eNeuro. 2021 Sep 9;8(5):ENEURO.0108-21.2021. doi: 10.1523/ENEURO.0108-21.2021. Print 2021 Sep-Oct. PMID: PMC8431824

SARS-CoV-2 in infant urine and fecal samples after in utero COVID-19 exposure.
Boateng JO, Wachman EM, Turcinovic J, Devera J, Jain M, Jean-Sicard S, Woodard E, Cruikshank A, Sinha B, Bartolome R, Barnett ED, Parker MG, Yarrington C, **Connor JH**, Taglauer E, Sabharwal V. Pediatr Res. 2021 Oct 30:1-5. doi: 10.1038/s41390-021-01822-x. Online ahead of print. PMID: PMC8556813

SARS-CoV-2 reinfection associates with unstable housing and occurs in the presence of antibodies.
Bean DJ, Monroe J, Turcinovic J, Moreau Y, **Connor JH**, Sagar M. Clin Infect Dis. 2021 Nov 10:ciab940. doi: 10.1093/cid/ciab940. Online ahead of print. PMID: PMC8689949

Acute and Chronic Cardiovascular Manifestations of COVID-19: Role for Endotheliopathy.
Cooke JP, **Connor JH**, Jain A. Methodist Debakey Cardiovasc J. 2021 Dec 15;17(5):53-62. doi: 10.14797/mdcvj.1044. eCollection 2021. PMID: PMC8680072

Efficacy of Pfizer-BioNTech in SARS-CoV-2 Delta cluster. *
Landsberg HE, Turcinovic J, Sullivan M, **Connor JH**, **Hamer DH**, Platt JT. Int J Infect Dis. 2022 Jan;114:62-64. doi: 10.1016/j.ijid.2021.10.053. Epub 2021 Oct 30. PMID: PMC8556072

Science, not speculation, is essential to determine how SARS-CoV-2 reached humans. *
Calisher CH, Carroll D, Colwell R, **Corley RB**, Daszak P, Drosten C, Enjuanes L, Farrar J, Field H, Golding J, Gorbalenya AE, Haagmans B, Hughes JM, **Keusch GT**, Lam SK, Lubroth J, Mackenzie JS, Madoff L, Mazet JK, Perlman SM, Poon L, Saif L, Subbarao K, Turner M. Lancet. 2021 Jul 17;398(10296):209-211. doi: 10.1016/S0140-6736(21)01419-7. Epub 2021 Jul 5. PMID: PMC8257054

Dengue Virus-2 Infection Affects Fecundity and Elicits Specific Transcriptional Changes in the Ovaries of Aedes aegypti Mosquitoes. *
Feitosa-Suntheimer F, Zhu Z, Mameli E, Dayama G, Gold AS, Broos-Caldwell A, Troupin A, Rippee-Brooks M, **Corley RB**, **Lau NC**, **Colpitts TM**, Londoño-Rentería B. Front Microbiol. 2022 Jun 23;13:886787. doi: 10.3389/fmicb.2022.886787. eCollection 2022. PMID: PMC9260120

SARS-CoV-2 Disrupts Proximal Elements in the JAK-STAT Pathway. *
Chen DY, Khan N, Close BJ, Goel RK, Blum B, Tavares AH, Kenney D, Conway HL, Ewoldt JK, Chitalia VC, **Crossland NA**, Chen CS, Kotton DN, Baker SC, Fuchs SY, **Connor JH**, **Douam F**, Emili A, **Saeed M**. J Virol. 2021 Sep 9;95(19):e0086221. doi: 10.1128/JVI862-21. Epub 2021 Sep 9. PMID: PMC8428404

Cellular tropism of SARS-CoV-2 in the respiratory tract of Syrian hamsters and B6.Cg-Tg(K18-ACE2)2PrImn/J transgenic mice. *
Yen HL, Valkenburg S, Sia SF, Choy KT, Peiris JSM, Wong KHM, **Crossland NA**, **Douam F**, Nicholls JM. Vet Pathol. 2022 Jul;59(4):639-647. doi: 10.1177/03009858211043084. Epub 2021 Sep 1. PMID: PMC8721337

Association of vaping with decreased vascular endothelial growth factor expression and decreased microvessel density in cutaneous wound healing tissue in rats.
Jaleel Z, Blasberg E, Troiano C, Montanaro P, Mazzilli S, Gertje HP, **Crossland NA**, Platt M, Spiegel J. Wound Repair Regen. 2021 Nov;29(6):1024-1034. doi: 10.1111/wrr.12945. Epub 2021 Jun 15. PMID: 34129265

An outbreak of systemic chlamydiosis in farmed American alligators (Alligator mississippiensis).
Carossino M, Nevarez JG, Sakaguchi K, Paulsen DB, Langohr IM, Strother K, Ferracone J, Roy A, **Crossland NA**, Del Piero F. Vet Pathol. 2022 May 3:3009858221095269. doi: 10.1177/03009858221095269. Online ahead of print. PMID: 35502797

A Naturally Occurring Polymorphism in the Base of Sudan Virus Glycoprotein Decreases Glycoprotein Stability in a Species-Dependent Manner
Lennemann NJ, Dillard J, Ruggio N, Cooney AL, Schaack GA, **Davey RA**, Maury W. J Virol. 2021 Aug 25;95(18):e0107321. doi: 10.1128/JVI.01073-21. Epub 2021 Aug 25. PMID: PMC8387054.

Non-canonical proline-tyrosine interactions with multiple host proteins regulate Ebola virus infection.

Batra J, Mori H, Small GI, Anantpadma M, Shtanko O, Mishra N, Zhang M, Liu D, Williams CG, Biedenkopf N, Becker S, Gross ML, Leung DW, **Davey RA**, Amarasinghe GK, Krogan NJ, Basler CF. *EMBO J.* 2021 Sep 15;40(18):e105658. doi: 10.15252/embj.2020105658. Epub 2021 Aug 2. PMID: PMC8441301 (available on 2022-09-15)

Synthesis and antiviral activity of fatty acyl conjugates of remdesivir against severe acute respiratory syndrome coronavirus 2 and Ebola virus.

El-Sayed NS, Jureka AS, Edwards MR, Lohan S, Williams CG, Keiser PT, **Davey RA**, Totonchy J, Tiwari RK, Basler CF, Parang K. *Eur J Med Chem.* 2021 Dec 15;226:113862. doi: 10.1016/j.ejmech.2021.113862. Epub 2021 Sep 21. PMID: PMC8454092

Adipocytes are susceptible to Ebola Virus infection.

Gourronc FA, Rebagliati MR, Kramer-Riesberg B, Fleck AM, Patten JJ, Geohegan-Barek K, Messingham KN, **Davey RA**, Maury W, Klingelutz AJ. *Virology.* 2022 Aug;573:12-22. doi: 10.1016/j.virol.2022.057. Epub 2022 May 27. PMID: 35690007

Monoclonal antibodies binding data for SARS-CoV-2 proteins.

Mishra N, Teyra J, Boytz R, Miersch S, Merritt TN, Cardarelli L, Gorelik M, Mihalic F, Jemth P, **Davey RA**, Sidhu SS, Leung DW, Amarasinghe GK. *Data Brief.* 2022 Aug;43:108415. doi: 10.1016/j.dib.2022.108415. Epub 2022 Jun 24. PMID: PMC9232259

Evaluation of Phenol-Substituted Diphyllin Derivatives as Selective Antagonists for Ebola Virus Entry.

Plescia CB, Lindstrom AR, Quintero MV, Keiser P, Anantpadma M, **Davey R**, Stahelin RV, Davisson VJ. *ACS Infect Dis.* 2022 May 13;8(5):942-957. doi: 10.1021/acscinfecdis.1c00474. Epub 2022 Mar 31. PubMed Central PMID: PMC9112336.

Characterization of SARS-CoV-2 Variants B.1.617.1 (Kappa), B.1.617.2 (Delta), and B.1.618 by Cell Entry and Immune Evasion.

Ren W, Ju X, Gong M, Lan J, Yu Y, Long Q, Kenney DJ, O'Connell AK, Zhang Y, Zhong J, Zhong G, **Douam F**, Wang X, Huang A, Zhang R, Ding Q. *mBio.* 2022 Apr 26;13(2):e0009922. doi: 10.1128/mbio099-22. Epub 2022 Mar 10. PMID: PMC9040861

Comparison of RNA synthesis initiation properties of non-segmented negative strand RNA virus polymerases.

Shareef AM, Ludeke B, Jordan P, Deval J, **Fearn R**. *PLoS Pathog.* 2021 Dec 16;17(12):e1010151. doi: 10.1371/journal.ppat.1010151. eCollection 2021 Dec. PMID: PMC8717993

Helical ordering of envelope-associated proteins and glycoproteins in respiratory syncytial virus.

Conley MJ, Short JM, Burns AM, Streetley J, Hutchings J, Bakker SE, Power BJ, Jaffery H, Haney J, Zanetti G, Murcia PR, Stewart M, **Fearn R**, Vijayakrishnan S, Bhella D. *EMBO J.* 2022 Feb 1;41(3):e109728. doi: 10.15252/embj.2021109728. Epub 2021 Dec 22. PMID: PMC8804925

Natural history of disease in cynomolgus monkeys exposed to Ebola virus Kikwit strain demonstrates the reliability of this non-human primate model for Ebola virus disease.

Niemuth NA, Fallacara D, Triplett CA, Tamrakar SM, Rajbhandari A, Florence C, Ward L, **Griffiths A**, Carrion R Jr, Goez-Gazi Y, Alfson KJ, Staples HM, Brasel T, Comer JE, Massey S, Smith J, Kocsis A, Lowry J, Johnston SC, Nalca A, Goff AJ, Shurtleff AC, Pitt ML, Trefry J, Fay MP. *PLoS One.* 2021 Jul 2;16(7):e0252874. doi: 10.1371/journal.pone.0252874. eCollection 2021. PMID: PMC8253449

Identification and Characterization of Defective Viral Genomes in Ebola Virus-Infected Rhesus Macaques.

Johnson RI, Boczkowska B, Alfson K, Weary T, Menzie H, Delgado J, Rodriguez G, Carrion R Jr, **Griffiths A**. *J Virol.* 2021 Aug 10;95(17):e0071421. doi: 10.1128/JVI714-21. Epub 2021 Aug 10. PMID: PMC8354239

Biomaterials and Oxygen Join Forces to Shape the Immune Response and Boost COVID-19 Vaccines.

Colombani T, Eggermont LJ, Rogers ZJ, McKay LGA, Avena LE, Johnson RI, Storm N, **Griffiths A**, Bencherif SA. *Adv Sci (Weinh).* 2021 Jul 20;8(18):2100316. doi: 10.1002/advs.202100316. eCollection 2021 Sep. PMID: PMC8209904

*2021 Taxonomic update of phylum Negarnaviricota (Riboviria: Orthornavirae), including the large orders Bunyavirales and Mononegavirales. **

Kuhn JH, ..., **Griffiths A**, ..., **Mühlberger E**, et al. *Arch Virol.* 2021 Dec;166(12):3513-3566. doi: 10.1007/s00705-021-05143-6. PMID: PMC8627462 (available on 2022-12-01)

Structural basis for continued antibody evasion by the SARS-CoV-2 receptor binding domain.

Nabel KG, Clark SA, Shankar S, Pan J, Clark LE, Yang P, Coscia A, McKay LGA, Varnum HH, Brusic V, Tolan NV, Zhou G, Desjardins M, Turbett SE, Kanjilal S, Sherman AC, Dighe A, LaRocque RC, Ryan ET, Tylek C, Cohen-Solal JF, Darcy AT, Tavella D, Clabbers A, Fan Y, **Griffiths A**, Correia IR, Seagal J, Baden LR, Charles RC, Abraham J. *Science.* 2022 Jan 21;375(6578):eabl6251. doi: 10.1126/science.abl6251. Epub 2022 Jan 21. PMID: PMC9127715

Characterization of an Anti-Ebola Virus Hyperimmune Globulin Derived From Convalescent Plasma.

Ciencewicki JM, Herbert AS, Storm N, Joselyn NM, Huie KE, McKay LGA, **Griffiths A**, Dye JM, Willis T, Arora V. *J Infect Dis.* 2022 Feb 15;225(4):733-740. doi: 10.1093/infdis/jiab432. PMID: 34448858

Secondary structural ensembles of the SARS-CoV-2 RNA genome in infected cells.

Lan TCT, Allan MF, Malsick LE, Woo JZ, Zhu C, Zhang F, Khandwala S, Nyeo SSY, Sun Y, Guo JU, Bathe M, Näär A, **Griffiths A**, Rouskin S. *Nat Commun.* 2022 Mar 2;13(1):1128. doi: 10.1038/s41467-022-28603-2. PMID: PMC8891300

Antibodies induced by ancestral SARS-CoV-2 strain that cross-neutralize variants from Alpha to Omicron BA.1.

Windsor IW, Tong P, Lavidor O, Sanjari Moghaddam A, McKay LGA, Gautam A, Chen Y, MacDonald EA, Yoo DK, **Griffiths A**, Wesemann DR, Harrison SC. *Sci Immunol*. 2022 May 10:eabo3425. doi: 10.1126/sciimmunol.abo3425. Online ahead of print. PMID: PMC9097876 **Free PMC article**

Calpain-1 inhibition attenuates thrombosis in a humanized model of sickle cell disease and inhibits SARS-CoV-2 Replication.

Schwake, C., McKay, L.G.A., Cartel, C., **Griffiths, A.**, Flaumen, R., Chishti, A. Accepted for publication in *Journal of Cellular and Molecular Medicine*.

Virus-Mimicking Polymer Nanoparticles Targeting CD169⁺ Macrophages as Long-Acting Nanocarriers for Combination Antiretrovirals.

Eshaghi B, Fofana J, Nodder SB, **Gummuluru S**, Reinhard BM. *ACS Appl Mater Interfaces*. 2022 Jan 19;14(2):2488-2500. doi: 10.1021/acsmi.1c17415. Epub 2022 Jan 7. PMID: PMC9126061

*Identification of druggable host targets needed for SARS-CoV-2 infection by combined pharmacological evaluation and cellular network directed prioritization both in vitro and in vivo. **

Patten JJ, Keiser PT, Gysi D, Menichetti G, Mori H, Donahue CJ, Gan X, do Valle I, Geoghegan-Barek K, Anantpadma M, Boytz R, Berrigan JL, Hulse-Stubbs S, Ayazika T, O'Leary C, Jalloh S, Wagner F, Ayehunie S, Elledge SJ, Anderson D, Loscalzo J, Zitnik M, **Gummuluru S**, Namchuk MN, Barabási AL, **Davey RA**. *bioRxiv*. 2022 Feb 1:2021.04.20.440626. doi: 10.1101/2021.04.20.440626. PMID: PMC8077576

*Comparison of Culture, Antigen Test, and Polymerase Chain Reaction for Pneumococcal Detection in Cerebrospinal Fluid of Children. **

Hasanuzzaman M, Saha S, Malaker R, Rahman H, Sajib MSI, Das RC, Islam M, **Hamer DH**, Darmstadt GL, Saha SK. *J Infect Dis*. 2021 Sep 1;224(12 Suppl 2):S209-S217. doi: 10.1093/infdis/jiab073. PMID: PMC8409532

Spatio-temporal visualisation of cutaneous leishmaniasis in an endemic, urban area in Iran.

Firouraghi N, Mohammadi A, **Hamer DH**, Bergquist R, Mostafavi SM, Shamsoddini A, Raouf-Rahmati A, Fakhar M, Moghaddas E, Kiani B. *Acta Trop*. 2022 Jan;225:106181. doi: 10.1016/j.actatropica.2021.106181. Epub 2021 Oct 20. PMID: 34678259

Validation of visual estimation of neonatal jaundice in low-income and middle-income countries: a multicentre observational cohort study.

Darmstadt GL, **Hamer DH**, Carlin JB, Jeena PM, Mazzi E, Narang A, Deorari AK, Addo-Yobo E, Chowdhury MA, Kumar P, Abu-Sarkodie Y, Yeboah-Antwi K, Ray P, Bartos AE, Saha SK, Foote E, Bahl R, Weber MW. *BMJ Open*. 2021 Dec 31;11(12):e048145. doi: 10.1136/bmjopen-2020-048145. PMID: PMC8720979

If we build it, will they come? Results of a quasi-experimental study assessing the impact of maternity waiting homes on facility-based childbirth and maternity care in Zambia.

Scott NA, Kaiser JL, Ngoma T, McGlasson KL, Henry EG, Munro-Kramer ML, Biemba G, Bwalya M, Sakanga VR, Musonda G, **Hamer DH**, Boyd CJ, Bonawitz R, Vian T, Kruk ME, Fong RM, Chastain PS, Mataka K, Ahmed Mdluli E, Veliz P, Lori JR, Rockers PC. *BMJ Glob Health*. 2021 Dec;6(12):e006385. doi: 10.1136/bmjgh-2021-006385. PMID: 34876457

Care-Seeking Behavior for Newborns in Rural Zambia.

Thairu L, Gehling H, Kafwanda S, Yeboah-Antwi K, **Hamer DH**, Lunze K. *Matern Child Health J*. 2022 Jun;26(6):1375-1383. doi: 10.1007/s10995-021-03329-7. Epub 2022 Jan 14. PMID: 35028891

Postpartum Mental Health in Rural South Africa: Socioeconomic Stressors and Worsening Mental Health.

Silverman DT, Killion JP, Evans D, Coetzee L, Rockers PC, **Hamer DH**. *Matern Child Health J*. 2022 Feb;26(2):434-440. doi: 10.1007/s10995-021-03268-3. Epub 2021 Oct 19. PMID: 34665355

Effect of maternal vitamin D supplementation on nasal pneumococcal acquisition, carriage dynamics and carriage density in infants in Dhaka, Bangladesh.

Taghivand M, Pell LG, Rahman MZ, Mahmud AA, Ohuma EO, Pullangyeum EM, Ahmed T, **Hamer DH**, Zlotkin SH, Gubbay JB, Morris SK, Roth DE. *BMC Infect Dis*. 2022 Jan 13;22(1):52. doi: 10.1186/s12879-022-07032-y. PMID: PMC8759256

Population-based rates, risk factors and consequences of preterm births in South-Asia and sub-Saharan Africa: A multi-country prospective cohort study.

Alliance for Maternal and Newborn Health Improvement (AMANHI) GA Study Group. *J Glob Health*. 2022 Feb 19;12:04011. doi: 10.7189/jogh.12.04011. eCollection 2022. PMID: PMC8850944

Association of COVID-19 Quarantine Duration and Postquarantine Transmission Risk in 4 University Cohorts.

Liu AB, Davidi D, Landsberg HE, Francesconi M, Platt JT, Nguyen GT, Yune S, Deckard A, Puglin J, Haase SB, **Hamer DH**, Springer M. *JAMA Netw Open*. 2022 Feb 1;5(2):e220088. doi: 10.1001/jamanetworkopen.202288. PMID: 35212750

Hair, serum and urine chromium levels in children with cognitive defects: A systematic review and meta-analysis of case control studies.

Islam GMR, Rahman MM, Hasan MI, Tadesse AW, Hamadani JD, **Hamer DH**. *Chemosphere*. 2022 Mar;291(Pt 2):133017. doi: 10.1016/j.chemosphere.2021.133017. Epub 2021 Nov 20. PMID: 34813844 Review.

*Minimal SARS-CoV-2 classroom transmission at a large urban university experiencing repeated into campus introduction.**

Kuhfeldt K, Turcinovic J, Sullivan M, Landaverde L, Doucette-Stamm L, **Hamer DH**, Platt J, Klapperich C, Landsberg HE, **Connor JH**. medRxiv. 2022 Mar 16:2022.03.16.22271983. doi: 10.1101/2022.03.16.22271983. Preprint. PMID: PMC8936094

Intended versus actual delivery location and factors associated with change in delivery location among pregnant women in Southern Province, Zambia: a prespecified secondary observational analysis of the ZamCAT.

Solomon H, Henry EG, Herlihy J, Yeboah-Antwi K, Biemba G, Musokotwane K, Bhutta A, **Hamer DH**, Semrau KEA. BMJ Open. 2022 Mar 7;12(3):e055288. doi: 10.1136/bmjopen-2021-055288. PMID: PMC8905985

Acute hepatitis A in international travellers: a GeoSentinel analysis, 2008-2020.

Balogun O, Brown A, Angelo KM, Hochberg NS, Barnett ED, Nicolini LA, Asgeirsson H, Grobusch MP, Leder K, Salvador F, Chen L, Odolini S, Díaz-Menéndez M, Gobbi F, Connor BA, Libman M, **Hamer DH**. J Travel Med. 2022 Mar 21;29(2):taac013. doi: 10.1093/jtm/taac013.PMID: 35134210

*Early introduction and rise of the Omicron SARS-CoV-2 variant in highly vaccinated university populations.**

Petros BA, Turcinovic J, Welch NL, White LF, Kolaczyk ED, Bauer MR, Cleary M, Dobbins ST, Doucette-Stamm L, Gore M, Nair P, Nguyen TG, Rose S, Taylor BP, Tsang D, Wendlandt E, Hope M, Platt JT, Jacobson KR, Bouton T, Yune S, Auclair JR, Landaverde L, Klapperich CM, **Hamer DH**, Hanage WP, MacInnis BL, Sabeti PC, **Connor JH**, Springer M. Clin Infect Dis. 2022 May 25:ciac413. doi: 10.1093/cid/ciac413. Online ahead of print. PMID: PMC9213864

Use of benznidazole to treat chronic Chagas disease: An updated systematic review with a meta-analysis.

Crespillo-Andújar C, Comeche B, **Hamer DH**, Arevalo-Rodriguez I, Alvarez-Díaz N, Zamora J, Pérez-Molina JA. PLoS Negl Trop Dis. 2022 May 16;16(5):e0010386. doi: 10.1371/journal.pntd.10386. eCollection 2022 May. PMID: PMC9135346

Planning for Maternity Waiting Home Bed Capacity: Lessons from Rural Zambia.

Vian T, Kaiser JL, Ngoma T, Juntunen A, Mataka KK, Bwalya M, Sakanga VIR, Rockers PC, **Hamer DH**, Biemba G, Scott NA. Ann Glob Health. 2022 May 24;88(1):37. doi: 10.5334/aogh.3691. eCollection 2022. PMID: PMC9138814

Real-time Feedback to Improve HIV Treatment Adherence in Pregnant and Postpartum Women in Uganda: A Randomized Controlled Trial.

Sabin LL, Simmons E, Halim N, **Hamer DH**, Gifford AL, West RL, Larson A, Bonawitz R, Aroda P, Banigbe B, Holderman AJ, Murray L, DeSilva MB, Gasuza J, Mukasa B, Messersmith LJ. AIDS Behav. 2022 Jun 15. doi: 10.1007/s10461-022-03712-7. Online ahead of print.PMID: 35704124

Buildout and integration of an automated high-throughput CLIA laboratory for SARS-CoV-2 testing on a large urban campus.

Landaverde L, McIntyre D, Robson J, Fu D, Ortiz L, Chen R, Oliveira SMD, Fan A, Barrett A, Burgay SP, Choate S, Corbett D, Doucette-Stamm L, Gonzales K, **Hamer DH**, Huang L, Huval S, Knight C, Landa C, Lindquist D, Lockard K, Macdowell TL, Mauro E, McGinty C, Miller C, Monahan M, Moore R, Platt J, Rolles L, Roy J, Schroeder T, Tolan DR, Zaia A, Brown RA, Waters G, Densmore D, Klapperich CM. SLAS Technol. 2022 Jun 17:S2472-6303(22)05163-9. doi: 10.1016/j.slast.2022.063. Online ahead of print. PMID: PMC9212990

*Viral dynamics of Omicron and Delta SARS-CoV-2 variants with implications for timing of release from isolation: a longitudinal cohort study.**

Bouton TC, Atarere J, Turcinovic J, Seitz S, Sher-Jan C, Gilbert M, White L, Zhou Z, Hossain MM, Overbeck V, Doucette-Stamm L, Platt J, Landsberg HE, **Hamer DH**, Klapperich C, Jacobson KR, **Connor JH**. Clin Infect Dis. 2022 Jun 23:ciac510. doi: 10.1093/cid/ciac510. Online ahead of print. PMID: PMC8996632 Updated.

*Barriers and facilitators to facility-based delivery in rural Zambia: a qualitative study of women's perceptions after implementation of an improved maternity waiting homes intervention Memory B cell repertoire for recognition of evolving SARS-CoV-2 spike **

Tong P, Gautam A, Windsor IW, Travers M, Chen Y, Garcia N, Whiteman NB, McKay LGA, Storm N, Malsick LE, **Honko AN**, Lelis FJN, Habibi S, Jenni S, Cai Y, Rennick LJ, Duprex WP, McCarthy KR, Lavine CL, Zuo T, Lin J, Zuiani A, Feldman J, MacDonald EA, Hauser BM, **Griffiths A**, Seaman MS, Schmidt AG, Chen B, Neuberg D, Bajic G, Harrison SC, Wesemann DR. Cell 2021 Sep 16;184(19):4969-4980.e15. doi: 10.1016/j.cell.2021.07.025. Epub 2021 Jul 23. PMID: PMC8299219

*Surface Glycan Modification of Cellular Nanosponges to Promote SARS-CoV-2 Inhibition.**

Ai, X., Wang, D., **Honko, A.**, Duan, Y., Gavriush, I., Fang, R. H., **Griffiths, A.**, Gao, W., & Zhang, L. (2021). Journal of the American Chemical Society, 143(42), 17615–17621. PMID: PMC8525344

*An AAV-based, room-temperature-stable, single-dose COVID-19 vaccine provides durable immunogenicity and protection in non-human primates.**

Zabaleta N, Dai W, Bhatt U, Hérate C, Maisonnasse P, Chichester JA, Sanmiguel J, Estelien R, Michalson KT, Diop C, Maciorowski D, Dereuddre-Bosquet N, Cavarelli M, Gallouët AS, Naninck T, Kahlaoui N, Lemaitre J, Qi W, Hudspeth E, Cucalon A, Dyer CD, Pampena MB, Knox JJ, LaRocque RC, Charles RC, Li D, Kim M, Sheridan A, Storm N, Johnson RI, Feldman J, Hauser BM, Contreras V, Marlin R, Tsong Fang RH, Chapon C, van der Werf S, Zinn E, Ryan A, Kobayashi DT, Chauhan R, McGlynn M, Ryan ET, Schmidt AG, Price B, **Honko A**, **Griffiths A**, Yaghmour S, Hodge R, Betts MR, Freeman MW, Wilson JM, Le Grand R, Vandenberghe LH. Cell Host Microbe. 2021 Sep 8;29(9):1437-1453.e8. doi: 10.1016/j.chom.2021.082. Epub 2021 Aug 7. PMID: PMC8346325

- A Modular Biomaterial Scaffold-Based Vaccine Elicits Durable Adaptive Immunity to Subunit SARS-CoV-2 Antigens.* *
Langellotto F, Dellacherie MO, Yeager C, Ijaz H, Yu J, Cheng CA, Dimitrakakis N, Seiler BT, Gebre MS, Gilboa T, Johnson R, Storm N, Bardales S, Graveline A, White D, Tringides CM, Cartwright MJ, Doherty EJ, **Honko A, Griffiths A**, Barouch DH, Walt DR, Mooney DJ. *Adv Healthc Mater.* 2021 Nov;10(22):e2101370. doi: 10.1002/adhm.202101370. Epub 2021 Oct 4. PMID: PMC8652677
- Surface Glycan Modification of Cellular Nanosponges to Promote SARS-CoV-2 Inhibition.* *
Ai X, Wang D, **Honko A**, Duan Y, Gavrish I, Fang RH, **Griffiths A**, Gao W, Zhang L. *J Am Chem Soc.* 2021 Oct 27;143(42):17615-17621. doi: 10.1021/jacs.1c07798. Epub 2021 Oct 14. PMID: PMC8346325
- Natural History of Aerosol-Induced Ebola Virus Disease in Rhesus Macaques.*
Downs I, Johnson JC, Rossi F, Dyer D, Saunders DL, Twenhafel NA, Esham HL, Pratt WD, Trefry J, Zumbun E, Facemire PR, Johnston SC, Tompkins EL, Jansen NK, **Honko A**, Cardile AP. *Viruses.* 2021 Nov 17;13(11):2297. doi: 10.3390/v13112297. PMID: PMC8619410
- Preclinical Efficacy of IMM-BCP-01, a highly active patient derived anti-SARS-CoV-2 antibody cocktail.*
Nitikin, P.A., DiMuzio, J.M., Dowling, J.P., Bingaman-Steele, J.L., Heimbach, B.C., Henriquez, N., Nicolescu, C., Polley, A., Sikorski, E.L., Howanski, R.J., Nath, M., Shukla, H., Scheaffer, S.M., Finn, J.P., Liang, L.F., Smith, T., Storm, N., McKay, L.G.A., Johnson, R.I., Malsick, L.E., **Honko, A.N., Griffiths, A.**, Diamond, M.S., Sarma, P., Geising, D.H., Morin, M.J., Robinson, M.K. *Sci Immunol.* 2022. eab19943. PMID: 35771946 Preprint.
- Detailed analysis of the pathologic hallmarks of Nipah virus (Malaysia) disease in the African green monkey infected by the intratracheal route.*
Cline C, Bell TM, Facemire P, Zeng X, Briese T, Lipkin WI, Shamblyn JD, Esham HL, Donnelly GC, Johnson JC, Hensley LE, **Honko AN**, Johnston SC. *PLoS One.* 2022 Feb 10;17(2):e0263834. doi: 10.1371/journal.pone.0263834. eCollection 2022. PMID: PMC8830707
- Fecal Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-Cov-2) RNA is associated with decreased coronavirus disease 2019 (COVID-19) Survival.* *
Das Adhikari U, Eng G, Farcasanu M, Avena LE, Choudhary MC, Triant VA, Flagg M, Schiff AE, Gomez I, Froehle LM, Diefenbach TJ, Ronsard L, Lingwood D, Lee GC, Rabi SA, Erstad D, Velmahos G, Li JZ, Hodin R, Stone JR, **Honko AN, Griffiths A**, Yilmaz ÖH, Kwon DS. *Clin Infect Dis.* 2022 Mar 23;74(6):1081-1084. doi: 10.1093/cid/ciab623. PMID: PMC8406863
- IMM-BCP-01, a patient-derived anti-SARS-CoV-2 antibody cocktail, is active across variants of concern including Omicron BA.1 and BA.2.* *
Nikitin PA, DiMuzio JM, Dowling JP, Patel NB, Bingaman-Steele JL, Heimbach BC, Henriquez N, Nicolescu C, Polley A, Sikorski EL, Howanski RJ, Nath M, Shukla H, Scheaffer SM, Finn JP, Liang LF, Smith T, Storm N, McKay LGA, Johnson RI, Malsick LE, **Honko AN, Griffiths A**, Diamond MS, Sarma P, Geising DH, Morin MJ, Robinson MK. *Sci Immunol.* 2022 Jun 30:eab19943. doi: 10.1126/sciimmunol.abl9943. Online ahead of print. PMID: PMC9273042
- Interferon-alpha or -beta facilitates SARS-CoV-2 pulmonary vascular infection by inducing ACE2.* *
Klouda T, Hao Y, Kim H, Kim J, Olejnik J, **Hume AJ**, Ayyappan S, Hong X, Melero-Martin J, Fang Y, Wang Q, Zhou X, **Mühlberger E**, Jia H, Padera RF Jr, Raby BA, Yuan K. *Angiogenesis.* 2022 May;25(2):225-240. doi: 10.1007/s10456-021-09823-4. Epub 2021 Oct 29. PMID: 34714440; PMID: PMC8554520.
- Reconstructed signaling and regulatory networks identify potential drugs for SARS-CoV-2 infection.* *
Ding J, Lugo-Martinez J, Yuan Y, Huang J, **Hume AJ**, Suder EL, **Mühlberger E**, Kotton DN, Bar-Joseph Z. *bioRxiv.* 2021 Dec 9:2020.06.01.127589. doi: 10.1101/2020.06.01.127589. Preprint. PMID: PMC7574259
- Human airway lineages derived from pluripotent stem cells reveal the epithelial responses to SARS-CoV-2 infection.* *
Wang R, **Hume AJ**, Beermann ML, Simone-Roach C, Lindstrom-Vautrin J, Le Suer J, Huang J, Olejnik J, Villacorta-Martin C, Bullitt E, Hinds A, Ghaedi M, Rollins S, Werder RB, Abo KM, Wilson AA, **Mühlberger E**, Kotton DN, Hawkins FJ. *Am J Physiol Lung Cell Mol Physiol.* 2022 Jan 12. doi: 10.1152/ajplung.00397.2021. Epub ahead of print. PMID: 35020534
- A virus-specific monocyte inflammatory phenotype is induced by SARS-CoV-2 at the immune-epithelial interface.* *
Leon J, Michelson DA, Olejnik J, Chowdhary K, Oh HS, **Hume AJ**, Galván-Peña S, Zhu Y, Chen F, Vijaykumar B, Yang L, Crestani E, Yonker LM, Knipe DM, **Mühlberger E**, Benoist C. *Proc Natl Acad Sci U S A.* 2022 Jan 4;119(1):e2116853118. doi: 10.1073/pnas.2116853118. PMID: 34969849.
- Recombinant Lloviu virus as a tool to study viral replication and host responses.* *
Hume AJ, Heiden B, Olejnik J, Suder EL, Ross S, Scoon WA, Bullitt E, Ericsson M, White MR, Turcinovic J, Thao TTN, Hekman RM, Kaserman JE, Huang J, Alysandratos KD, Toth GE, Jakab F, Kotton DN, Wilson AA, Emili A, Thiel V, **Connor JH**, Kemenesi G, Cifuentes D, **Mühlberger E**. *PLoS Pathog.* 2022 Feb 4;18(2):e1010268. doi: 10.1371/journal.ppat.1010268. eCollection 2022 Feb. PMID: PMC8849519
- The oral drug nitazoxanide restricts SARS-CoV-2 infection and attenuates disease pathogenesis in Syrian hamsters.* *
Miorin L, Mire CE, Ranjbar S, **Hume AJ**, Huang J, **Crossland NA**, White KM, Laporte M, Kehrer T, Haridas V, Moreno E, Nambu A, Jangra S, Cupic A, Dejez M, Abo KA, Tseng AE, Werder RB, Rathnasinghe R, Mutetwa T, Ramos I, de Aja JS, de Alba Rivas CG, Schotsaert M, **Corley RB**, Falvo JV, Fernandez-Sesma A, Kim C, Rossignol JF, Wilson AA, Zwaka T, Kotton DN, **Mühlberger E**, García-Sastre A, Goldfeld AE. *bioRxiv.* 2022 Feb 9:2022.02.08.479634. doi: 10.1101/2022.02.08.479634. Preprint. PMID: PMC8845418

Ebola virus infection induces a type I interferon response and the shutdown of key liver functions in human iPSC-derived hepatocytes *

Manhart W, Mancio L, Suder E, Villacorta-Martin C, Lindstrom-Vautrin J, Bernbaum J, Mazur S, Johnson R, Mithal A, Olejnik J, Hume AJ, Kaserman J, Wilson AA, Bhatia S, Mühlberger E, Mostoslavsky G. Accepted in Stem Cell Reports. <https://doi.org/10.21203/rs.3.rs-346554/v1>

Development of Neutralization Breadth against Diverse HIV-1 by Increasing Ab-Ag Interface on V2.

Gao N, Gai Y, Meng L, Wang C, Wang W, Li X, Gu T, Louder MK, Doria-Rose NA, Wiehe K, Nazzari AF, Olia AS, Gorman J, Rawi R, Wu W, Smith C, Khant H, de Val N, Yu B, Luo J, Niu H, Tsybovsky Y, Liao H, Kepler TB, Kwong PD, Mascola JR, Qin C, Zhou T, Yu X, Gao F. *Adv Sci (Weinh)*. 2022 May;9(15):e2200063. doi: 10.1002/advs.202200063. Epub 2022 Mar 23. PMID: PMC9130890

Channeling macrophage polarization by rocaglates increases macrophage resistance to Mycobacterium tuberculosis.

Chatterjee S, Yabaji SM, Rukhlenko OS, Bhattacharya B, Waligurski E, Vallavoju N, Ray S, Kholodenko BN, Brown LE, Beeler AB, Ivanov AR, Kobzik L, Porco JA Jr, Kramnik I. *iScience*. 2021 Jul 10;24(8):102845. doi: 10.1016/j.isci.2021.102845. eCollection 2021 Aug 20. PMID: PMC8333345

CXCL1: A new diagnostic biomarker for human tuberculosis discovered using Diversity Outbred mice.

Koyuncu D, Niazi MKK, Tavolara T, Abeijon C, Ginese ML, Liao Y, Mark C, Specht A, Gower AC, Restrepo BI, Gatti DM, Kramnik I, Gurcan M, Yener B, Beamer G. *PLoS Pathog*. 2021 Aug 17;17(8):e1009773. doi: 10.1371/journal.ppat.1009773. eCollection 2021 Aug. PMID: PMC8423361

Progression and Dissemination of Pulmonary Mycobacterium Avium Infection in a Susceptible Immunocompetent Mouse Model. *

Rosenbloom R, Gavriš I, Tseng AE, Seidel K, Yabaji SM, Gertje HP, Huber BR, Kramnik I, Crossland NA. *Int J Mol Sci*. 2022 May 26;23(11):5999. doi: 10.3390/ijms23115999. PMID: PMC9181083

Medium throughput protocol for genome-based quantification of intracellular mycobacterial loads and macrophage survival during in vitro infection.

Yabaji SM, Chatterjee S, Waligursky E, Gimelbrant A, Kramnik I. *STAR Protoc*. 2022 Mar 15;3(2):101241. doi: 10.1016/j.xpro.2022.101241. eCollection 2022 Jun 17. PMID: PMC8931439

Science, not speculation, is essential to determine how SARS-CoV-2 reached humans.

Calisher CH, Carroll D, Colwell R, Corley RB, Daszak P, Drosten C, Enjuanes L, Farrar J, Field H, Golding J, Gorbalenya AE, Haagmans B, Hughes JM, Keusch GT, Lam SK, Lubroth J, Mackenzie JS, Madoff L, Mazet JK, Perlman SM, Poon L, Saif L, Subbarao K, Turner M. *Lancet*. 2021 Jul 17;398(10296):209-211. doi: 10.1016/S0140-6736(21)01419-7. Epub 2021 Jul 5.

Should global financing be the main priority for pandemic preparedness? - Authors' reply.

Lurie N, Keusch GT, Dzau VJ. *Lancet*. 2021 Jul 31;398(10298):388-389. doi: 10.1016/S0140-6736(21)01382-9. PMID: 34332679.

An appeal for an objective, open, and transparent scientific debate about the origin of SARS-CoV-2 - Authors' reply.

Keusch GT, Lam SK, Mackenzie JS, Saif L, Turner M. *Lancet*. 2021 Oct 16;398(10309):1404-1405. doi: 10.1016/S0140-6736(21)02013-4. Epub 2021 Sep 17. PMID: PMC8448489

Transposable element landscapes in aging Drosophila.

Yang N, Srivastav SP, Rahman R, Ma Q, Dayama G, Li S, Chinen M, Lei EP, Rosbash M, Lau NC (2022). *PLoS Genetics*. 2022 Mar 3;18(3):e1010024. doi: 10.1371/journal.pgen.1010024. eCollection 2022 Mar. PMID: 35239675

Extending and Running the Mosquito Small RNA Genomics Resource Pipeline.

Dayama G, Bulekova K, Lau NC. *Methods Mol Biol*. 2022;2509:341-352. doi: 10.1007/978-1-0716-2380-0_20. PMID: 35796973

Transcriptomic and small RNA response to Mayaro virus infection in Anopheles stephensi mosquitoes.

Henderson C, Brustolin M, Hegde S, Dayama G, Lau N, Hughes GL, Bergey C, Rasgon JL. *PLoS Negl Trop Dis*. 2022 Jun 28;16(6):e0010507. doi: 10.1371/journal.pntd10507. eCollection 2022 Jun. PMID: PMC9273063

An Integrative Genomic Strategy Identifies sRAGE as a Causal and Protective Biomarker of Lung Function.

Keefe J, Yao C, Hwang SJ, Courchesne P, Lee GY, Dupuis J, Mizgerd JP, O'Connor G, Washko GR, Cho MH, Silverman EK, Levy D. *Chest*. 2022 Jan;161(1):76-84. doi: 10.1016/j.chest.2021.06.053. Epub 2021 Jul 6. PMID: PMC8783029 (available on 2023-01-01)

Understanding the Host in the Management of Pneumonia. An Official American Thoracic Society Workshop Report.

Dela Cruz CS, Evans SE, Restrepo MI, Dean N, Torres A, Amara-Elori I, Awasthi S, Caler E, Cao B, Chalmers JD, Chastre J, Cohen TS, Cohen AH, Crothers K, Di YP, Egan ME, Feldman C, Gautam S, Halstead ES, Herold S, Jones BE, Luna C, Niederman MS, Mendez R, Mizgerd JP, Nusrat R, Ramirez J, Shindo Y, Waterer G, Yeligar SM, Wunderink RG. *Ann Am Thorac Soc*. 2021 Jul;18(7):1087-1097. doi: 10.1513/AnnalsATS.202102-209ST. PMID: PMC8328365

Liver-Dependent Lung Remodeling during Systemic Inflammation Shapes Responses to Secondary Infection.

Odom CV, Kim Y, Burgess CL, Baird LA, Korkmaz FT, Na E, Shenoy AT, Arafa EI, Lam TT, Jones MR, Mizgerd JP, Traber KE, Quinton LJ. *J Immunol*. 2021 Oct 1;207(7):1891-1902. doi: 10.4049/jimmunol.2100254. Epub 2021 Sep 1. PMID: PMC8631467

Antigen presentation by lung epithelial cells directs CD4+ TRM cell function and regulates barrier immunity.

Shenoy AT, Lyon De Ana C, Arafa EI, Salwig I, Barker KA, Korkmaz FT, Ramanujan A, Etesami NS, Soucy AM, Martin IMC, Tilton BR, Hinds A, Goltry WN, Kathuria H, Braun T, Jones MR, Quinton LJ, Belkina AC, **Mizgerd JP**. *Nat Commun*. 2021 Oct 5;12(1):5834. doi: 10.1038/s41467-021-26045-w. PMID: PMC8492657

Comprehensive phenotyping of murine lung resident lymphocytes after recovery from pneumococcal pneumonia.

Shenoy AT, Lyon De Ana C, Barker KA, Arafa EI, Martin IMC, **Mizgerd JP**, Belkina AC. *Cytometry A*. 2021 Dec 2;10.1002/cyto.a.24522. doi: 10.1002/cyto.a.24522. Online ahead of print. PMID: PMC9160214 (available on 2023-06-02)

Stimulation of a subset of natural killer T cells by CD103+ DC is required for GM-CSF and protection from pneumococcal infection.

Murray MP, Crosby CM, Marcovecchio P, Hartmann N, Chandra S, Zhao M, Khurana A, Zahner SP, Clausen BE, Coleman FT, **Mizgerd JP**, Mikulski Z, Kronenberg M. *Cell Rep*. 2022 Jan 11;38(2):110209. doi: 10.1016/j.celrep.2021.110209. PMID: PMC8934033

Update on the Features and Measurements of Experimental Acute Lung Injury in Animals: An Official American Thoracic Society Workshop Report.

Kulkarni HS, Lee JS, Bastarache JA, Kuebler WM, Downey GP, Albaiceta GM, Altemeier WA, Artigas A, Bates JHT, Calfee CS, Dela Cruz CS, Dickson RP, Englert JA, Everitt JJ, Fessler MB, Gelman AE, Gowdy KM, Groshong SD, Herold S, Homer RJ, Horowitz JC, Hsia CCW, Kurahashi K, Laubach VE, Looney MR, Lucas R, Mangalmurti NS, Manicone AM, Martin TR, Matalon S, Matthay MA, McAuley DF, McGrath-Morrow SA, **Mizgerd JP**, Montgomery SA, Moore BB, Noël A, Perlman CE, Reilly JP, Schmidt EP, Skerrett SJ, Suber TL, Summers C, Suratt BT, Takata M, Tudor R, Uhlig S, Witzernath M, Zemans RL, Matute-Bello G. *Am J Respir Cell Mol Biol*. 2022 Feb;66(2):e1-e14. doi: 10.1165/rcmb.2021-0531ST. PMID: PMC8845128

Epithelial LIF signaling limits apoptosis and lung injury during bacterial pneumonia.

Na E, Allen E, Baird LA, Odom CV, Korkmaz FT, Shenoy AT, Matschulat AM, Jones MR, Kotton DN, **Mizgerd JP**, Varelas X, Traber KE, Quinton LJ. *Am J Physiol Lung Cell Mol Physiol*. 2022 Apr 1;322(4):L550-L563. doi: 10.1152/ajplung.2021. Epub 2022 Feb 9. PMID: PMC8957336 (available on 2023-04-01)

Recruitment and training of alveolar macrophages after pneumococcal pneumonia.

Arafa EI, Shenoy AT, Barker KA, Etesami NS, Martin IM, Lyon De Ana C, Na E, Odom CV, Goltry WN, Korkmaz FT, Wooten AK, Belkina AC, Guillon A, Forsberg EC, Jones MR, Quinton LJ, **Mizgerd JP**. *JCI Insight*. 2022 Mar 8;7(5):e150239. doi: 10.1172/jci.insight.150239. PMID: PMC8983128

Selection of a SARS-CoV-2 Surrogate for Use in Surface Disinfection Efficacy Studies with Chlorine and Antimicrobial Surfaces.

String G. M., White M. R., Gute D. M., **Mühlberger E.**, Lantagne D. S. 2021. *Environ Sci Technol Lett*. acs.estlett.1c00593. published online 01 Oct 2021. PMID: PMC8491555.

Extracellular vimentin is an attachment factor that facilitates SARS-CoV-2 entry into human endothelial cells.

Amraei R, Xia C, Olejnik J, White MR, Napoleon MA, Lotfollahzadeh S, Hauser BM, Schmidt AG, Chitalia V, **Mühlberger E**, Costello CE, Rahimi N. *Proc Natl Acad Sci U S A*. 2022 Feb 8;119(6):e2113874119. doi: 10.1073/pnas.2113874119. PMID: PMC8833221

*CD169-mediated restrictive SARS-CoV-2 infection of macrophages induces pro-inflammatory responses. **

Jalloh S, Olejnik J, Berrigan J, Nisa A, Suder EL, Akiyama H, Lei M, Tyagi S, Bushkin Y, **Mühlberger E**, **Gummuru S**. *bioRxiv*. 2022 Mar 30:2022.03.29.486190. doi: 10.1101/2022.03.29.486190. Preprint. PMID: PMC8978933

*Isolation of infectious Lloviu virus from Schreiber's bats in Hungary. **

Kemenesi G, Tóth GE, Mayora-Neto M, Scott S, Temperton N, Wright E, **Mühlberger E**, **Hume AJ**, Suder EL, Zana B, Boldogh SA, Görföl T, Estók P, Lanszki Z, Somogyi BA, Nagy Á, Pereszlényi CI, Dudás G, Földes F, Kurucz K, Madai M, Zeghib S, Maes P, Vanmechelen B, Jakab F. *Nat Commun*. 2022 Mar 31;13(1):1706. doi: 10.1038/s41467-022-29298-1. PMID: 35361761

*Distinctive features of the respiratory syncytial virus priming loop compared to other non-segmented negative strand RNA viruses. **

Cressey TN, Shareef AM, Kleiner VA, Noton SL, Byrne PO, McLellan JS, **Mühlberger E**, **Fearns R**. *PLoS Pathog*. 2022 Jun 22;18(6):e1010451. doi: 10.1371/journal.ppat.1010451. eCollection 2022 Jun. PMID: PMC9255747

Profiling SARS-CoV-2 HLA-I peptidome reveals T cell epitopes from out-of-frame ORFs.

Weingarten-Gabbay S, Klaeger S, Sarkizova S, Pearlman LR, Chen DY, Gallagher KME, Bauer MR, Taylor HB, Dunn WA, Tarr C, Sidney J, Rachimi S, Conway HL, Katsis K, Wang Y, Leistriz-Edwards D, Durkin MR, Tomkins-Tinch CH, Finkel Y, Nachshon A, Gentili M, Rivera KD, Carulli IP, Chea VA, Chandrashekar A, Bozkus CC, Carrington M; MGH COVID-19 Collection & Processing Team, Bhardwaj N, Barouch DH, Sette A, Maus MV, Rice CM, Clauser KR, Keskin DB, Pregibon DC, Hacohen N, Carr SA, Abelin JG, **Saeed M**, Sabeti PC. *Cell*. 2021 Jul 22;184(15):3962-3980.e17. doi: 10.1016/j.cell.2021.05.046. PMID: PMC8173604

*DNA templates with blocked long 3' end single-stranded overhangs (BL3SSO) promote bona fide Cas9-stimulated homology-directed repair of long transgenes into endogenous gene loci. **

Bandyopadhyay S, Douglass J, Kapell S, Khan N, Feitosa-Suntheimer F, Klein JA, Temple J, Brown-Culbertson J, Tavares AH, **Saeed M**, **Lau NC**. *G3 (Bethesda)*. 2021 Aug 7;11(8):jkab169. doi: 10.1093/g3journal/jkab169. PMID: PMC8496256

Developing a SARS-CoV-2 Antigen Test Using Engineered Affinity Proteins.

Kim S, Yee E, Miller EA, Hao Y, Tay DMY, Sung KJ, Jia H, Johnson JM, **Saeed M**, Mace CR, Yüksel Yurt D, Sikes HD. ACS Appl Mater Interfaces. 2021 Aug 25;13(33):38990-39002. doi: 10.1021/acscami.1c08174. Epub 2021 Aug 11. PMID: 34379400

Discovery of Quinoxaline-Based P1-P3 Macrocyclic NS3/4A Protease Inhibitors with Potent Activity against Drug-Resistant Hepatitis C Virus Variants.

Nageswara Rao D, Zephyr J, Henes M, Chan ET, Matthew AN, Hedger AK, Conway HL, **Saeed M**, Newton A, Petropoulos CJ, Huang W, Kurt Yilmaz N, Schiffer CA, Ali A. J Med Chem. 2021 Aug 26;64(16):11972-11989. doi: 10.1021/acs.jmedchem.1c00554. Epub 2021 Aug 18. PMID: PMC9228641

Susceptibilities of Human ACE2 Genetic Variants in Coronavirus Infection.

Ren W, Zhu Y, Lan J, Chen H, Wang Y, Shi H, Feng F, Chen DY, Close B, Zhao X, Wu J, Tian B, Yuan Z, Zhou D, **Saeed M**, Wang X, Zhang R, Ding Q. J Virol. 2022 Jan 12;96(1):e0149221. doi: 10.1128/JVI.01492-21. Epub 2021 Oct 20. PMID: PMC8754202

Proteomic elucidation of the targets and primary functions of the picornavirus 2A protease.

Serganov AA, Udi Y, Stein ME, Patel V, Fridy PC, Rice CM, **Saeed M**, Jacobs EY, Chait BT, Rout MP. J Biol Chem. 2022 Jun;298(6):101882. doi: 10.1016/j.jbc.2022.101882. Epub 2022 Mar 31. PMID: PMC9168619

Surgical Strikes on Host Defenses: Role of the Viral Protease Activity in Innate Immune Antagonism.

Chin CV, **Saeed M**. Pathogens. 2022 Apr 28;11(5):522. doi: 10.3390/pathogens11050522. Review. PMID: PMC9145062

Guidelines for nucleic acid template design for optimal cell-free protein synthesis using an Escherichia coli reconstituted system or a lysate-based system.

Asahara H, Magnelli P, Shi X, Tuckey C, Zhou Y, **Samuelson JC**. Methods Enzymol. 2021;659:351-369. doi: 10.1016/bs.mie.2021.075. Epub 2021 Sep 2. PMID: 34752294

Regulation of Pkc1 Hyper-Phosphorylation by Genotoxic Stress.

Liu L, Veis J, Reiter W, Motari E, Costello CE, **Samuelson JC**, Ammerer G, Levin DE. J Fungi (Basel). 2021 Oct 17;7(10):874. doi: 10.3390/jof7100874. PMID: PMC8541566

FY22 Funded Research

The work which resulted in the publications listed in the preceding pages would not have been possible without the ability of our faculty to competitively seek funding to support their research activities. NEIDL faculty members - both core faculty (Table 1) and affiliate faculty (Table 2) - received over **\$ 38 Million** in funding in FY22 for the following projects:

Table 1

AWARD TITLE	PI / CO-PI	SPONSOR (PRIME SPONSOR)	PROJECT START DATE - PROJECT END DATE	ADDL FUNDS FY-22
DEVELOPMENT OF A RVSV VECTORED VACCINE FOR LASSA VIRUS: NONHUMAN PRIMATE EFFICACY AND IMMUNOGENICITY	CONNOR JOHN	UTMB Galveston (DOD/DTRA)	09/01/21 - 08/31/22	423,390
DEEP SEQUENCING OF PATHOGENS TO PRECISELY DEFINE TRANSMISSION NETWORKS USING RARE VARIANTS	CONNOR JOHN	Harvard College (NIH/NIAID)	06/01/21 - 05/31/22	110,731
CULTURE OF SARS-COV-2 VARIANTS OF INTEREST FROM THE MASS REGION	CONNOR JOHN	Harvard College (China Evergrande)	10/01/21 - 09/30/22	178,250
ADVANCEMENT OF A POXVIRUS INHIBITOR	CONNOR JOHN	NIH/NIAID	03/12/20 - 02/28/25	593,447
MODULAR POINT-OF-CARE PLATFORM FOR DIFFERENTIAL DIAGNOSIS OF VIRAL HEMORRHAGIC FEVERS	CONNOR JOHN	RedBud Labs (NIH/NIAID)	02/14/22 - 06/01/22	80,342
GEOSENTINEL: THE GLOBAL SURVEILLANCE NETWORK OF ISTM AND CDC	CONNOR JOHN	ISTM (HHS/CDC)	05/01/22 - 04/30/23	120,540
DETERMINANTS OF COVID19-INDUCED VENOUS THROMBOSIS AND TARGETED THERAPY ASSESSED WITH BIOENG VEIN-CHIP	CONNOR JOHN	NIH/NHLBI	05/01/21 - 04/30/25	665,322
NATIONAL EMERGING INFECTIOUS DISEASES LABORATORIES OPERATIONS	CORLEY B RONALD	NIH/NIAID	07/08/21 - 06/30/26	11,500,000
VECTRA POLARIS QUANTITATIVE PATHOLOGY IMAGING SYSTEM	CROSSLAND NICK	NIH/Office of the Director	09/20/21 - 09/19/22	402,440
ASSAY DEVELOPMENT AND EXECUTION OF LIVE VIRUS TESTING IN SUPPORT OF THE HARVARD-ABBVIE RESEARCH COLLAB	DAVEY ROBERT	Harvard College (AbbVie)	03/01/21 - 02/28/24	151,138
ANTIVIRAL LEAD IDENTIFICATION TO TREAT FILOVIRUS INFECTIONS	DAVEY ROBERT	Purdue University (NIH/NIAID)	08/12/19 - 07/31/23	136,394
MODELING FILOVIRUS INFECTION OF AND TRAFFICKING THROUGH SKIN	DAVEY ROBERT	The Univ of Iowa (NIH/NIAID)	08/01/18 - 07/31/22	111,807
STTR PHASE I: AMINOMETHYL BENZAMIDES AS NOVEL ANTI EBOLA AGENTS	DAVEY ROBERT	ChicagoBio Solutions (NIH/NIAID)	08/16/19 - 07/31/22	107,114
EVALUATION OF SARS-COV-2 NUCLEASE CONJUGATES FOR TREATMENT IN THE RODENT MODEL OF SARS-COV-2 DISEASE	DAVEY ROBERT	MIT	07/01/21 - 12/31/21	11,365
SMALL MOLECULE INHIBITORS OF EBOLA VIRUS POLYMERASE FUNCTION	DAVEY ROBERT	Icahn School at Mt Sinai (NIH/NIAID)	02/01/22 - 01/31/23	194,935

AWARD TITLE	PI / CO-PI	SPONSOR (PRIME SPONSOR)	PROJECT START DATE	PROJECT END DATE	ADDL FUNDS FY-22
EVALUATION OF MRNA-BASED VACCINE APPROACHES AGAINST ENDEMIC CORONAVIRUSES	DOUAM FLORIAN	Moderna Therapeutics	12/07/21	05/31/22	297,066
CHARACTERIZATION OF A HUMAN-SPECIFIC POSITIVE REGULATOR OF FLAVIVIRUS INFECTION	DOUAM FLORIAN	NIH/NIAID	04/12/21	03/31/23	106,438
THERAPEUTIC EFFECTS OF NAPC2 IN SARS-COV-2 INFECTION OF K18-HACE2 TRANSGENIC MICE	BOSMANN / DOUAM	ARCA Biopharma	03/15/21	12/31/21	49,170
DEFINING THE IMPACT OF PER/POLYFLUOROALKYL SUBSTANCE EXPOSURE ON SUSCEPTIBILITY TO SARS-COV-2 INFECTION AND DISEASE	SCHLEZINGER / DOUAM	NIH/NIES	03/02/21	02/28/23	201,700
DEVELOPING COMBINATION THERAPIES AGAINST PNEUMO- AND PARAMYXOVIRUSES CAUSING SEVERE RESPIRATORY INFECTION	FEARNS RACHEL	Georgia State Univ (NIH/NIAID)	07/01/18	06/30/22	16,500
STUDIES TO ASSESS THE MECHANISM OF ACTION OF AN L INHIBITOR	FEARNS RACHEL	Merck, Sharp & Dohme	02/15/22	08/14/22	36,682
MECHANISMS OF MARBURG VIRUS GENE EXPRESSION	FEARNS / MUHLBERGER	NIH/NIAID	05/08/18	04/30/23	529,563
PILOT STUDY TO ASSESS PROTECTION AGAINST FILOVIRUS CHALLENGE BY AN INFUSED MONOCLONAL ANTIBODY	GRIFFITHS ANTHONY	Leidos (NIH/NCI)	10/31/19	09/23/23	1,676,229
RATIONALLY DESIGNED PAN-EBOLAVIRUS VACCINE	GRIFFITHS ANTHONY	Integrated BioTherapeutics (NIH/NIAID)	12/01/19	05/31/22	243,450
IMMUNOGENICITY AND EFFICACY TESTING OF MEDICAL COUNTERMEASURES (VACCINES AND OTHER BIOLOGICS) AGAINST BSL-4 PATHOGENS IN NHPS	GRIFFITHS ANTHONY	Battelle Memorial (NIH/NIAID)	06/23/20	02/28/22	1,160
EVALUATION OF PROTECTIVE EFFECT AGAINST SARS-COV-2 CHALLENGE OF AAVCOVID19-1 (AC1) AND AAVCOVID19-3 (AC3) VACCINES	GRIFFITHS ANTHONY	Mass Eye & Ear (Albamunity)	07/01/20	03/30/21	81,662
DISCOVERY AND DEVELOPMENT OF ANTIBODY THERAPEUTICS FOR SARS-COV-2 AND OTHER HIGH-RISK EMERGING VIRUSES	GRIFFITHS ANTHONY	Harvard College (AbbVie)	10/30/20	10/29/23	485,192
ACCELERATED DEVELOPMENT OF A SARS-COV-2 VACCINE BASED ON THE LIVE VSVG CHIMERIC VIRUS PLATFORM	GRIFFITHS ANTHONY	IAVI (DOD/DTRA)	02/26/21	10/04/22	1,065,498
MODEL DEVELOPMENT/ REFINEMENT STUDY EBOLA VIRUS MAKONA INTRANASAL CHALLENGE	GRIFFITHS ANTHONY	Janssen Vaccines	04/16/21	04/16/23	383,240
EFFICACY OF UMASS TEST ARTICLE IN HAMSTER MODEL OF SARS-COV-2	GRIFFITHS ANTHONY	UMass Worcester	05/01/21	12/31/21	19,608
CELLULAR NANOSPONGES AS A MEDICAL COUNTERMEASURE TO EMERGING VIRUS THREATS	GRIFFITHS ANTHONY	UCSD (DOD/DTRA)	05/13/21	05/12/26	372,832

AWARD TITLE	PI / CO-PI	SPONSOR (PRIME SPONSOR)	PROJECT START DATE - PROJECT END DATE	ADDL FUNDS FY-22
STIMULATING HOST INNATE IMMUNITY TO PROTECT AGAINST EBOLA VIRUS	GRIFFITHS ANTHONY	Bolder Biotech (NIH/NIAID)	07/02/21 - 06/30/22	78,052
VACCINES FOR PREVENTION OF RG3 AND RG4 EMERGING TICKBORNE VIRAL DISEASES	GRIFFITHS ANTHONY	UConn (NIH/NIAID)	08/20/21 - 07/31/26	124,383
MIT FIUNDED PROJECT OF SARS-COV-2 INFECTION OF VACCINATED MICE	GRIFFITHS ANTHONY	MIT	09/01/21 - 12/31/21	13,310
DISCOVERING DURABLE PAN-CORONAVIRUS IMMUNITY	GRIFFITHS ANTHONY	BWHI (NIH/NIAID)	09/16/21 - 08/31/24	481,635
SEROLOGIC AND MOLECULAR STUDIES OF HUMAN ANTI-HCOV ANTIBODY CROSS-IMMUNITY AND PROTECTIVE RESPONSES AMONG ENDEMIC HCOVS AND SARS-COV2	GRIFFITHS ANTHONY	DFCI (NIH/NIAID)	09/17/21 - 08/31/24	71,719
IN VIVO STUDIES OF SARS-COV-2 VARIANTS	GRIFFITHS ANTHONY	Harvard College	10/15/21 - 09/30/22	218,500
DETERMINE THE NEUTRALIZATION POTENCY OF IMM20253 +/- A SERIES OF ANTIBODIES AGAINST THE CLINICAL ISOLATE OF THE OMICRON VARIANT VS THE WA1/2020 D614G REFERENCE SARS-COV-2 STRAIN	GRIFFITHS ANTHONY	Immunome, Inc.	01/15/22 - 06/30/22	182,656
NEUTRALIZATION OF SARS-COV-2 WITH RATIONALLY DESIGNED ANTIBODY-BASED THERAPEUTICS	GRIFFITHS ANTHONY	Cidara Therapeutics	02/15/22 - 07/30/22	96,063
MODEL DEVELOPMENT AND MEDICAL COUNTER MEASURES (MCMS) FOR BSL-4 AGENTS IN NHPS	GRIFFITHS ANTHONY	NIH/NIAID	03/16/22 - 03/15/23	1,956,086
SO1: INFRASTRUCTURE AND INSTALLATION OF CAGING TO SUPPORT CEPI HIGH CONTAINMENT ANIMAL STUDIES	GRIFFITHS ANTHONY	CEPI	04/01/22 - 11/30/22	618,660
SO2: PURCHASE AND HOUSING OF AFRICAN GREEN MONKEYS FOR USE IN CEPI HIGH CONTAINMENT ANIMAL STUDIES	GRIFFITHS ANTHONY	CEPI	04/01/22 - 08/31/23	1,980,082
ASSAYS IN SUPPORT OF FILOVIRUS NONHUMAN PRIMATE STUDIES FOR ADVANCEMENT OF MEDICAL COUNTERMEAS	GRIFFITHS ANTHONY	Battelle Memorial (HHS/ASPR/BARDA)	04/14/22 - 09/02/24	342,871
COMPANION DIAGNOSTICS AND ANTIVIRALS FOR COVID-19 USING CRISPR-CAS13	HONKO ANNA	Harvard College (Gates Foundation)	12/01/21 - 11/30/22	221,700
SIDEROPHER-DEPEDENT INHIBITORS OF MYCOBACTERIUM TUBERCULOSIS	KRAMNIK IGOR	UAB (NIH/NIAID)	06/22/20 - 05/31/22	104,211
NECROSIS IN PULMONARY TB GRANULOMAS: DYNAMICS, MECHANISMS, AND THERAPIES	KRAMNIK IGOR	NIH/NLBI	03/04/22 - 02/28/26	724,795
ANALYSIS OF ANTIBODY-DEPENDENT ENHANCED INFECTION BY SARS COV-2	MUHLBERGER ELKE	Regeneron (HHS/ASPR/BARDA)	07/01/21 - 07/01/22	30,012

AWARD TITLE	PI / CO-PI	SPONSOR (PRIME SPONSOR)	PROJECT START DATE	PROJECT END DATE	ADDL FUNDS FY-22
THWARTING INFLUENZA WITH RNA-POWERED MODULATORS (THIRM)	MUHLBERGER ELKE	Georgia Tech (DOD/DARPA)	03/01/22	03/31/23	27,920
ELUCIDATING THE IMMUNE RESPONSE OF SCHREIBER'S BATS TO LLOVIU VIRUS INFECTION IN VITRO AND IN VIVO	MUHLBERGER ELKE	NIH/NIAID	02/24/22	01/31/24	243,800
INVESTIGATING INTERFERON ANTAGONISTS IN DELAYING INNATE IMMUNE RESPONSES TO SARS-COV-2	SAEED MOHSAN	Loyola Univ, Chicago (NIH/NIAID)	07/09/21	06/30/26	206,250
DEFINING THE ROLE OF SITE-SPECIFIC PROTEOLYSIS IN INNATE DEFENSE SIGNALING	SAEED MOHSAN	NIH/NIGM	07/01/22	04/30/27	412,500
TOTAL NEIDL CORE FACULTY AWARDS					\$ 28,488,409

Table 2

AWARD TITLE	PI / CO-PI	SPONSOR	PROJECT START DATE	PROJECT END DATE	ADDL FUNDS FY-22
AUSTERE ENVIRONMENTS CONSORTIUM FOR ENHANCED SEPSIS OUTCOMES (ACESO) UGANDA	BHADELIA NAHID	Henry M. Jackson Foundation (DOD)	08/01/19	06/30/22	20,658
NATIONAL EBOLA TRAINING AND EDUCATION CENTER	BHADELIA NAHID	Emory University (HHS/Assist. Sec. for Preparedness)	06/30/21	12/31/21	85,959
MASSCPR PASC: AUTOIMMUNITY AND INFLAMMATORY RESEARCH WORKING GROUP	BHADELIA / CAPPIONE	Harvard College (China Evergrande)	03/01/22	02/28/23	103,790
BACTERIAL POLYPHOSPHATES IN SEPSIS	BOSMANN MARKUS	NIH/NIAID	02/22/21	01/31/25	537,125
NEW GENETIC MODELS FOR C5A RECEPTORS	BOSMANN MARKUS	NIH/NLBI	07/01/18	06/30/23	412,500
ROLE REVERSAL OF MAVS IN BACTERIAL SEPSIS	BOSMANN MARKUS	NIH/NLBI	08/15/18	06/30/23	482,381
BIOSENSOR ENGINEERING FROM METAGENOMIC SCREENING	GALAGAN JAMES	DOD/ONR	09/14/21	09/13/22	179,231
NOVEL BIOSENSORS BASED ON MINING BACTERIAL TRANSCRIPTION FACTORS	GALAGAN / KLAPPERICH	NIH/NIBIB	07/01/20	03/31/25	655,799
BIOSENSORS FOR PHYSIOLOGICAL MONITORING FROM BACTERIAL MINING	GALAGAN JAMES	DOD/ONR	05/01/21	04/30/24	177,000
GM3 NANOPARTICLES FOR SUSTAINED DELIVERY OF ANTI-RETRO VIRALS TO LYMPHATIC TISSUES	GUMMULURU/ REINHARD	NIH/NIAID	11/08/17	10/31/22	624,640
SYNERGISTIC MECHANISMS OF CHRONIC INNATE IMMUNE ACTIVATION IN MICROGLIA BY OPIATES AND HIV INFECTION	GUMMULURU/ CHENG	NIH/NIDA	07/15/20	04/30/25	689,563

AWARD TITLE	PI/CO-PI	SPONSOR (PRIME SPONSOR)	PROJECT START DATE	PROJECT END DATE	ADDL FUNDS FY-22
PERSISTENT HIV EXPRESSION INDUCED TYPE 1 IFN RESPONSES AND INFLAMMAGING	GUMMULURU RAHM	NIH/NIA	08/01/18	04/30/23	771,666
PERSISTENT HIV-1 EXPRESSION AND MICROGLIA DYSFUNCTION	GUMMULURU RAHM	BMC Corp (NIH/NIDA)	08/01/21	05/31/26	277,494
SINGLE CELL TRANSCRIPTOMICS OF THE OPIOID USE DISORDER AD HIV SYNDEMIC IN THE HUMAN BRAIN	GUMMULURU RAHM	J. Craig Venter Inst (NIH/NIDA)	11/05/20	08/31/25	140,023
PLASMONIC INACTIVATION OF VIRUS AND MYCOPLASMA CONTAMINANTS	GUMMULURU / REINHARD	NIH/NIGMS	08/01/21	05/31/25	330,000
FOGARTY GLOBAL HEALTH TRAINING FELLOWSHIP PROGRAM	HAMER DAVIDSON	Harvard SPH (NIH/FIC)	07/01/17	06/30/22	19,513
GEOSENTINEL THE GLOBAL SURVEILLANCE NETWORK OF ISTM AND CDC	HAMER DAVIDSON	ISTM (HSS/CDC)	09/01/21	08/30/26	450,043
STRUCTURE-FUNCTION ANALYSIS OF INFECTION- AND VACCINE-INDUCED B-CELL REPERTOIRES	KEPLER THOMAS	Children's Hospital (NIH/NIAID)	08/01/17	07/31/22	87,000
IMMUNE MECHANISMS OF PROTECTION AGAINST MYCOBACTERIUM TUBERCULOSIS CENTER (IMPAC-TB)	KEPLER THOMAS	Harvard College (NIH/NIAID)	09/30/19	06/30/22	5,239
THE INTERPLAY BETWEEN TRANSPOSONS AND PIRNA PATHWAYS	LAU NELSON	NIH/NIGMS	08/10/20	04/30/24	448,981
LUNG-RESIDENT ANTIBACTERIAL HETEROTYPIC IMMUNITY	MIZGERD JOSEPH	NIH/NIAID	07/01/19	06/30/24	956,343
PNEUMONIA BIOLOGY	MIZGERD JOSEPH	NIH/NLBI	01/11/17	12/31/23	838,786
PULMONARY PATHOPHYSIOLOGY SUB-PHENOTYPES OF PNEUMONIA	MIZGERD JOSEPH	NIH/NIAID	02/01/22	01/31/27	856,279
THE BIOCHEMISTRY AND CELL BIOLOGY OF THE SPINDLY O-FUCOSYLTRANSFERASE OF TOXOPLASMA	SAMUELSON JOHN	NIH/NIGMS	01/01/20	11/30/23	464,666
TOTAL AFFILIATED FACULTY AWARDS					\$ 9,614,679

Additional Support for Research from the NEIDL Director's Strategic Initiatives Fund

One way Boston University supports the NEIDL is through the NEIDL Director's Research Initiatives fund. These funds are designed to enhance the research activities in the NEIDL. As the NEIDL research programs have evolved and the existing instrumentation has aged, so the way the Director's Research Initiatives have evolved as well. Operational support of the BSL-2 and BSL-3, remains a large expense (almost \$80,000), which also involves maintaining the pathogen repository. As instrumentation has aged, and the need for replacement shared equipment has accelerated, around \$160,000 was committed to this. Shared equipment repairs and maintenance (\$40,000) and contracted services and common supplies (\$12,000) helped support BSL-2 work. Organoid and stem cell work remains a high priority, and almost \$30,000 was committed to this function. Other areas included support for the insectary (\$8,000) and Veterinary Pathology services (\$34,000), and support for the BSL-4 (\$7,000). Finally, support of CEID remains an important strategic objective of the NEIDL, and \$80,000 was committed for this purpose.

Faculty and Staff Recognition

Conference - Invited Speaker Engagements (National & International)

Markus Bosmann

- Polyphosphates – ‘The Forgotten Polymer’ as a New Modulator of Inflammation. Research Training Group 2336, ‘Resolution of Inflammation – Mediators, Signaling and Therapeutic Options’, funded by the German Research Foundation, Johann Wolfgang Goethe-University, Germany. November 2021

John Connor

- Contact Tracing Summit (6 University virtual conference) “*Relentless Outside Pressure Arrival and Spread of Variants of Concern through an Urban University*”. July 2021
- University Texas Health San Antonio, San Antonio TX “*Biomarkers of Outcome in High Consequence Viral Infection*”. August 2021
- Partners-In-Health Outbreaks Response Roundtable (COVID Academy) “*Test, Track, Trace: hybrid epidemiology at Boston University*” December 2021
- Massachusetts Consortium of Pathogen Readiness “*SARS-CoV-2 Genomic Analysis at Boston University*”. March 2022
- CDC Spheres meeting (300 participants) “*Test, trace, sequence: Disease transmission tracking in a university environment*”. May 2022
- Council of State and Territorial Epidemiologists (CSTE) special meeting (200 participants) “*Test, trace, sequence: Disease transmission tracking in a university environment*”. June 2022

Nick Crossland

- NCPL Annual Initiative Update. NEIDL. Invited speaker, March 2022
- Bridging the gap of Morphology and Molecular Biology through Advanced Tissue Based Technologies. Microbiology Retreat, BUSM. Invited speaker. April 2022.

Florian Douam

- Humanized mouse models to investigate immunological mechanisms defining effective control of SARS-CoV-2 infection in the human lung. Univ of North Carolina at Greensboro. October 2021
- Humanized mouse models to investigate immunological mechanisms defining effective control of SARS-CoV-2 infection in the human lung. ARC COVID-19 meeting. Boston University School of Medicine, October 2021.
- Humanized mouse models to investigate immunoregulations defining effective control of SARS-CoV-2 infection in the human lung. Nature/PerkinElmer Webinar. October 2021.
- It’s all about the journey: Setting up a virology lab in the 2020s. ACAV panel. American Society for

Tropical Medicine and Hygiene Annual Symposium. November 2021

Anthony Griffiths

- Advanced development of Ebola virus countermeasures yields insights into viral pathogenesis, University of Connecticut, Storrs, CT
- Advanced development of Ebola virus countermeasures yields insights into viral pathogenesis, Italian Society for Virology (Invited)

David Hamer

- “Careers in global health” presented to the National University of Ireland, Galway for Prof. Gerard Flaherty’s medical school class October 2021
- “Dengue epidemiology and clinical manifestations” presented in “Dengue in the times of COVID-19: Focus on Travelers”, ISTM January 2022
- “Bites, envenomation, and seafood poisoning” presented at ISTM Travel Medicine Review and Update Course February 2022
- “Adventure travel, altitude and diving” presented at the virtual ISTM Travel Medicine Review and Update Course, February 2022
- “Les arboviruses” presented with Dr. Alix Miauton in the tropical health course at the Université de Lausanne, Lausanne, Suisse. March 2022
- “High and cold – prevention and management of acute mountain sickness and frostbite” presented during the annual general meeting of the Society of Travel Medicine of Ireland, April 2022
- “Evolving epidemiology of arboviruses”, Formation Continue for Médecine Tropicale et de Médecine des Voyages for the Swiss Society of Tropical and Travel Medicine in Bern, Switzerland, June 2022
- “Warmer climate, rising dengue risk” presented in symposium entitled “Mosquitoes are not impressed by face masks” at the 8th Northern European Conference, June 2022
- “COVID pandemic, lessons learned” presented in the 7th Annual British Columbia Infectious Disease Symposium, Vancouver, BC, Canada, June 2022

Adam Hume

- “Nuclear responses to SARS-CoV-2 infection”. Massachusetts Consortium on Pathogen Readiness TechTalk July 2021
- “Productive infection of pluripotent stem cell derived intestinal organoids with SARS-CoV-2 generates an epithelial intrinsic inflammatory response”. American Society for Virology Annual Meeting. July 2021
- Pathogens and Immune Response” Biotechnology in the World of Medicine, BioPharmaceutical Technology Center Institute, Madison, WI March 2022
- “Generation of a reverse genetic system and recombinant virus to study Lloviu virus, a bat-borne European filovirus” Viruses 2022 - At the Leading Edge of Virology Research April 2022

- “Generation and use of recombinant Lloviu virus as a tool to study the bat-borne European filovirus”. American Society for Virology Annual Meeting, Madison, WI July 2022

Igor Kramnik

- “Mechanisms of pulmonary TB progression” University of Arkansas Department of Microbiology and Immunology invited seminar speaker. December 2021
- “Post-primary pulmonary TB: a model and mechanisms” The Many Hosts of Mycobacteria conference, Columbus, Ohio. July 29/22

Nelson Lau

- Transposable element landscape changes are buffered by RNA silencing in aging *Drosophila*.”, TRANSPOSABLE ELEMENTS AT THE CROSSROADS OF EVOLUTION, HEALTH AND DISEASE Keystone Symposia 2022, Whistler, Canada [*Conference Presentation Abstract Selected but Conference Postponed from COVID19 Omicron Surge*] February 2022
- Genomics of piRNAs and small RNAs in mosquito and man: harbingers of persistent viruses and transposon activation.” MBO Workshop: Piwi proteins and piRNAs. April 2022

Jay Mizgerd

- Webinar expert panelist for society journal club (Section on Genetics and Genomics, ATS)
- Distinguished Scientist Seminar Series, Emory University (Immunology & Molecular Pathogenesis PhD program)
- Expert panelist for society journal club (Allergy, Immunology & Inflammation Assembly, ATS)
- Novel Vaccine Approaches for Pulmonary Infection Symposium, International Conference of the ATS
- New England Immunology Conference (Woods Hole, MA)
- Keynote Address, Celebration of Professor Joseph D. Brain, Harvard Chan School of Public Health (Dept of Environmental Health)
- University of Toronto, Canada (Grand Rounds, Critical Care Medicine)
- Webinar journal club, senior author for paper discussed (Allergy, Immunology & Inflammation Assembly, ATS)
- Immune Response in Respiratory Infection, International Conference of the Japanese Respiratory Society, Kyoto, Japan, April 22-24, 2022
- University of Florida, Gainesville, FL (Pulmonary, Critical Care and Sleep Medicine)
- Symposium on The Aftermath of Lung Infection, International Conference of the ATS, San Francisco, CA, May 13-18, 2022
- Webinar expert panelist for society journal club (cosponsored by Allergy, Immunology & Inflammation Assembly and Respiratory Cell and Molecular Biology Assembly, ATS 2022)
- Gordon Research Conference, Biology of Acute Respiratory Infection. Ventura, CA

- One Pathology: The Model Organism Approach to Disease Research, American College of Veterinary Pathologists Annual Meeting . Boston, MA

Elke Mühlberger

- Department of Infectious Diseases, University of Georgia, Athens, GA. “iPSC-derived cells and organoids to study SARS-CoV-2 and Ebola virus infections”
- ASV 2021. Annual Meeting of the American Society of Virology (convener, filovirus session)
- Gordon Conference on Fluids in Disease Transmission and Contamination, Holyoke, MA “Frontier challenges in the study of the mechanisms of transmission of highly pathogenic viruses (invited speaker August 2022)
- International ITU Molecular Biology and Genetics Student Congress’21, Istanbul Technical University. “iPSC-derived cells and organoids to study Ebola virus and SARS-CoV-2 infections”. October 2021

Conferences – Chairs and Meeting Organizers

Ronald Corley

- Co-Chair, NBL-RBL Annual Meeting, Boston, MA March 2022

David Hamer

- Co-Chair (with Monica Lu), Scientific Session 122: Pneumonia, Respiratory Infections and Tuberculosis for the 70th annual meeting of ASTMH November 2021

Jay Mizgerd

- Session Chair “Beyond Alum and Capsular Polysaccharides: Novel Vaccine Approaches for Pulmonary Infection,” International Conference of the ATS. virtual symposium, Fall 2021
- Session Chair “The Aftermath of Lung Infection: Recovered Lungs Are Different Lungs,” International Conference of the ATS 2022
- Session Chair “What’s New in Pneumonia?” Mini-Symposium, International Conference of the ATS
- Chair - “Lung Immunity and Respiratory Infection” Postgraduate Course, International Conference of the ATS (Co-Chair w/H. Koziel 2022)

Mohsan Saeed

- Co-chaired session “Animal models, organoids, and replication systems” at the International Symposium on HCV and related viruses, July 2021. (co-chair: Alex Ploss, Princeton Univ.)
- Keynote speaker at the California Northstate University Research Symposium. 2021

Committee activities

Robert Davey

- NIH NIAID Board of Scientific Counselors. Review of Rocky Mountain Laboratories. Ad hoc member

Anthony Griffiths

- Advisory Board Member, WHO Marburg Virus Advisory Panel
- MARVAC World Health Organization advisory group for the development of Marburg Virus vaccines

David Hamer

- Chair, Scientific Program Committee, CISTM 18, 2022

Elke Mühlberger

- Member, Board of Scientific Counselors, NIH Vaccine Research Center April 2022-June 2026
- Member, Medical Research Council (MRC), UK, March-April 2022

Editorial Boards

Markus Bosmann

- Associate Editor, Frontiers in Immunology, section inflammation, 2022

Anthony Griffiths

- Board of Reviewers, Journal of Gen Virology, 2022

Adam Hume

- Review board member, Microorganisms 2021-22

Honors

Mark Bosmann

- Recognized as an Expertscape Expert in Sepsis. Markus Bosmann placed in top 1% of scholars writing about Sepsis over the past 10 years, 2021

David Hamer

- Boston Medical Center Be Exceptional Award to the Infectious Diseases COVID-19 team, 2021
- Honorary Member, Society of Travel Medicine of Ireland, 2022

Joseph Mizgerd

- Scientific Achievements Award, AII, ATS 2022
- Pneumonia Advisory Committee, PI-TB,ATS 2022

Mohsan Saeed

- 2022 Recipient of the NIH Maximizing Investigators' Research Award (MIRA)

Memberships

David Hamer

- Member, Nominating Committee, ASTMH, 2022

Adam Hume

- Member, International Committee on Taxonomy of Viruses (ICTV) Filoviridae Study Group 2021-2022

NIH Study Sections

Marcus Bosmann

- Special Emphasis Panel (SEP ZRG1CB-L(55)R), R35 ESI MIRA applications, PAR-20-117, 2022

John Connor

- Reviewer NIAID review panel IHD, 2021
- Reviewer NIAID ZRG1 AIDC-V, 2022
- Co-Chair NIAID F30/F31 IIHD review panel, 2022
- Panel member, Peer Reviewed Medical Research Program of the Department of Defense, 2022

Florian Douam

- NIH SuRE award R16 - study section review panel, member. November 2021

Anthony Griffiths

- Vaccines Against Microbial Diseases, October 2021

Igor Kramnik

- ZRG1 F07A A20 study section, April 2022

Nelson Lau

- ZRG1 Special Emphasis Panel Review of F30/F31/F32 NRSA Fellowship Grants, 2022

Joseph Mizgerd

- IHD Study Section
- Board of Scientific Counselors (Site Visit), NHLBI Division of Intramural Research
- Investigator Initiated Program Project Applications (P01), NIAID
- Investigator Initiated Program Project Applications (P01), NHLBI
- Host Interactions with Bacterial Pathogens (HIBP) Study Section

Mohsan Saeed

- NIH emergency awards: Antiviral Drug Discovery (AViDD) Centers for Pathogens of Pandemic Concern (2022)

Patents

John Connor

- US 17/431,584 Detection of Fibrin Formation or Removable at the Nano-Scale. Patent Date: 8/21/21 pending
- US 17/469,827 Polyamine Transport Inhibitors as Antivirals. Patent Date: 9/8/21 pending
- PCT/US21/42008 Viral Detection Systems and Uses Thereof. Patent Date: 7/21/21 pending

Florian Douam

- Jacob Schneiderman, Florian Douam. Novel Therapy for Acute Damage to Lung Tissue. 2021. US application 17235968

Review Panels

Markus Bosmann

- Reviewer, Division Biology and Medicine, Swiss National Science Foundation, Switzerland 2022.

John Connor

- Reviewer French National Research Agency (ANR) grant proposal, 2021
- Reviewer (invited) European Research Council (ERC) 2022
- Review panel member, Rapid Response grant call Canada (CIHR/IDRC) 2022

Ronald Corley

- Chair, Review Panel for Canada Foundation for Innovation Bioscience Research Infrastructure Fund. May, June 2022

Florian Douam

- Honk Kong Research Grant council, General research fund. Reviewer. March 2022.
- Guest editing: “Latest research in flavivirus vaccines” Vaccines, 2022

Faculty Promotions

Associate Research Professor

Adam Hume, PhD

During his 12 year tenure at BU, Adam has developed into one of our most passionate virologists focused on improving our understanding of replication and pathogenesis of the highly pathogenic viruses filoviruses. One focus of Dr. Hume’s research has been the use of filoviral reverse genetics systems, including minigenomes and virus rescue systems for Ebola, Reston, and Marburg viruses. Minigenomes allow for the safe study of viral genome replication and transcription at BSL-2 as well as facilitate antiviral drug screening.

Dr. Hume leveraged his extensive experience with filoviral minigenome systems to study a lesser-known emerging filovirus called Lloviu. Using Lloviu virus minigenomes, Dr. Hume has attempted to compare its pathogenicity to that of Ebola and Reston Viruses and its zoonotic potential to cause disease in humans. Another of Adam’s particular research interests is studying bat-borne zoonotic viruses and comparing the immune responses of human cells and cells from the bat reservoir species, like the Egyptian fruit bats, the host species of Marburg, and the Schreiber’s bats, the host species of Lloviu. To study these processes, Dr. Hume has developed many tools, including identifying cross-reacting antibodies and RT-PCR primers, that work with these relatively unstudied species, allowing us to gain our first insights into how these bats respond to these viruses.

Dr. Hume demonstrated a strong commitment to establishing BSL-4 research at the NEIDL. As a member of the Mühlberger laboratory, he played a critical role in launching BSL-4 work. He was integrally involved in establishing scientific SOPs for BSL-4 and has become one of our most experienced BSL-4 laboratory trainers and mentors.

Dr. Hume’s work has been published in prestigious journals, reported on by multiple news sources, and presented at national and international scientific meetings. Dr. Hume has a strong publication record, particularly after the NEIDL BSL-4 laboratories became active in August 2018. Since his promotion to Senior Research Scientist in March 2019, he has coauthored 8 publications. Some of the most notable reflected his recent work on SARS-CoV-2 with collaborators in the Center of Regenerative Medicine, published in *Cell Stem Cell* and *Molecular Cell*. Other papers have been published or are in review.

Dr. Hume has been invited and presented his work at nationally and international events from the beginning of his postdoctoral career at BU. Notably, he presented his work on the Lloviu Virus Minigenome Systems at the Keystone Symposia: Framing the Response to Emerging Virus Infections, which took place in Hong Kong. More recently, he presented his work on SARS-CoV-2 at Cold Spring Harbor Laboratories.

Education



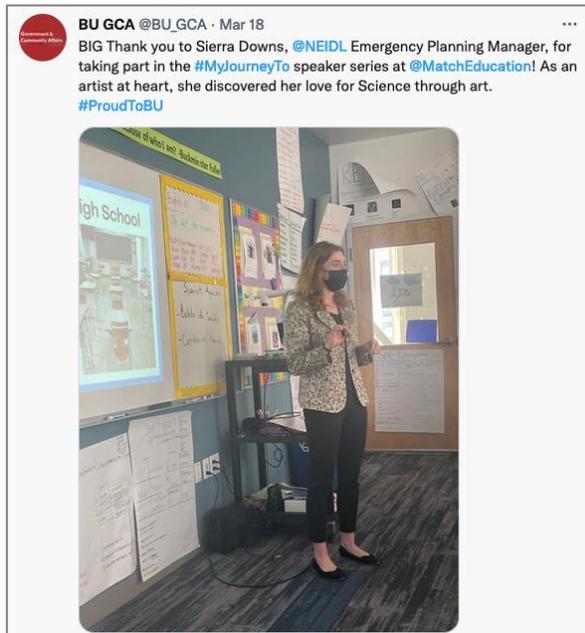
In partnership with the Department of Microbiology and the GMS Immunology Training Program, the NEIDL co-sponsors the Microbial Pathogenesis and Immunology Seminar Series. Below is a list of virology guest speakers who presented at the NEIDL.

Date	Speaker	Title	Program & Host
October 20, 2021	David Martinez, PhD University of North Carolina at Chapel Hill	Immunologic approaches to preventing and treating Sarbecovirus infections	Microbiology Lee Wetzler
November 3, 2021	Craig Cameron, PhD University of North Carolina at Chapel Hill	Antiviral therapy: towards the personal and the precise	Microbiology Andy Henderson
November 10, 2021	Sebla Kutluay, PhD Washington University	The unexpected role of HIV-1 integrase in particle maturation	Microbiology Rahm Gummuru
December 1, 2021	Shawn Lyons, PhD Boston University	The RNA biology of stress response	Microbiology Rachel Fearn
January 19, 2022	Faculty candidate: Shailabh Kumar, PhD Stanford University	Nanointerfaces for molecules to mosquitoes: mapping vector-pathogen dynamics at the planetary scale	BME-Micro/NEIDL joint recruitment campaign John Connor
January 26, 2022	Student-invited speaker: Faith Osier, PhD Imperial College of London	Fc-dependent IgG parasite clearance as a guide for vaccine development against <i>P. falciparum</i> malaria	Microbiology Josiane Fofana
February 1, 2022	Faculty candidate: Adriana Tomic, PhD University of Oxford, UK	Simon says: revealing immunity to influenza using systems immunology	BME-Micro/NEIDL joint recruitment campaign John Connor
February 8, 2022	Faculty candidate: Yvon L. Woappi, PhD Harvard Medical School	Learning how to regenerate tissue from DNA viruses	BME-Micro/NEIDL joint recruitment campaign John Connor
February 9, 2022	Faculty candidate: Connie Chang, PhD Montana State University	Single-cell infection studies of Influenza virus using droplet microfluidics	BME-Micro/NEIDL joint recruitment campaign John Connor
February 16, 2022	Oliver Fregoso, PhD University of California Los Angeles	Understanding the host DNA damage response in HIV replication and cure	Microbiology Rahm Gummuru
February 17, 2022	Faculty candidate: Abraam Yakoub, PhD University of North Dakota	Virus and virus engineering in disease pathogenesis and therapy	BME-Micro/NEIDL joint recruitment campaign John Connor
February 23, 2022	Faculty candidate: Jeffrey Grabowski, PhD NIH/NIAID	Virus interactions with ticks: tick cultures and translation towards countermeasure development	BME-Micro/NEIDL joint recruitment campaign John Connor
March 30, 2022	Claudia Jakubzick, PhD Dartmouth Geisel School of Medicine	Natural antibodies are the keys that start the engine to T cell-mediated antitumor immunity... and pulmonary macrophages	Immunology Training Prog Jay Mizgerd
April 6, 2022	Alexander Ploss, PhD Princeton University	Looking under the hood of a killer virus: new insights into the molecular biology of Hepatitis B virus	NEIDL Florian Douam
April 13, 2022	Jie Sun, PhD University of Virginia	Respiratory viral infection: from acute morbidity to chronic sequelae	Immunology Training Prog Jay Mizgerd
April 20, 2022	Margaret Scull, PhD University of Maryland	Mucin-mediated defense against Influenza virus infection	NEIDL Mohsan Saeed
April 27, 2022	Student-invited speaker: Emmie de Wit, PhD NIH/NIAID	A rapid response to the emergence of SARS-CoV-2: making past experiences count	Microbiology Callie Donahue

Community Outreach

As research activity increased at the NEIDL, so has our commitment to our role as a community resource for information and education. This has led us to try different, and creative methods to introduce community members of all ages to the exciting world of science and research. As outlined in more detail below, NEIDL researchers and staff actively participated in community engagement activities to share their knowledge and expertise.

This past spring Dr. Corley hosted the Spring, the 12th Annual NBL/RBL Network Meeting in Boston and opened the facility for in-person guided tours. This in person event, well attended by over 100 network members, was a welcome respite from the pandemic blues.



We have an ongoing relationship to bring our scientists and staff to the Match High School in Brookline through our Speaker Series. The “My Journey To...” Speaker Series is a career exploration program focused on exposing students to careers they may not be familiar with. Featuring various BU professionals including the NEIDL, the Speaker Series allows for students to engage with experts around college preparedness, job skills, and their personal roadway to their respective careers. The goal is to show students that no two paths are the same, and the road takes hard work, relationship building, and sometimes a bit of luck.

As BU opened its doors for the summer session, the NEIDL had its first in-person tour with high school students since the pandemic started. Our female professionals hosted a panel with the **Greater Research Opportunities for Girls (GROW)**, Followed by a tour of the Simulation lab, the suit room and the Animal Science Center. The NEIDL thanks Aditi Broos-Caldwell, Judith Olejnik, Fabiana Feitosa-Suntheimer, Colleen Thurman and Jacqueline Turcinovic for contributing to this program and inspiring the next generation of women scientists.

Another group to visit the NEIDL was BU SPH-PopHealthExperience 2022. This year, students came prepared with insightful questions. Not only did we discuss careers, but also climate change and government policy. According to Francisco Patino, Assistant Director of Lifelong Learning at Boston University School of Public Health, the NEIDL continues to be a favorite piece of the BU SPH-PopHealthExperience program. NEIDL contributors to this year’s PopHealthExperience program were Anna Tseng, Nafisah Nakhid, and Judy Yen.

For the final NEIDL tour of the Summer, we tried a new format. The Summer Training as Research Scholars (STaRS) cohort of students at the BU School of Medicine, were placed in small groups and researchers moved from group to group, like “Research Speed Dating”. This arrangement allowed students to be more comfortable asking questions in a smaller group. Researchers got to interact directly with students and each student got to ask questions of each researcher. The feedback we received from the cohort was that the tour was very insightful and informative. The NEIDL Research Speed Daters were: Anthony Griffiths, Anna Tseng, Fabiana Feitosa-Suntheimer, Yulianela Diaz Perez, Mohamed Ouizougoun-Oubari and Jacquelyn Turcinovic.



Our Faculty in the News

COVID-19

BU's Nahid Bhadelia Joins White House COVID-19 Response Team

Bhadelia, an infectious diseases physician and global leader in pandemic preparedness, named senior policy advisor for global COVID response

Original article from The Brink by Andrew Thurston. June 13, 2022

Nahid Bhadelia, an infectious diseases physician and founder of Boston University's Center for Emerging Infectious Diseases Policy & Research (CEID), has joined the White House COVID-19 Response Team as senior policy advisor for global COVID response. The team's founding goal was to create a unified national response to the pandemic.



Nahid Bhadelia, an infectious diseases physician and founder of Boston University's Center for Emerging Infectious Diseases Policy & Research (CEID), has joined the White House COVID-19 Response Team as senior policy advisor for global COVID response. The team's founding goal was to create a unified national response to the pandemic.

A BU School of Medicine associate professor of infectious diseases, Bhadelia is taking a sabbatical for the duration of the full-time position; David Hamer, a BU School of Public Health professor of global health, will serve as interim head of CEID.

"Dr. Bhadelia is widely known as an international expert and leader in highly communicable and emerging infectious diseases," says Gloria Waters, BU vice president and associate provost for research. Bhadelia is also an associate director of BU's National Emerging Infectious Diseases Laboratories (NEIDL) and helped launch and develop the Special Pathogens Unit at Boston Medical Center, BU's primary teaching hospital.

"She has extensive clinical, field, academic, and policy experience in pandemic preparedness," says Waters. "Her background in health and

human security, international affairs, and her training in infectious disease, coupled with her extensive experience in health system response to emerging infectious diseases—such as Ebola, Zika, and more recently, COVID-19—have provided her with invaluable insights into the underlying challenges to pandemic preparedness."

Bhadelia brings a broad global health background to the White House position. She worked in West and East Africa during multiple Ebola virus outbreaks, contributed to pandemic preparedness in Liberia and Uganda, and codirects the BU and University of Liberia Emerging and Epidemic Virus Research Program, which is funded by the Fogarty International Center. She's also a member of the World Health Organization's Technical Advisory Group for Universal Health and Preparedness Review, which is working to improve the metrics used to measure pandemic preparedness.

Since founding CEID in 2021, Bhadelia has grown it into a hub for research and actionable policies—the center has contributed to congressional hearings, advised legislative offices, prepared policy briefs, and provided input on pandemic preparedness bills.

That work will continue under Hamer's direction. An infectious diseases specialist, Hamer says Bhadelia has been a leader not just for CEID, but for the rest of the nation during the COVID-19 pandemic.

"Since the beginning of the SARS-CoV-2 pandemic, Nahid has been a dynamic speaker, helping to effectively communicate about all aspects of the pandemic—from epidemiology to vaccination—to colleagues at Boston University and Boston Medical Center, as well as the broader US population," he says. "Her insight into the disease, strategies for prevention, including vaccination, and input on policies to mitigate the adverse public health consequences of COVID-19 in the United States—as well as low- and middle-income countries worldwide—have resulted in national and international recognition of her expertise as an emerging infectious disease specialist."

Draft bill would ban CDC, NIH from funding lab research in China

House of Representatives measure catalyzed in part by suspicion that pandemic virus escaped from Wuhan laboratory

Original article from Science by Jocelyn Kaiser. June 12, 2022

A proposal moving through Congress to bar the National Institutes of Health (NIH) and the Centers for Disease Control and Prevention (CDC) from funding research laboratories in China is sparking concern among scientists. If signed into law, the measure could cut off millions of dollars of U.S. funds flowing to collaborative research projects in several areas, including HIV/AIDS, cancer, mental health, and flu surveillance.

The proposed ban, part of a 2023 spending bill approved by the U.S. House of Representatives Committee on Appropriations on 30 June, grew out of suspicions among some lawmakers, so far unsupported by evidence, that the



The Wuhan Institute of Virology in China. A bill moving through Congress would bar U.S. biomedical science agencies from funding researchers at the institute and all other laboratories in China. HECTOR RETAMAL/AFP VIA GETTY IMAGES

Wuhan Institute of Virology (WIV) in China released the coronavirus that started the current pandemic, as well as objections to other potentially risky biomedical experiments involving animals. Specifically, the measure would bar the Department of Health and Human Services (the parent agency of NIH and CDC) from funding WIV or “any other laboratory” in China, Russia, or any country the U.S. government has designated a foreign adversary, a list that currently includes Iran and North Korea.

The measure’s sponsor, Representative Chris Stewart (R-UT), says the ban is aimed at ensuring the United States does not fund “dangerous research” in “uncontrolled environments” overseas.

Some scientific organizations are concerned by the proposal’s expansive scope. “It seems a bit

extreme,” says Eva Maciejewski, spokesperson for the Foundation for Biomedical Research, which advocates for animal research. “In theory it’s good to have oversight over biosafety and animal welfare, but in practice there may be better ways than blocking all NIH funding to foreign countries.”

The microbiology community is also troubled, says Mary Lee Watts, director of federal affairs for the American Society for Microbiology. “International collaboration is essential to allowing our scientists to ... understand disease threats wherever in the world they exist, in order to protect public health,” Watts says. An NIH spokesperson said the agency does not comment on pending legislation. But **Gerald Keusch** of Boston University, a former director of the NIH Fogarty International Center, believes “most of the senior leadership [of NIH’s 27 institutes] will be deeply concerned to have Congress interfering in the review and awarding of grants.” (It is unusual for lawmakers to adopt such countrywide bans on research funding.)

Backing the measure is the White Coat Waste Project, an animal rights group that 2 years ago publicized NIH’s funding of WIV. Justin Goodman, the group’s senior vice president, says that “taxpayers shouldn’t be forced to fund ... cruel, wasteful, and dangerous animal experiments in hostile countries ... where there’s no real transparency and accountability.” (The proposed measure, however, does not specifically mention animal studies.) The ban’s potential impact isn’t clear. WIV is largely funded by the Chinese government, and researchers there have received no U.S. funding since NIH, citing compliance issues, suspended a small subcontract for studying bat coronaviruses in July 2021. But NIH supports other research in China, with grants totaling \$8.9 million in 2021 and \$5.6 million this year, according to federal databases.

Projects that do not involve laboratory work—such as a long-running NIH-funded survey on health and retirement in China—could be spared. But many others would likely be vulnerable, including three projects headed by Chinese investigators studying influenza and the mosquito-borne diseases dengue and malaria, and dozens of subawards to Chinese groups participating in clinical trials of drugs, studies of the health effects of heavy metals, and neuroscience research. The U.S. leader of one clinical trial in Shanghai—who asked for anonymity—said his Chinese partner is a former trainee and “close collaborator,” and it would not be possible to recruit enough patients at a single site in the United States.

The ban would likely have a smaller impact on research in Russia. NIH and CDC appear to have just two active grants there, and they may already be subject to recent White House guidance winding down U.S. research funding to Russia because of its war against Ukraine. There are no NIH or CDC grants to researchers in Iran or North Korea.

To become law, the ban would need to survive negotiation of a final bill with the Senate. Some research groups hope lawmakers will remove the provision before any bill goes to President Joe Biden for final approval, likely late this year.

After the Infection Is Gone

MassCPR experts discuss the knowns and unknowns of long COVID

Original article from Harvard Medical School News by Ekaterina Pesheva. June 9, 2022

As the vast majority of the world's population continues to encounter SARS-CoV-2 virus and become infected, one question looms ever larger: What will be the long-term physiological repercussions of having had COVID?

Experts from the Harvard Medical School-led Massachusetts Consortium on Pathogen Readiness discuss the emerging science, latest knowledge, and critical unknowns of the novel syndrome known as long COVID.



Elena Fomina/iStock / Getty Images Plus

- **Nahid Bhadelia**, founding director, Boston University Center for Emerging Infectious Diseases Policy and Research; associate professor of infectious diseases, Boston University School of Medicine; visiting fellow, the White House Office of Science & Technology Policy; long COVID research group co-lead for MassCPR
- Bruce Levy, HMS Parker B. Francis Professor of Medicine and division chief of pulmonary and critical care medicine at Brigham and Women's Hospital; long COVID research group co-lead for MassCPR
- Linda Sprague Martinez, associate professor, Boston University School of Social Work; co-director, Community Engagement Program, BU Clinical & Translational Science Institute; long COVID research group health disparities lead for MassCPR
- Jake Lemieux, instructor in medicine, Harvard Medical School, infectious disease specialist at Mass General; viral variants program co-lead for MassCPR

Harvard Medicine News: Set the stage for us and give us the big-picture view on long COVID.

Bhadelia: First, it is important to keep in mind that aside from long COVID, there are many other post-acute infectious syndromes, so the notion that infections can cause long-term post-acute symptoms is not new.

Some of these syndromes develop after viral infections like Ebola, Epstein-Barr, polio, dengue, H1N1 flu, and others, while others develop after infection with a bacterial pathogen. We do not understand many of these syndromes, and their scope is not well defined. For many of these, our understanding is limited by research capacity and clinical infrastructure and our ability to capture good, quality data in order to understand the biological phenomena that underpin them.

Levy: Long COVID is such a novel entity that even its definitions vary. The World Health Organization and the Centers for Disease Control and Prevention define it slightly differently, but, broadly speaking, long COVID involves a constellation of symptoms that develop in the aftermath of acute infection—hence the term post-acute sequelae of SARS-CoV-2 infection, or PASC. The WHO says long COVID starts three months from the onset of COVID with symptoms, that these symptoms last at least two months and are not explained by alternative diagnoses. The CDC starts the clock earlier, at four weeks after original infection. Important to note, as of July 1, 2021, post-COVID conditions can be considered a disability under the Americans with Disabilities Act.

HMNews: What is the current profile of long COVID in terms of prevalence and symptoms?

Levy: Keeping in mind that our knowledge is evolving, and this is a moving target, here's what we know so far. Overall, the current CDC estimate is that one in five adults over the age of 18 may have a condition related to COVID, but the estimate is uncertain. According to the CDC, 13 percent of people diagnosed with COVID meet the definition of long COVID one month after infection, and this number drops to 2.5 percent three months after infection.

Among those hospitalized with COVID, more than 30 percent have symptoms suggestive of long COVID six months after infection. Hospitalization does appear to drive risk for long COVID. It appears the condition is more common in women, with at least 60 percent of long COVID diagnoses occurring among women, but this could also be a factor of who is more likely to seek care.

Fatigue is the most prevalent symptom, with two-thirds of patients reporting it as part of their symptom constellation. Yet, there is a very wide variety of symptoms associated with this condition—neurologic, neurocognitive, respiratory, sleep disturbances, mental health, cardiovascular, rheumatologic, digestive, and more. The long-term effect of long COVID remains to be seen over time. Long-term, will long COVID lead to more vascular illness, atherosclerosis, heart failure? These are all questions that remain to be answered.

HMNews: How much do we know about what drives the development of long COVID? What is known and unknown about its pathophysiology?

Bhadelia: One of the overarching questions in this unfolding mystery is whether these symptoms and conditions are truly caused by COVID itself or are being picked up more just because patients are coming to seek care who weren't before their SARS-CoV-2 diagnoses, and their preexisting conditions are coming to attention. It's very difficult to tease apart. If we don't know biological mechanisms are causing people to be sick, then it's hard to tease apart what's attributable to the virus itself versus other factors.

Another challenge in gleaning answers is the heterogenous nature of studies of long COVID, how differently they are designed, which makes it tricky to compare patient populations across studies. Another complicating factor is that there are many different phenotypes of long COVID, meaning that some people will develop more neurocognitive symptoms, while others more pulmonary symptoms. Underpinning these phenotypes are potential clues, none of which are slam dunk, but just different angles through which researchers are approaching figuring out the pathophysiology of long COVID.

Some of the hypotheses about what drives long COVID include the presence of persistent reservoirs of SARS-CoV-2 in the body—we've seen this in other infections: reactivation of other dormant viruses such as Epstein-Barr; the presence of predisposing conditions such as diabetes and obesity; the development of a maladaptive autoimmune response against the body's own tissues; chronic inflammation that fails to go away after the infection; blood vessel inflammation and damage; and direct organ injury from the initial severe disease.

In terms of who's at risk: So far, according to CDC data, it looks like the highest risk groups for developing long COVID include those who are not vaccinated, people with medical comorbidities, those with severe initial infections, those who developed multi-system inflammatory syndrome, either adult or pediatric. Ultimately, we want to know enough to create a uniform understanding of what long COVID is by taking away all the confounding factors. But it is important to note that regardless of the cause, there are many people who are suffering and need care, and we need to address that as a health care system.

HMNews: In addition to the science and the research that are so fundamental to understanding long COVID, what are some of the broader health care, policy, and societal implications of long COVID?

Sprague Martinez: Health inequities were pervasive pre-COVID and amplified with the pandemic. Some of the highest infection rates in Massachusetts occurred among immigrant communities, working class residents, and families of color.

As in COVID, health equity in long COVID is of paramount concern. People of color will likely be disproportionately impacted by long COVID. Long COVID has the potential to further widen existing gaps in health and getting ahead of it is critical.

The MassCPR long COVID research group includes a health equity core of investigators that span disciplines and are embedded at hospitals that conduct clinical trials as part of the NIH Recover Initiative. Our goal is to define the impact of long COVID on diverse communities in Massachusetts and to identify barriers to proper diagnosis and care of long COVID for those communities. We want to increase both awareness of and access to long COVID treatment both among those communities and the primary care providers serving them.

We are also interested in influencing relevant policies at the local, state, and national levels. Right now, we are focused on Black and Latinx communities, but plan to expand and engage other people of color and other marginalized groups in the state. Some of the key elements in advancing health equity will involve direct community engagement to increase diversity and representation in clinical trials that study long COVID and support of community health centers and community health workers. Other elements include engaging the primary care providers to ensure they are getting the latest research and engaging their patients in meaningful conversations that go beyond symptoms to understand the social factors impacting their health and referrals to job retraining programs for patients who cannot continue to do their work because of long COVID symptoms.

Also important will be policies related to eviction moratoriums, many of which have sunset, rental assistance, unemployment insurance, and more. Factors that lead and sustain inequities are caused by racialized policies that create inequitable living conditions. Tackling inequity will require an understanding of the structural determinants of health.

HMNews: Where are we on the treatment front for long COVID?

Lemieux: First a word on prevention and minimizing risk: Early data suggest that whatever we can do to limit the severity of acute disease will pay dividends on the other side in limiting the incidence of long COVID. That still needs to be explored further, and some therapies will be better than others. We have seen from just about every angle that vaccination limits the severity of disease, and since the risk of long COVID is linked to the severity of acute infection, we believe that vaccination will be partly, but not completely, protective.

Once a patient develops long COVID, that's a very different story. Right now, we are in a situation of having to treat and manage a complicated clinical syndrome that affects multiple systems in different ways. Every patient is different, every patient's symptoms will be different, and potentially the treatment of every patient will be different. That's hard on so many levels—it's hard to establish which treatments are best for which patients and, of course, it's hard for patients.

We are going to be in a period—an unenviable position, frankly—of having to feel our way, of having to treat patients symptomatically and having to conduct clinical trials and investigations to get at therapies that are safe and effective and reverse the mechanisms of disease to the extent that this is possible, while recognizing we don't yet know the mechanisms of disease for many manifestations of long COVID.

Sprague Martinez: That question of diagnosis and treatment is even more complicated among marginalized communities. For many people it's a privilege to take the day off from work and to seek care and treatment, especially in the context of employment instability which continues to impact industries such as service, which was very hard hit during the pandemic.

Then there's the question of awareness. Have people heard of Paxlovid? Are they aware it's out there? I can say that many people are not aware. How do we get the word out about it? Then there's testing. Access to at-home testing is not equitable. Many people may not even know they have COVID. We can't assume access to health information or care.

HMNews: Where does the pandemic stand in Massachusetts?

Lemieux: Things are getting better, but transmission of SARS-CoV-2 is still quite high if you look at both at the percentage of positivity and at the absolute case counts. The wastewater RNA levels are high. Although we are probably past the peak of the BA.2.12.1 wave, we are starting to see BA.4 and BA.5 variants starting to show up in a few percentage points of our sequenced cases here in the region. They are more transmissible and likely to cause some surge in cases. So, while we are dealing with the effects of post-acute COVID, we are also likely going to be seeing a lot of ongoing acute COVID into the summer, and there may be a resurgence in the fall.

MONKEYPOX

How Worried Should We Be about Monkeypox?

The country's history with smallpox vaccination might help, but it's not yet a cause for alarm, says NEIDL virologist John H. Connor

Original article from *The Brink* by Doug Most. May 25, 2022

As if COVID-19 is not enough to worry about, now there is monkeypox. In Boston, one patient hospitalized with the monkeypox virus came in contact with 200 people (most of them healthcare workers). And the World Health Organization (WHO) has identified more than 100 suspected and confirmed cases of the virus in an outbreak across Europe and North America. Like similar pox viruses, monkeypox is most often transmitted through close skin-to-skin contact, and can, less easily, be transmitted through respiratory droplets when a patient has lesions in the mouth. Monkeypox symptoms include a fever, headache, exhaustion, and a rash or lesions—about 1 in 10 who contract the disease die from it, according to the Centers for Disease Control and Prevention.

And on Monday even President Joe Biden weighed in, reassuring Americans already exhausted from the COVID-19 experience that the country was ready if it had to take on a new virus. Still, he advised people to be careful.

For some perspective on monkeypox and the level of concern it's causing, *The Brink* spoke with **John H. Connor**, a virologist at Boston University's National Emerging Infectious Diseases Laboratories (NEIDL) and a BU School of Medicine associate professor of microbiology.

Q&A WITH JOHN H. CONNOR



The Brink: What kind of virus is monkeypox?

Connor: An orthopoxvirus. It carries its genome as double-stranded DNA. It is similar to other known human pathogens like cowpox, and it's also similar to vaccinia, which is the vaccine that was used to eradicate smallpox circulation on this planet.

We've heard that the smallpox vaccine is effective in protecting people from monkeypox. But are most people vaccinated against smallpox?

So, the answer is that Americans—and people in many places around the world—used to automatically get vaccinated against smallpox. But then in 1980, the WHO declared smallpox had stopped circulating. And there was a key transition that occurred. There had not been a single case of it, maybe [one] in 1977, and very few cases before then. The effort to eradicate smallpox from the planet was successful. It was worth vaccinating people when there was a threat. Then when the threat completely disappeared, the benefit of being vaccinated against a virus that was not actively circulating was highly debatable. And so vaccination largely disappeared, except for individuals in specific circumstances. Part of the reasoning for why regular vaccination stopped is that the vaccine does have side effects. There was a population health effect of the vaccine. A small number of people, small percent, will have a very strong reaction to vaccination.

So, for me, born in the late 1960s, would I likely be vaccinated?

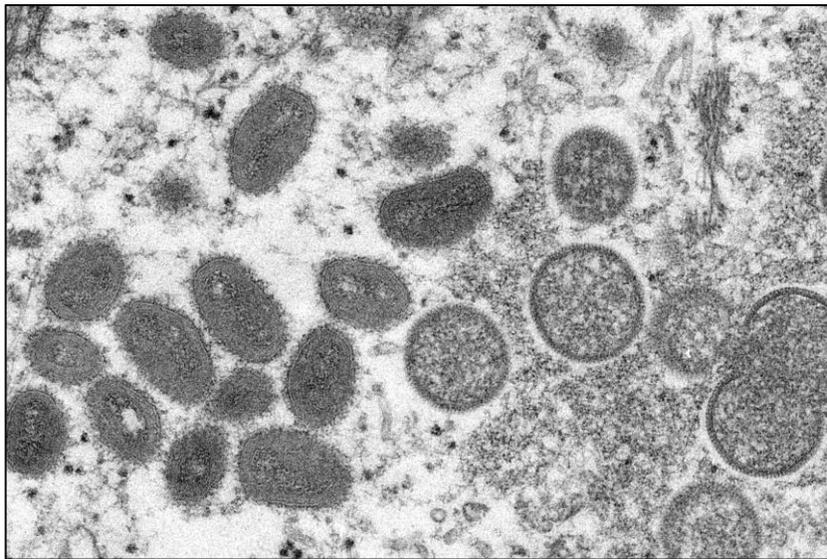
Do you have a scar on your shoulder?

I think that I do, yes. But what percentage of the world is vaccinated against smallpox?

I'm not positive, but I know that number is out there. [According to Pfizer, 80 percent of the world's population had been vaccinated by 1967. Since smallpox's eradication, vaccines have typically been held in stockpiles; the United States ended routine vaccination in 1972.]

OK, so knowing what we know about monkeypox, and knowing that not everybody today is vaccinated against smallpox, how concerned should we be as we read about this new virus in our midst?

From my standpoint, people should be aware. But I am not packing the car and heading for the hills. The basis for that is while monkeypox can be transmitted person to person, it is not something that has been described as having the real wildfire transmission we have seen with SARS-CoV-2. There has been an ongoing outbreak of monkeypox in Nigeria



An image of various monkeypox virus particles taken from human skin. According to BU virologist John H. Connor, monkeypox "is not something that has been described as having the really wildfire transmission we have seen with SARS-CoV-2." Courtesy CDC/Goldsmith. Photo via Smith Collection/Gado/Getty Images

since 2017. There are only 500 or 600 cases. Compare that, over five years, with SARS-CoV-2—we are a little over two years in and we are at how many hundred million cases? Of the two current infection threats, I think SARS-CoV-2 is the one that people should be concerned about right now.

For people who do get it, how serious is monkeypox?

It can be a very serious disease. But if it is caught early, and depending on which strain, it might not be. Right now, the current cluster of cases is from that milder strain. But there have been deaths reported from monkeypox infections.

Have NEIDL researchers been interested in pox viruses before this latest news?

We have been thinking about this since 2010, 2011. We are currently supported by the National Institutes of Health grant to research a small molecule that blocks poxviruses, including monkeypox. We are working with colleagues in the chemistry department to take an

interesting initial “hit” and make it more potent and more likely to be a successful broad-spectrum anti-poxviral treatment.

Knowing that the smallpox vaccination is effective against pox viruses like monkeypox, in your mind, is there any chance of smallpox vaccinations coming back?

It’s a good question. You may remember this was tried once before. After the anthrax attacks [in 2001], there was very real concern about weapons of mass destruction and biological agents being used as weapons of terror, and smallpox came up as something that would be really horrible. And I agree with that. The armed forces began a vaccination campaign. But I believe they stopped it because there were some severe adverse reactions. It came down to, if there is not a clear and present danger, is the cost of starting up again worth it? That answer could shift if monkeypox becomes an endemically transmitted event. But from my perspective, we’re not there yet.

From everything you are saying, you sound confident that monkeypox could be contained?

Yes. We have evidence of being prepared. Monkeypox has been imported into the United States recently: into Texas in July 2021, into Maryland in the fall of 2021. In both of these instances, importation did not then lead to lots of additional monkeypox infections. In the future, if there is evidence of community transmission, the United States has several options: postexposure treatment and vaccination. The country has prepared in ways that we weren’t prepared for COVID-19. We could react in a big way.

Checking in on the state of the pandemic, 2 years after Mass. emergency declaration

Original article from WBUR by Rupa Shenoy & Hafsa Quraishi. March 10, 2022

Thursday marks the two-year anniversary of when Gov. Charlie Baker declared Massachusetts under a state of emergency due to COVID-19.



At the time, there were 92 known or presumptive cases in the state. Baker eliminated non-essential travel by state workers, called for state employees to work remotely and urged private companies to do the same.

Now, two years later, communities across the state are loosening mask mandates, lifting capacity limits and easing restrictions.

WBUR’s *Morning Edition* host Rupa Shenoy checked in on the state of the pandemic with infectious disease specialist Dr. **David Hamer**, professor of global health and medicine at Boston University.

INTERVIEW HIGHLIGHTS

On the pandemic over the past few years

It’s been a roller coaster ride. Back when Gov. Baker made the announcement two years ago, we knew so little about this virus and about how it was spread. And I think over time, we’ve learned a lot of lessons about what can be done safely using prevention measures.

If you look at the numbers ... the total number of new cases, hospitalizations — everything has been going down very quickly over the last month. I think there still is some transmission of SARS-CoV-2 in the community, but it’s at much lower levels and the risk is gradually subsiding.

On the possibility of a case surge following loosening restrictions

I think that we’re in a good place right now. There’s a lot of both natural immunity from infection as well as vaccine-associated immunity. The only way we’re going to have a really major increase in cases, I think, is if a new variant arises.

On keeping ourselves safe and healthy

I think it really is going to be a new world ... individuals really need to balance the risk based on [both] their personal risk and also what’s going on in the community. And because all the community measures in Massachusetts look very good right now, I think that the risk is progressively subsiding.

I think we need to keep a careful eye on children. Certainly, if they have symptoms, they should be tested and isolated until their test result is back. Beyond that, if there's less virus overall being transmitted in the community, then the risk of children becoming infected and transmitting to other children or family members should be much lower.

"The only way we're going to have a really major increase in cases, I think, is if a new variant arises."

On what the COVID-19 pandemic might look like in a year

That's a tough question. The optimistic scenario is that we will not have any more waves and we'll just have periodic clusters, maybe because of transmission to groups that have not had the vaccine at all, or importation of the virus from outside of the country. That sort of transmission may become seasonal and so, we may be looking at late fall and winter, when we typically have influenza season and certain other respiratory viruses. I think we will be doing a lot more dual testing for influenza and SARS-CoV-2 at the same time until we have a better grasp of the epidemiology of the disease.

The worst case scenario is that we have another successor to omicron come along, and if it's different enough that there's not great protective immunity, which is essentially what happened with delta and omicron, then we could have another wave and we might have to restrict movements and bring back mask use in certain places and do enhanced testing and so forth. But I'm cautiously optimistic that's not going to happen.

This segment aired on March 10, 2022.

COVID-19

Here's what to do after being exposed to omicron

Dr. David Hamer, a professor of global health and medicine, explains why rapid tests are effective at indicating when someone is infectious.

Original article from Boston.com By Gwen Egan. January 24, 2022

In the wake of the omicron wave, the steps to take after being exposed to COVID-19 seem increasingly complicated. If someone gets exposed, do they test immediately? Or do they test after 5 days? Should they isolate for 5 days? Or 7? Or even 10?

Below is a timeline of what to do after being exposed to omicron and what should be done at each juncture with the help of Dr. **David Hamer**, a professor of global health and medicine at the Boston University School of Public Health and School of Medicine.

Right after being exposed:

First off, don't get tested right away.

"With omicron there's at least a suggestion that you become infected and symptomatic within two or three days," said Hamer. "If you have had no symptoms and no other exposure, testing the same day as an exposure is a waste of time."

3-5 days after exposure:

Get tested.

According to the most recent **Center for Disease Control guidelines**, if someone exhibits symptoms of COVID-19, they should get tested. Therefore, over the 3-5 days following exposure, that person should be monitoring for signs of symptoms.

What symptoms to look for within those 3-5 days:

The symptoms associated with omicron are slightly different than previous variants, aligning more with influenza or the common cold according to Hamer. Someone with **omicron can expect to experience symptoms** like: runny nose, headache, fatigue, sneezing, sore throat.

This is according to the Zoe COVID-19 Symptom Study.

What kind of test to get and why:

During Governor Baker’s **Jan. 11 press conference** concerning testing and exposure, he emphasized that rapid tests are a reliable way to get results.

This is because of something called viral load.

Hamer explained viral load as “the concentration of virus in some bodily fluid.”

Rapid tests are a strong option because they are quick, cheap, easy to find and use, and can usually tell someone that — not only are they positive — but also infectious.

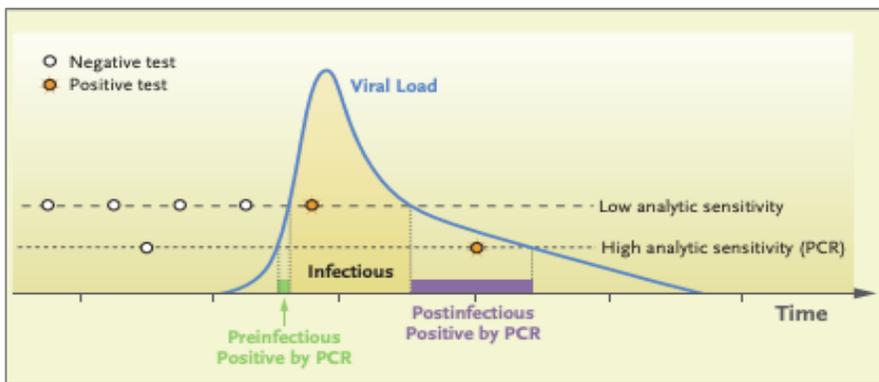
A PCR test will indicate a positive result if someone has “really tiny particles” of virus left in their system, according to Hamer. However, rapid tests will only show a positive result if there is a large amount of virus concentrated in the body, enough to make that individual clearly infected and infectious.

When someone is initially infected with COVID-19, said Hamer, they are likely to be infectious because there is a large amount of virus in the body. However, as that infected person starts to recover, their viral load quickly decreases.

“SARS-CoV-2 is a virus that grows quickly inside the body, so by the time a benchmark PCR test becomes positive, the virus is well into exponential growth,” read a study published in the **New England Journal of Medicine**. “At that point, it is probably hours, not days, before the virus grows by orders of magnitude, reaching the detection thresholds of currently available cheap and rapid point-of-care tests.”

For a more visual interpretation, the **New England Journal of Medicine** created a graph.

“[A rapid test] does tell you that you’re infected, but also, it has a pretty good association or sort of correlation with infectiousness,” Hamer said.



Graph from *Rethinking Covid-19 Test Sensitivity — A Strategy for Containment*.

After testing positive:

Current CDC guidelines recommend isolating for five days. However, Hamer has thoughts.

“The CDC recommends five days. It’s not based on a lot of evidence...day five about half or less people are no longer infectious, and by the seventh day it’s gone down to an even better, lower level of risk.”

Hamer recommends abiding by the official isolation guidelines,

but advises it’s good practice to continue wearing a well-fitted mask for a few days beyond the five-day mark.

Why it’s not suggested to get a PCR test to return to normal activity:

“The PCR is much more sensitive, so it can detect really tiny particles of the RNA of the virus.” he said, “Some people will have bits and pieces of virus spread that goes out for weeks.”

Therefore, getting a rapid or even an at-home test to provide to workplaces, schools, and other facilities is a much better method. This is also why most organizations that do mandatory testing of their community, such as universities, will exempt those who have already been infected from the testing requirement for 90 days.

We work with dangerous pathogens in a downtown Boston biocontainment lab – here’s why you can feel safe about our research

Original article from The Conversation, authored by Ronald Corley. July 14, 2021



Security precautions, thoughtful facilities design, careful training and safe lab practices help keep pathogens isolated. Boston University Photography, CC BY-ND

Microbiologist **Ronald Corley** has gone to work every day throughout the pandemic as director of the National Emerging Infectious Diseases Laboratories. Within this secure lab facility in Boston, scientists study pathogens as diverse as tuberculosis, Ebola virus, yellow fever virus and Zika virus. Many investigators there quickly turned their attention in 2020 to SARS-CoV-2, the virus that causes COVID-19.

Here Corley answers some of the most frequently asked questions about this kind of biosecure lab and the work researchers do inside it.

What is the purpose of a biocontainment facility?

A newly emerging or reemerging human pathogen is detected somewhere around the globe every 12 to 18 months.

Infectious diseases don’t respect borders. Because of the global economy and unprecedented mobility, everyone on the planet is vulnerable to potentially devastating infectious diseases that may have originated halfway across the world. In this age of high-speed travel, we are as little as 36 hours away from any outbreak.

As with SARS-CoV-2, scientists may know little about emerging pathogens or the diseases they cause. Studying these germs – whether bacteria, viruses or other microorganisms – in the safe environment of a biocontainment laboratory is the best protection humankind has against these diseases. In the lab, researchers can safely test new diagnostics, therapeutics and vaccines. The more scientists learn about these new diseases, the better prepared we are for the ones that will come after.



This is where labs like the NEIDL, and our stringent safety measures, are important. I feel safer from infection working in the NEIDL than I do in my apartment building. We know what we’re working with in the lab and how to keep ourselves and others safe. But outside, I don’t know who I might pass who could have a transmissible pathogen, including the coronavirus.

This is not to say that there is no risk working within the laboratory – there is. But we minimize it through a series of safety measures – including building systems, laboratory design, personal protective equipment, training and safety protocols – that have been tried and tested in laboratories across the world.

How do you try to minimize risk?

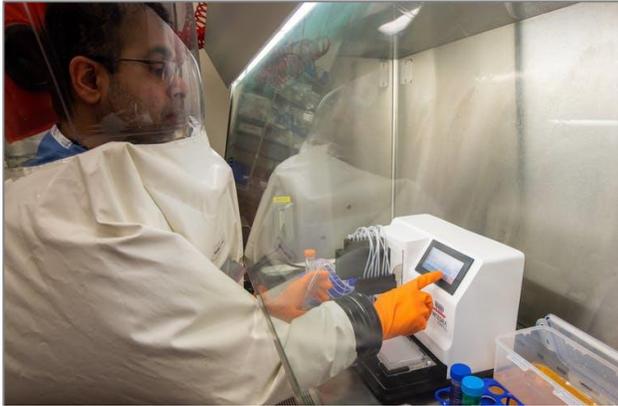
Our biosafety manual sets the standards for all work with biological material in the NEIDL. Requirements increase in complexity from Biosafety Level 2 (BSL-2) on to BSL-3 and BSL-4.

In the U.S., the Centers for Disease Control and Prevention determines each pathogen’s biocontainment level, based on what’s known about how it infects its host, the severity of the disease it causes, how easily transmissible the pathogen may be and the nature of the work itself – does it potentially create aerosols, for example. The biosafety levels require different types of engineering controls – such as the building materials the space uses, directional air flow to ensure pathogens can’t get out, HEPA filtration so that only sterile air is discharged from the lab space and so on.

The administrative controls required vary by biosafety level, as well – safety protocols, requirements for personnel training, limiting access and so forth.

Each level requires different types of personal protective equipment: gloves and lab coats in a BSL-2 laboratory, protective lab wear and N95 or PAPR respirators in BSL-3 or a fully encapsulating suit in a BSL-4 laboratory.

“Safety First” is not just a bumper-sticker phrase at the NEIDL. Everyone from public safety officers to support staff to researchers has fully bought into the culture of safety. It informs the way we’re trained and drilled, the way pathogens are transported to the facility, and policies that govern our employees. We know the risks of the work, train on protective measures, and ensure every member of our staff follows our protocols.



Working with pathogens only within a specially ventilated biosafety cabinet provides another layer of security. Boston University

What does containment look like with these safety strategies in place?

Everyone undergoes annual background checks, medical clearances and training. Only cleared staff can enter the building alone.

There are limited ways into the space, one for pedestrians, and one for vehicles, like delivery trucks. Entry requires access via biometric or card access or both, and screening by security. Access controls limit staff members to entering spaces where they have permission to work, based on their training, clearances and biosafety protocols. A network of security systems and closed-circuit cameras monitors the facility.

Entering laboratories requires that workers don the appropriate PPE for the area. Within the labs, we know

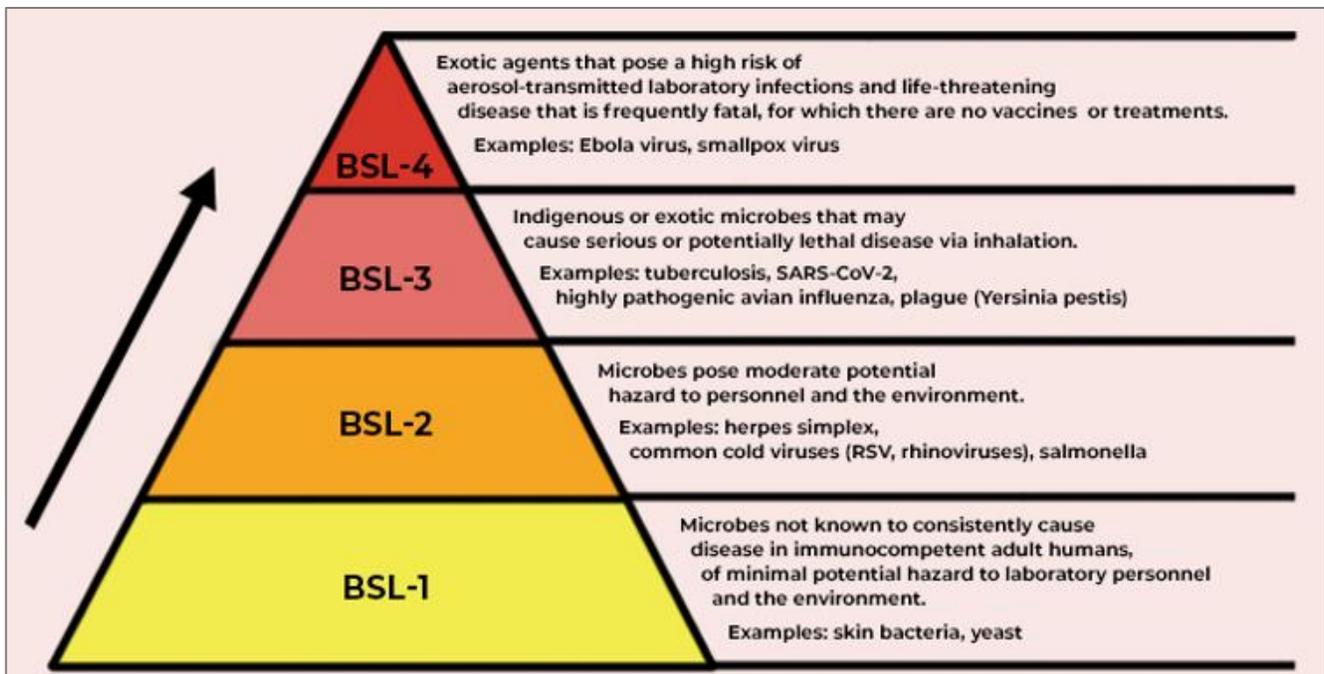
what pathogen we are working with and how it is being used and are confident staff are following the safety measures required to keep them safe. This ensures the safety of others in the building as well as the surrounding community.

Importantly, the biosafety practices ensure that each pathogen we’re studying is restricted to the appropriate spaces. Researchers work at biosafety cabinets that sterile-filter the air before releasing it back into the lab.

What kinds of regulation and oversight are there?

Biocontainment laboratories do not function in a vacuum. The building and laboratory designs, and the PPE and operating procedures that protect staff, meet the guidelines set by the CDC and by the 574-page book “Biosafety in Microbiological and Biomedical Laboratories” from the CDC and National Institutes of Health.

To carry out a project, the lead scientist begins with an application to the Institutional Biosafety Committee. Experts in biosafety and science review the application, as do laypersons who provide a community perspective. These



Biosafety levels are defined by how much risk is involved in working with particular pathogens. The Conversation, CC BY-ND

deliberations are open and transparent thanks to public participation on the committee. Its minutes are posted online. Safety professionals also inspect the laboratory facilities before work gets underway.

In the city of Boston, projects that involve any BSL-3 or BSL-4 work require review and approval from the Boston Public Health Commission, one of the only local public health departments with this type of oversight. Work with certain types of pathogens called “select agents” that pose a severe threat is further regulated by the Division of Select Agents and Toxins within the CDC.

Here at the NEIDL, both city and federal officials inspect the laboratories, interviewing personnel and reviewing records, including maintenance records. They also inspect pathogen inventories. Inspections can be announced or unannounced.

What would happen if something went wrong?



The safety of a facility like the NEIDL benefits from the expertise and resources available in a densely populated area. Boston University Photography, CC BY-ND

An important aspect of safety is making sure everyone knows what to do in an emergency. Three trainings per year involve first responders from the city as well as from Boston University. These are done as either live drills or tabletop exercises with experts walking through what an emergency would look like. Afterward we review how we did and develop plans for improvement.

Community members are also part of the exercises, and this keeps our neighbors involved and hopefully provides assurance of our ability to handle accidents, keeping ourselves and the community safe.

At Boston University, we post all laboratory incidents, including those at the NEIDL, on a quarterly basis to ensure that we remain transparent in our activities. Depending on what

went wrong, we may also report to the BPHC and the CDC.

Why place these high-security labs in urban environments with lots of neighbors instead of the middle of nowhere?

Scientific research is a communal activity, and advances happen in places where diverse expertise is concentrated. It's no different for research on emerging pathogens. Research on pathogens relies on faculty with expertise in not only the pathogens themselves but chemistry, engineering, stem cell biology, structural biology, immunology and more.

Biocontainment research also requires facilities engineers, safety professionals and security personnel. You can find personnel with diverse experience and expertise in metropolitan areas that are already home to biomedical research.

The original permitting process of the NEIDL mandated a comprehensive risk assessment to determine any potential danger for the community. After two years and independent review by two scientific panels, we ended up with the most extensive analysis of risk for any BSL-3 or BSL-4 facility in the U.S. It considered hundreds of possible scenarios that might result in exposure of a worker to a pathogen, or the release of a biological agent. The report concluded that it's as safe, or even safer, to have such a facility in an urban environment than in a rural or suburban environment.

“Near misses” have occurred at these kinds of labs within the U.S. and Europe. A near miss might, for example, involve glove tears and a potential exposure to a pathogen during laboratory work, but these have never resulted in any community infections. At the NEIDL, we intend to maintain this track record.

What are the risks of not doing this research?

Science builds on what's been learned before, accelerating our ability to respond to new outbreaks. The data we generate speeds progress on other pathogens as well, and informs how we develop and test potential therapeutics and vaccines.



The risk of not doing this work is to leave ourselves more vulnerable to emerging pathogens as they arise.

Professionals working on emerging infectious diseases are interested in solving problems that benefit the public's health. We take pride in our work and are serious about our responsibility to perform our work safely and securely. We recognize that this research is often viewed skeptically and thus strive to keep the trust of the public by ensuring transparency around the work we do.

Scientists put their intellectual curiosity to work on problems that challenge public health. Boston University Photography, [CC BY-ND](#)

People

Scientific Leadership



Ronald B. Corley, PhD
Chair, Dep of Microbiology
Director, NEIDL
Director, Immunology Core

Dr. Corley's Research interests:

- Innate and adaptive immunity to human pathogens



Gerald T. Keusch, MD
Professor of Medicine & Intl Health
Associate Director, NEIDL
Director, Collaborative Research

Dr. Keusch's research interests:

- Global impact of infectious diseases on economic development and public health



Nahid Bhadelia, MD, MALD
Associate Professor, Medicine / ID
Associate Director, NEIDL
Founding Director, CEID

Dr. Bhadelia's research interests:

- International pandemics strategy & policy
- Disaster preparedness training for healthcare workers

Faculty



John H. Connor, PhD
Associate Professor, Microbiology
Lead, Viral Genomic Tracking & Single Cell Omics Resource

Dr. Connor's research interests:

- Virus-host interaction
- Viral domination of protein synthesis
- Novel approaches to virus detection



Markus Bosmann, MD
Associate Professor of Medicine, Pathology & Laboratory Medicine

Dr. Bosmann's research interests:

- Acute respiratory distress syndrome
- Infection-associated inflammation
- Macrophages, neutrophils, T cells, lung epithelial cells



Nicholas Crossland, DVM ACVP
Assistant Professor, Pathology
Director, Comparative Pathology Lab

Dr. Crossland's research interests:

- *Borrelia burgdorferi* and mechanisms of persistence
- Comparative pathology using animal models



Robert Davey, PhD
Professor, Microbiology
Lead, High Throughput Screening Service Unit, NEIDL

Dr. Davey's research interests:

- Host factor-based therapy development
- Infection mechanism for filoviruses and other high containment viruses



Florian Douam, PhD
Assistant Professor, Microbiology

Dr. Douam's research interests:

- Viral immunogenicity and pathogenicity mechanisms in vivo
- Advanced humanized mouse systems



Rachel Fearns, PhD
Professor, Microbiology
Director of Graduate Studies

Dr. Fearns' research interests:

- Negative strand RNA virus polymerase activities
- Control of respiratory syncytial virus RNA synthesis



Anthony Griffiths, PhD
Associate Professor, Microbiology
Director, Nonclinical Studies Unit

Dr. Griffiths' research interests:

- Multiple aspects of filovirus biology
- Development of vaccines and therapeutics



Anna Honko, PhD
Research Assoc Prof, Microbiology
Assoc Director, Nonclinical Studies Unit

Dr. Honko's research interests:

- Immunology and vaccine development
- Characterization of infectious disease animal models using implantable radiotelemetry



Thomas B Kepler, PhD
Professor, Microbiology, Math & Statistics

Dr. Kepler's research interests:

- Quantitative Systems Immunology
- Vaccine Development



Rahm Gummuluru, PhD
Professor and Vice-Chair, Department of Microbiology

Dr. Gummuluru's research interests:

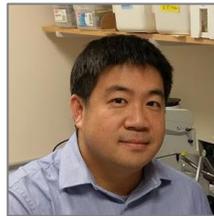
- Multiple aspects of filovirus biology
- Development of vaccines and therapeutics



Adam J Hume, PhD
Research Associate Professor, Microbiology

Dr. Hume's research interests:

- Replication and pathogenesis of filoviruses using minigenomes and reverse genetics
- Pathogenicity and zoonosis of Lloviu virus



Nelson Lau, PhD
Associate Professor, Biochemistry
Director, Genome Science Institute

Dr. Lau's research interests:

- Transposable element regulation
- RNAi and Regulatory RNAs
Gene silencing



Davidson Hamer, MD
Professor, Global Health & Medicine
Interim Director, CEID

Dr. Hamer's research interests:

- Tropical Infectious diseases
- Multi-site disease surveillance
- Molecular epidemiological studies to predict outbreaks



Igor Kramnik, MD, PhD
Assoc Professor, Medicine and Microbiology

Dr. Kramnik's research interests:

- Genes controlling host resistance and susceptibility to TB
- Mechanisms of macrophage activation and differentiation



Joseph Mizgerd, PhD
Professor, Medicine, Microbiology & Biochemistry
Director, Pulmonary Center

Dr. Mizgerd's research interests:

- Pulmonary immunity
- Cell biology of the lung
- Mechanisms of inflammation

Core Faculty (blue font)
Affiliate Faculty (black font)

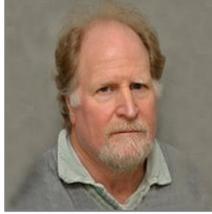


Elke Mühlberger, PhD

Professor, Microbiology
Director, NEIDL Virology Services

Dr. Mühlberger's research interests:

- Host response to filovirus infection
- Molecular mechanisms of filovirus replication and transcription



John C. Samuelson, MD, PhD

Professor of Molecular and Cell Biology
Professor of Microbiology

Dr. Samuelson's research interests:

- Pathogenesis of protozoan parasites
- Structures of parasite walls & glycoprotein



Mohsan Saeed, PhD

Assistant Professor, Biochemistry

Dr. Saeed's research interests:

- Role of viral proteases in shaping virus-host interactions

Core Faculty (blue font) *
Affiliate Faculty (black font)

Laboratory Staff and Trainees

Connor Lab

Hashimi, Marziah *

Postdoctoral Associate, Microbiology

Hossain, Mofazal

Research Technician, Microbiology

Nguyen, Michelle

Graduate Student, Pharmacology & ET

Overbeck, Victoria *

Graduate Student, Microbiology

Seitz, Scott

Postdoctoral Associate, Microbiology

Sher-Jan, Cole

Research Technician, Microbiology

Strampe, Jamie Michelle

PhD Student, Bioinformatics

Turcinovic, Jacquelyn

PhD Student, Bioinformatics

Velasquez-Reyes, Joseline

PhD Student, Microbiology

Crossland Lab

Gertje, Hans

Lab Manager, Histotechnology, NCPL

Kothari, Jeet *

Graduate Student, Pathology

O'Connell, Aoife *

Research Tech, Histotechnology, NCPL

Tseng, Anna

Graduate Student, Pathology

Davey Lab

Boytz, Ruthmabel *

Research Technician, Microbiology

Close, Brianna *

Graduate Student, Biochemistry

Donohue, Callie

PhD Student, Microbiology

Geoghegan-Barek, Kathleen

Lab Manager, Microbiology

Keiser, Patrick

Graduate Student, Microbiology

Mori, Hiroyuki *

Postdoctoral Associate, Microbiology

Patten, Justin

Sr. Research Technician, Microbiology

Stubbs, Sarah Hulsey

Research Scientist, Microbiology

Douam Lab

Adams, Scott

PhD Student, Microbiology

Cervone, Francis *

Graduate Student, Pathology

Chavez, Elizabeth

PhD Student, Microbiology

Gold, Alexander

PhD Student, Microbiology

Kenney, Devin

PhD Student, Microbiology

Lebner, Tyler *

Graduate Student, Pathology

Mameli, Enzo

Visiting Researcher, HMS

Matsuo, Mau *

Undergraduate Student, Biology, CAS

Muwowo, Suwlanji *

Undergrad Student, Biology, CAS

Nono, Evans T *

Undergraduate Student, Biology, CAS

Sheikh, Amira

Lab Manager, Microbiology

Soto Albrecht, Yentli *

Visiting Scientist, UPenn

Tamura, Tomokazu *

Visiting Scientist, Princeton University

Unali, Giulia *

Postdoctoral Associate, Microbiology

Fearns Lab

Breen, Michael

PhD Student, Microbiology

Kim, Heesu *

Research Technician, Microbiology

Klein, Piers

Undergraduate Student, UROP

Kleiner, Victoria

PhD Student, Microbiology

Ludeke, Barbara PhD

Sr. Research Scientist, Microbiology

Ouizougun-Oubari, Mohamed

Postdoctoral Associate, Microbiology

Powell, Kaila *

Undergraduate Student, Biology, CAS

Griffiths Lab

Dunn, Molly DVM, MPH *

Project Manager, Nonclinical Studies Unit

Gavrish, Igor * *

Research Technician, Microbiology

Jegado, Brice *

Postdoctoral Associate, Microbiology

Johnson, Rebecca

Postdoctoral Associate, Microbiology

McKay, Lindsay

Postdoctoral Associate, Microbiology

Reynolds, Brooke

Lab Manager, Microbiology

Silva Ayala, Daniela *

Postdoctoral Associate, Microbiology

Staples, Hilary

Compliance Manager, Nonclinical Studies

Storm, Nadia

Postdoctoral Associate, Microbiology

Kramnik Lab

Yabaji, Shivraj M

Postdoctoral Associate, Microbiology

Mühlberger Lab

Flores, Elizabeth

PhD Student, Microbiology

Heiden, Baylee

Research Technician, Microbiology

Hu, Xiaoyi

Visiting Scientist, MIT

Olejnik, Judith

Sr. Research Scientist, Microbiology

Ross, Stephen

PhD Student, Microbiology

Suder, Ellen Lee

PhD Student, Microbiology

White, Mitchell

Lab Manager, Microbiology

Saeed Lab

Chen, Da-Yua

Postdoctoral Associate, Biochemistry

Chin, Chue (Alice)

Postdoctoral Associate, Biochemistry

Conway, Hasahn *

Research Technician, Biochemistry

Khan, Nazimuddin *

Postdoctoral Associate, Biochemistry

Seeman, Marc *

Undergraduate Student, Biochemistry

Tavares, Alexander *

Graduate Student, Biochemistry

Lab Operations Staff

Broos-Caldwell, Aditi

Lab Operations Manager, BSL-2 & BSL-3

Feitosa-Suntheimer, Fabiana

ACL Insectary Manager

Yen, Judy

Sr Lab Operations Manager, BSL-4

Executive Leadership



Ronald B. Corley, PhD
Director, NEIDL

Administration

Durkop, Betina A
Executive Coordinator

Forman, Lora
Administrative Manager, Operations

Spell, Virginia
Research Contracts Manager

Trevino, Richard , MPH
Director, Finance & Research
Administration

Community Relations

Britton, Valeda J JD
Executive Director, Community Relations

Idiokitas, Chimel *
Assistant Director, Community Relations

Facilities Maintenance & Operations

Amadio, Paul
Facilities Engineering Operations Manager

Ananian, David
General Mechanic

Baires, J Victoria
Custodian

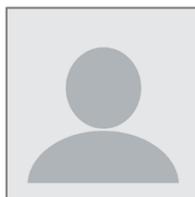
Cardoso, Jonathan
Control Technician I

Connell, Jeffrey *
General Mechanic

Corbett, Joseph
Controls Manager

Cyr, Brandon
Control Technician II

Fonseca, Paulo *
Control Technician II



David D. Flynn *
Director of Operations

Kjersgard, Eric J
Control Technician II

Madden, Lance
General Mechanic

Minacapilli, Salvatore
General Mechanic

Mosca, Derek
Maintenance Mechanic

Moylan, John *
General Mechanic

Nakhid, Nafisah
Assistant Engineer

Rodriguez, Mario
Custodian, NEIDL

Rusk, Scott
Director, NEIDL Facilities

Santana, Jorge *
General Mechanic

Sousa, Daniel
NEIDL Shipper & Receiver

Solimini, Michael **
General Mechanic

Tucker, Daniel
Maintenance Mechanic

Tupe, Michael T
Maintenance Mechanic

Wynne, Paul M
Control Technician II

Information Technology

McCall, John
Director, Information Technology Core

Slutzky, Ben
IT Operations Administrator



Kevin Tuohey
Chief Safety Officer

Environmental Health & Safety

Benjamin, Shannon, MBA CBSP
Assoc. Director, Research Safety
for High Containment (BSL-3)

Ahmad, Anwaar *
Assoc. Director, Research Safety
for Maximum Containment (BSL-4)

Downs, Sierra Nicole
Prog Mgr, Emergency Response Planning

Ellis, Andrew W
Sr. Research Safety Specialist

Flynn, Nick
Biocontainment Operations Manager

Madico, Guillermo, MD PhD
Scientific Safety Officer

Nigro, Nick *
Sr. Specialist, High Containment

Olinger, Gene G, PhD
Assoc. Director, Training in High
Containment

Penner-Hahn, Julianne *
Quality Assurance Officer

Randall-Hlubek, Deborah *
Quality Assurance Specialist (contractor)

Rando (Harrington), Patrice *
Senior Specialist, High Containment

Tuohey, Kevin M
Executive Director, Research Compliance
Chief Safety Officer

Wallenstein, Adam
Senior Specialist, High Containment

Joined during FY22 (green *)
Left during FY22 (red *)

Animal Research Support

Adams, Tarik *
Veterinary Research Technician

Alicea, Anthony *
Veterinary Research Technician

Carvalho, Mariah
Veterinary Research Technician

Diaz-Perez, Yulianela
Veterinary Research Technician

Furtado, Oscar M
Veterinary Research Technician

Grosz, Kyle
Operations Manager

Harrington, Patrice *
Veterinary Research Technician

Laprise, Ambre
Veterinary Research Technician

Lemoine, Kyle
Veterinary Research Technician

Mazur, Michelle *
Research Clinical Veterinarian

McAndrews, Ryan *
Veterinary Research Technician

Mclaughlin, Robert J
Veterinary Research Technician

Niemi, Steven *
ASC Director & Attending Veterinarian

Nickel, Ashley
Veterinary Research Technician

Nunes, Corey
Assistant Director of Operations

Renon, Dominique *
Veterinary Research Technician

Roberts, Matthew *
Veterinary Research Technician

St Denis, Lauren *
Veterinary Research Technician

Thurman, Coleen
Research Clinical Veterinarian

Public Safety

Annese, Rae
Public Safety Officer

Barros, Christopher L
Public Safety Officer

Barros, Jeffrey P
Public Safety Officer

Derosa, Paul *
Public Safety Officer

Duffy, Joseph M
Public Safety Officer

Estrella, David
Public Safety Officer

Estrella, Jesse *
Public Safety Officer

Gallivan, John
Public Safety Officer

Maldonis, Joseph
Public Safety Officer

Paparo, Scott
Systems Integrator

Perrotta, Thomas *
Staff Operations Manager

Phelps, Justin
Public Safety Officer

Saad, Jacob
Public Safety Officer

Salhi, Adil
Public Safety Officer

Souza, Joshua
Public Safety Officer

Taranto, Stephen
Director of Public Safety, BUMC

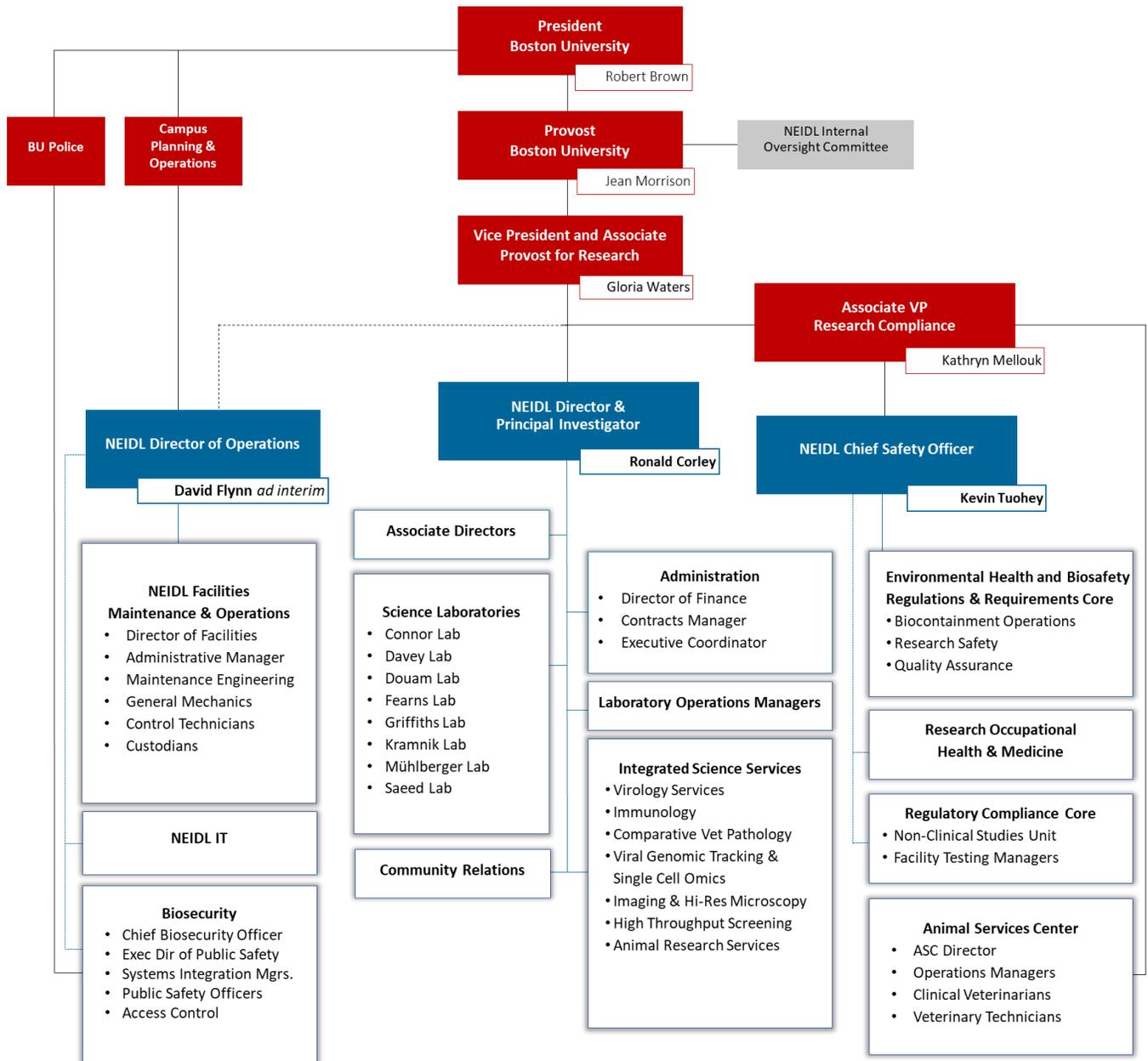
Taverna, Michelle
Access Control Officer

Tracy, Harris
Systems Integrator

Wynne, Sean C
Public Safety Officer

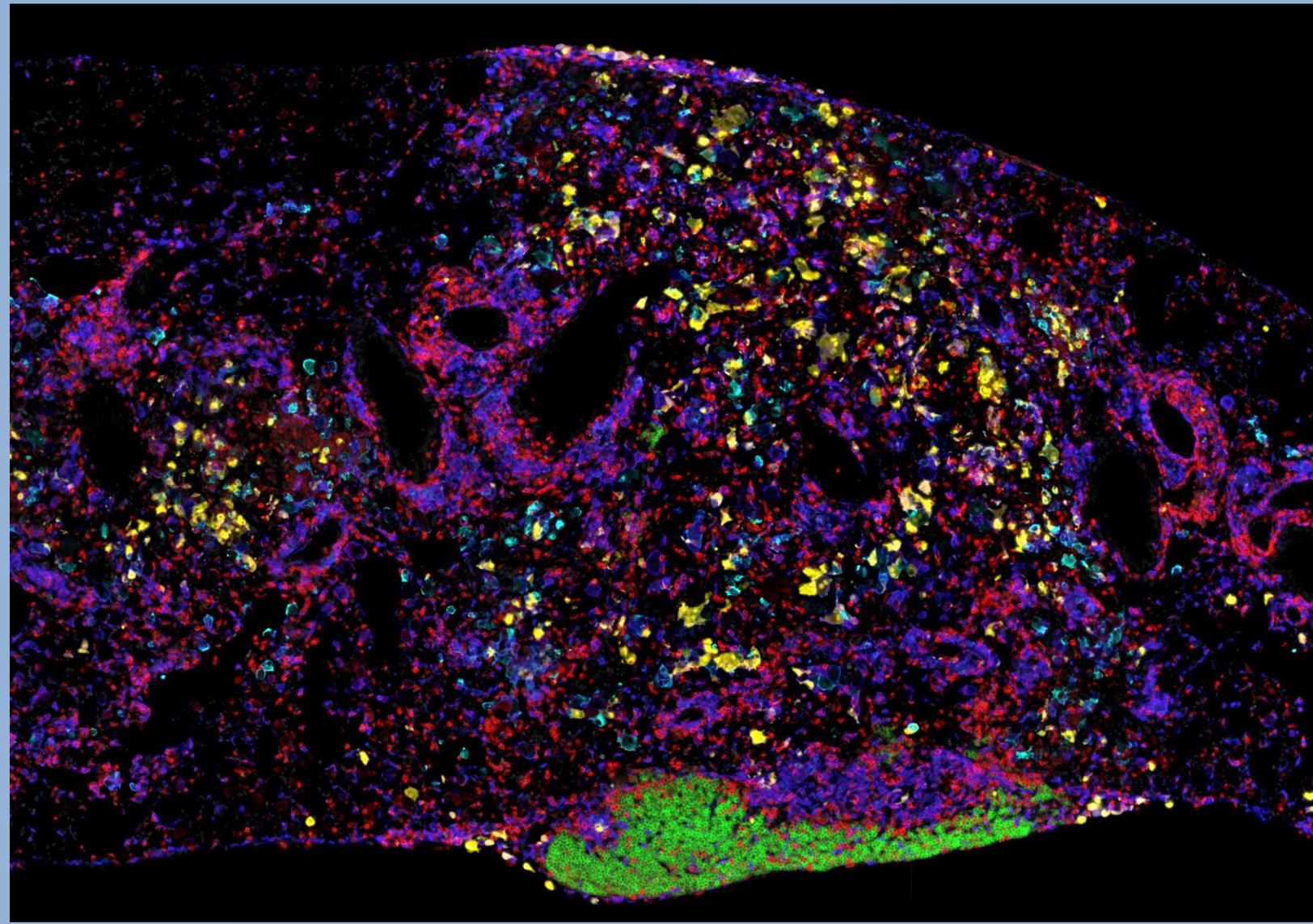
Joined during FY22 (green *)
Left during FY22 (red *)

NEIDL Organizational Chart



Picture on front cover: SARS-CoV-2 neurodissemination in the transgenic humanized K18hACE2 mouse brain at the level of the hippocampus: SARS-CoV-2 Spike (orange); neurons-NeuN (teal); microglia-Iba1 (magenta); astrocytes-GFAP (green); blood vessels-CD31 (red); DAPI-grey. Douam and Crossland labs.. Students: Anna Tseng and Devin Kenney

Picture on back cover: Post-primary tuberculosis in the immunocompetent but susceptible C3H.B6-sst1 mouse lung: pan-macrophage-Iba1 (blue), M1 macrophages-iNOS (teal), M2 macrophages-Arg1 (yellow); T cells-CD3epsilon; B cells-CD19-green; DAPI-grey. Kramnik and Crossland labs. Student: Anna Tseng. Post-doc: Shivraj Yabaji



Boston University National Emerging
Infectious Diseases Laboratories

