COVID-19 has unfortunately halted lab work, conferences, and in-person networking, which is especially detrimental to researchers just starting their labs. Through social media and our reviewer networks, we met some early-career stem cell investigators impacted by the closures. Here, they introduce themselves and their research to our readers.

**Of Mice and Children**

Eszter Posfai
Princeton University

I started as an assistant professor at Princeton in 2019. My lab studies how cell fate decisions and morphogenesis are coupled in a self-organizing framework that underlies the formation of the early mammalian embryo. We engineer mouse embryos with exciting tools to visualize and manipulate developmental mechanisms in ways that were previously not possible and seek to establish novel stem cell-based models to gain simplified access to embryonic events.

Everyone’s lives have been impacted by COVID-19. For me it has turned the volume up on concerns any starting PI faces. We shut down the lab. How do we produce that critical preliminary data needed to get the lab’s first grants? We reduced mice to maintenance levels. Mice take time. Even if we reopen, how long until we recover from this setback? With three young kids and an essential worker husband I have turned into a stay-at-home mom for the past 3 months. How do I give my lab members and the students I teach the support they need? Especially under these circumstances I am very grateful for my department’s understanding and for my fantastic team who have been soldiering on while their PI has only sparsely been on the grid. I am excited by news that we may be resuming research with limited capacity soon. While the ripples of this pandemic will be felt for a long time in the scientific community, I believe that with our dedication to science, baked with some compassion for one another, we will overcome these challenges.

**Neighbors: Friends or Foes**

Joo-Hyeon Lee
Wellcome-MRC Cambridge Stem Cell Institute

Our lungs are host to a multitude of epithelial cell types, with an array of neighboring stromal and immune cells that support them in maintaining the lung barrier. Careful communication between stem cells and their neighbors is essential for proper tissue repair following injury. My lab is interested in the critical role of the neighboring cell in influencing stem cells during the repair process. We are fascinated by how neighbors can act as either friends or foes during regeneration. We ask fundamental questions involving how stem cells sense environmental changes and determine their cell fate and how niches develop and remodel the local environment during lung regeneration and the early stages of disease progression. To answer these questions, we combine in vitro organoids, in vivo genetic models, single-cell profiling, and clonal biophysical modeling.

This pandemic poses a uniquely challenging time to all and reminds us of the things that we are grateful for. The contribution of my dedicated team, who remain healthy and passionate about their research, and the extension of my funding support as a Wellcome Trust Fellow are maintaining our research efforts. Our expertise in human lung organoid models has enabled us, with collaborators, to contribute swiftly to COVID-19 research. During this period of uncertainty, our ultimate goal to provide therapeutic interventions in lung diseases of unknown cause and without cure remains in sharp focus.

**Modeling Stem Cell Fate**

Adam L. MacLean
University of Southern California

Our lab at USC began in 2019. We study stem cell fate using mathematical and computational models. We are interested in fundamental questions regarding the regulation of stem cell identity and stem cell differentiation and how these processes become dysregulated in cancer.

In many cases stemness is not cell-autonomous, but rather is an active process involving signals received from the microenvironment. We model these dynamics and investigate the gene regulatory networks that control stem cell differentiation focusing on particular systems, such as hematopoietic or epithelial lineages. We develop methods to analyze the wealth of data that single-cell technologies offer—integrated with mathematical modeling and Bayesian parameter inference—to gain new insight into cell fate decisions. In cancer, we seek to resolve controversies around cell fate, for example regarding the identity of cancer stem cells, or the impact on tumor growth of EMT.

The impact of COVID-19 on our group has been less severe than others since we are a dry lab and can work remotely. However, we share big challenges with other junior faculty including the impact of working from home on our ability to work closely with students and a dramatically altered funding climate. In the face of recent crises, caused not only by the pandemic but also by systemic racism, our hope is to emerge with greater means and will to make academia more just and equitable—a prerequisite for great science.
The early mouse embryo as well as the pluripotent cells derived from it are excellent tools to study changes in developmental potential and cell fate specification. In our lab, we leverage these in vitro and in vivo models to gain better comprehension of embryonic development and cancer. I have always been fascinated by how a single cell has the potential to generate a whole organism. During the first days of development, the zygote undergoes a series of orchestrated cell divisions to generate a blastocyst, a self-organized structure consisting of three spatially segregated cell lineages and a fluid-filled cavity. This is arguably the most important period of embryonic development but still has many unknowns. We aim to understand the molecular mechanisms deployed to restrict cell potency and establish embryonic identity during early development. Our ultimate goal is to evaluate how these mechanisms influence cell fate decisions.

A year and a half after starting my lab, we were hit by COVID-19. This period has been extremely challenging for my group. However, I feel privileged for having an amazing team that has adjusted well with the difficulties associated with virtual communication. In addition, the NIH leadership has been extremely supportive and understanding to all tenure-track investigators during this time. With some light appearing on the horizon, we look forward to finally restarting our research activities. To quote Queen, “The show must go on!”

My lab focuses on the development and diseases of the cardiac conduction system that regulates the heartbeat. We are using human pluripotent stem cells as a developmental model system to obtain new insights into human heart development. Specifically, we are establishing developmental-biology-based approaches to differentiate stem cells into cell types of the conduction system, including sinoatrial node and atrioventricular node pacemaker cells. We are testing the ability of these pacemaker populations to function as biological pacemakers and to regenerate the conduction system. In addition, we are establishing new in vitro models for conduction system diseases to explore disease mechanisms and identify potential drug treatment targets.

Like many start-up labs, we have been working hard to generate data for a first publication and have lost valuable experiments due to the lab shutdown caused by the COVID-19 pandemic. On the bright side, the research community has come together to solve the unique challenges we are facing during this unprecedented time. As a result, I believe this event will serve as a reminder of the importance of science and strengthen the scientific community. My team is eager to return to the lab to pick up where we left off and we are currently looking to hire a postdoctoral fellow. Therefore, I am optimistic that COVID-19 won’t stop us from continuing our mission to develop novel therapies for patients suffering from heart rhythm disorders.

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My lab seeks to understand the complex genetic and cellular processes that underlie the cardiomyocyte cell cycle and myocardial regeneration. We are following up on an observation made during my postdoc that the capacity to regenerate one’s heart is not “one size fits all.” Instead, distinct genetic backgrounds display variable competence to respond to an injury. My growing team is working to validate novel candidate genes that influence the cardiomyocyte cell cycle and understand how these genetic players interact. We are expanding our toolshed to examine additional cell types contributing to variable outcomes after injury, alongside expansion to other species and tissues.

2018–2019 was a huge year for my family. In just a one-year period, my husband (also a scientist) and I moved across the country, started two new academic labs, welcomed our second child, and bought our first home. It was a whirlwind, but totally worth it! I thought that was the hardest year of my life, but then COVID-19 came. My emotions have run the gamut: fear, anxiety, jealousy, inadequacy, acceptance, hope, and gratitude. For 10 weeks, my husband and I “worked” from home, juggling two careers and two children under 5. I have been mentoring my young lab entirely virtually; their resilience and enthusiasm inspire me daily. I am grateful to my colleagues and mentors that have encouraged me, included me, and taken me under their wings. Perhaps this experience has only reinforced that I am in the right place.