footnotes are in fact true telegraphic jewels cloaking critical assessments on important topics, such as optogenetics, engrams, and Jennifer Aniston neurons. Such a complex journey through the bewitching mainstream heterodoxy tends a constellation of crucial neural matters that shall spur apposite discussions within neuroscience writ large10. “You may or may not agree with me,” Buzsáki says, “but at least you will experience a different perspective”. Indeed, one can recognize good thinking without needing to accord with the thoughts. As always, and in the end, the major (philosophical) questions remain unanswered. Why the brain11 and only the brain? Psychology might one day be to neuroscience what alchemy was to chemistry. And yet, like Newton, perhaps we should practice them both. Anyhow, it is enlightening to inquire into the psychological significance of neural reductions. There are probably more insights about the human mind in a Dostoevsky novel than in any neuroscience treatise. Proud of what neuroscientists have, can, and will achieve, let us not forget the other two main Delphic maxims, “nothing in excess” and “surety brings ruin”. 

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Q & A

Erin Goley

Erin Goley is an Associate Professor of Biological Chemistry at the Johns Hopkins University School of Medicine. She earned her BA at Hood College in Frederick, Maryland, and her PhD in Molecular and Cell Biology at the University of California, Berkeley. After training as a eukaryotic cell biologist with Matt Welch at Berkeley, she pursued postdoctoral research in bacterial cell biology with Lucy Shapiro at Stanford. Her laboratory at Hopkins focuses on understanding the mechanisms of bacterial morphogenesis and the regulation of bacterial growth in changing environments. 

What turned you on to biology in the first place? I took a pretty naïve route to biological research, but looking back my biggest early influence was my stepdad, who was a pediatrician. He ran his own small practice out of the old nineteenth-century school building in our town and I spent a lot of time at his office as a kid and early teen doing little odd jobs. I remember being fascinated by how my stepdad would swab kids’ throats for suspected strep infection, streak out the samples on blood agar plates, and incubate them on top of the fridge (the warmest spot in the building). A day or two later, he’d look for the halo around bacterial colonies, indicating strep, and prescribe a course of penicillin. This was my first exposure to microbiology and, though it didn’t immediately turn me on to research, it certainly piqued my interest in the biological world. I majored in biochemistry and math as an undergrad at Hood College, a small liberal arts college in Maryland, and after a couple of summers in labs I was hooked on being at the bench. It still took me a while to learn that I could get a PhD and do something other than teach — i.e. I could run a lab for a living.

And what drew you to your specific field of research? Some of my earliest research experiences were with microbes: my undergraduate thesis and work as a technician at the USDA post undergrad were focused on fungal and viral plant pathogens. The diversity of the microbial world and the myriad ways that microbes interact with each other and with host organisms were fascinating to me. When I started graduate school in the Molecular and Cell Biology program at UC-Berkeley, thinking that I wanted to be a microbiologist, I did rotations in three labs that each looked at the interactions between pathogens and the actin cytoskeleton of their host (Listeria monocytogenes actin-based motility in Dan Portnoy and Matt Welch’s labs and nuclear actin assembly induced by baculoviruses in Loy Volkman’s lab). At the time, Matt’s lab was building on what they’d learned about how Listeria activates the host Arp2/3 complex to mediate its motility to understand more about how eukaryotic cells normally activate Arp2/3 for localized actin assembly. Though initially drawn in by the microbes, I joined Matt’s lab and got hooked on the cytoskeleton during my time at Berkeley. While there, I was trained broadly as a eukaryotic cell biologist in large part through our joint group meetings with the labs of Rebecca Heald (focused on mitotic spindle assembly) and Karsten Weis (studying nucleocytoplasmic transport). When the time came to choose a postdoctoral lab, the bacterial homologs of actin, tubulin, and intermediate filament proteins had recently been revealed, and I was excited by the opportunities for discovery in their regulation and function. As an aside, there’s been debate in the field about whether bacterial polymers are actually ‘cytoskeletal’ — they may not be, but if they hadn’t been called ‘the
cytoskeleton’ at the time when I was looking for postdoc labs I may never have been drawn to them. My colleague Arash Komeili at Berkeley has made this argument for calling bacterial organelles ‘organelles’, even though they’re distinct from the canonical membrane-bound organelles of eukaryotes. Using common terminology brings people into the field and highlights the deep roots and similar strategies of cell biology across kingdoms.

The field of bacterial cell biology ended up being a perfect blend of my long-standing interest in microbes and my nascent love of cell biology. I joined Lucy Shapiro’s lab at Stanford for my postdoc and began using Caulobacter crescentus as a model to dig into the mechanisms that the tubulin homolog FtsZ uses to direct cell division, something we still study in my lab at Hopkins. I still primarily identify as a cell biologist, as that’s the field in which I was trained. Recently, I was being introduced by a student during a seminar in a microbiology department and she noted that I was among the few who could really claim the title ‘microbiologist’. I was confused until she talked about how I’d published research on fungi, viruses, and bacteria. Turns out it’s true — but I always look at microbes through the lens of cell biology.

**Do you have a favorite science book?**

One that stood out recently was *I Contain Multitudes* by Ed Yong. It’s a beautifully written, well-informed dive into the complex relationships between microbes and their hosts. Another is Carl Zimmer’s *She Has Her Mother’s Laugh*, which is a comprehensive, captivating history and exploration of heredity, from the Habsburg family to CRISPR engineering.

**What is the best advice that you’ve been given?**

This may not be advice exactly, but a couple of lessons that my mom instilled in me growing up have shaped the way that I’ve pursued my science: specifically, work hard and be generous. My mom raised six kids and was one of the single most generous people I know and taught me to always try to lift up those around you when you can. Hard work in science isn’t anything novel, but it’s certainly been necessary as I’ve gone through grad school, postdoc, and now in my independent position. I will say though that I try to work efficiently and for quality and not for quantity of hours. It took some time for me to learn in grad school that, just because my neighbor was in the lab 16 hours a day, it didn’t mean that they were any more productive than I was (still true as a PI). The ‘be generous’ part just makes science more open, fun, and inclusive for everyone. I’ve benefited from the generosity of colleagues who’ve nominated me for leadership positions or awards, invited me to talk at seminars, and shared their expertise and/or unpublished findings or reagents with me. I’ve tried to pay this forward to others, and I am very open in pursuing and sharing my science. I reach out to colleagues to share projects that we’ve initiated — or are thinking about initiating — to make sure that we are not competing, my lab regularly shares unpublished data in talks, and we pre-print everything we ultimately publish. I also aim to be generous to trainees with my time — whether they are my own or those in other labs or institutions — to share my experiences and expertise when it may be helpful to them. Being open and generous as a scientist has not hurt me and has benefited me as well as my trainees in countless ways.

**What’s your favorite experiment?**

An overall approach that I really love is the use of pathogens to inform our understanding of eukaryotic cell biology: that is, the idea that pathogens are the best cell biologists because they’ve been evolving to manipulate their hosts far longer than we’ve been studying cell biology. I’m showing my bias here, but my favorite example of this is the use of *Listeria* actin-based motility to uncover principles of eukaryotic actin regulation and function. First, there’s that classic electron microscopy study from Lew Tilney and Dan Portnoy showing that *Listeria* associate with host actin filaments and likely use host actin to move around and spread cell to cell (J. Cell Biol. (1989) 109, 1597–1608). (As an aside, Dan wrote a retrospective of this work that’s a fun and informative read (Mol. Biol. Cell (2012) 23, 1141–1398.).) Julie Theriot’s subsequent work in Tim Mitchison’s lab showing that the rate of actin incorporation into the comet tail of *Listeria* matched the rate of motility helped to solidify the hypothesis that actin polymerization itself could drive movement of cargo. Building on this as a postdoc in the Mitchison lab, Matt Welch purified the Arp2/3 complex from platelet extracts as the host factor required (in addition to actin) to assemble actin on the *Listeria* surface, providing experimental support for the idea of actin nucleation by Arp2/3. He then showed that ActA from *Listeria* was required to activate robust actin nucleation by Arp2/3, laying the groundwork for identification of host proteins (e.g. WASP, N-WASP, and SCAR/WAVE) with Arp2/3-activating functions. I’m a sucker for a biochemical purification of protein activity, so Matt’s paper isolating Arp2/3 as an actin-nucleating factor is right up there as one of my favorite experiments ever (Nature (1997) 385, 265–269).

**What is your favorite conference?**

I’ll make a shameless plug here for two conferences that I’ve been involved in organizing — roles I took on because I love these meetings! The first is the long-standing ‘Plant and Microbial Cytoskeleton’ Gordon Research Conference (GRC). Originally conceived to explore plant and fungal cytoskeletal regulation and function, the conference was expanded in 2010 to include non-fungal microbes. What I love about this conference is the breadth of systems explored — it’s all focused on cytoskeletal function and regulation but in plants, yeasts, bacteria, archaea, protists, and so on. The diversity is stunning and I always learn something new. It’s also a great venue to compare different mechanisms of cytoskeletal function in diverse organisms with a cell wall. In these cells the cytoskeleton is typically not ‘skeletal’ at all but regulates intracellular transport and directs deposition and metabolism of the cell wall.

The second is a new GRC that I’m planning with Mark Buttnar on ‘Bacterial Cell Biology and Development’, to be held in the summer of 2023. It aims to cover the organization as well as dynamics of the bacterial cell and includes everything from chromosome structure to organelles to morphogenesis to inter-species
interactions. I’m particularly excited for this conference because the field has lacked a regular, dedicated meeting ever since the American Society for Microbiology canceled their portfolio of conferences, including the much-loved ‘Prokaryotic Cell Biology and Development’ meeting.

Any strong views on social media and science? I am a huge advocate for using social media for science communication and for building scientific networks — particularly Twitter and Slack. Not only are these platforms fantastic for rapidly sharing scientific findings, networking with other scientists, and advancing one’s career, they are really democratizing in determining who has a voice. This can be especially powerful for trainees and for individuals from historically marginalized groups in science. A recent, inspiring example of this on Twitter has been the emergence of ‘Black In...’ events (for example, ‘Black In Microbiology’ week) to highlight and promote Black scientists and their contributions to various STEM fields. These events are empowering for the Black scientists who organize and participate in them but also draw the attention of others in the field and beyond to these amazing scientists. Personally, I was overwhelmed during #BlackInMicro week by the impressive science, advocacy, and creativity of Black microbiologists and now have a much longer list of individuals I can turn to for seminars, jobs, panels, collaborations, and so on.

Slack is another useful platform for connecting to other scientists. Shout out to Prachee Avasthi at Dartmouth for starting ‘New PI Slack’ as a venue for new faculty to share experiences and resources, build their networks, and just feel connected to their peers. This group inspired a number of others (e.g. ‘Future PI Slack’ and ‘Mid-Career Slack’) that are collectively influencing the careers of huge numbers of academic scientists. In another example, my friend Peter Chien and I set up a Slack community a couple of years ago for those who study Caulobacter and other alphaproteobacteria (appropriately called ‘CauloSlackers’). It’s been a fantastic venue for trainees to interact and for everyone to share resources, info, and expertise. We used CauloSlackers to plan and execute two virtual Caulobacter conferences during COVID-19!

What do you think is one of the biggest problems that the scientific enterprise is facing today? An issue that I’ve become increasingly interested in is how we, in academia, can address failures in mentoring relationships (specifically between a PI and the students and postdocs in their laboratory). These range from a poor fit or poor communication in a mentor–mentee match to more extreme failures, such as verbal abuse and Title IX violations on the part of the PI. The former may be remedied by implementing multilayered mentoring structures, early and regular monitoring of mentor–mentee matches, and training in best practices for mentors (all of which are easier to implement for graduate students because of existing graduate program structures than for postdoctoral fellows). The latter is harder to deal with, particularly if trainees don’t feel safe or supported in coming forward. I think that we’ve all heard through whisper networks about “that PI” who has a track record of treating trainees as a disposable workforce, of verbal abuse, or of inappropriate comments to trainees of color, women, or international trainees. When offenders remain in their positions, receive awards, continue to recruit students and postdocs, and/ or are put in positions of leadership despite bad behavior, it sends the message that this kind of behavior is tolerable, as long as the science is exceptional. Trainees shouldn’t have to rely on a whisper network to avoid joining a toxic lab, and they should have clear and effective avenues to seek institutional intervention if they experience abuse. I don’t know what the answers are, but I think that it will take a cultural change in academia where we all see effective mentoring as a prerequisite for hiring, career advancement, honorifics, and continued recruitment of trainees, and where toxic PIs lose their jobs.

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Quick guide
Synaptonemal complex

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What is the synaptonemal complex? The synaptonemal complex (SC) is a meiosis-specific multiprotein complex that forms between homologous chromosomes during prophase of meiosis I. Once assembled, the SC mediates and maintains synapsis along the full length of each pair of homologous chromosomes. Back in 2003, Scott Page and I (R.S.H.) described the synaptonemal complex as “the elephant in the meiotic living room”.

What does the SC do? The SC is required for the maturation of programmed DNA double-strand breaks into crossovers (COs) — the reciprocal exchanges of DNA between parental chromosomes. COs provide the physical link that holds homologous chromosomes together until they segregate from each other at the end of meiosis I.

Furthermore, the SC is likely required to mediate at least some aspects of CO control, such as CO interference. Interference acts to reduce the number of COs on the same chromosome and to cause COs to be more widely spaced than expected by random chance if two or more COs are formed. In some cases, failure to reduce the number of CO events may result in impaired segregation. Indeed, interference may explain why even though organisms make a large number of DNA double-strand breaks, only a fraction of those breaks become COs.

In some organisms, the SC also facilitates homologous segregation at anaphase I. In these cases, modified forms of the SC or perduring SC components appear to facilitate this function. For example, perdurance of SC at budding yeast centromeres is required for centromere pairing and/ or centromere clustering, as well as for promoting faithful segregation of non-CO chromosomes. Or, in the case of silkworm females, a modified...