# Scientific Storytelling Workshop

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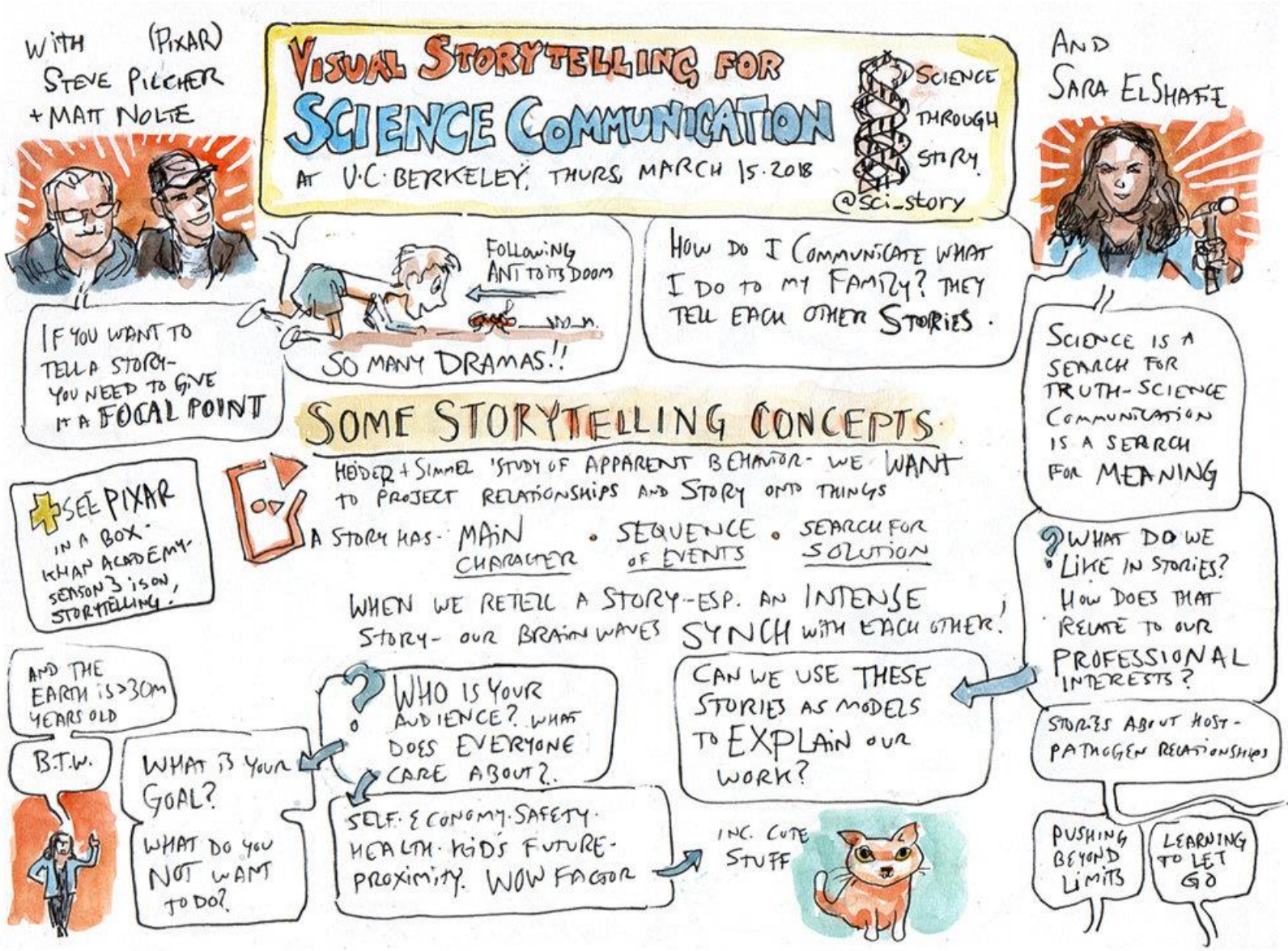


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# Scientific Storytelling Workshop Goals

- Storytelling value: Why this approach?
- Discussion of some basic concepts in a “story” using the Hollywood screenwriter’s approach (What compelling question are you pursuing as a “character” in the story?  What is the nature of the journey? What is at stake? What is the next act?)
- Discussion of how the story changes depending on the audience
- Workshop breakout task 1: Transform the abstract into the first parts of a scientific story for an oral presentation
- Discussion of task 1 ideas
- Workshop breakout task 2: Transform the abstract into the first parts of a scientific story for an oral presentation
- Discussion of task 2 ideas
- Just for Fun
- Wrap up

For oral presentations

## Open with a question

"start with a compelling hook, locking audiences in from the start"

"short anecdote to help set the stage and generate interest"

## It's all about the journey

"The research process lends itself perfectly to this narrative concept; it can be depicted as a winding road of plot twists that reshapes the lives of scientists and our understanding of the world. "

"Invite others to join you for the ups and downs of your research. Instead of just presenting your findings, describe how you reacted to those discoveries. What was most exciting? What did you get wrong?"

## Explain what's at stake

"Why should my audience care?"

…"Use the 'And [**Momentum**], But [**Conflict**], Therefore [**Resolution**]' (ABT) framework [outlined by scientist-filmmaker Randy Olson in his book *Houston, We Have a Narrative* (2015)] to construct a punchy, logical narrative rather than a recitation of fact, after fact, after fact."

## The next act

What's next in the research pursuit?

Adapted from Josh Ettinger (PhD student at University of Oxford, UK: "What Hollywood can teach researchers about scientific storytelling" https://www.nature.com/articles/d41586-020-01731-9

## Workshop breakout task 1:
## Transform the abstract into the first parts of a scientific story for an oral presentation

What are the basic elements of the research described here?  First, consider listing several facts from the abstract you deem important for the story.

Then, write up to 5 sentences that transform the abstract into the heart of the story for an oral presentation.

Then, let’s share.

# Multifunctional Ribosomal Proteins: Evidence for Extraribosomal Functions within the eRpL22 Family in *Drosophila melanogaster*


## Abstract

The eRpL22 family includes Rps that, based on known interactions with an array of cellular components, may have extraribosomal roles in cellular pathways and may be implicated in disease phenotypes. In *Drosophila melanogaster*, tissue-specific expression of Rp family paralogues in the adult testis contributes to ribosome heterogeneity by forming populations of specialized ribosomes containing eRpL22 or eRpL22-like with differential mRNA translation specificities. Here we report that tissue-specific overexpression of eRpL22, but not eRpL22-like, in *D. melanogaster* causes cellular overproliferation in the testis. The overproliferation phenotype can be recapitulated in *Drosophila* S2 cell lines. *In vivo* testicular overproliferation is partially phenocopied when eRpL22 overexpression is confined to somatic cyst cells and accessory glands using cell type-specific GAL4 drivers. The mechanism underlying this remains unknown but overexpression phenotypes are abolished in both S2 cells and testes when only the rRNA binding, C-terminal end of eRpL22 (lacking the *Drosophila*-specific histone H1-like domain) is overexpressed, suggesting a non-ribosomal role for the N-terminal domain. Additional evidence suggests that upregulation of eRpL22 also causes an increase in the cyclin-dependent kinase 7 (CDK7), without noticeable changes in cyclin A or B levels. Taken together, these data support the hypothesis that eRpL22 functions extraribosomally, through its N-terminal domain, in cellular pathways involving CDK7 in *Drosophila*. Experiments are ongoing to determine if direct interactions between eRpL22 and cell cycle components contribute to cell and tissue overgrowth.

Anecdote to “open” the story about multifunctional proteins that may also imprint the take home message with a nonscientific audience

- Anecdote: Personal Ballroom dancing story description

- **Take home message [AND]** Some proteins can carry out different functions depending on what proteins they bind to. If bound to protein A, they work in pathway A. But if bound to protein B, they function in a totally different pathway. Multifunctional proteins carry out different functions depending on their protein binding partners. [**BUT]** Disruption of binding to a partner can have consequences [**THEREFORE**] that cause disease because the normal pathway is not working.

## **Workshop breakout task 2**:

What are the basic elements of the research described here? First, consider listing several facts from the abstract you deem important for the story.  List the facts in the order in which they appear in the abstract (number the facts).

Then, write up to 5 sentences that transform the abstract into the beginning of a story for an oral presentation.

Then, let’s share.

# Investigation of homologous recombination between heterotypic phages within a prophage-mediated defense system


## Abstract

Comparative analyses of mycobacteriophage genomes reveals extensive genetic diversity in genome organization and gene content, contributing to widespread mosaicism. The evolutionary history of phages has been shaped by both homologous and illegitimate recombination mechanisms that drive horizontal gene transfer between phages and their bacterial host genomes. These mechanisms have largely been viewed as stochastic processes that occur given the presence of recombineering enzymes and sites of recombination. Relatively little is known about other factors that contribute to or drive recombination to effect genomic changes. Using phage infection assays, bacteriophage recombineering of electroporated DNA (BRED), and comparative genome analysis, we have discovered a prophage system within a Butters (cluster N mycobacteriophage) lysogen that constrains the outcome of infection by heterotypic phage Island3 (subcluster I1 mycobacteriophage). We have previously reported that a Butters prophage provides defense against infection by Island3 (Mageeney *et al.*, 2020). In an effort to isolate Island3 defense escape mutants, we isolated phages with recombinant genomes comprised of regions of Butters and Island3 arranged from left arm to right arm as Butters-Island3-Butters (BIBs). Recombination occurs within two distinct homologous regions that encompass *lysin A, lysin B,* and *holin* genes in one segment, and *RecE* and *RecT* genes in the other. Structural genes of the mosaic BIB genome are contributed by Butters while the immunity cassette is derived from Island3. Consequently, BIBs are morphologically identical to Butters (as shown by transmission electron microscopy) but are homoimmune with Island3. All plaques recovered from infections show evidence of hybrid phages called BIBs. A reverse experiment where an Island3 lysogen was infected with Butters yielded Butters phages and no recombinants, demonstrating a directionality to the recombination phenomenon. We hypothesize that Butters prophage-mediated defense is instrumental in directing recombination between an invading Island3 genome and the resident Butters prophage. To test this hypothesis that defense and recombination are linked, we scored outcomes of Island3 infection in mutant lysogens where specific prophage-expressed genes were deleted using BRED strategies. Of the mutant lysogens tested, we found that deletion of Butters *gene 57r* abolishes defense against Island3 and reduces the incidence of recombination by 90%. The certainty of recombination in the Butters prophage system suggests that anti-phage defense systems may contribute significantly to widespread mosaicism within phage genomes. To our knowledge, this work is the first empirical demonstration of a link between an anti-phage defense system and recombination between heterotypic phages. While the mechanism underlying this observed prophage-mediated recombination is unknown, our results highlight potential selective pressures that may drive recombination events leading to phage genome reshuffling and phage genome evolution.

# JUST For FUN

Find the connection.  Battle Phages: Phage against phage and the connection to  “Chang-Chi and the Legend of the Ten Rings”.

When is the sequence “3 1 2 4 5” better than “1 2 3 4 5”? Or does another sequence serve a better purpose?

Wrap up:

Comments/Questions?