Reflection Activity

1. Why might we care about scientific communication?
   When will we need to communicate science?

2. What makes you feel that any communication has been successful?
   As a receiver? As a sender?

We often blame ourselves for struggling to understand talks or papers…

> "I got stuck here. I feel like there was a huge logical leap I couldn't follow."

> "There's way too much going on in this plot. What am I supposed to be looking at?"

but poor communication is often the barrier, not your scientific understanding.

Science communication is complex

Many models, but some basic rules

For any task, try to find clear examples, dissect those examples, and apply those rules to your work

Build your own style, within the confines of basic design and writing expectations

In these workshops, we'll turn your instincts as a reader of science into tools for identifying…

WHEN scientific communication is confusing

WHY it's confusing

HOW to fix the problem

…and start applying these tools to your 20.109 work.

Here's how workshops will go

1. Look at examples from the field
2. Derive principles and strategies
3. Practice strategies
4. Go home with a checklist/rubric

…and our workshops are interactive,
so bring your energy and critical thinking,
and snacks.

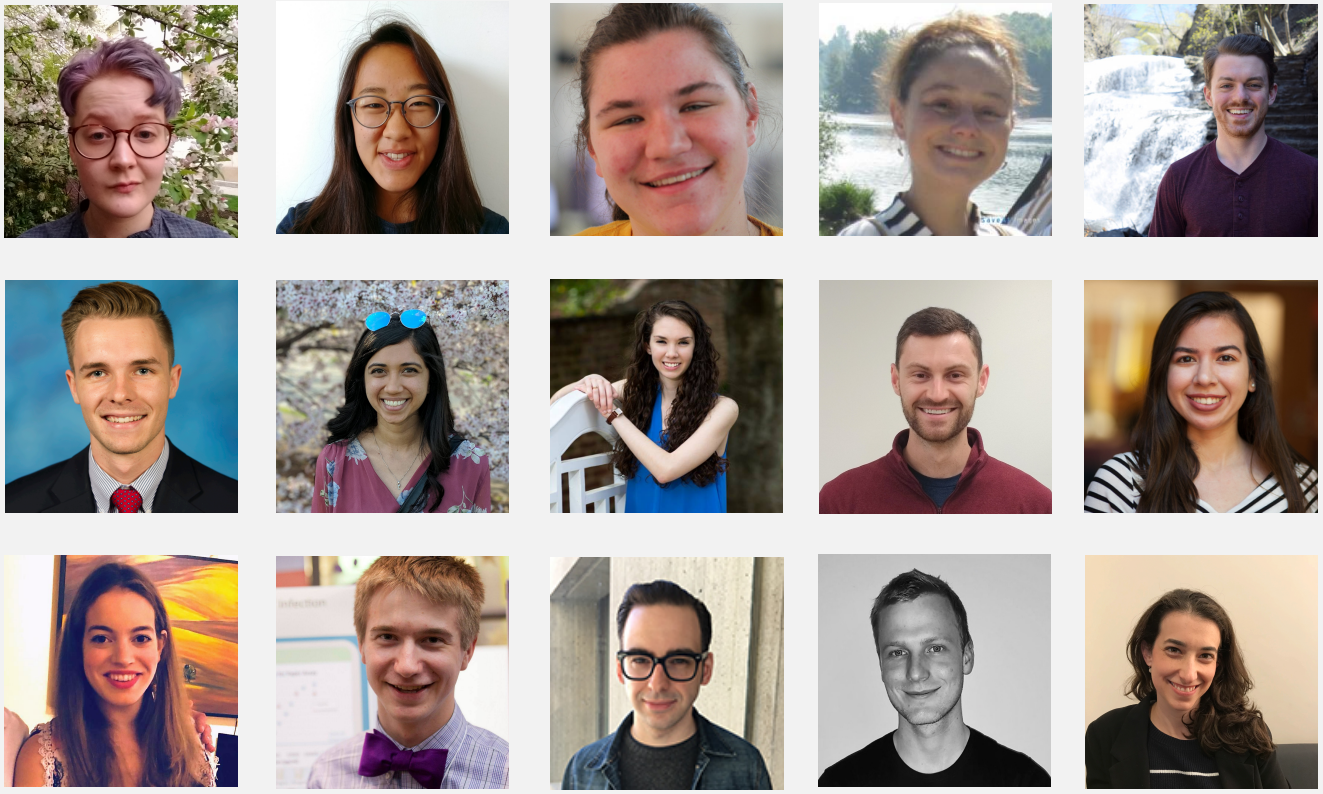
MIT BIOLOGICAL ENGINEERING COMMUNICATION LAB

- Science communication is discipline specific
- Needs to be learned by doing and getting feedback

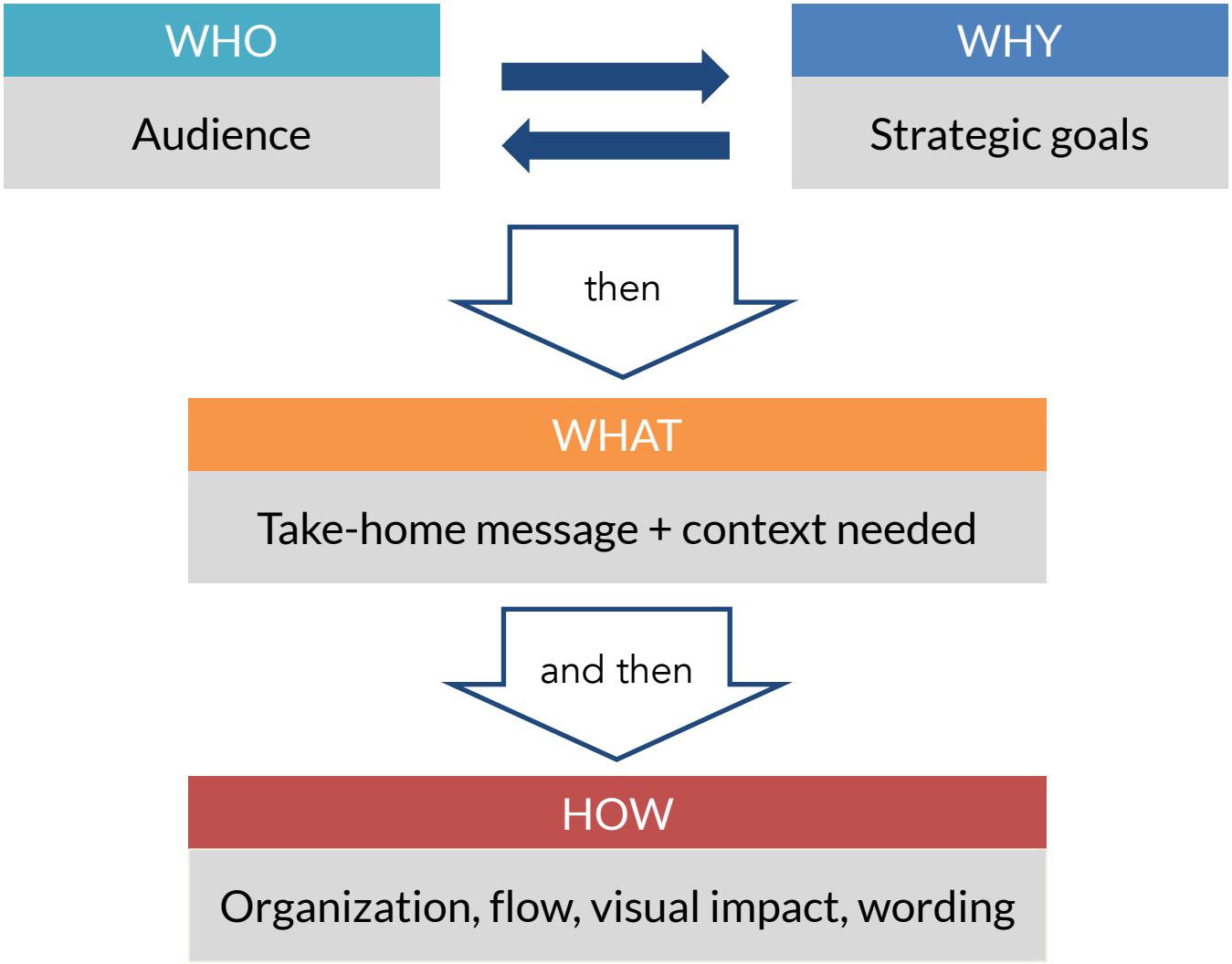
Dr. Prerna Bhargava
BE Communication Lab Manager and Instructor

Dr. Sean Clarke
Communication Instructor
Biotech Industry Liaison

Comm Lab Fellows are available to help you revise, rehearse, and get fresh perspective



We approach all communication tasks with a focus on **message**



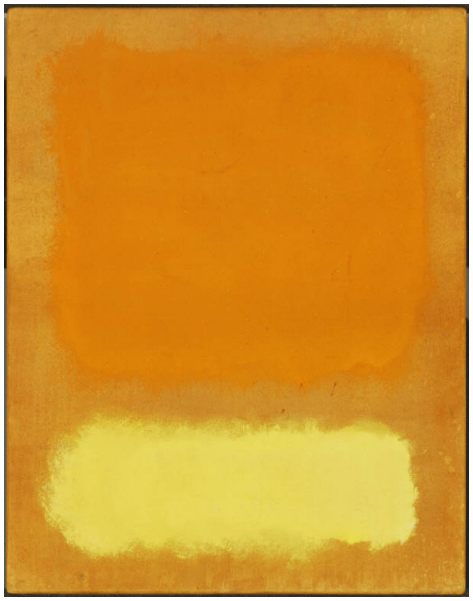
What to expect at an appointment

- Coaches will mostly ask questions and help you come up with answers
- Think about what you hope to get out of an appointment
- Do some prethinking and bring something thoughts, a figure, some text – something the fellow can work with

20.109 Communication Workshop 1: Titles and Abstracts

Dr. Prerna Bhargava

Dr. Sean Clarke



Untitled
Mark Rothko, 1968
Phillips Collection (Washington, DC)

MIT BIOLOGICAL ENGINEERING COMMUNICATION LAB

Helping you communicate effectively.
mitcommlab.mit.edu/be

Titles & Abstracts

WHO
Audience

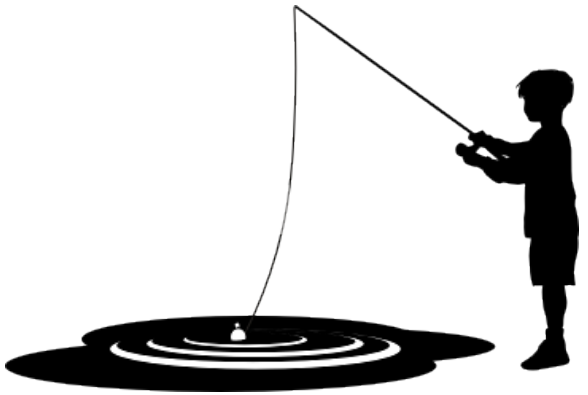
WHY
Strategic goals

Why do they matter?

Attracting your audience: first judgment

Influencing whether someone will read or cite your paper

Indexing – Will readers even find your paper?



Think about your last literature search

Google Scholar

protein runners

Articles

About 46,900 results (**0.15** sec)

Any time
Since 2020
Since 2019
Since 2016
Custom range...

Sort by relevance
Sort by date

include patents
include citations

Create alert

[HTML] Increased serum levels of non-collagenous matrix proteins (cartilage oligomeric matrix **protein** and melanoma inhibitory activity) in marathon **runners**

M Neidhart, U Müller-Ladner, W Frey… - Osteoarthritis and …, 2000 - Elsevier

Objective Marathon **runners** have an increased risk of developing joint disease. During and after a 42-km run, elevation of multiple cytokines occurs in the blood, reflecting inflammatory processes. We compared this cytokine response with serum levels of cartilage oligomeric …

☆ Cited by 177 Related articles All 10 versions Web of Science: 107

… troponin-I with creatine kinase, creatine kinase-MB isoenzyme, tropomyosin, myoglobin and C-reactive **protein** release in marathon **runners**: cardiac or skeletal …

P Cummins, A Young, ML Auckland… - European journal of …, 1987 - Wiley Online Library

Problems arise in distinguishing skeletal from cardiac muscle trauma on the basis of serum enzyme tests following severe muscle exercise. The contributions of cardiac and skeletal sources have been assessed in eleven marathon **runners** by measuring pre-and post-race …

☆ Cited by 173 Related articles All 4 versions Web of Science: 119 »

Metabolic effects of a **protein**-supplemented carbohydrate drink in marathon **runners**

PC Colombani, E Kovacs… - … Journal of Sport …, 1999 - journals.humankinetics.com

A field study was performed to investigate the acute influence of a milk **protein** hydrolysate supplemented drink (CHO+ PRO) on metabolism during and after a marathon run compared to the same drink without **protein** (CHO). Carbohydrate metabolites and hormones were not …

☆ Cited by 43 Related articles All 7 versions Web of Science: 20

Nutrient intake of endurance **runners** with ovo-lacto-vegetarian diet and regular western diet

M Eisinger, M Plath, K Jung, C Leitzmann - Zeitschrift für …, 1994 - Springer

… Also no differences were observed in the total intake of fat, **protein** and carbohydrates … of body weight and the increasing energy intake towards the end of the race signify that the **runners**, irrespective of … No **runner** reported stomach upsets or other problems of the digestive tract …

☆ Cited by 46 Related articles All 6 versions Web of Science: 9

PubMed protein runners

Create RSS Create alert Advanced

Format: Summary **Sort by**: Most Recent **Per page**: 20 **Send to**

Best matches for protein runners:

Exercise-induced bronchoconstriction, temperature regulation and the role of heat shock **proteins** in non-asthmatic recreational marathon and half-marathon **runners**.
Bekos C et al. Sci Rep. (2019)

Exercise-induced Changes in Soluble ST2 Concentrations in Marathon **Runners**.
Aengevaeren VL et al. Med Sci Sports Exerc. (2019)

Salivary immunity and lower respiratory tract infections in non-elite marathon **runners**.
Cantó E et al. PLoS One. (2018)

Switch to our new best match sort order

Search results

Items: 1 to 20 of 1389

<< First < Prev Page 1 of 70 Next > Last >>

1. Improvements in Skeletal Muscle Can Be Detected Using Broadband NIRS in First-Time Marathon **Runners**.
Jones S, Kinsella M, Torlasco C, Kaynezhad P, de Roever I, Moon JC, Hughes AD, Bale G.
Adv Exp Med Biol. 2020;1232:245-251. doi: 10.1007/978-3-030-34461-0_31.
PMID: 31893417
Similar articles

2. The Short Tandem Repeat of the *DMT1* Gene as a Molecular Marker of Elite Long-Distance **Runners**.
Wuyun G, Hu Y, He Z, Li Y, Yan X.
Int J Genomics. 2019 Nov 23;2019:7064703. doi: 10.1155/2019/7064703. eCollection 2019.
PMID: 31871928 **Free PMC Article**
Similar articles

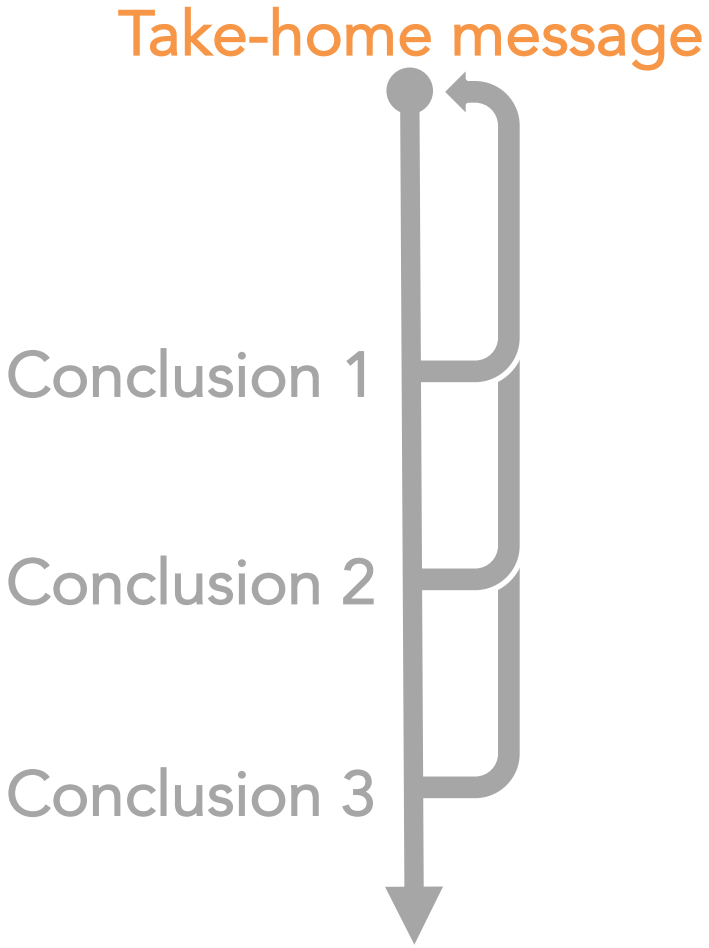
Titles & Abstracts

Who is your audience?

- People in your field
- Editors, reviewers
- Researchers outside your field
- Students like you!
- Reporters
- Funders, politicians
- Anyone looking for information

Your title and abstract convey your take-home message

WHAT
Take-home message



Why was this an important study?

How does it further scientific thinking?

Why should anyone read your paper?

Titles

Let’s take a look at these titles

Nanopore long-read RNAseq reveals widespread transcriptional variation among the surface receptors of individual B cells

Small-molecule inhibitor targeting the HSP90-Cdc37 protein-protein interaction in colorectal cancer

Effective titles are messages

What did you find? So what?

A survey of small molecules with ligand binding activity

vs.

Conserved hydroxyl and carbonyl ligand structures are implicated in high-affinity receptor binding

Build and simplify your title with key terms

KEY NOUNS

KEY VERBS

Novel methods for early prediction of undesirable interference by microbial inhabitants of the human gut with metabolism of the cardiac drug digoxin give rise to strategies for alleviating drug inactivation

NEW AND IMPROVED TITLE

Predicting and alleviating drug interference by human gut microbiome

TOO SIMPLIFIED = LESS INFORMATIVE

Novel methods for prediction of drug interference

Frame titles for your audience

The level of detail can vary for the same paper

Inulin modulates conspecific antagonism towards vancomycin-resistant *B. subtilis* strain BF819 in the human gut microbiome

vs.

A human gut commensal exhibits targeted antagonism towards an antibiotic-resistant clinical counterpart

Remember your audience to condense jargon for concision

Surveying somatic mutations in P53, EGFR, BRCA1, and HRAS for impact on MCF7 tumors with heterogeneous cell composition.

Replace jargon to attract a broader audience

Surveying the impact of breast cancer oncogenes on tumor heterogeneity

What if your story doesn't seem conclusive?

- Tell your story in a different way:
  - focus on the technology?
  - what did you learn?
- Convey negative results

A Raf-Competitive K-Ras Binder Can Fail to Functionally Antagonize Signaling.

Brief Communications Arising | 19 September 2018

Evidence that CD32a does not mark the HIV-1 latent reservoir

- Make a descriptive title that's clear and interesting

Would you change anything about these titles?

Nanopore long-read RNAseq reveals widespread transcriptional variation among the surface receptors of individual B cells

Small-molecule inhibitor targeting the HSP90-Cdc37 protein-protein interaction in colorectal cancer

Abstracts

Unscramble this real abstract

In 5 minutes:

- Read all the sentences
- Look for signaling language
- Number the sentences in logical order

Clonal dynamics of native haematopoiesis.

Nature. 2014 Oct 16; 514(7522): 322–327.
Sun J, Ramos A, Chapman B, Johnnidis JB, Le L, Ho YJ, Klein A, Hofmann O, Camargo FD.

Assemble this abstract

1. It is currently thought that life-long blood cell production is driven by the action of a small number of multipotent haematopoietic stem cells.

2. Evidence supporting this view has been largely acquired through the use of functional assays involving transplantation.

3. However, whether these mechanisms also govern native non-transplant haematopoiesis is entirely unclear.

4. Here we have established a novel experimental model in mice where cells can be uniquely and genetically labelled *in situ* to address this question.

5. Using this approach, we have performed longitudinal analyses of clonal dynamics in adult mice that reveal unprecedented features of native haematopoiesis.

6. In contrast to what occurs following transplantation, steady-state blood production is maintained by the successive recruitment of thousands of clones, each with a minimal contribution to mature progeny.

7. Our results demonstrate that a large number of long-lived progenitors, rather than classically defined haematopoietic stem cells, are the main drivers of steady-state haematopoiesis during most of adulthood.

8. Our results also have implications for understanding the cellular origin of haematopoietic disease.

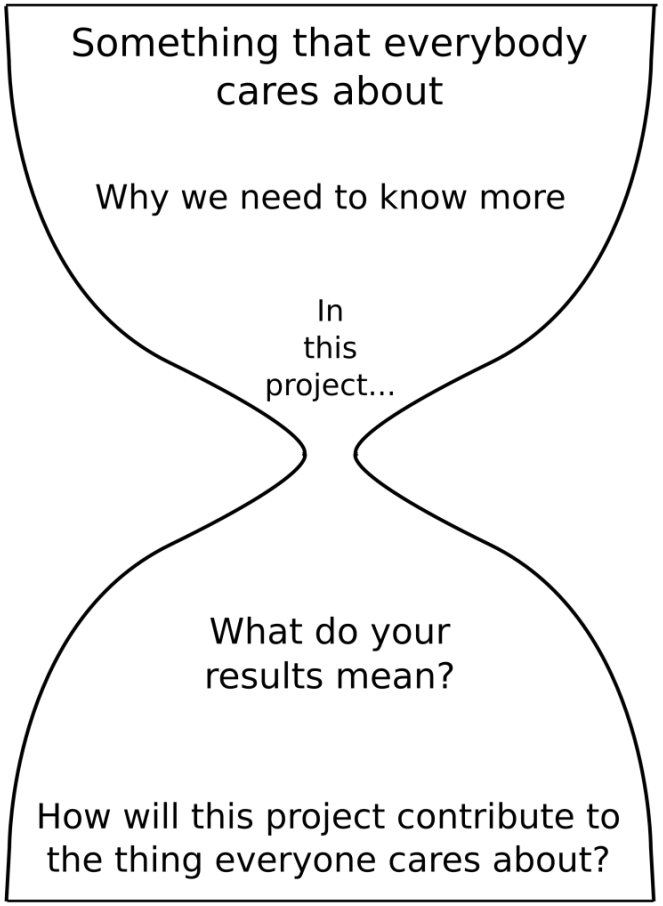
Clonal dynamics of native haematopoiesis.

Sun J, Ramos A, Chapman B, Johnnidis JB, Le L, Ho YJ, Klein A, Hofmann O, Camargo FD.

Abstract

It is currently thought that life-long blood cell production is driven by the action of a small number of multipotent haematopoietic stem cells. Evidence supporting this view has been largely acquired through the use of functional assays involving transplantation. However, whether these mechanisms also govern native non-transplant haematopoiesis is entirely unclear. Here we have established a novel experimental model in mice where cells can be uniquely and genetically labelled in situ to address this question. Using this approach, we have performed longitudinal analyses of clonal dynamics in adult mice that reveal unprecedented features of native haematopoiesis. In contrast to what occurs following transplantation, steady-state blood production is maintained by the successive recruitment of thousands of clones, each with a minimal contribution to mature progeny. Our results demonstrate that a large number of long-lived progenitors, rather than classically defined haematopoietic stem cells, are the main drivers of steady-state haematopoiesis during most of adulthood. Our results also have implications for understanding the cellular origin of haematopoietic disease.

An effective abstract is an hourglass-shaped message.



General background

Specific background

Knowledge gap, Unknown

HERE WE SHOW…

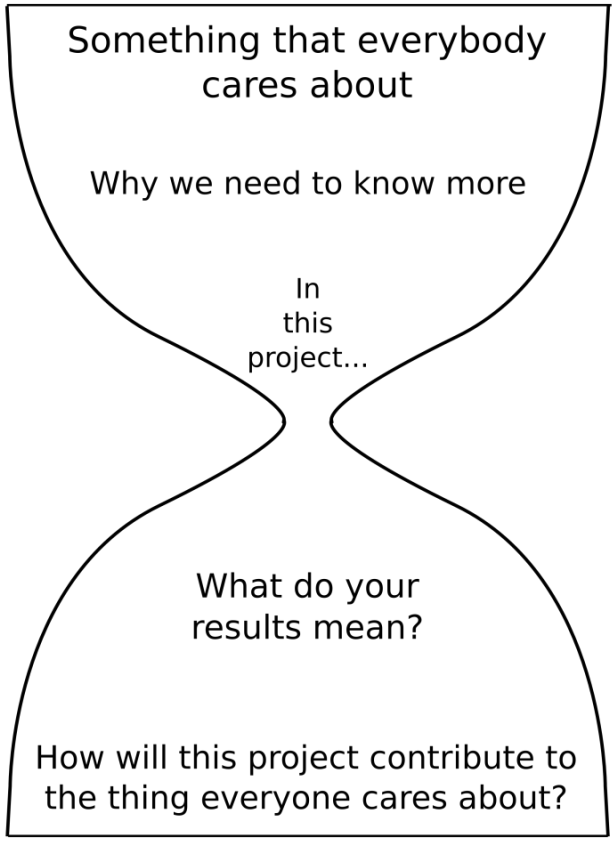
Results

Implication

Significance

Remember to answer these questions for your reader in your abstract

1. What is the **problem**?
2. Where is the **gap**?
3. What did you **do**?
4. What is the **implication**?



The hourglass structure mapped onto our abstract

1. It is currently thought that life-long blood cell production is driven by the action of a small number of multipotent haematopoietic stem cells.

General background

2. Evidence supporting this view has been largely acquired through the use of functional assays involving transplantation.

Specific background

3. However, whether these mechanisms also govern native non-transplant haematopoiesis is entirely unclear.

Knowledge gap, Unknown

4. Here we have established a novel experimental model in mice where cells can be uniquely and genetically labelled *in situ* to address this question.

HERE WE SHOW…

5. Using this approach, we have performed longitudinal analyses of clonal dynamics in adult mice that reveal unprecedented features of native haematopoiesis.

Results

6. In contrast to what occurs following transplantation, steady-state blood production is maintained by the successive recruitment of thousands of clones, each with a minimal contribution to mature progeny.

Results

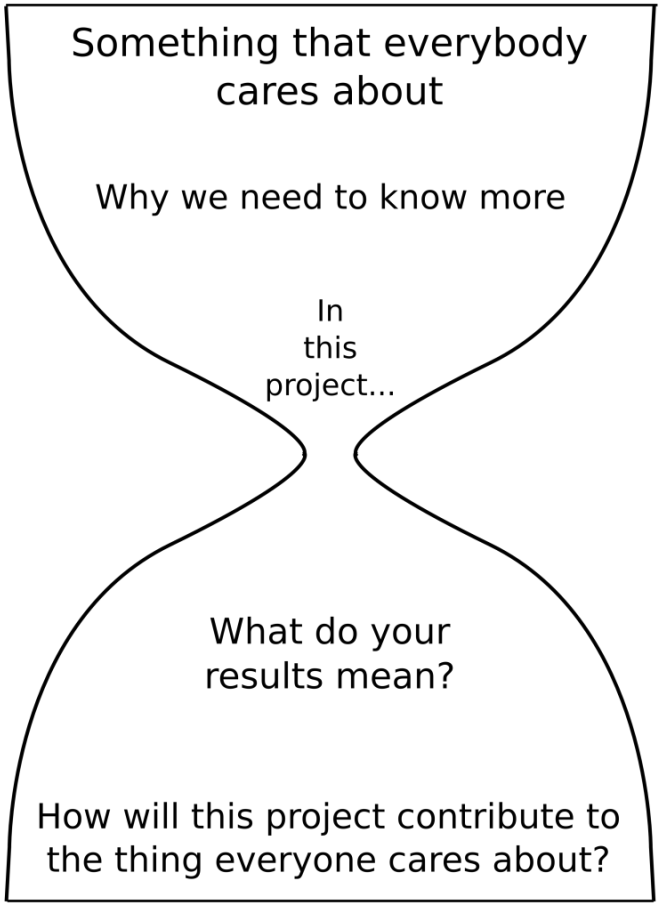
7. Our results demonstrate that a large number of long-lived progenitors, rather than classically defined haematopoietic stem cells, are the main drivers of steady-state haematopoiesis during most of adulthood.

Implication

8. Our results also have implications for understanding the cellular origin of haematopoietic disease.

Significance

Create an argument to convince readers that your work is important



General background

Specific background

Knowledge gap, Unknown

HERE WE SHOW…

Results

Implication

Significance

argument = claim + evidence + reasoning

Claim	A statement of our understanding about a phenomenon, about the outcome of a study, or about the author's view of the field
Evidence	Data to support the claim
Reasoning	Justification of the claim that shows **how** the evidence specifically supports the claim

Your abstract should contain at least one claim, which is your take home message

Here we have established a novel experimental model in mice where cells can be uniquely and genetically labelled *in situ* to address this question.

HERE WE SHOW… (CLAIM)

Using this approach, we have performed longitudinal analyses of clonal dynamics in adult mice that reveal unprecedented features of native haematopoiesis.

Results (Evidence)

In contrast to what occurs following transplantation, steady-state blood production is maintained by the successive recruitment of thousands of clones, each with a minimal contribution to mature progeny.

Results (Evidence)

Our results demonstrate that a large number of long-lived progenitors, rather than classically defined haematopoietic stem cells, are the main drivers of steady-state haematopoiesis during most of adulthood.

Implication (Reasoning)

The knowledge gap and "here we show" are typically next to each other, creating a logical flow for the reader.

However, whether these mechanisms also govern native non-transplant haematopoiesis is entirely unclear.

Knowledge gap, Unknown

Here we have established a novel experimental model in mice where cells can be uniquely and genetically labelled *in situ* to address this question.

HERE WE SHOW…

From this "here we show" statement, what would you expect the title of the paper to be?

The title and “here we show” statement reflect the same content

Clonal dynamics of native haematopoiesis.

However, whether these mechanisms also govern native non-transplant haematopoiesis is entirely unclear.

Knowledge gap, Unknown

Here we have established a novel experimental model in mice where cells can be uniquely and genetically labelled *in situ* to address this question.

HERE WE SHOW…

Your results should reflect your take home message

Technology Focus

Here we show that RNA-seq can be used to identify mechanisms of drug action within a cell.

1. What data did you use?
2. What analysis tools?
3. Did you find any interesting pathways?

Biology Focus

Here we use a cell viability assay and analysis of RNA-seq data to understand the mechanism through which target cells have increased survival after drug treatment.

1. What did you learn about the mechanism from these assays?
2. What can you do next?

Be quantitative about the results that you include

Signaling words help guide the reader

Question + Experiment	Results	Answer/ Conclusion	Implication
To determine whether…, we…	We found…	We conclude that…	These results suggest that…
We asked whether…	Our results show…	Thus,…	These results may play a role in…
To answer this question, we…	Here we report…	These results indicate that…	Y can be used to…
X was studied by…			

Read lots of abstracts and collect useful phrases, choose clarity over originality.

Tense in abstracts is a little tricky

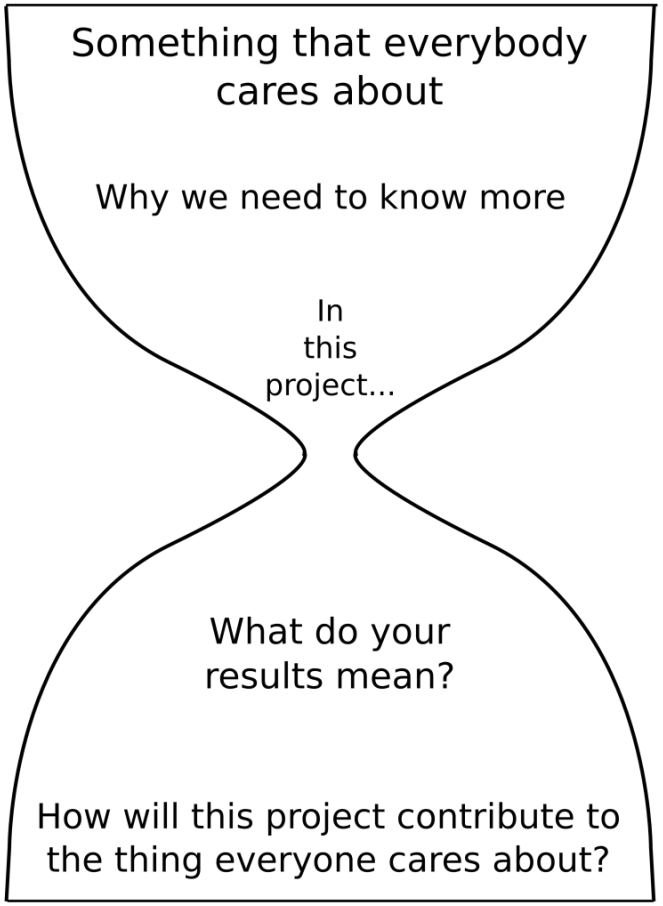
Present Tense | Past Tense

Disrupting the interactions between Hsp90 and Cdc37 is emerging as an alternative and specific way to regulate the Hsp90 chaperone cycle in a manner not involving adenosine triphosphatase inhibition. Here, we identified DDO-5936 as a small-molecule inhibitor of the Hsp90-Cdc37 protein-protein interaction (PPI) in colorectal cancer. DDO-5936 disrupted the Hsp90-Cdc37 PPI both in vitro and in vivo via binding to a previously unknown site on Hsp90 involving Glu47, one of the binding determinants for the Hsp90-Cdc37 PPI, leading to selective downregulation of Hsp90 kinase clients in HCT116 cells. In addition, inhibition of Hsp90-Cdc37 complex formation by DDO-5936 resulted in a remarkable cyclin-dependent kinase 4 decrease and consequent inhibition of cell proliferation through Cdc37-dependent cell cycle arrest. Together, our results demonstrated DDO-5936 as an identified specific small-molecule inhibitor of the Hsp90-Cdc37 PPI that could be used to comprehensively investigate alternative approaches targeting Hsp90 chaperone cycles for cancer therapy.

Protip: Avoid novelty claims.

- Unless you've read every paper, you don't really know if you're the first to discover something.
- A surprising result: unanticipated, or against common dogma, but not unprecedented
- Appropriately qualified, there are certain "firsts" you do know…

An effective abstract is an hourglass-shaped message.



General background

Specific background

Knowledge gap, Unknown

HERE WE SHOW…

Results

Implication

Significance

Activity

Read the abstracts from the two papers.

What questions did the authors address in their abstracts?

Nanopore long-read RNAseq reveals widespread transcriptional variation among the surface receptors of individual B cells

Understanding gene regulation and function requires a genome-wide method capable of capturing both gene expression levels and isoform diversity at the single-cell level. Short-read RNAseq is limited in its ability to resolve complex isoforms because it fails to sequence full length cDNA copies of RNA molecules. Here, we investigate whether RNAseq using the long-read single-molecule Oxford Nanopore MinION sequencer is able to identify and quantify complex isoforms without sacrificing accurate gene expression quantification. After benchmarking our approach, we analyze individual murine B1a cells using a custom multiplexing strategy. We identify thousands of unannotated transcription start and end sites, as well as hundreds of alternative splicing events in these B1a cells. We also identify hundreds of genes expressed across B1a cells that display multiple complex isoforms, including several B cell specific surface receptors. Our results show that we can identify and quantify complex isoforms at the single cell level.

Problem

Gap

Here we show

Implication

Small-molecule inhibitor targeting the Hsp90-Cdc37 protein-protein interaction in colorectal cancer

Disrupting the interactions between Hsp90 and Cdc37 is emerging as an alternative and specific way to regulate the Hsp90 chaperone cycle in a manner not involving adenosine triphosphatase inhibition. Here, we identified DDO-5936 as a small-molecule inhibitor of the Hsp90-Cdc37 protein-protein interaction (PPI) in colorectal cancer. DDO-5936 disrupted the Hsp90-Cdc37 PPI both in vitro and in vivo via binding to a previously unknown site on Hsp90 involving Glu47, one of the binding determinants for the Hsp90-Cdc37 PPI, leading to selective downregulation of Hsp90 kinase clients in HCT116 cells. In addition, inhibition of Hsp90-Cdc37 complex formation by DDO-5936 resulted in a remarkable cyclin-dependent kinase 4 decrease and consequent inhibition of cell proliferation through Cdc37-dependent cell cycle arrest. Together, our results demonstrated DDO-5936 as an identified specific small-molecule inhibitor of the Hsp90-Cdc37 PPI that could be used to comprehensively investigate alternative approaches targeting Hsp90 chaperone cycles for cancer therapy.

Problem

Gap

Here we show

Implication

Take-homes for Titles and Abstracts:

- **Highlight your take-home** message: identify your research question & your contribution
- Focus on **findings**, not methods.
- Be **succinct**.
- Be **quantitative**.

Unscramble this abstract

Evidence supporting this view has been largely acquired through the use of functional assays involving transplantation.

Using this approach, we have performed longitudinal analyses of clonal dynamics in adult mice that reveal unprecedented features of native haematopoiesis.

Our results demonstrate that a large number of long-lived progenitors, rather than classically defined haematopoietic stem cells, are the main drivers of steady-state haematopoiesis during most of adulthood.

It is currently thought that life-long blood cell production is driven by the action of a small number of multipotent haematopoietic stem cells.

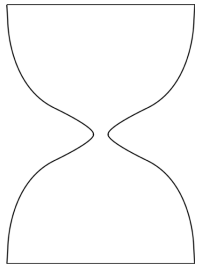
Our results also have implications for understanding the cellular origin of haematopoietic disease.

Here we have established a novel experimental model in mice where cells can be uniquely and genetically labelled in situ to address this question.

However, whether these mechanisms also govern native non-transplant haematopoiesis is entirely unclear.

In contrast to what occurs following transplantation, steady-state blood production is maintained by the successive recruitment of thousands of clones, each with a minimal contribution to mature progeny.

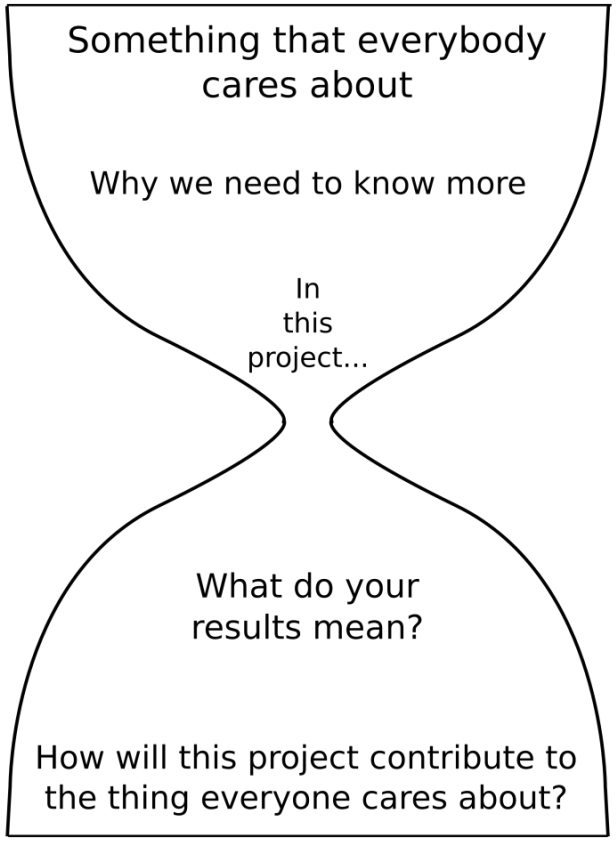
Here are the components of an effective abstract



General background	Something everyone in your audience cares about
Specific background	Zoom in from General Background toward what you did
Knowledge gap, Unknown	Question that will be answered by your research, or a problem, phenomenon that is not understood
HERE WE SHOW	Conclusion, answer to the Unknown
Results	Brief summary of approach + very high-level results. Common pitfall = too much of Methods/Results
Implication, Significance	So what? What do your results mean for the thing everyone cares about? Next steps?

Remember to answer these questions for your reader in your abstract

1. What is the **problem**?
2. Where is the **gap**?
3. What did you **do**?
4. What is the **implication**?



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Small-molecule inhibitor targeting the Hsp90-Cdc37 protein-protein interaction in colorectal cancer

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Include a minimal description of your key methods, (if it aligns with your message)

We first used small molecule microarrays to screen for ligands that could bind to FKBP12. Next, PPIase (peptidyl-prolyl cis-trans isomerase) assay was conducted to determine the level of FKBP12 protein activity. Then, a DSF (differential scanning fluorimetry) assay was conducted to confirm ligand binding to FKBP12.

Too much for an abstract

We further evaluated two potential binders, ligands 18 and 28, using peptidyl-prolyl isomerase (PPIase) and differential scanning fluorimetry (DSF) assays to confirm ligand binding.