How to write scientific papers

P. A. Wiggins for Phys 494
w/ content from Cin-Ty Lee
& cartoons from the internet!

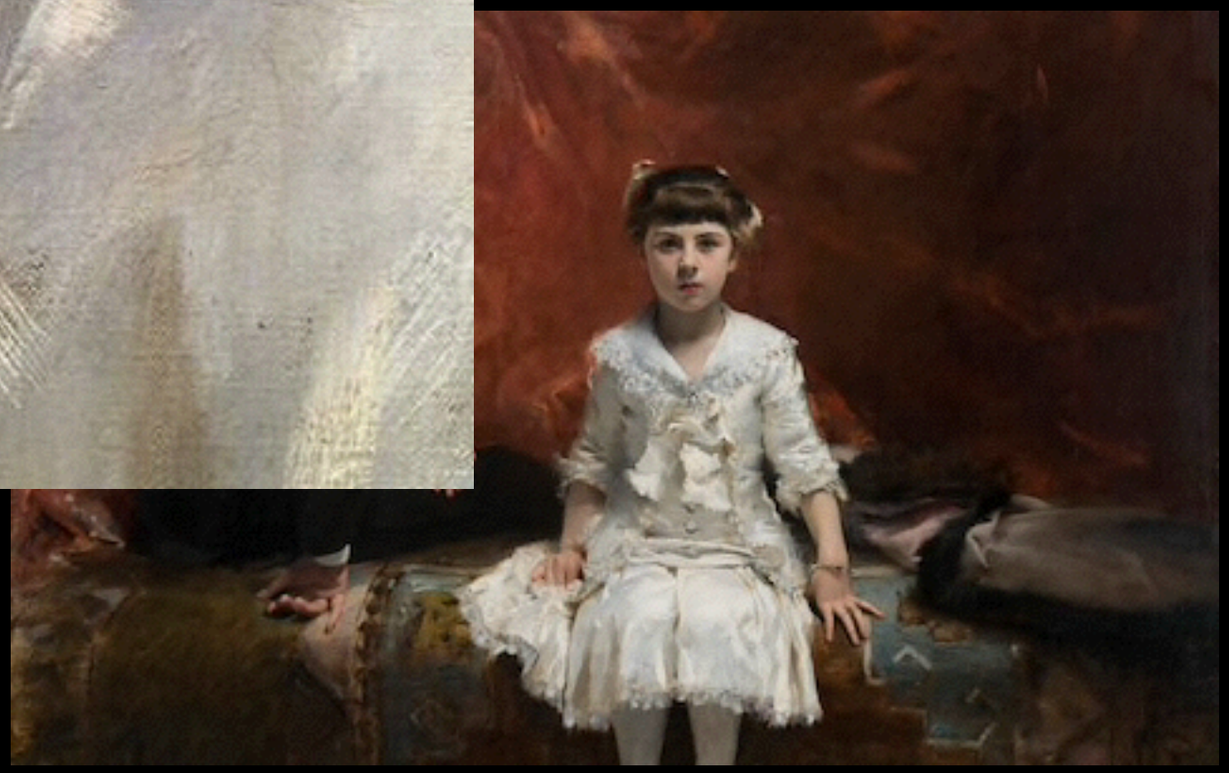
What makes a good writer?

- Knowledge of the subject or topic
- Passion for the subject
- A logical and creative mind
- A desire to communicate
- Appreciation for who the audience is
- **A willingness to revise and rewrite**

Your writing is *a form of art*…

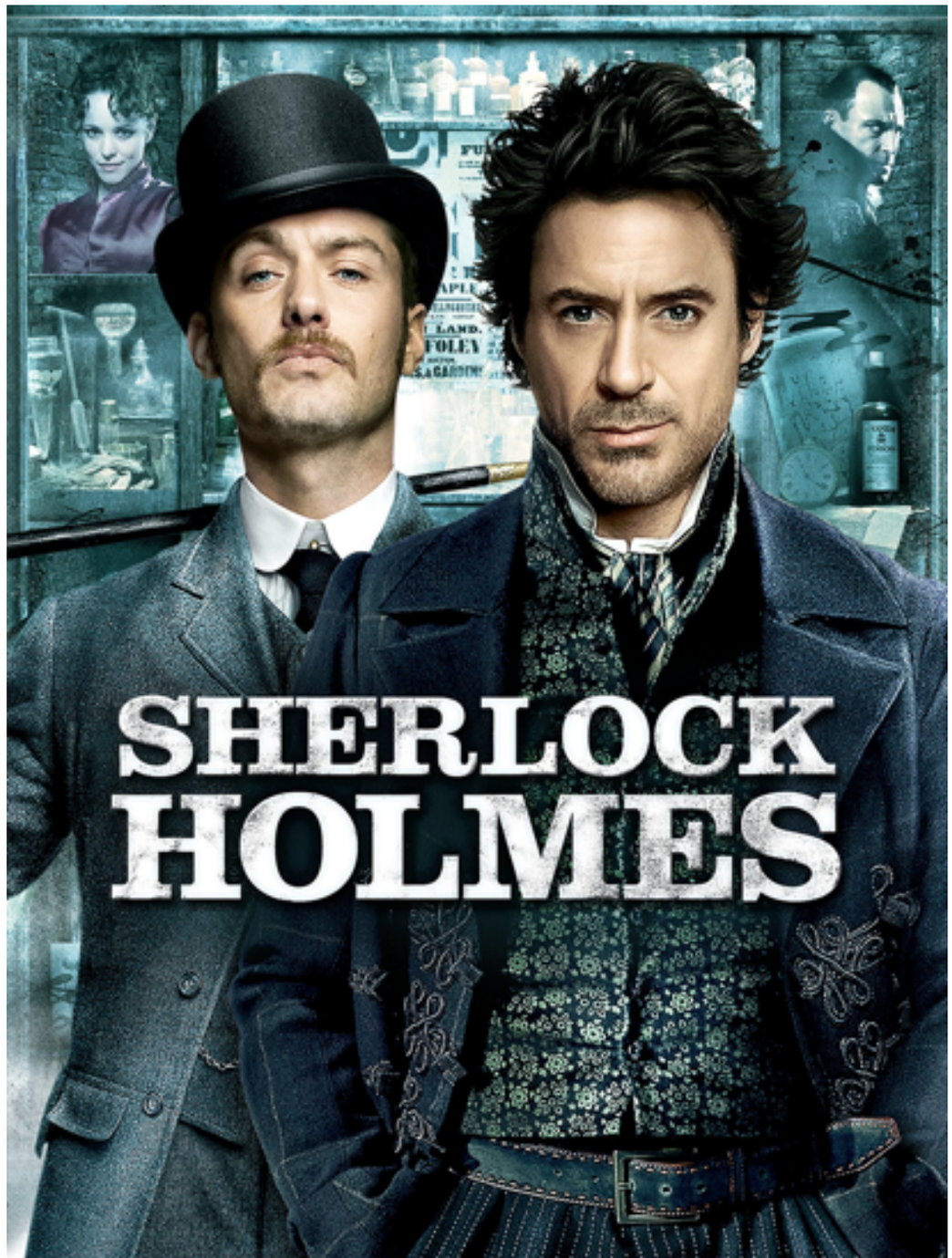
- Creative expression
  - never copy someone else's work (plagiarism)
- good writer & artist require similar skills
  - You need to be your own editor

… just as many words as are necessary, but not a word more…



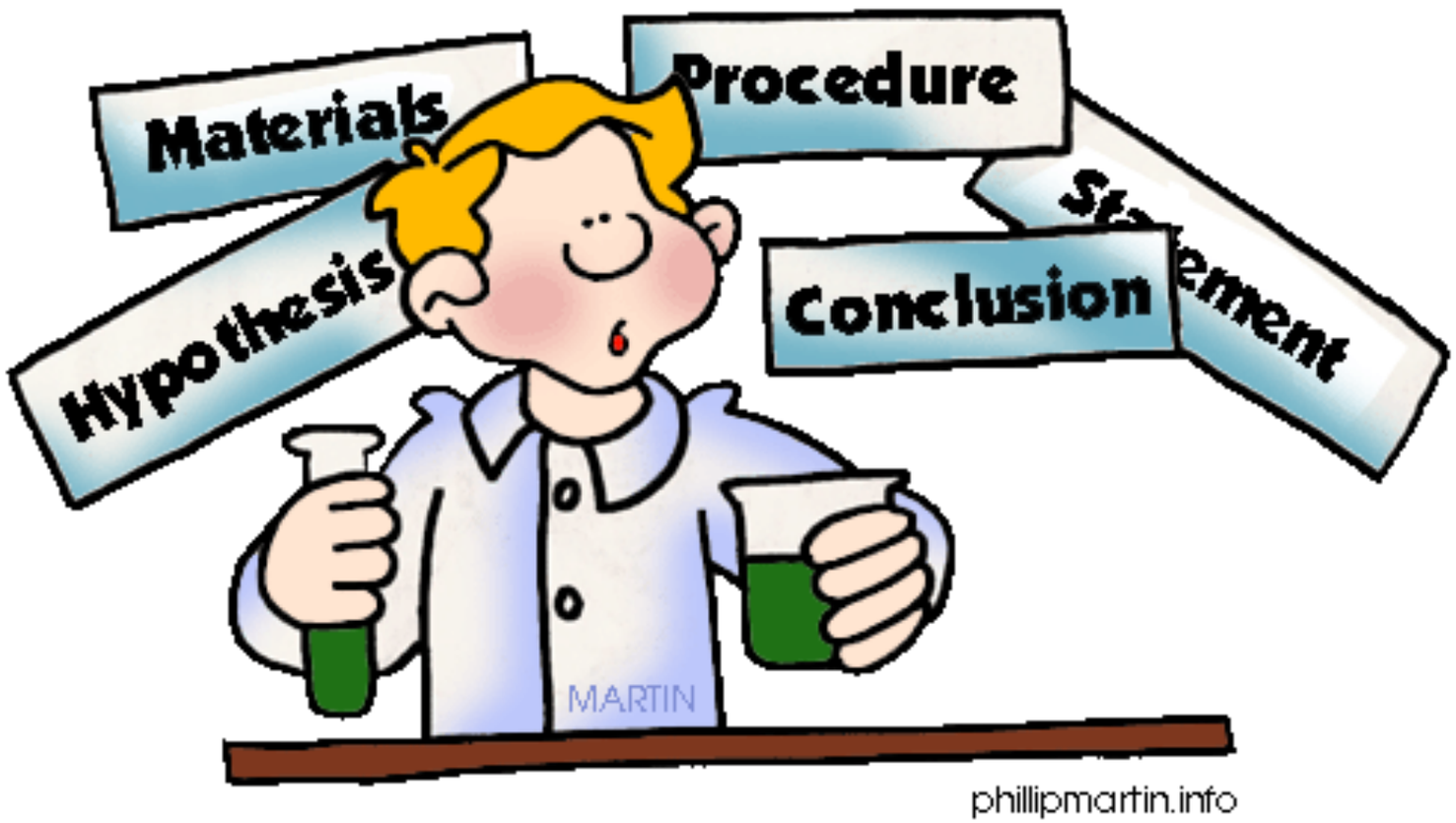
Scientific papers ~ mystery novels

- Identify the mystery or problem to be solved
- Describe the methods used to gather data (evidence).
- Present the data (evidence) in an unbiased way
- Analyze the data
- Interpret and discuss the data
- Conclude



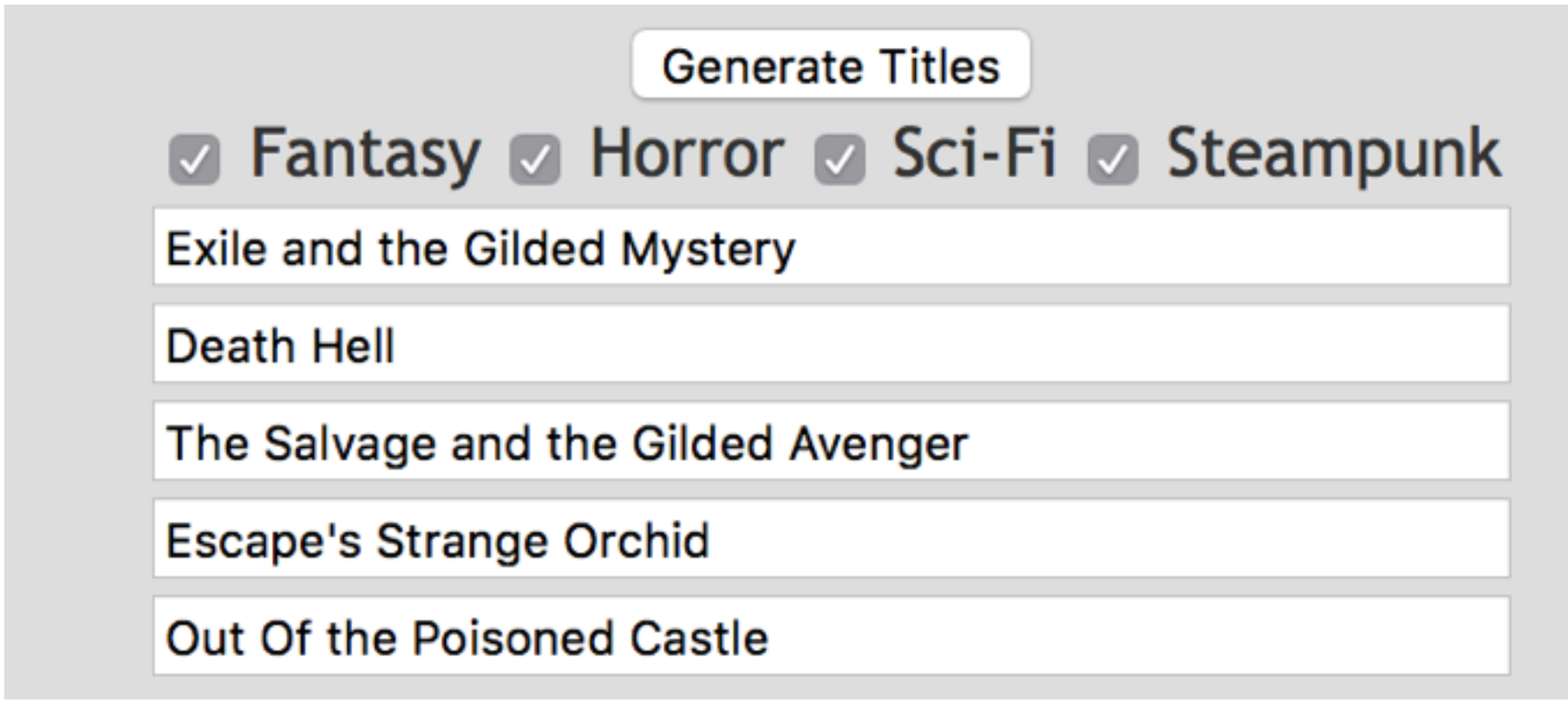
Structuring a scientific paper….

- **Follow this recipe to the letter!**
- Structure:
  - Title & abstract
  - Introduction
  - Methods
  - Results
  - Discussion
  - Conclusion



Title

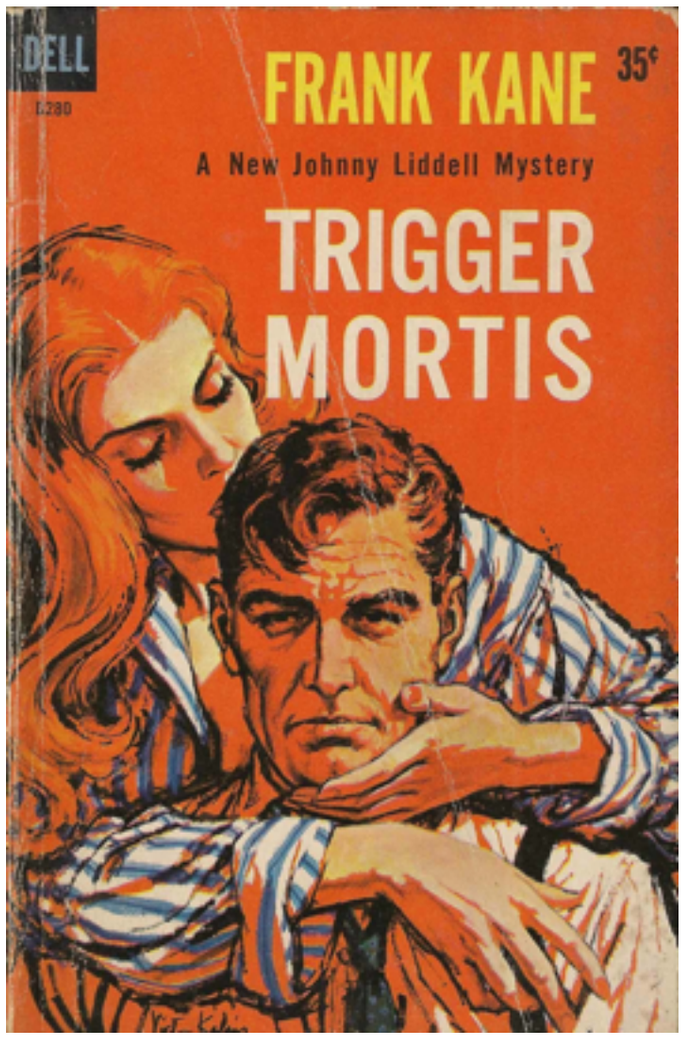
- The title must be a concise and informative phrase.
- This is the first thing a potential reader will see.



Example:

Structural basis for ArfA–RF2–mediated translation termination on mRNAs lacking stop codons

Paul Huter[1]*, Claudia Müller[1]*, Bertrand Beckert[1,2], Stefan Arenz[1], Otto Berninghausen[1], Roland Beckmann[1] & Daniel N. Wilson[1,2]



Abstract

- One paragraph
- summarizes the paper.
- Consists of the following:

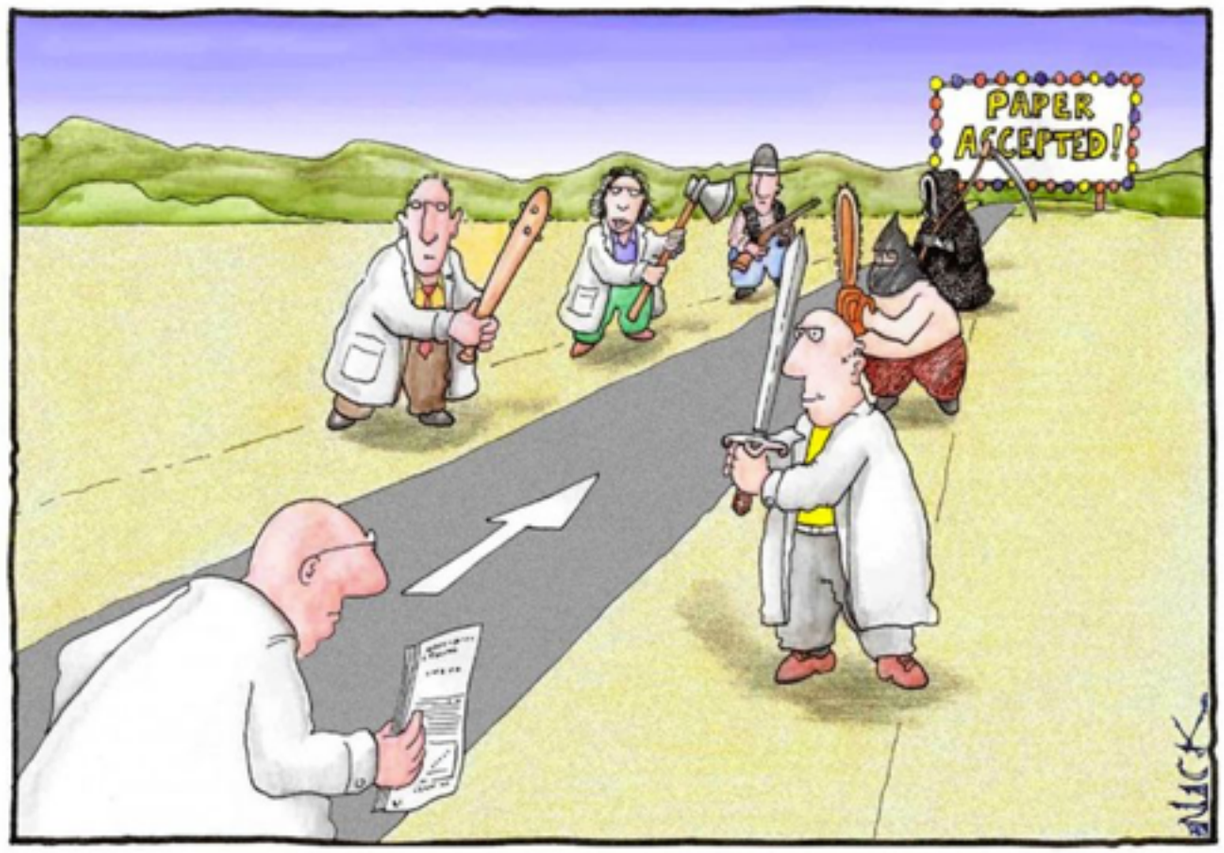
1. Motivate the problem
2. Methods (establishing credibility),
3. Summarize important results
4. Conclusions

Example:

In bacteria, ribosomes stalled on truncated mRNAs that lack a stop codon are rescued by the transfer-messenger RNA (tmRNA), alternative rescue factor A (ArfA) or ArfB systems[1]. Although tmRNA–ribosome and ArfB–ribosome structures have been determined[2-7], how ArfA recognizes the presence of truncated mRNAs and recruits the canonical termination release factor RF2 to rescue the stalled ribosomes is unclear. Here we present a cryo-electron microscopy reconstruction of the *Escherichia coli* 70S ribosome stalled on a truncated mRNA in the presence of ArfA and RF2. The structure shows that the C terminus of ArfA binds within the mRNA entry channel on the small ribosomal subunit, and explains how ArfA distinguishes between ribosomes that bear truncated or full-length mRNAs. The N terminus of ArfA establishes several interactions with the decoding domain of RF2, and this finding illustrates how ArfA recruits RF2 to the stalled ribosome. Furthermore, ArfA is shown to stabilize a unique conformation of the switch loop of RF2, which mimics the canonical translation termination state by directing the catalytically important GGQ motif within domain 3 of RF2 towards the peptidyl-transferase centre of the ribosome. Thus, our structure reveals not only how ArfA recruits RF2 to the ribosome but also how it promotes an active conformation of RF2 to enable translation termination in the absence of a stop codon.

Introduction

- Motivate the reader by identifying the question or problem
- Explaining broader significance
- Provide necessary background information
- Present your approach to solving the problem
- Concluded with a brief summary of the important results
- Write for the non-specialist. Keep jargon to a minimum.



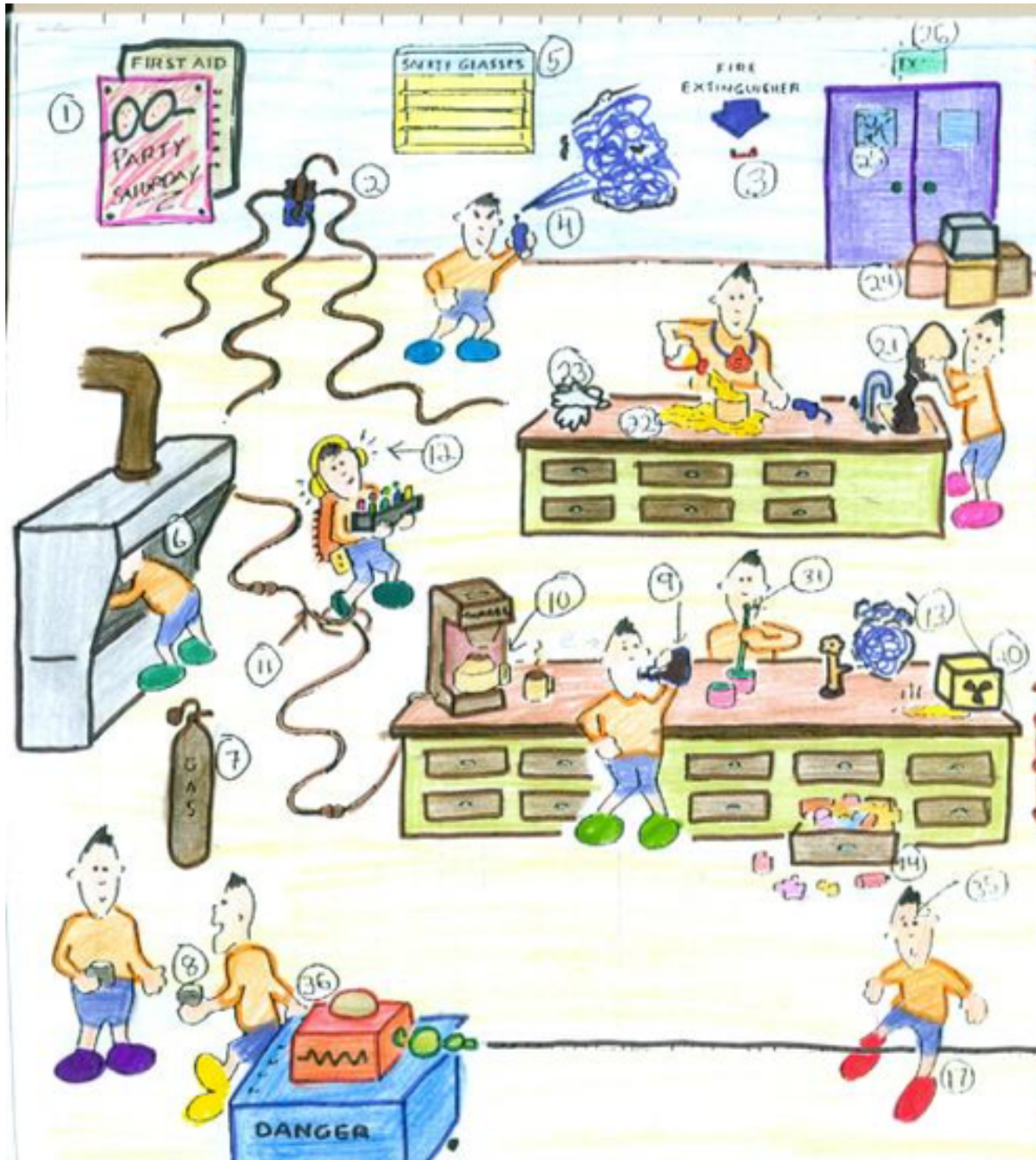
Most scientists regarded the new streamlined peer-review process as "quite an improvement."

Example:

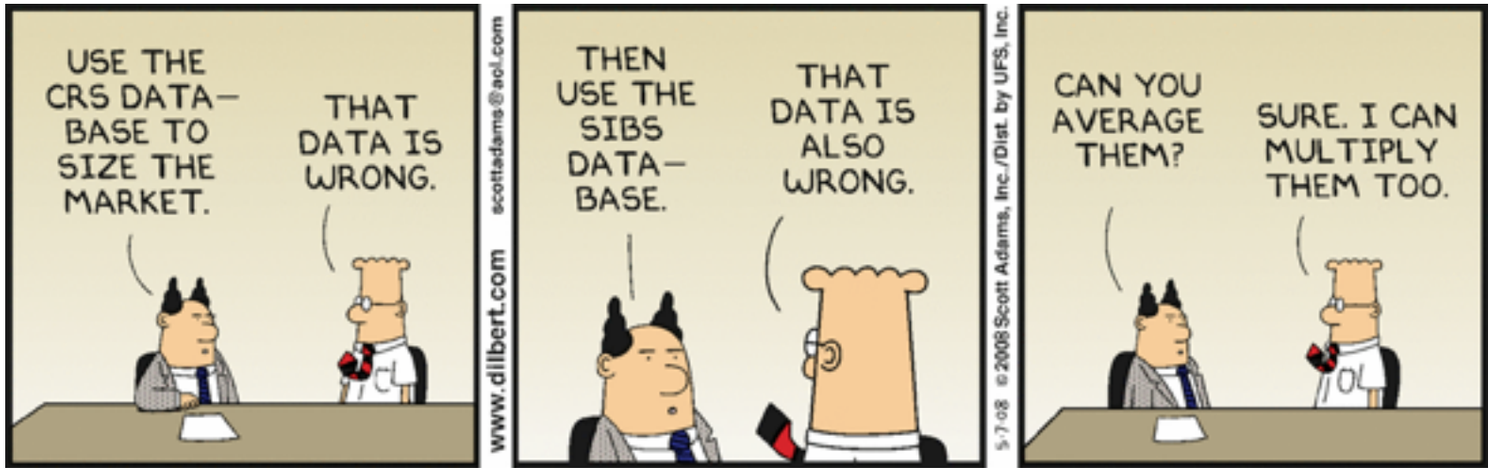
Premature transcription termination or truncation of a full-length mRNA can lead to mRNAs lacking a stop codon. Ribosomes translating these truncated mRNAs become trapped at the 3′ end of the mRNA because translation elongation or termination cannot occur. In bacteria, these stalled ribosomes are recognized and recycled by the tmRNA rescue system (reviewed in ref. 1). A subset of bacteria, such as *E. coli*, can survive without the tmRNA system owing to the presence ArfA[8]. The synthetic lethality arising from inactivation of both the tmRNA and ArfA rescue systems can be alleviated by overexpression of ArfB[9]. Collectively, these studies illustrate the physiological importance that the rescue of stalled ribosomes has for cell viability. Structural studies have revealed how ribosomes stalled on truncated mRNA are recognized and recycled by the tmRNA–SmpB complex[6,7] or ArfB[5]. In the case of ArfB, the empty mRNA channel of the ribosome is probed by the C-terminal helix, positioning the N-terminal catalytic GGQ-containing domain at the peptidyl-transferase centre (PTC) to trigger peptidyl-tRNA hydrolysis[5]. Similarly, in the tmRNA–SmpB complex, the C-terminal helix of SmpB recognizes the empty mRNA channel and positions the tRNA-like domain of tmRNA at the PTC to enable

Methods

- Establishes the credibility of your data and results
- Present the details of the method
- experimental / theoretical / analytic details of method



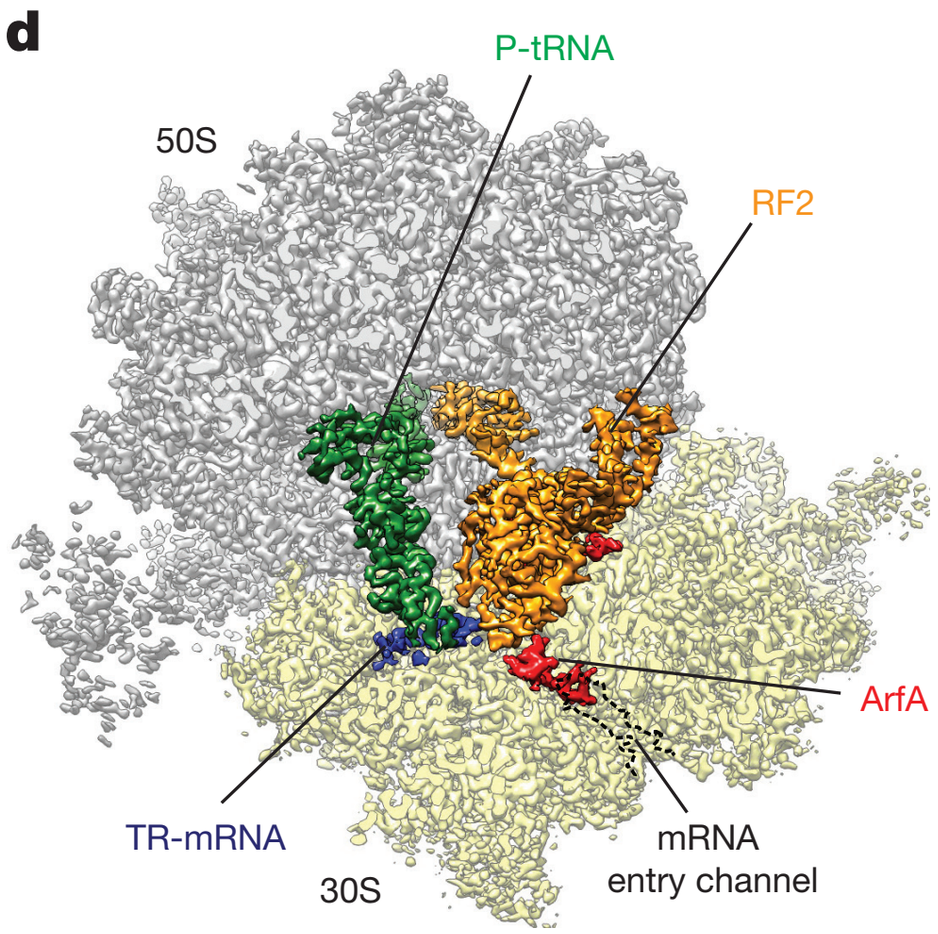
Results



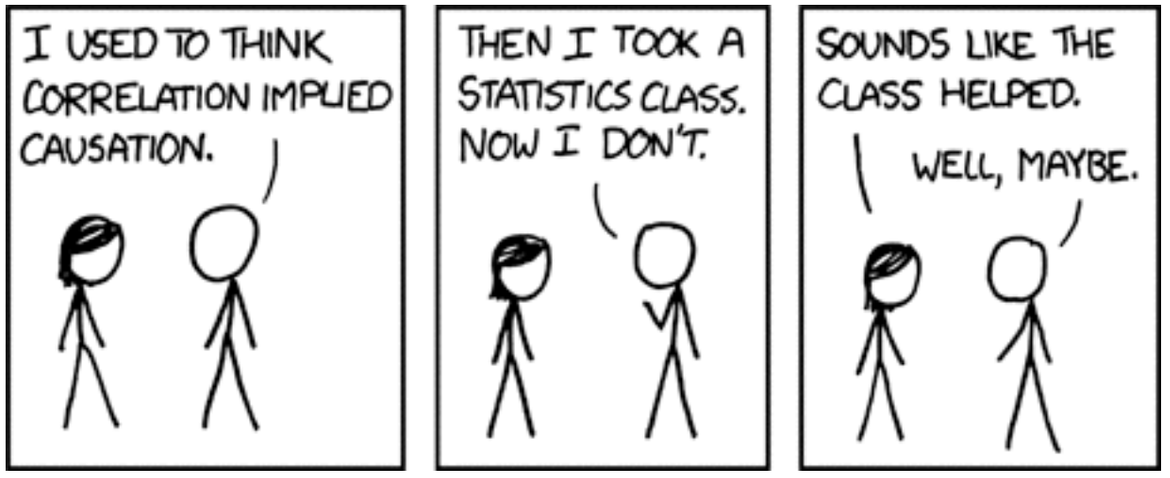
- Unbiased presentation of evidence and observations
- All the results: positive, negative or neutral
- Data should stand the test of time
- Does not include opinions, biases, interpretations
- … but discuss
  - quality of the data or show correlations
  - compare your results with other published results
- Everything you present in this section must be addressed in the Discussion

Example:

To generate a suitable complex for structural analysis, *in vitro* translation reactions were performed with a truncated mRNA in the presence and absence of ArfAΔ17 (lacking residues 56–72) and/or RF2. As reported previously[13,14], the presence of both ArfA and RF2 was required for efficient recycling of the peptidyl-tRNA (Extended Data Fig. 1). By contrast, replacing wild-type RF2 with the catalytically inactive RF2-GAQ mutant (in which the tripeptide Gly-Gly-Gln is converted to Gly-Ala-Gln) prevented peptidyl-tRNA hydrolysis and recycling (Extended Data Fig. 1), as described previously[13]. Cryo-electron microscopy (cryo-EM) analysis of the ArfAΔ17–RF2-GAQ–stalled ribosomal complex (hereafter referred to as ArfA-RF2-SRC) and *in silico* sorting of this dataset yielded a major subpopulation of ribosomal particles that contained stoichiometric occupancy of P-tRNA, ArfA and RF2 (Extended Data Fig. 2). Subsequent refinement resulted in a final reconstruction of ArfA-RF2-SRC (Fig. 1d) with an average resolution of 3.1 Å (Extended Data Fig. 3 and Extended Data Table 1). The electron density for most of ArfA was well-resolved with local resolution mostly within the range of 3.0 to 3.5 Å (Fig. 1e), enabling a molecular model to be built *de novo* for residues 2–46 of ArfA (Fig. 1f, g). The lack of density for the C-terminal 9 amino acids of ArfA prevented these residues from being included in the final model.



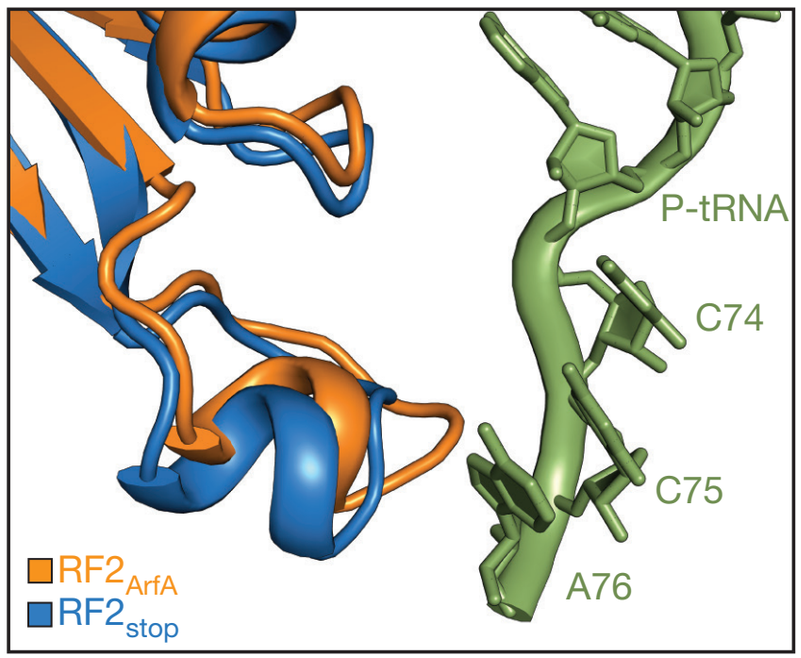
Discussion:



- Present your interpretation of the data
    - Address the hypothesis or question in the Intro
    - How do your results compare to published studies?
    - Be fair and honest
    - Lay out the logic of your conclusions.
    - Point out the limitations of your interpretations

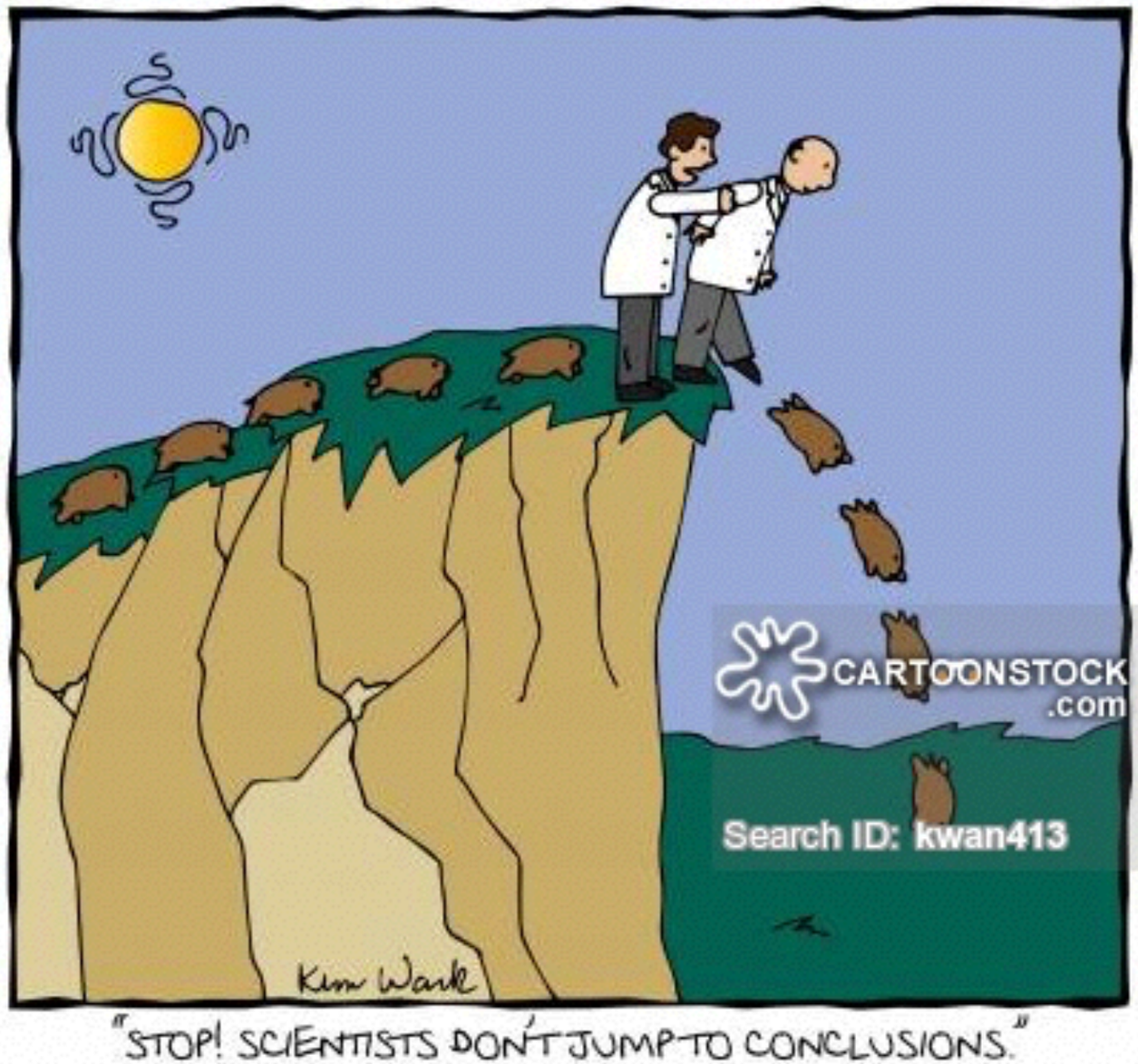
Example:

between the switch loop and A1492 (Fig. 4a, b). Instead, ArfA itself appears to stabilize a distinct conformation of the switch loop in RF2 that extends the α-helix α7 of domain 3 of RF2 by three helical turns when compared to the crystal structure of the free (closed) form of RF2 (ref. 22) (Fig. 4c, d, Supplementary Video 1). The extension of helix α7 is analogous to that observed during canonical translation termination with RF2 (refs 17, 18) (Fig. 4e, Supplementary Video 2). As observed for canonical termination[17,18], the open conformation of RF2 on the ribosome in the presence of ArfA also directs the GGQ motif of domain 3 into the PTC (Fig. 4f), although the density for the GAQ motif is poorly resolved, possibly owing to the inactivity of the mutation. The A18T mutation that led to the discovery of ArfA does not interfere with ribosome binding[8] or RF2 recruitment, but prevents peptidyl-tRNA hydrolysis[14]. This can be rationalized on the basis of the ArfA-RF2-SRC structure since the A18T mutation is not located at the ArfA–ribosome or ArfA–RF2 interfaces, but would rather perturb the conformation of the N terminus of ArfA and thereby interfere indirectly with the correct placement of domain 3 of RF2 at the PTC (Extended Data Fig. 7).



Conclusions

- Final delivery
- Re-iterate the most important messages in your paper.
  - Do not motivate the problem
  - Do not discuss the methods
- Summarize the most important
  - results &
  - conclusions derived from the data.



Example:

In conclusion, our findings indicate that ArfA not only provides an interface to recruit RF2 to the ribosome in the absence of a stop codon, but also, by interacting with the switch loop of RF2, induces conformational changes that lead to the accurate placement of domain 3 at the PTC. Structurally, the bacterial recycling systems are similar in that they use tmRNA–SmpB[6,7], ArfB[5] or ArfA (Extended Data Fig. 8) to monitor the mRNA channel and release the nascent polypeptide before ribosome splitting. This contrasts with the eukaryotic recycling of ribosomes stalled on truncated mRNAs, in which ribosome splitting by Dom34–Hbs1 and ABCE1 occurs before nascent polypeptide chain release[23,24].