The *Science* family of journals

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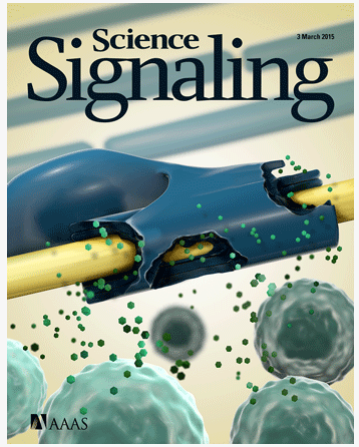
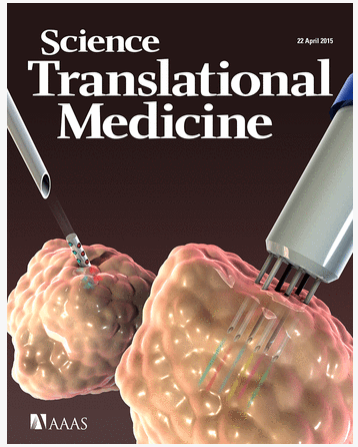
Where to submit?

- Journal scope and audience
- Review process and criteria
- Article types and format
- Editorial board
- Sister journals
- Access

Review process





Professional editors

- Staff editors & Board of Reviewing Editors - review or reject?
- External in-depth review
- Reject, revise, accept
- Assist authors in communicating research

Academic editors

- Review or reject?
- Can serve as reviewers or send for external in-depth review
- Reject, revise, accept

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Value of professional editors

- Have the power to overrule reviewers (and Board members)
- Skilled in finding appropriate reviewers
- Broad knowledge of the field and literature
- Not distracted by their "real" job
- Enhance accessibility of papers
- Invite commentary featuring research
- Help authors communicate scientific findings effectively

Gold versus Green Open Access

Gold Open Access

- Authors pay full publication costs*
- Articles are available immediately upon publication without fee to the reader

Science Advances

Green Open Access

- Authors pay only a small part of the publication costs
- Subscribers (institutional or individual) pay for immediate access
- Nonsubscribers can pay to access individual articles

Science

Science Signaling

Science Translational Medicine

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Access to *Science*, *Science Signaling*, and *Science Translational Medicine*

- **Immediate access**: Research authors receive a link to their article immediately after publication that can be put on a web page to allow free access to the article.
- **6 months after publication**: Accepted version of peer-reviewed content can be posted in authorized public repositories (such as PubMed Central).
- **12 months after publication**: Research content is freely available at the journal's website.

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Submit to *Science*?

- Is your finding a big step forward with broad implications?
- Is your paper cross disciplinary?
- Did you apply a new technique to investigate difficult scientific questions?
- Is your research in the biological, physical, or social sciences?
- Is your study self-contained and suitable for the *Science* format?

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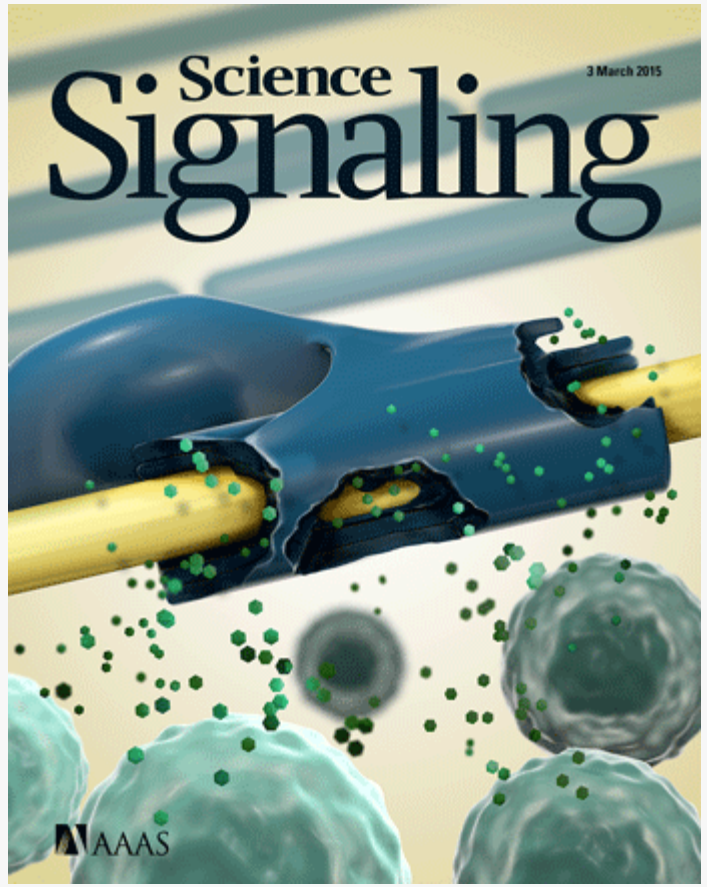
Submit to *Science Signaling?*

- Do you study cellular or organismal regulation:
  - with implications for understanding physiology and pathophysiology?
  - with implications for treating disease?
  - with mechanistic insight? (for regulation of cellular processes)
  - with computational or modeling analysis leading to experimentally tested predictions?
- Was your paper recommended from *Science* or *Science Translational Medicine*?



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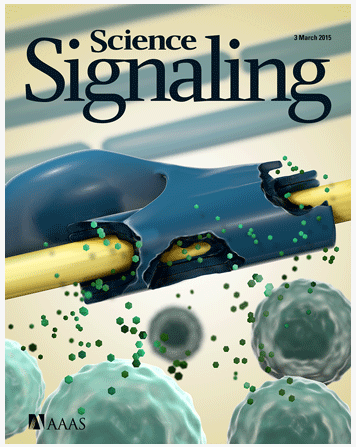
Research Article or Research Resource?

- Is the study hypothesis-driven and are the hypotheses tested?
- Does the study provide a significant advance in understanding biological regulation?

Research Article



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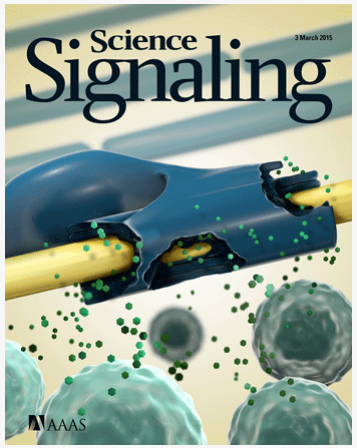
Research Article or Research Resource?

- Is the study not testing a specific hypothesis?
- Does the study present a novel technique or tool with validation, without investigating a biological question?
- Does the study provide a validated data set or describe applications of a validated database?

Research Resource



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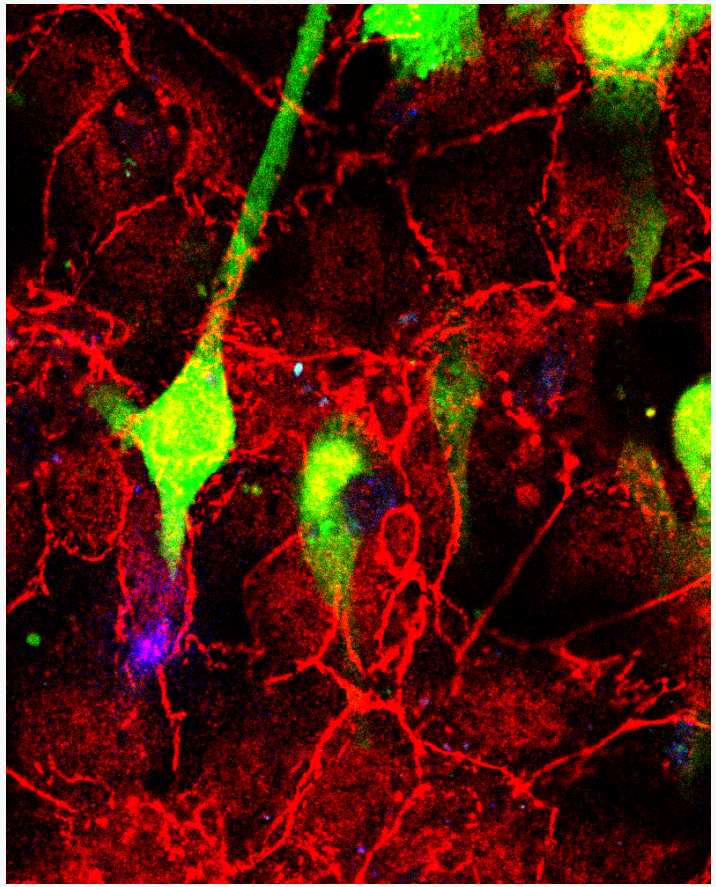

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RESEARCH ARTICLE

CANCER IMMUNOLOGY

Invasive breast carcinoma cells from patients exhibit Mena^{INV}- and macrophage-dependent transendothelial migration

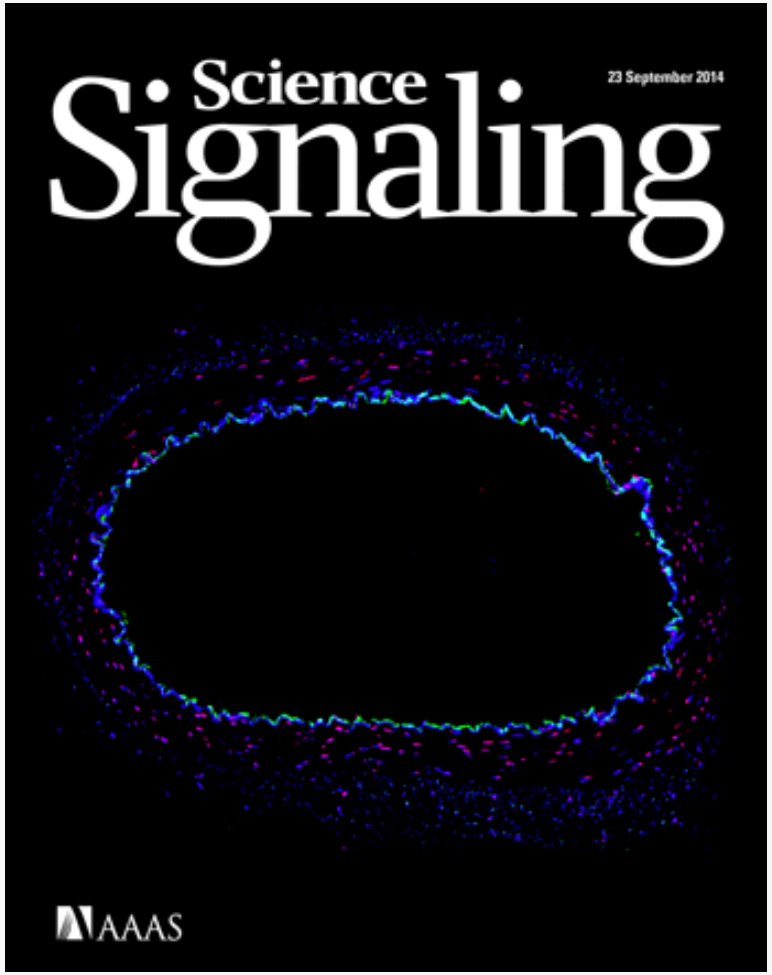
Jeanine Pignatelli,[1*] Sumanta Goswami,[1,2] Joan G. Jones,[1,3,4,5] Thomas E. Rohan,[3] Evan Pieri,[2] Xiaoming Chen,[1] Esther Adler,[4] Dianne Cox,[1] Sara Maleki,[4] Anne Bresnick,[6] Frank B. Gertler,[7] John S. Condeelis,[1,5,8*] Maja H. Oktay[4*]

RESEARCH ARTICLE

CANCER

The Secreted Protein ANGPTL2 Promotes Metastasis of Osteosarcoma Cells Through Integrin $\alpha_5\beta_1$, p38 MAPK, and Matrix Metalloproteinases

Haruki Odagiri,[1,2*] Tsuyoshi Kadomatsu,[1*] Motoyoshi Endo,[1] Tetsuro Masuda,[1,2] Masaki Suimye Morioka,[3] Shigetomo Fukuhara,[4] Takeshi Miyamoto,[5] Eisuke Kobayashi,[5] Keishi Miyata,[1] Jun Aoi,[1] Haruki Horiguchi,[1] Naotaka Nishimura,[1] Kazutoyo Terada,[1] Toshitake Yakushiji,[2] Ichiro Manabe,[3] Naoki Mochizuki,[4] Hiroshi Mizuta,[2] Yuichi Oike[1,6†]



RESEARCH ARTICLE

VASCULAR BIOLOGY

Fibroblast growth factor receptor 1 is a key inhibitor of TGFβ signaling in the endothelium

Pei-Yu Chen,[1*†] Lingfeng Qin,[2*] George Tellides,[2] Michael Simons[1,3†]

RESEARCH ARTICLE

BIOCHEMISTRY

Tandem phosphorylation within an intrinsically disordered region regulates ACTN4 function

Timothy Travers,[1] Hanshuang Shao,[2] Brian A. Joughin,[3] Douglas A. Lauffenburger,[4] Alan Wells,[2] Carlos J. Camacho[1*]

RESEARCH ARTICLE

DEVELOPMENTAL BIOLOGY

Temporal and spatial regulation of epsin abundance and VEGFR3 signaling are required for lymphatic valve formation and function

Xiaolei Liu,[1,2] Satish Pasula,[1] Hoogeun Song,[1] Kandice L. Tessneer,[1] Yunzhou Dong,[1] Scott Hahn,[1] Tadayuki Yago,[1] Megan L. Brophy,[1,2] Baojun Chang,[1] Xiaofeng Cai,[1] Hao Wu,[1] John McManus,[1] Hirotake Ichise,[3] Constantin Georgescu,[4] Jonathan D. Wren,[2,4] Courtney Griffin,[1,5] Lijun Xia,[1,2] R. Sathish Srinivasan,[1] Hong Chen[1,2*]

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Call for Papers
› Submit a Research Article

Connections Maps & Data
› Canonical Pathways
› Specific Pathways
› Canonical Components
› Specific Components

More Information
› Pathway Authorities
› Terms, Software Tips & Data Attributes
› Commercial Products
› Request for Data

Database of Cell Signaling

This database provides information on the components of cellular signaling pathways and their relations to one another, which are organized into pathways called Connections Maps, which serve as the graphical interface into the database. Scientists with expertise in a given field, designated as Pathway Authorities, provide the information. With canonical or general data about cell signaling, as well as specific data about particular signaling processes in specific organisms and cells, there is information for both novices to cell signaling and experts. Access to the database is free with registration (which is also free) or can be accessed without registration through an institutional site license to *Science Signaling*. More about Connections Maps ›

Browse Connections Maps Pathways

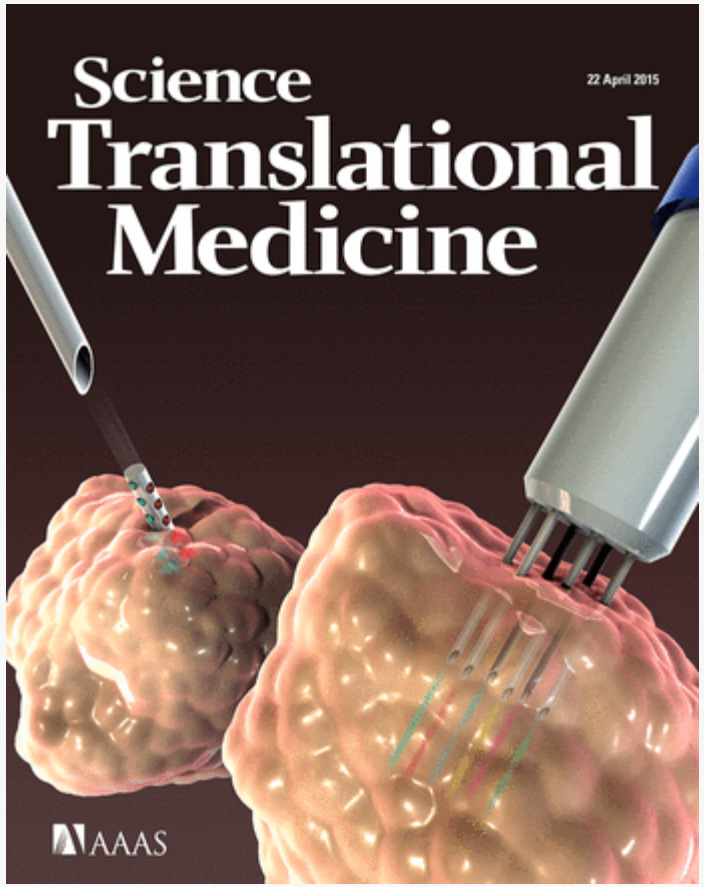
Expandable lists of pathways

- By Subject
- By Scope
- By Model Organism
- By *Science* Issue

Browse Connections Maps Components

Submit to *Science Translational Medicine?*

- Does your research advance preclinical biomedical discoveries toward improved patient care?
- Does your research advance engineering discoveries toward improved patient care?
- Does your research take clinical observations back to the lab for mechanistic studies?
- Does your research inform health policy?
- Was your paper recommended by *Science* or *Science Signaling*?



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Submit to *Science Advances*?

- Is your finding a step forward with a broad implication?
- Is your paper cross disciplinary?
- Is your research in the biological, physical, or social sciences?
- Does your funding agency require publication in an open access journal?
- Was your paper recommended from a *Science* journal?

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From lab to editor

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Who am I?
My life in the lab

Education

- Bachelors Degree in Microbiology and Biochemistry – Madras University
- Masters degrees in Biotechnology – Jawaharlal Nehru University
- Graduate Degree in Systems Neuroscience Cambridge University

Career experience

- Postdoctoral fellow at Department of Systems Biology, HMS
- Lecturer in Tufts University (Systems Biology)

Publication record

- 10 primary literature publications
- 1 review article
- 10 as first author
- Over 1000 citations

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What do I do as a (professional) editor?

- READ, READ, READ
- Solicit articles
- Perform technical editing
- Manage peer review
- Make final decision about acceptance or rejection
- Work with artist to create figures
- Write summaries or commentary articles
- Attend scientific meetings

Editor's role for Research Articles and Resources

Read cover letter and manuscript. Assign to an appropriate member of the Board of Reviewing Editors.

Discuss manuscript and Board member evaluations with the other *Science Signaling* editors. Reject or send out for in-depth review.

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STRUCTURED REVIEW

Include the following information in your review:

1) **Rate the quality of the study, using the terms below, in your comments to the editor**
   - **Excellent**: exceptional research design, conclusions are fully supported by the data
   - **High**: strong research design, minimal additional experiments needed to support the conclusions
   - **Average**: strong experimental design, some additional experiments or controls needed to support the conclusions
   - **Fair**: uneven quality in experimental design, many additional experiments or controls needed
   - **Poor**: serious weaknesses in experimental design; conclusions are not adequately supported by the data
2) **Rate the impact of the study, using the terms below, in your comments to the editor**
   - **Very high**: Major advance with impact in several fields
   - **High**: Potential to advance one or more fields
   - **Average**: Important advance for a defined field
   - **Low**: Limited advance over previous studies or advance limited by flaws
3) **Indicate if any of the following deficiencies are present and provide details in the comments to the authors:**
   - Lacks mechanism
   - Conclusions not supported by the data
   - Missing or inappropriate quantification and statistical analysis
   - Too descriptive or phenomenological
   - Not within journal scope
   - Inappropriate or missing references
4) **Indicate if the manuscript requires any of the following and provide details in the comments to the authors:**
   - Additional controls
   - Additional experiments
   - Quantification and statistical analysis
   - Revision for language usage or clarity of presentation
   - Additional references

Editor's role for Research Articles and Resources

Discuss reviews with the *Science Signaling* editors. Reject or perform a preliminary edit and ask for revisions in response to editorial and referee comments.

- N values
- Are the statements consistent with the data? Are quantification and statistical analysis necessary to support conclusions drawn?
- Accuracy, clarity, and conciseness
- Statistics

Editor's role for Research Articles and Resources

Read authors' response to the referees. Send (or not) revised manuscript to re-review.

Reject manuscript or perform technical edit. Evaluate author revisions. For papers of particular interest, solicit commentary or podcast interview. Prepare manuscript for copyediting and layout. Schedule manuscript for publication. Check galleys.

Practical tips for scientific writing

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5 Golden Rules

- Know your audience
- Write clearly
- Write concisely
- Write accurately
- Follow instructions

≠

When writing, think like…

- They're intelligent.
- They want to understand.
- They don't know your experiments.

Your family

The funder

- She gets lots of requests for money.
- She is not an expert in your field.
- She has 5 minutes before the next meeting.
- She wants to support research.

- He has 30-50 active manuscripts.
- He handles multiple presubmission inquiries a day.
- He fields multiple inquiries from authors a day.
- He wants to find the best papers.

The editor

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Audience

- Scientists in your field
- Scientists outside of your field
- Reviewers
- Editors

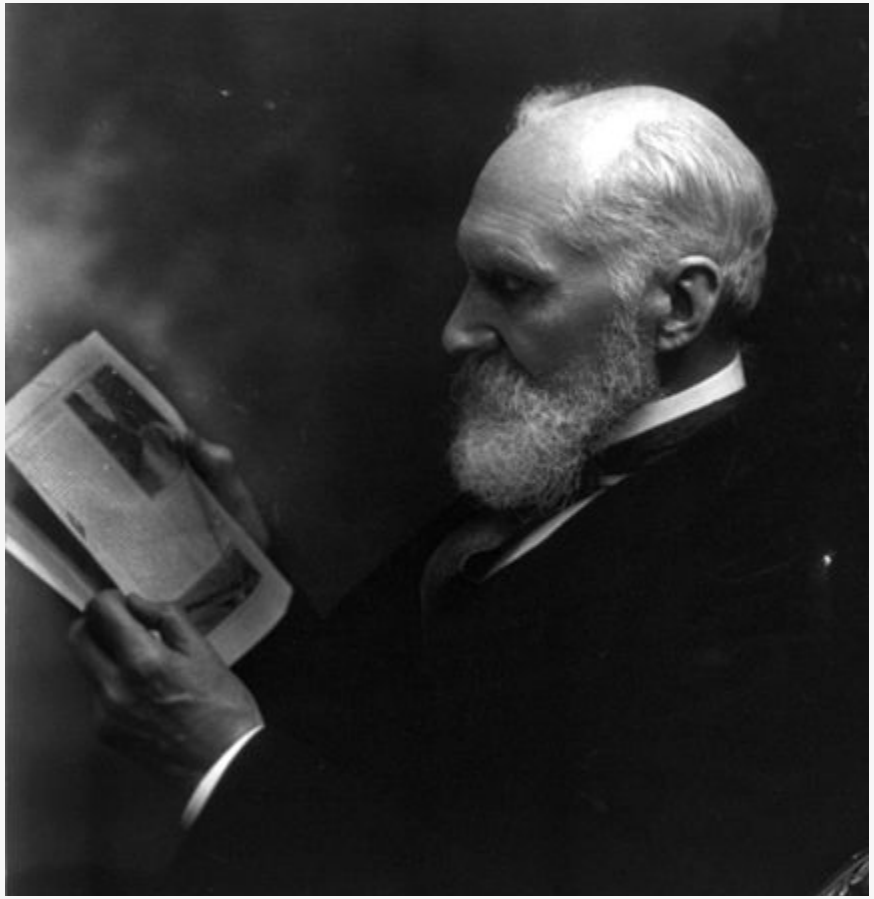
These people are drowning in information and very busy!

- Press and general public

Think like a reviewer

Run your own review process

- A scientist in your own specialty
- A scientist in an unrelated specialty
- A good editor for the English language



Reviewing the manuscript

The research

- Is the result important?
- Do the conclusions advance new concepts?
- Is the approach original?
- Are the data reliable and reproducible (how many n)?
- Are the data properly quantified and statistically evaluated?
- Are experiments properly controlled?

The presentation

- Are errors and typos eliminated?
- Does the text read smoothly?
- Are needlessly convoluted sentences avoided?
- Are the results described and not just a written statement of the data?
- Are the figures clear, well labeled, and selected to show the most critical information?
- Does the discussion appropriately account for other research?

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The cover letter

- This is your chance to speak directly to the editor.
  - Explain the overall context of your results. What problem do they solve?
  - Explain why you are excited about your work.
    (Don't forget to do these in the paper, also!)
- Keep it short, preferably 1 page.
- Have someone else proofread and edit it, especially if English is not your first language.
- If you are re-using your cover letter from submission of the paper to another journal, don't forget to change the name of the journal!!!

Think like an editor

- Are there good reasons to reject this paper?
- Are there good reasons to proceed with this paper?
- Is the paper within the journal's scope?
- Is the paper competitive?
- Can I convince my fellow editors of the value of this paper?

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Think like an editor

- Can I grasp the main finding and importance from the abstract and cover letter?
- Is the title reflective of the main findings of the study?
- If I am having trouble grasping the message because of awkward writing or poorly presented figures, how will reviewers react?

Rewrite and edit for clarity, conciseness, and accuracy

Make sure that every word in every sentence says what you really mean!

Think like a reviewer for a *Science* journal

- If correct, would this paper be interesting enough for a *Science* journal?
- If you saw this paper in *Science*, *Science Signaling*, *Science Translational Medicine*, or *Science Advances* would you say
  - **"Cool!"**
  - **"This changes my entire way of thinking!"**
  - **"I can't wait to share this with my lab!"**
  - **"What were those idiot editors thinking?"**

Write clearly

- Avoid imprecise words
  - Regulates
  - Alters
  - Influence
- Avoid words with multiple meanings
  - Levels
  - Elevates
  - Significant
- Avoid lab jargon

- Precise
  - Stimulates the activity
  - Increases the abundance
  - Represses the gene's expression
- Unambiguous
  - Amount, abundance, or concentration
  - Increase
  - Substantial or important
- Genes, RNAs, and proteins
  - Use italics for genes and transcripts
  - Use plain text for proteins and active RNA molecules

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Write concisely

- Avoid convoluted sentences with multiple clauses
- Avoid presenting published results as a historical review
- Use simple declarative sentences; divide into two sentences if necessary
- If published work is not in dispute, present it as a fact

Don't say in 4 words what can be said in 1!

Write accurately

- Avoid claims of novelty
- Avoid speculation
- Avoid superlatives
- Correlations ≠ cause and effect
- Written ≠ spoken language
- Don't anthropomorphize
  - Proteins are not people!

Your paper is not a used car.
Don't overinterpret, overstate, or oversell!

Examples

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Writing samples: Before

Abstract 109 words

Pathogenic mutations in leucine-rich repeat kinase 2 (LRRK2) are the most common genetic cause of late onset Parkinson's disease (PD). There was mounting evidence that the intrinsic kinase activity of LRRK2 is required for LRRK2-mediated PD pathogenesis. However, recent studies suggested that LRRK2 kinase activity is dispensable for neuron survival and its protective activity against the neurotoxin. It was hypothesized that LRRK2 kinase domain might have a scaffolding role independent of its intrinsic kinase activity in the assembly of signaling complexes. In this regard, the intrinsic kinase activity of LRRK2 appears to be a Trojan horse for PD and modulation of its kinase activity could be potentially therapeutically beneficial.

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Edited

Abstract

Pathogenic mutations in leucine-rich repeat kinase 2 (LRRK2) are ~~the most~~ common genetic causes of late onset Parkinson's disease (PD). ~~There was mounting evidence~~Initial studies indicated that the intrinsic kinase activity of LRRK2 is associated ~~with~~~~required for~~ LRRK2-mediated PD pathogenesis. However, ~~recent studies suggested that~~ LRRK2 kinase activity may be~~is~~ dispensable for neuron survival and may not be required for its protective activity against ~~the~~ neurotoxicity~~n~~. ~~It was hypothesized that~~ Thus, ~~LRRK2 kinase domain might have a scaffolding role independent of its intrinsic kinase activity in the assembly of signaling complexes. In this regard~~, the intrinsic kinase activity of LRRK2 appears to be a Trojan horse for PD and ~~modulation~~ inhibition of its kinase activity could be potentially therapeutically beneficial.

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Final

Abstract 83 words

Pathogenic mutations in leucine-rich repeat kinase 2 (LRRK2) are common genetic causes of late onset Parkinson's disease (PD). Initial studies indicated that the intrinsic kinase activity of LRRK2 is associated with LRRK2-mediated PD pathogenesis. However, the kinase activity of LRRK2 may be dispensable for neuron survival and may not be required for its protective activity against neurotoxicity. Thus, the intrinsic kinase activity of LRRK2 appears to be a Trojan horse for PD and inhibition of its kinase activity could be potentially therapeutically beneficial.

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Research Article Abstract: Before (224 words)

Cells derived from ataxia telangiectasia (A-T) patients exhibit defective cell cycle checkpoints following ionizing radiation (IR), profound radiosensitivity and high levels of chromosome aberrations. We have shown that transient ATM kinase inhibition from +15 to +75 min following IR is sufficient to radiosensitize cells and accumulate persistent chromosome aberrations. We show here that DNA-PK kinase inhibition from +15 to + 75 min is also sufficient to radiosensitize cells and accumulate persistent chromosome aberrations. The ATM kinase-dependent mechanisms that ensure cell survival and suppress chromosome aberrations during this interval are independent of DNA-PK kinase activity. Neither the activation nor the recovery of the IR-induced G2/M cell cycle checkpoint are affected by ATM kinase inhibition from +15 to +75 min, indicating that 15 min of ATM kinase signaling is sufficient to induce this cell cycle checkpoint. Surprisingly, ATM kinase inhibition from +15 to +75 min abrogates IR-induced sister chromatid exchange (SCE), a phenotype attributed to the repair of damaged replication forks. Further, ATM kinase inhibition using either KU55933 or KU60019 is sufficient to disrupt camptothecin-induced SCE. Since DNA damage-induced SCE is maintained A-T cells that express no ATM protein, and the ATM kinase inhibitors have no effect on DNA damage-induced SCE in A-T cells, these data reveal that the consequences of acute ATM kinase inhibition and adaptation to ATM protein disruption are distinct in S-phase cells.

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Did your eyes glaze over?

- Lack of context
- Too much methodological detail
- Imprecise language
- Too many undefined terms

Problems

Cells derived from ataxia telangiectasia (A-T) patients exhibit defective cell cycle checkpoints following ionizing radiation (IR), profound radiosensitivity and high levels of chromosome aberrations.

We have shown that transient ATM kinase inhibition from +15 to +75 min following IR is sufficient to radiosensitize cells and accumulate persistent chromosome aberrations.

We show here that DNA-PK kinase inhibition from +15 to + 75 min is also sufficient to radiosensitize cells and accumulate persistent chromosome aberrations.

The ATM kinase-dependent mechanisms that ensure cell survival and suppress chromosome aberrations during this interval are independent of DNA-PK kinase activity.

- What is radiosensitivity?
- What is the relationship of radiosensitivity to chromosome aberrations?

- ATM kinase = The kinase that phosphorylates ATM? NO

- What are ATM and DNA-PK?
- DNA-PK kinase = The kinase that phosphorylates DNA-PK? NO

- ATM kinase-dependent = Mechanisms that rely on phosphorylation of ATM? NO
- How does cell survival relate to radiosensitivity?

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More problems

Neither the activation nor the recovery of the IR-induced G2/M cell cycle checkpoint are affected by ATM kinase inhibition from +15 to +75 min, indicating that 15 min of ATM kinase signaling is sufficient to induce this cell cycle checkpoint.

Surprisingly, ATM kinase inhibition from +15 to +75 min abrogates IR-induced sister chromatid exchange (SCE), a phenotype attributed to the repair of damaged replication forks.

Further, ATM kinase inhibition using either KU55933 or KU60019 is sufficient to disrupt camptothecin-induced SCE.

- What is the IR-induced G2/M checkpoint?
- Why is this surprising? What do damaged replication forks have to do with IR-induced damage?
- Too much experimental detail.
- What is camptothecin?

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And more problems

Since DNA damage-induced SCE is maintained A-T cells that express no ATM protein, and the ATM kinase inhibitors have no effect on DNA damage-induced SCE in A-T cells, these data reveal that the consequences of acute ATM kinase inhibition and adaptation to ATM protein disruption are distinct in S-phase cells.

- Since should be because.
- Genes are expressed, not proteins.
- The information about S-phase is out of context.

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Clean edited version with editorial queries (182 words)

ABSTRACT

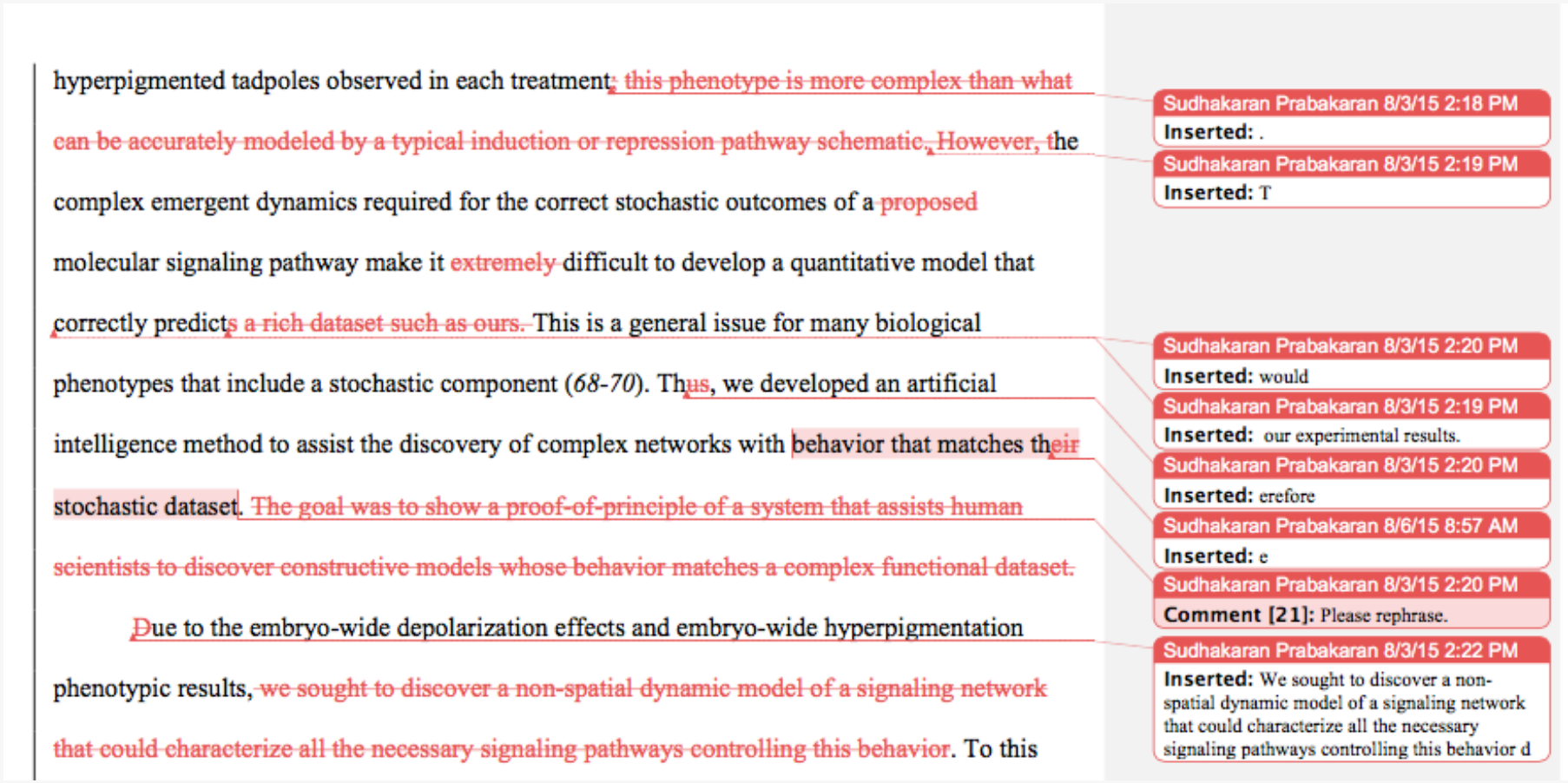
Cells derived from ataxia telangiectasia (A-T) patients exhibit defective cell cycle checkpoints due to mutations in the gene encoding ATM (ataxia telangiectasia mutated). Following exposure to ionizing radiation (IR), A-T cells exhibit sensitivity to IR-induced cellular damage (radiosensitivity), resulting in abundant chromosome aberrations. ATM is a member of a family of kinases that become activated in response to DNA damage, and exposure of cells to IR triggers ATM activity and subsequent transient inhibition ATM causes radiosensitivity. We show that, despite activation and recovery from the G_2/M checkpoint following transient inhibition of ATM 15 minutes after cellular irradiation, the cells exhibited radiosensitivity and accumulation of persistent chromosome aberrations. With reversible inhibitors of DNA-PK (DNA-dependent protein kinase), another kinase involved in responding to DNA damage, and ATM, we show that these two kinases acted through distinct DNA repair mechanisms: ATM resolved DNA damage through a mechanism involving sister chromatid exchange (SCE), whereas DNA-PK acted through nonhomologous end joining. Furthermore, DNA damage-induced SCE occurred in A-T cells, suggesting that A-T cells have adapted to the loss of ATM and have alternative mechanisms to initiate SCE.

Comment [NG1]: Abstract cannot exceed 250 words. I have substantially revised this to make it more accessible and to put the new results into a broader context. I thought including the details of the methods and timing made the abstract difficult to follow.

Comment [NG2]: Are the last two the consequence of IR or are these present in the patients cells all the time? Correct as edited?

Comment [NG3]: We try to reserve "levels" for positions within a hierarchy. Please use abundant or concentration or amount as appropriate.

Comment [NG4]: Introduce the kinases in one sentence. Is this correct?


hyperpigmented tadpoles observed in each treatment; this phenotype is more complex than what can be accurately modeled by a typical induction or repression pathway schematic. However, the complex emergent dynamics required for the correct stochastic outcomes of a proposed molecular signaling pathway make it extremely difficult to develop a quantitative model that correctly predicts a rich dataset such as ours. This is a general issue for many biological phenotypes that include a stochastic component (68-70). Thus, we developed an artificial intelligence method to assist the discovery of complex networks with behavior that matches their stochastic dataset. The goal was to show a proof-of-principle of a system that assists human scientists to discover constructive models whose behavior matches a complex functional dataset.
Due to the embryo-wide depolarization effects and embryo-wide hyperpigmentation phenotypic results, we sought to discover a non-spatial dynamic model of a signaling network that could characterize all the necessary signaling pathways controlling this behavior. To this
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Sudhakaran Prabakaran 8/3/15 2:20 PM
Comment [21]: Please rephrase.
Sudhakaran Prabakaran 8/3/15 2:22 PM
Inserted: We sought to discover a non-spatial dynamic model of a signaling network that could characterize all the necessary signaling pathways controlling this behavior d

Practice

- Although most PD cases are sporadic, at least seven genes have been reported to be implicated in the pathogenesis of familial PD (*1*).

(hint: too many words)

- *In vitro* studies indicated that several pathogenic mutations in LRRK2 caused an increase in the kinase activity, such as mutations R1441C in ROC GTPase domain and G2019S in kinase domain (*4-6*).

(hint: multiple problems, including a misplaced clause)

- While the physiological function of LRRK2 remains largely unknown, recent studies indicated a dispensable role of the intrinsic kinase activity of LRRK2 in neuron survival and its protective activity against neurotoxin (*10-12*).

(hint: multiple problems, especially temporal words)

- The current paper reports for the first time a sex reversal in transsexual people in the interstitial nucleus of the anterior hypothalamus (INAH) 3, a sexually dimorphic hypothalamic nucleus that was previously shown to be related to sexual orientation (citation 1, citation 2).

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Practice while you read

As you read for Journal Club, consider these best practices and ‘pencil’-edit the papers.

As you read background materials for your research, find the errors and think about how the writing could be improved.

Help each other. Read each other’s manuscripts.

Don’t be afraid of the red pen!

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Final thoughts and questions

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