



<http://www.edanzediting.com/mexico2014>

# Insights into Publication Success

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**Entre pares**  
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**Mexico**

**22 September 2014**



# A little about me...



THE UNIVERSITY  
OF AUCKLAND

NEW ZEALAND

Te Whare Wānanga o Tāmaki Makaurau



UNIVERSITY OF  
CAMBRIDGE



nature publishing group



# Be an effective communicator

- ✓ **Write effectively**
- ✓ **Choose the best journal to reach your target audience**
- ✓ **Logically present your research in your manuscript**
- ✓ **Convey the significance of your work to journal editors**
- ✓ **Properly revise your manuscript after peer review**

<http://www.edanzediting.com/mexico2014>

# Section 1

***Effective writing***

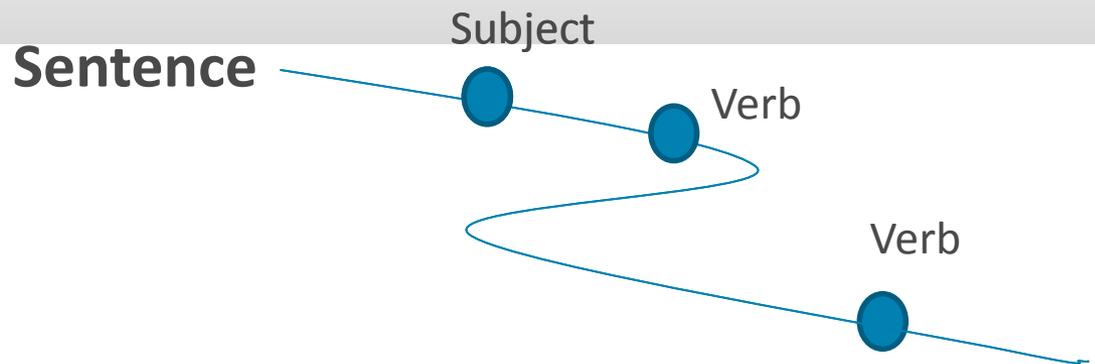
# Reader expectations

- Information is easier to understand when placed where most readers expect to find it
- Good writers are aware of these expectations
- Readability



# 1. Verb placement

- Readers expect verbs to closely follow subjects



# Active voice

Sentences written in the active voice are:

*simple*

*direct*

*clear*

*easy to read*

## Passive

The mechanisms regulating tumor growth were investigated.

## Active

We investigated the mechanisms regulating tumor growth.

# Active voice is preferred

## *ACS Style Guide*

“Use the **active voice** when it is less wordy and more direct than the passive”. (3<sup>rd</sup> ed., pg. 42)

## *APA Style*

“Use the **active voice** rather than the passive voice...”.  
[www.apastyle.org/learn/faqs/effective-verb-use.aspx](http://www.apastyle.org/learn/faqs/effective-verb-use.aspx)

## *Chicago Style Guide*

“As a matter of style, passive voice is typically, but not always, inferior to **active voice**”. (15<sup>th</sup> ed., pg. 177)

## *AMA Manual of Style*

“In general, authors should use the **active voice**...”.  
(10<sup>th</sup> ed., pg. 320)

# Stress position

Which sentence suggests that you  
will get a raise?

1. You deserve a raise, but the budget is tight.
2. The budget is tight, but you deserve a raise.

*Readers focus at the **end of the sentence** to determine what is important.*

# Stress position

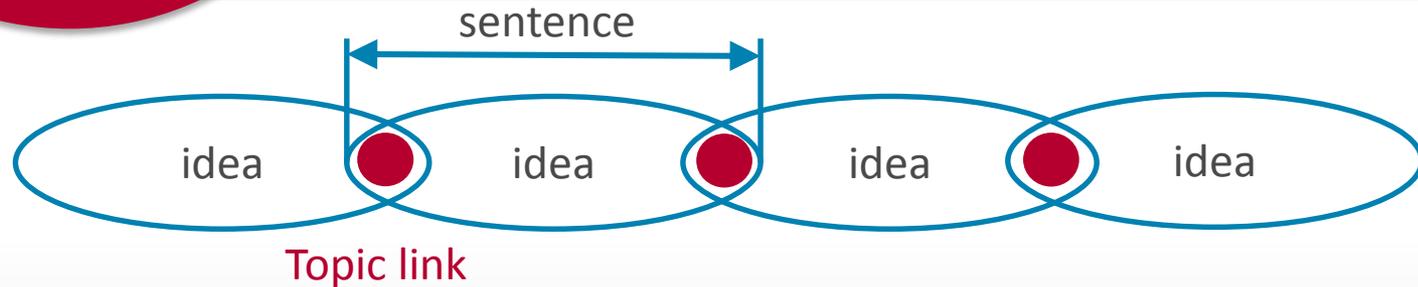
The stress position also introduces  
the topic of the next sentence

The budget is tight, but **you deserve a raise.** **Your salary**  
**Stress position** **Topic position**  
will increase at the beginning of next year.



*The topic position introduces the idea of the current sentence*

# Topic position



*The patient* went to the hospital to see a *gastroenterologist*. *The doctor* then performed a series of *diagnostic tests*. *The results* showed the patient suffered from a *bacterial infection*. *Antibiotics* were prescribed to treat the infection before the patient developed an *ulcer*.

# Linking your ideas in your manuscript

Marine biofouling is the accumulation of marine species onto submerged surfaces within the ocean. It imposes significant cost to the maritime industries and as a result has been the subject of a considerable number of preventative strategies. Until recently all of these have involved toxic coatings containing heavy metals such as copper and tin. More recently, environmental considerations such as bioaccumulation have led to the ban on **Stress sentence** This has resulted in a demand for non-toxic alternatives.



Current non-toxic antifouling strategies are driven by interfacial architecture and fall into two main behavioral categories, foul-**Topic sentence** aces and attachment-inhibiting (AI) surfaces. FR...

# Simple is best

- Simple language *is* best
- Makes *your* science more relevant
- Minimizes confusion – maximizes understanding
- Science is complex
- Use simple language to help more people understand your work

# Simple words

## Avoid

Additional

Adequate

Apparent

Attempt

Demonstrate

Endeavor

Exceedingly

## Preferred

More

Enough

Clear

Try

Show

Try

Very

## Section 2

***Journal selection***

# Factors to consider when choosing a journal

Aims & scope

Readership

Open access

Impact factor

*Varies by field*

***Which factor is most important to you?***

# Choosing a target journal

Journal selection *must* be based on an honest evaluation of *your* findings



Significance

Aims and Scope  
Impact

## JOURNAL SELECTOR

### Get started - find the journal that's right for you

#### Enter your abstract or article description

ATZ (50 µg/mL) were also prevented on co-treatment with QT (50 µM). Furthermore, ATZ-induced 3β- and 17β-hydroxysteroid dehydrogenase activities and NF-κB-expressions at the mRNA and protein levels were also recovered to control value on co-treatment with QT. These data showed that QT protected against ATZ-induced ILCs toxicity by restoring the expression of NF-κB and steroidogenic activity and by preventing the oxidative stress.

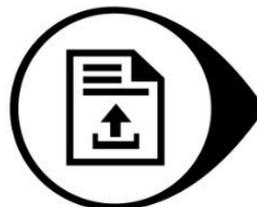
[Questions about entering your abstract or article description?](#)

#### Find your target journal

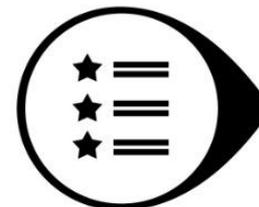
- Only journals with an Impact Factor
- Only journals with Open Access options

**Find your target journal**

The Journal Selector uses cutting-edge semantic technology to help you achieve publication success. Enter your abstract or sample text and the Journal Selector returns a list of journals that publish in related areas. You can refine your results based on the factors that matter to you, like publication frequency, Impact Factor or publishing model, including open access.



Enter your abstract



Find relevant journals



Select your target journal

**Insert your proposed abstract**

# Journal selection

# Journal Selector – [www.edanzediting.com/journal\\_selector](http://www.edanzediting.com/journal_selector)

## Recommended journals

### JOURNAL SELECTOR

### JOURNAL SELECTOR RESULTS

#### You can edit your abstract and refine your results

Quercetin testicular cells oxidative induced environmental chemicals. study, isolated interstitial Leydig cells (ILCs) immature rats, - ILCs culture, - cells atrazine (ATZ) quercetin (QT), toxicity, enzymes nuclear -kappaB (NF-) steroidogenic enzymes. ATZ ILCs 10 /mL reactive oxygen species, malondialdehyde (MDA), glutathione . ATZ glutathione peroxidase, glutathione reductase, glutathione--transferase superoxide dismutase-1

[Questions about refining or filtering your results?](#)

#### Filter your results to find the best match

Please use the options below to filter your results.

Impact Factor  Show only journals with an Impact Factor

0 0.5 1 1.5 2 3 5 7 10+

Frequency

Publishing model  Any  Open Access  Hybrid

**Refine Journal Results**

#### We recommend the following journals

#### SORT RESULTS BY

▼ [Match](#) | [Title](#) | [Impact Factor](#) | [Frequency](#) | [Model](#)

	<b><a href="#">Molecular and Cellular Biochemistry</a></b>	Impact Factor : 2.329	Frequency : Continuous	Model: Hybrid
	<b><a href="#">Toxicology in Vitro</a></b>	Impact Factor : 2.546	Frequency : Bimonthly	Model: N/A
	<b><a href="#">Free Radical Research</a></b>	Impact Factor : 2.805	Frequency : Monthly	Model: N/A
	<b><a href="#">Toxicology</a></b>	Impact Factor : 4.017	Frequency : Bimonthly	Model: N/A
	<b><a href="#">Food and Chemical Toxicology</a></b>			

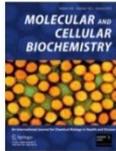
**Filter by:**  
Impact factor  
Publishing frequency  
Open access

Journal selection

# Journal Selector –

[www.edanzediting.com/journal\\_selector](http://www.edanzediting.com/journal_selector)

Semantic matching terms



## Molecular and Cellular Biochemistry

### Aims & Scope :

Molecular and Cellular Biochemistry: An International Journal for Chemical Biology in Health and Disease publishes original, full-length research papers and short communications in all areas of the biochemical sciences, the emphasis being on those papers which present novel findings relevant to the biochemical basis of cellular function and disease processes, as well as the mechanics of action of hormones and chemical agents.

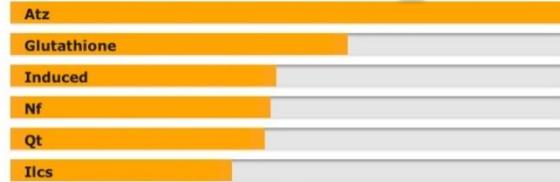
Investigations directed towards molecular biology and gene expression are encouraged. Membrane transport, secretory response, secretory systems, and related biochemical processes are areas of great interest. Studies examining the molecular and cellular basis of normal and pathological processes are also encouraged.

In addition to research, the journal publishes state of the art reviews. Specific subjects that are covered by Molecular and Cellular Biochemistry are: cellular metabolism, cellular pathophysiology, enzymology, ion transport, lipid biochemistry, membrane biochemistry, molecular biology, nuclear structure and function, and protein chemistry.

[Hide full aims and scope](#)

Impact Factor  
Frequency: Continuous

### Match Analysis :



[What does match analysis mean?](#)

Journals IF, Aims & Scope, and Frequency

Similar published articles

**Similar articles from this journal**

- [+ Quercetin decreases steroidogenic enzyme activity, NF-κB expression, and oxidative stress in cultured Leydig cells...](#)  
Published 2012 - Oct
- [+ Quercetin decreases steroidogenic enzyme activity, NF-κB expression, and oxidative stress in cultured Leydig cells...](#)  
Published 2012 - Dec
- [+ Ameliorative action of melatonin on oxidative damage induced by atrazine toxicity in rat erythrocytes.](#)  
Published 2011 - Jun
- [+ Causal relationship between Hexachlorocyclohexane cytotoxicity, oxidative stress and Na, K-ATPase in Ehrlich Asc...](#)  
Published 2006 - May

Have they published similar articles recently?

Have you cited some of these articles?

Journal selection

# Visit journal websites

The screenshot shows the Springer website for the journal *Molecular and Cellular Biochemistry*. The journal title is circled in green. The page includes the Springer logo, navigation menus, and a sidebar with various links. The journal's impact factor is 2.329, and its frequency is continuous. The page also features a 'Free Preview' button and social media sharing options.

**Molecular and Cellular Biochemistry**

**Aims & Scope**  
Molecular and Cellular Biochemistry: An International Journal for Chemical Biology in Health and Disease publishes original, full-length research papers and short communications in all areas of

**Impact Factor:** 2.329 \*  
**Frequency:** Continuous  
**Match Analysis:** Atz

Japan » Change

Springer

» New User  
LOGIN

HOME | MY SPRINGER | SUBJECTS | SERVICES | PUBLISHERS | ABOUT US

» *Biochemistry & Biophysics* Home > Life Sciences > Biochemistry & Biophysics

Advanced Search

SUBDISCIPLINES | JOURNALS | BOOKS | SERIES | TEXTBOOKS | REFERENCE WORKS

READ THIS JOURNAL ON SPRINGERLINK

- Online First Articles
- All volumes & issues
- Free: Sample Articles

FOR AUTHORS AND EDITORS

2012 Impact Factor 2.329

Aims and Scope

Submit Online

Open Choice - Your Way to Open Access

Instructions for Authors

Ethical Standard

ABOUT THIS JOURNAL | EDITORIAL BOARD

Like 26 Tweet 0 +1 3

# Tips to identify the most suitable journal

Identify the interests of the *journal editor*

- Editorials
- Review articles
- Special issues

# Tips to identify the most suitable journal

## Journal editor's interests

### Journal A

- Editorials
- Review articles
- Special issues

### Journal B

- Editorials
- Review articles
- Special issues

### Journal C

- Editorials
- Review articles
- Special issues

Manuscript

# Tips to identify the most suitable journal

Identify the interests of the *journal editor*

- Editorials
- Review articles
- Special issues

Identify the interests of the *readers*

- Most viewed
- Most cited

# Tips to identify the most suitable journal

## Reader's interests

### Journal A

- Most viewed
- Most cited

### Journal B

- Most viewed
- Most cited

### Journal C

- Most viewed
- Most cited

## Manuscript

## Section 3

### *Manuscript structure*

# Use your figures to structure your manuscript

## Where to start?

- ❖ Your *findings* are why you want to publish your work
- ❖ Form the basis of your manuscript
- ❖ First step, is to logically organize your findings

Figure 1

Table 1

Figure 2

?

Figure 3

Logical  
presentation

Is anything  
missing?

Additional  
analyses?

# Use your figures to structure your manuscript

## Where to start?

- ❖ Your *findings* are why you want to publish your work
- ❖ Form the basis of your manuscript
- ❖ First step, is to logically organize your findings

Figure 1

Table 1

Figure 2

Figure 3

Figure 4

Logical  
presentation

New data

# Prepare an outline

- I. **Introduction**
  - A. General background
  - B. Related studies
  - C. Problems in the field
  - D. Aims
- II. **Methods**
  - A. Subjects/Samples/Materials
  - B. General methods
  - C. Specific methods
  - D. Statistical analyses
- III. **Results**
  - A. Key points about Figure 1
  - B. Key points about Table 1
  - C. Key points about Figure 2
  - D. Key points about Figure 3
  - E. Key points about Figure 4
- IV. **Discussion**
  - A. Major conclusion
  - B. Key findings that support conclusion
  - C. Relevance to published studies
  - D. Unexpected/negative findings
  - E. Limitations
  - F. Implications
  - G. Future directions

## *Introduction*

**What background information  
you will introduce**

# Prepare an outline

- I. **Introduction**
  - A. General background
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  - G. Future directions

## *Introduction*

What background information  
you will introduce

## *Methods*

What analyses you will describe

# Prepare an outline

- I. Introduction**
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  - G. Future directions

## *Introduction*

What background information  
you will introduce

## *Methods*

What analyses you will describe

## *Results*

What findings you will present

# Prepare an outline

- I. Introduction**
  - A. General background
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  - E. Limitations
  - F. Implications
  - G. Future directions

## *Introduction*

What background information  
you will introduce

## *Methods*

What analyses you will describe

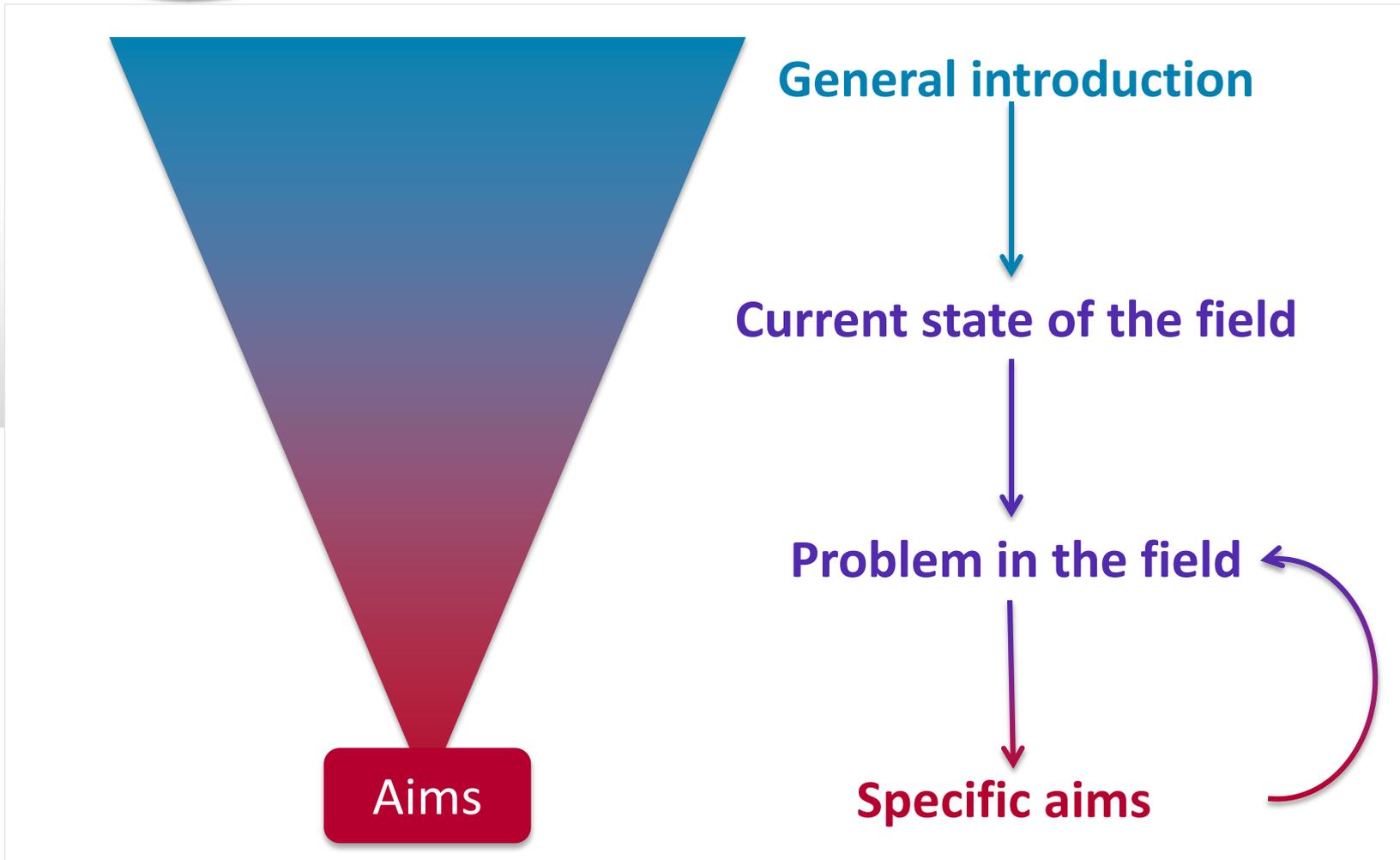
## *Results*

What findings you will present

## *Discussion*

What interpretations, limitations,  
and implications you will discuss

# Introduction



# Introduction – flow of information

Lung cancer is the leading cause of cancer mortality for men and women. Despite smoking prevention and cessation programs and advances in early detection, the 5-year survival rate for lung cancer is only 16% with current therapies. Although lung cancer incidence rates have recently declined in the United States, more lung cancer is now diagnosed in nonsmokers than in current smokers. Thus, even if all of the national anti-smoking campaign goals are met, lung cancer will remain a major public health problem for decades. New ways to treat or prevent lung cancer are therefore needed.

## General introduction

One potential therapeutic target for lung cancer is the Wnt signaling pathway. The canonical Wnt signaling pathway in mammals consists of a family of secreted lipid-modified Wnt protein ligands that bind to a family of 7-pass transmembrane Frizzled (Fzd) receptors, as reviewed. In brief, in the absence of ligand, glycogen synthase kinase-3 (GSK3), in complex with axin and adenomatous polyposis coli (APC), constitutively phosphorylates  $\beta$ -catenin, the primary Wnt signaling effector, targeting it for ubiquitination and proteasomal destruction. Ligand binding engages a pathway involving Dishevelled (Dvl) that inhibits GSK3, allowing  $\beta$ -catenin to accumulate in a hypophosphorylated form. This stabilized form of  $\beta$ -catenin can translocate to the nucleus, where it activates target gene transcription by complexing with T cell factor (TCF) and lymphoid enhancer-binding factor (LEF). In addition to key mediators of embryonic development, these target genes include critical growth-regulators such as *myc* and *cyclin D1*.

Aberrant Wnt signaling due to mutations in *\beta-catenin* or *APC* drives deregulated growth in both familial and non-hereditary colorectal cancers. However, in non-small cell lung cancers (NSCLC), the most common cause of lung cancer, the most common mutations are in *KRAS* and *EGFR*. Rather, aberrant Wnt activity in lung cancer is linked to increased expression of upstream Wnt signaling effectors such as Dvl or decreased expression of Wnt antagonists such as Wnt-inhibitory factor 1 (Wif-1).

## Specific introduction

Effective pharmacological inhibitors of the Wnt pathway have only recently become available. Screens for small-molecule antagonists of the Wnt pathway found two enzymes to be key mediators of Wnt signaling. These are poly-ADP-ribose polymerase (PARP) enzymes, tankyrase (TNKS) 1 and TNKS2, which attach poly-ADP-ribose (PAR) onto substrate proteins. Their roles in regulating telomerase function and mitotic spindle formation are known, but their role in PARsylating axin so as to maintain the optimal level for canonical Wnt signaling has only recently been recognized. The compounds identified in these screens, XAV939, IWR-1 exo, and IWR-1 endo, act by specifically inhibiting the PARP activity of TNKS1 and TNKS2. IWR-exo is a stereoisomer of IWR-1 endo with ~14-fold lower  $EC_{50}$ . PARP inhibition is a tractable pharmacological target *in vivo*, as antagonists of other PARP homologs exert antineoplastic responses in breast and ovarian cancer, as reviewed.

This study explored the hypothesis that inhibition of TNKS by pharmacological or genetic means would inhibit lung cancer growth *in vitro* and *in vivo* in clinically-relevant transgenic mouse models of lung cancer that were previously developed, as reviewed.

# Writing the Introduction

*Beginning* should demonstrate  
relevance/interest

## Interest

Lung cancer is the leading cause of cancer mortality for men and women. Despite smoking prevention and cessation programs and advances in early detection, the 5-year survival rate for lung cancer is only 16% with current therapies. Although lung cancer incidence rates have recently declined in the United States, more lung cancer is now diagnosed when considered together in former- and never-smokers than in current smokers. Thus, even if all of the national anti-smoking campaign goals are met, lung cancer will remain a major public health problem for decades. New ways to treat or prevent lung cancer are therefore needed.

*Important problem in the field*

# Writing the Introduction

Your *aims* should directly  
address this problem

**New ways to treat or prevent lung cancer are therefore needed.**

This study explored the hypothesis that inhibition of TNKS by pharmacological or genetic means would inhibit lung cancer growth *in vitro* and *in vivo*...

## *Experimental Design*

**What was  
used**

**Samples or participants  
Materials**

**How it was  
done**

**General methods  
Specific techniques  
(discuss controls)**

**How it was  
analyzed**

**Data analysis  
Quantification methods  
Statistical tests**

# Results

**Logical presentation**

**Key points relating to  
each display item**

**Subsections**

**Each subsection  
corresponds to  
*one figure***

**Factual description**

**What you found, *not*  
what it means**

# Display items

Present large amount  
of data *quickly* and  
*efficiently*

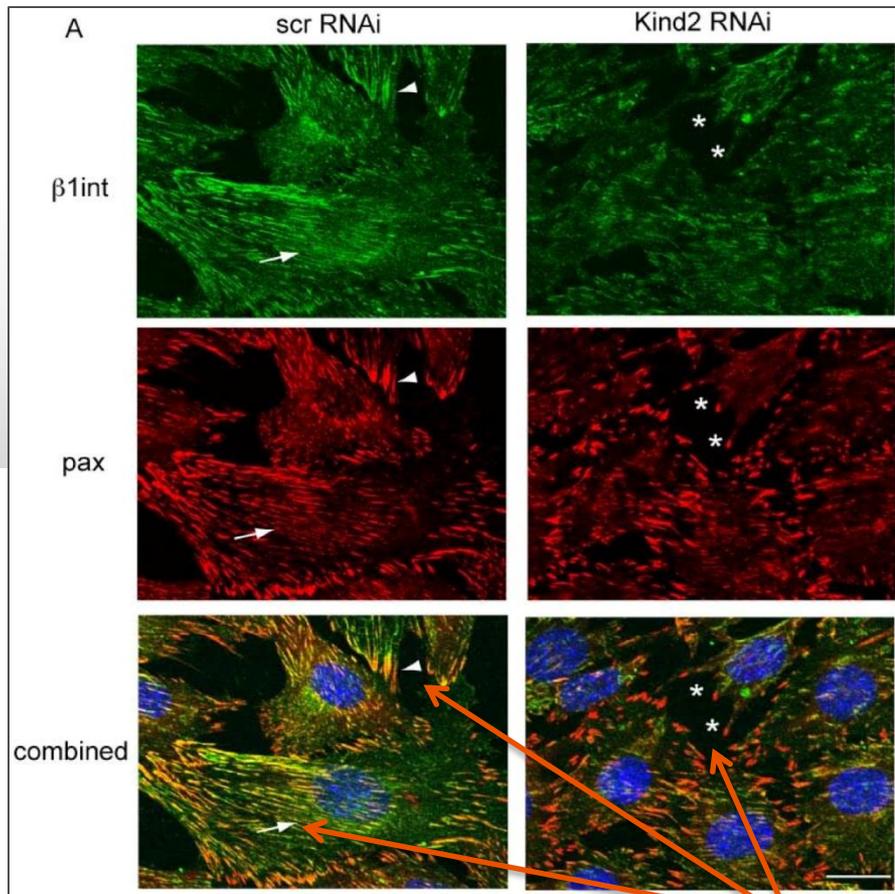
Usually the *first* thing  
readers will look at

## Figures, graphs & tables

Keep it *simple*: use  
separate panels if  
necessary

Must be able to *stand  
alone*: clear labels  
and figure legends

Clear figure legend



**Kindlin-2 knockdown and focal adhesion localization.** Confocal immunofluorescent microscopy with anti- $\beta$ 1 integrin and anti-paxillin on C2C12 cells transfected with RNAi and then changed to differentiation media for 2 days. Control cells show linear staining consistent with localization to costameres (arrows), as well as punctate focal contact staining (arrowheads). Focal contact proteins in the kindlin-2 RNAi cells fail to form linear structures and instead are concentrated in unusual appearing puncta (\*). (Scale bar = 20  $\mu$ M).

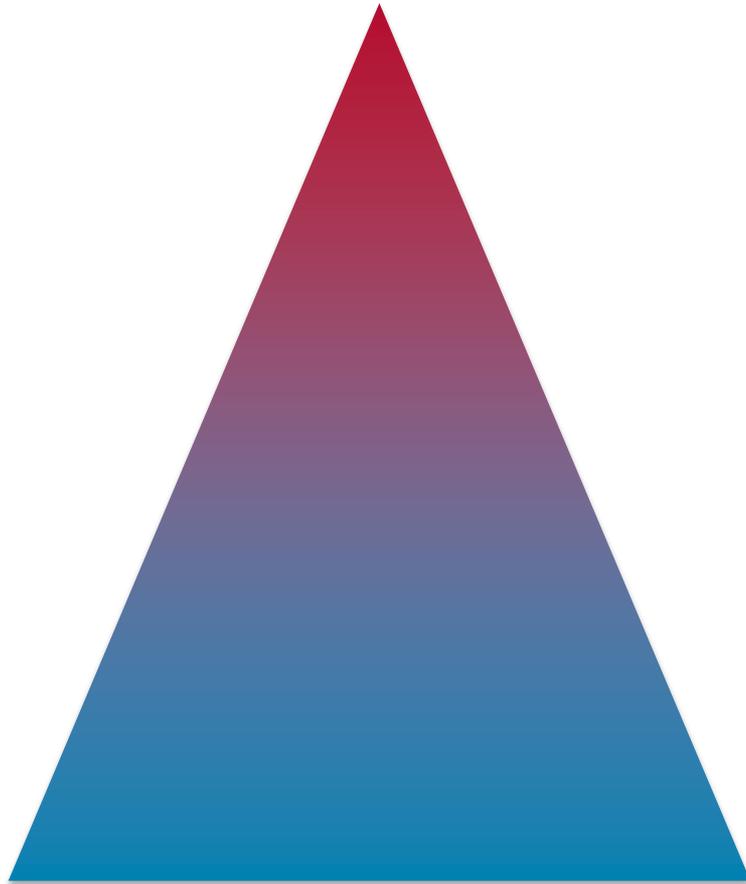
Title of the experiment

Brief methodology

Key findings

Clear indicators

# Discussion



Summary of findings



Relevance of  
findings



Implications for  
the field

# Discussion – flow of information

GPER is an E<sub>2</sub> binding, G-protein coupled membrane receptor that was reported to be overexpressed in breast endometrial, ovarian and thyroid cancers. The results presented here extend these observations to show that different types of lung cancers including adenocarcinomas, squamous cell carcinoma and large cell carcinomas express higher GPER than normal lung tissue. Here, we demonstrate for the first time that GPER is overexpressed in lung tumors and lung adenocarcinoma cell lines relative to normal lung and immortalized normal lung cell lines, although the expression of GPER transcript in HPL1D cells is higher than HBECS.

GPER has been postulated to be involved in E<sub>2</sub>-activation of EGFR. Filardo's group showed a link between GPER expression and tumor progression and increased tumor size in breast cancer patients. Recently, GPER overexpression was reported to be independent of ER $\alpha$  expression in breast cancer patient samples, indicating the importance of GPER in ER $\alpha$  negative tumors. GPER and EGFR expression were correlated in endometrial adenocarcinoma. Further, overexpression of GPER in advanced stage endometrial adenocarcinoma correlated with poor survival. Other studies also suggest increased GPER in breast, ovarian and endometrial cancers correlates with disease severity and reduced survival. These results are in agreement with studies demonstrating association of GPER overexpression in other cancers, although the scoring patterns and correlation of expression levels to disease state may vary among these studies.

A limitation of our study is that the average GPER staining scores among different lung cancer grades (I (10 cases), II (30 cases), III (16 cases)) were not significantly different. One other limitation of the current study is that we cannot conclude at this time whether GPER overexpression is cause or consequence of cancer. It is also possible that overexpression of GPER in lung cancers may reflect a defense mechanism to counteract excessive proliferation. Indeed, a recent report by Krakstad *et al.* showed that loss of GPER in ER $\alpha$ -positive endometrial cancers is associated with poor prognosis. Another study showed that the GPER agonist G-1 inhibited E<sub>2</sub>-induced uterine epithelial cell proliferation in mice by repressing MAPK activation, indicating that GPER effects are tissue specific. Because our studies were performed on commercial TMAs, the results cannot be extrapolated to correlate GPER expression levels to disease outcomes. Clearly, this is a next logical step in light of the novel findings.

We observed no differences in GPER expression between adenocarcinoma cell lines or tumors from male and female patients, similar to the previous findings of no difference in ER $\alpha$  or ER $\beta$  expression in NSCLC cells and tumors based on gender. In Western blots, rather than rely on one GPER antibody in our study, we used 3 different commercial antibodies to determine the correlation between mRNA and protein levels. It is indeed evident from our Western blot data that GPER appears to have different MW forms, likely due to glycosylation, dimerization, and interaction with other membrane proteins, and levels in the lung adenocarcinoma cell lines. More trivial explanations for the different staining patterns of GPER in Western blots may be due to differential purity/affinity of the three GPER antibodies as well as their capacity to bind to secondary antibodies. It will be important to determine the nature of these forms by proteomic analysis and gene sequencing to evaluate their biological significance.

Mechanism-based studies showed that GPER transactivates EGFR in breast cancer cells as well as in thyroid, endometrial and ovarian cancer cell lines. Inhibitors of EGFR tyrosine kinase (gefitinib) and ER (fulvestrant, ICI 182,780) were reported to synergize their anti-proliferative effects in NSCLC. Given the importance of EGFR signaling as a therapeutic target in lung cancer, further examination of the effect of EGF, heregulin, and amphiregulin on GPER expression and function in lung cancer may provide new insights into resistance to EGFR inhibitors and or how estrogens stimulate lung cancer.

In conclusion, the data presented in this manuscript demonstrate that GPER expression is higher in lung tumors compared to normal lung tissue. While it is not yet clear that elevated GPER expression is a cause of or consequence from lung cancer progression. Functional analysis of the effect of GPER expression will facilitate further delineation of the role of GPER in lung cancer.

Summary

Key findings

Relevance

Previous studies

Unexpected results

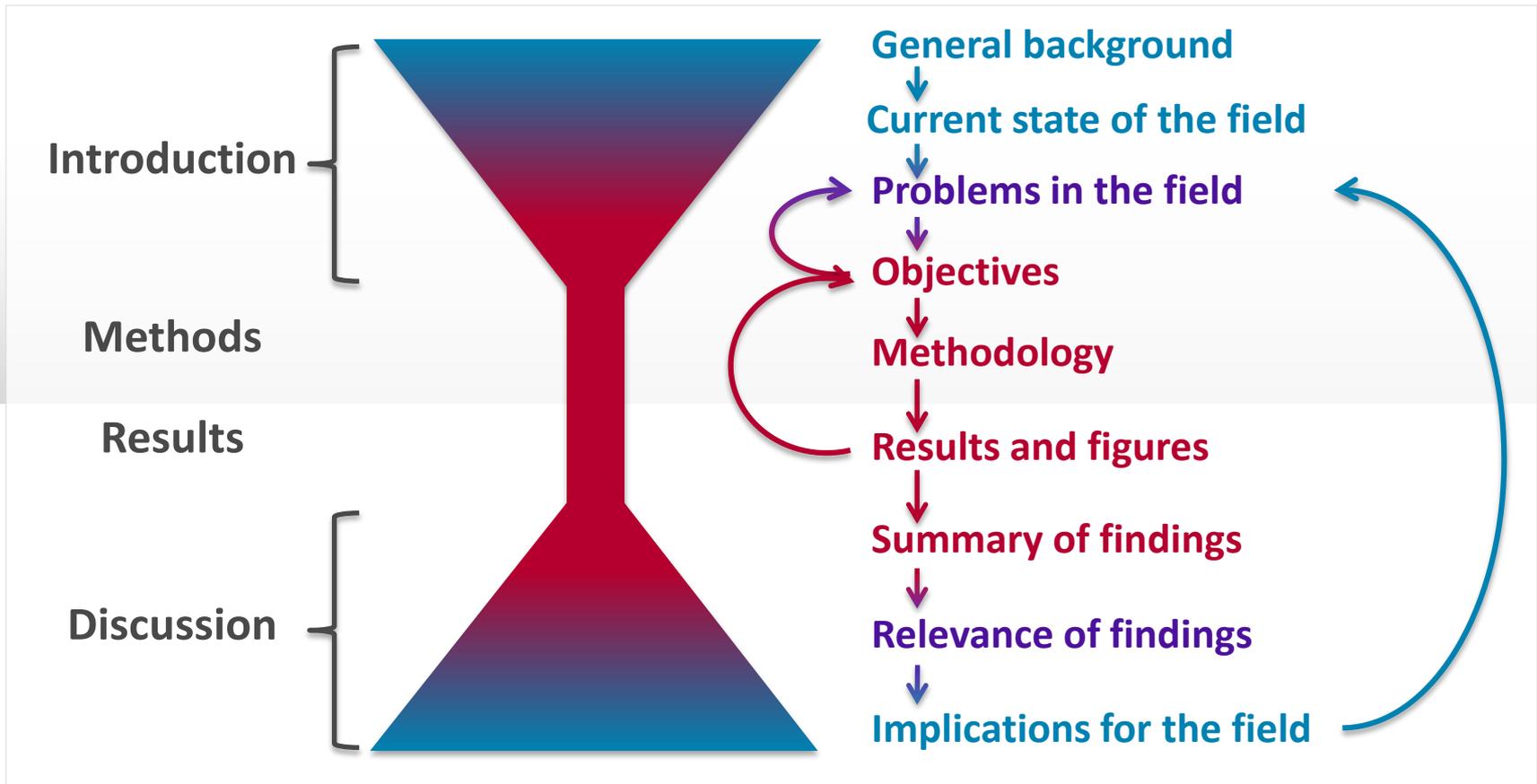
Limitations

Conclusions

Major conclusion

Implications

# Linking your ideas



*Logically link your ideas throughout your manuscript*

# Linking your ideas

## *Introduction*

New ways to treat or prevent lung cancer are therefore needed.

This study explored the hypothesis that inhibition of TNKS...would inhibit lung cancer growth...

## *Discussion*

Pharmacological or genetic inhibition of TNKS1 and TNKS2...reduces lung cancer proliferation...

Problem

Objectives

Conclusion

## Section 4

### *Titles and abstracts*

# Effective titles

## Important points

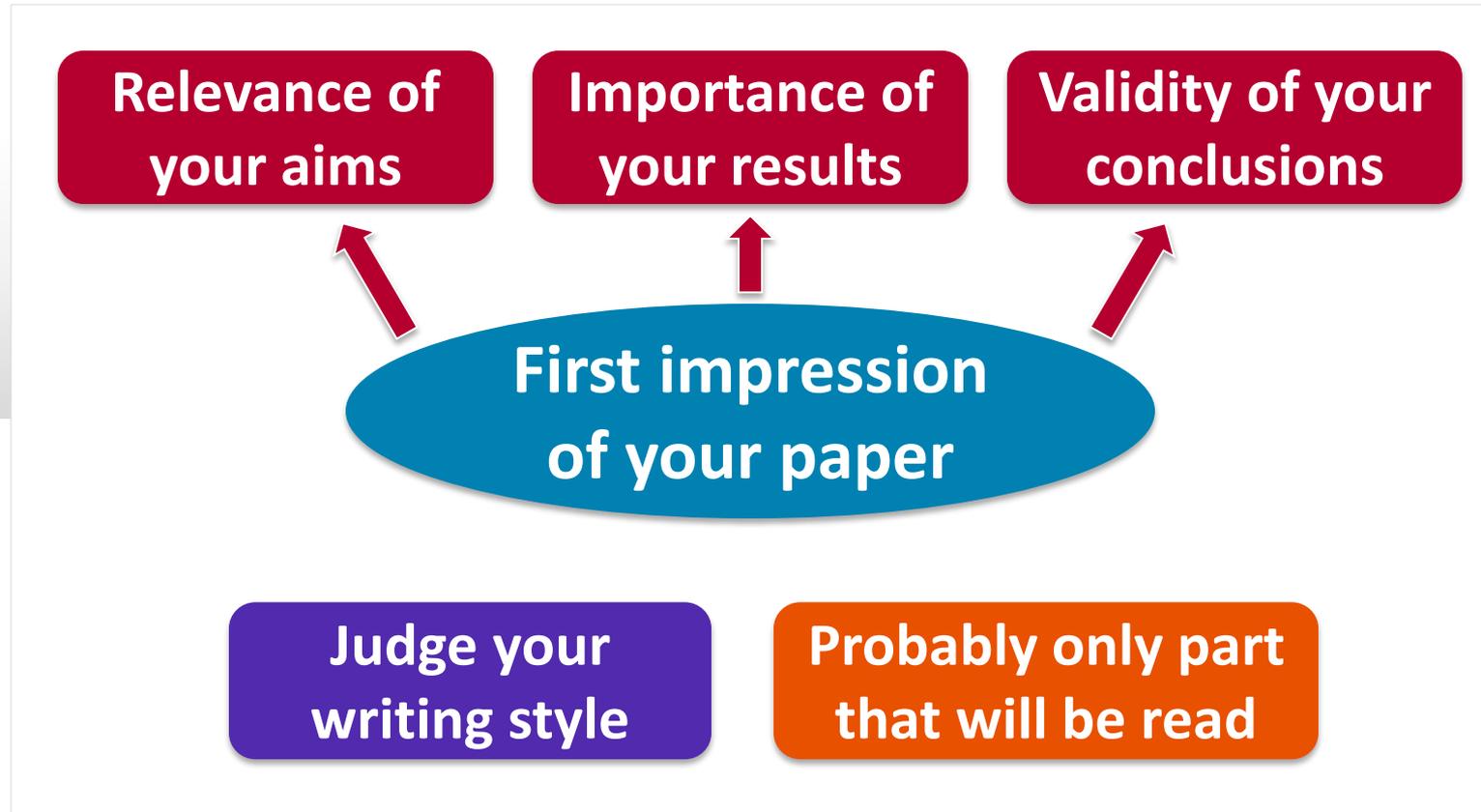
- ✓ Summarize key finding
- ✓ Contains keywords
- ✓ Less than 20 words

## Avoid

- ✗ Questions
- ✗ Abbreviations
- ✗ “New” or “novel”

*Your title should be a concise summary of your most important finding*

# Abstract



# Sections of an abstract

*Concise summary of your research*

**Background**



**Why the study was done**

**Aims**



**Your hypothesis**

**Methods**



**Techniques**

**Results**



**Most important findings**

**Conclusion**



**Conclusion/implications**

# Unstructured abstract

Our understanding of the mechanisms by which ducts and lobules develop is derived from model organisms and three-dimensional (3D) cell culture models wherein mammalian epithelial cells undergo morphogenesis to form multicellular spheres with a hollow central lumen. However, the mechanophysical properties associated with epithelial morphogenesis are poorly understood. We performed multidimensional live-cell imaging analysis to track the morphogenetic process starting from a single cell to the development of a multicellular, spherical structure composed of polarized epithelial cells surrounding a hollow lumen. We report that in addition to actively maintaining apicobasal polarity, the structures underwent rotational motions at rates of 15–20  $\mu\text{m}/\text{h}$  and the structures rotated 360° every 4 h during the early phase of morphogenesis. Rotational motion was independent of the cell cycle, but was blocked by loss of the epithelial polarity proteins Scribble or Pard3, or by inhibition of dynein-based microtubule motors. Interestingly, none of the structures derived from human cancer underwent rotational motion. We found a direct relationship between rotational motion and assembly of endogenous basement membrane matrix around the 3D structures, and that structures that failed to rotate were defective in weaving exogenous laminin matrix. Dissolution of basement membrane around mature, nonrotating acini restored rotational movement and the ability to assemble exogenous laminin. Thus, coordinated rotational movement is a unique mechanophysical process observed during normal 3D morphogenesis that regulates laminin matrix assembly and is lost in cancer-derived epithelial cells.

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Our understanding of the mechanisms by which ducts and lobules develop is derived from model organisms and three-dimensional (3D) cell culture models wherein mammalian epithelial cells undergo morphogenesis to form multicellular spheres with a hollow central lumen. However, the mechanophysical properties associated with epithelial morphogenesis are poorly understood.

**Background**

We performed multidimensional live-cell imaging analysis to track the morphogenetic process starting from a single cell to the development of a multicellular, spherical structure composed of polarized epithelial cells surrounding a hollow lumen.

**Methods**

We report that in addition to actively maintaining apicobasal polarity, the structures underwent rotational motions at rates of 15–20  $\mu\text{m}/\text{h}$  and the structures rotated 360° every 4 h during the early phase of morphogenesis. Rotational motion was independent of the cell cycle, but was blocked by loss of the epithelial polarity proteins Scribble or Pard3, or by inhibition of dynein-based microtubule motors. Interestingly, none of the structures derived from human cancer underwent rotational motion. We found a direct relationship between rotational motion and assembly of endogenous basement membrane matrix around the 3D structures, and that structures that failed to rotate were defective in weaving exogenous laminin matrix. Dissolution of basement membrane around mature, nonrotating acini restored rotational movement and the ability to assemble exogenous laminin.

**Results**

Thus, coordinated rotational movement is a unique mechanophysical process observed during normal 3D morphogenesis that regulates laminin matrix assembly and is lost in cancer-derived epithelial cells.

**Conclusion**

# Link ideas in your abstract

Titles and abstracts

Our understanding of the mechanisms by which ducts and lobules develop is derived from model organisms and three-dimensional (3D) cell culture models wherein mammalian epithelial cells undergo morphogenesis to form multicellular spheres with a hollow central lumen. However, the mechanophysical properties associated with epithelial morphogenesis are poorly understood.

**Background**

**However, the mechanophysical properties associated with epithelial morphogenesis are poorly understood.**

**Problem**

**Thus, coordinated rotational movement is a unique mechanophysical process...**

**Answer**

Thus, coordinated rotational movement is a unique mechanophysical process observed during normal 3D morphogenesis that regulates laminin matrix assembly and is lost in cancer-derived epithelial cells.

**Conclusion**

# Journal editors are busy!



<http://www.edanzediting.com/mexico2014>

## Section 5

### *Cover letters*

# Cover letters

**Significance  
Relevance**



**Is your work  
important?**

**Cover letters are the first impression for  
the journal editor**

**Interesting to  
their readers?**

**Level of English**

## Cover letters

# A good cover letter

Dear Dr Graeber,

**Editor's name**

**Manuscript title**

Please find enclosed our manuscript entitled "Amyloid-like inclusions in the brains of Huntington's disease patients" by McGowan et al., which we would like to submit for publication as a Research Paper in *Neurogenetics*.

**Publication type**

Recent immunohistochemical studies have revealed the presence of neuronal inclusions containing an N-terminal portion of the mutant huntingtin protein and ubiquitin in the brain tissues of Huntington's disease (HD) patients; however, the role of these inclusions in the disease process has remained unclear. One suspected disease-causing mechanism in Huntington's disease and other polyglutamine disorders is the potential for the mutant protein to undergo a conformational change to a more stable anti-parallel  $\beta$ -sheet structure...

**Give the background to the research**

To confirm if the immunohistochemically observed huntingtin- and ubiquitin-containing inclusions display amyloid features, we performed Congo red staining and both polarizing and confocal microscopy on post-mortem human brain tissues obtained from five HD patients, two AD patients, and two normal controls. Congo red staining revealed a small number of amyloid-like inclusions showing green birefringence by polarized microscopy, in a variety of cortical regions.... ..detected inclusions observed in parallel sections, suggesting that only a relatively small proportion of inclusions in HD adopt an amyloid-like structure.

**What was done and what was found**

We believe our findings will be of particular interest to the readership of *Neurogenetics*, which includes researchers and clinicians studying the genetic and molecular mechanisms underlying neurodegenerative diseases. Therefore, we feel that your journal provides the most suitable platform for the dissemination of our work to the research community.

**Interest to journal's readers**

We would also like to suggest the following reviewers for our manuscript...

**Recommend reviewers**

# Disclaimers about publication ethics

Original and unpublished

Not submitted to other journals

Authors agree on paper/journal

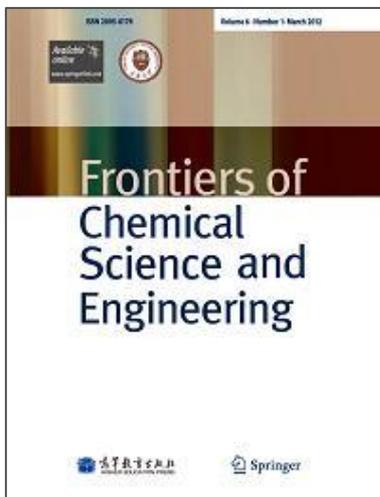
**“Must-have” statements**

No conflicts of interest

Source of funding

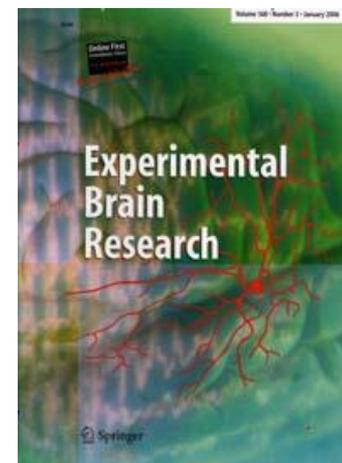
Authorship contributions

# Recommending reviewers



“When submitting a paper authors are requested to suggest **5** potential referees, supplying the full name, address, e-mail and research field in each case.

“When submitting your paper, you must provide the names, affiliations, and valid e-mail addresses of **five (5)** reviewers. If you do not do so, your paper will be returned, **unreviewed.**”



# Recommending reviewers

**Where to find them?**

From your reading/references, networking at conferences

**How senior?**

Aim for mid-level researchers

**Who to avoid?**

Collaborators (past 5 years), researchers from same institution

**International list:**

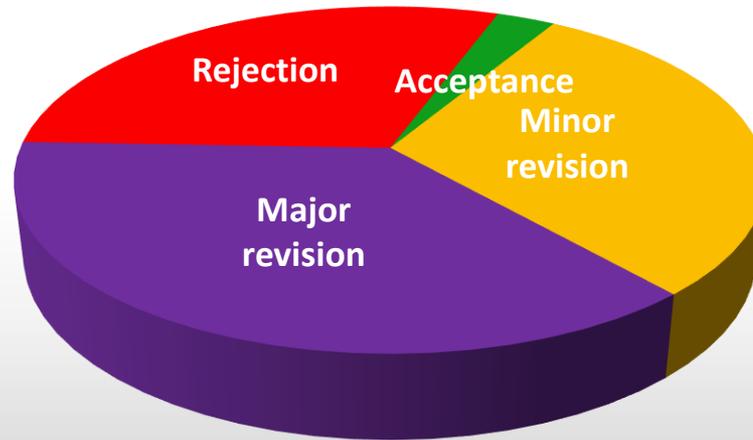
1 or 2 from Asia, 1 or 2 from Europe, and 1 or 2 from the Americas

<http://www.edanzediting.com/mexico2014>

## Section 6

***Peer Review***

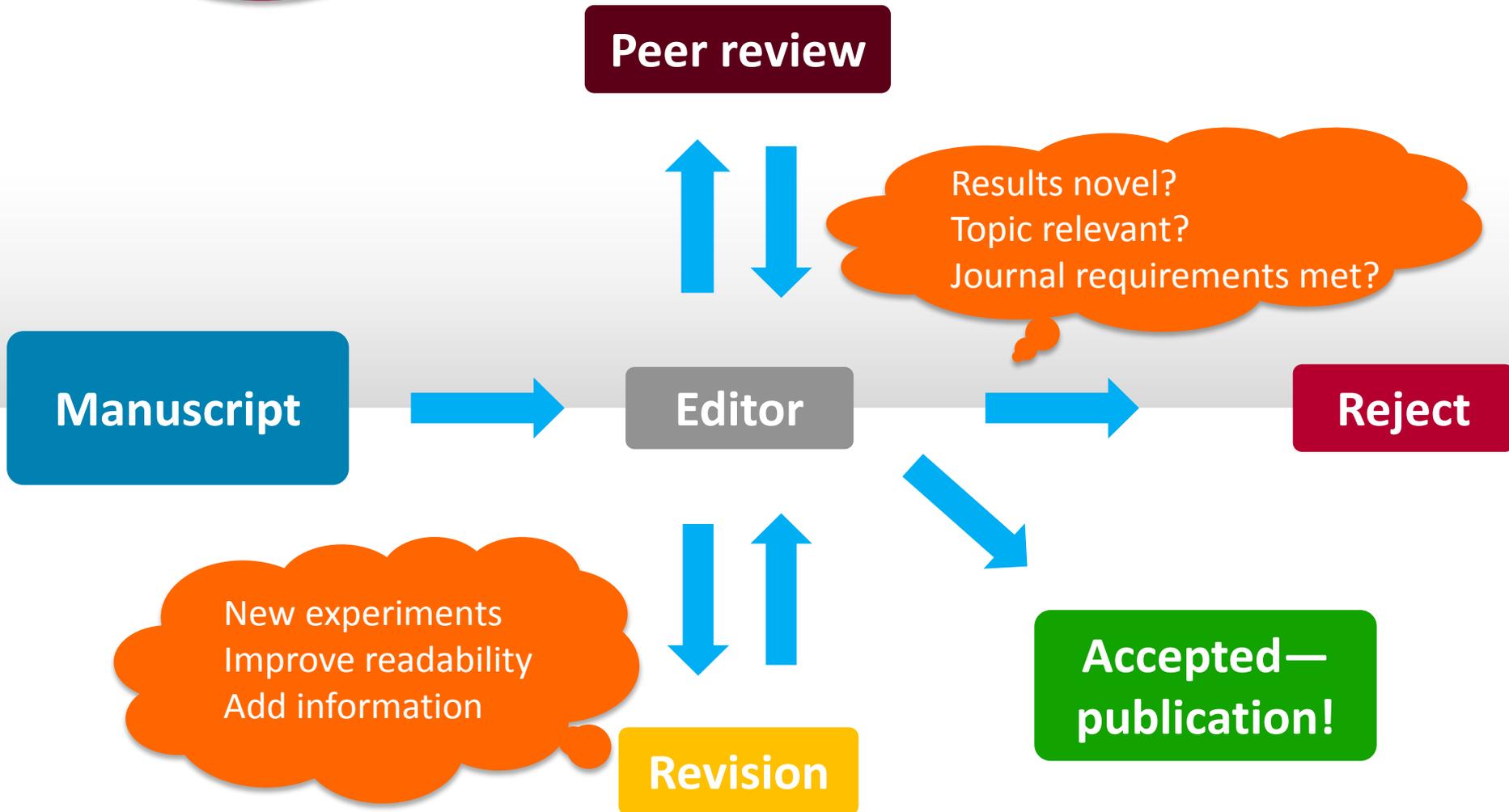
# Peer review improves your manuscript



- Few papers are accepted without revision
- Rejection and revision are integral
- Peer review should be a positive process

Peer review

# Submission process and time frames



# What reviewers are looking for

## The science

- ✓ Relevant hypothesis
- ✓ Good experimental design
- ✓ Appropriate methodology
- ✓ Good data analysis
- ✓ Valid conclusions

## The manuscript

### Discussion

- ✓ High readability

Formatting

# Revision

Respond to *every* reviewer comment

Response  
letter

*Easy* to see  
changes

Refer to line and page numbers

Use a different color font

Highlight the text

# Writing a response letter

Marc Lippman, MD  
Editor-in-Chief  
*Breast Cancer Research and Treatment*

3 September 2013

Dear Dr Lippman,

Re: Resubmission of manuscript reference No. WJS-07-5739

Please find attached a revised version of our manuscript originally entitled "Evaluation of the Glasgow prognostic score in patients undergoing curative resection for breast cancer liver metastases," which we would like to resubmit for consideration for publication in the *Breast Cancer Research and Treatment*.

The reviewer's comments were highly insightful and enabled us to greatly improve the quality of our manuscript. In the following pages are our point-by-point responses to each of the comments.

Revisions in the manuscript are shown as underlined text. In accordance with the first comment, the title has been revised and the entire manuscript has undergone substantial English editing.

We hope that the revisions in the manuscript and our accompanying responses will be sufficient to make our manuscript suitable for publication in the *Breast Cancer Research and Treatment*.

Address editor personally

Manuscript ID number

Thank reviewers



Highlight major changes

# Agreeing with reviewers

***Reviewer Comment:** In your analysis of the data you have chosen to use a somewhat obscure fitting function (regression). In my opinion, a simple Gaussian function would have sufficed. Moreover, the results would be more instructive and easier to compare to previous results.*

**Response:** We agree with the reviewer's assessment of the analysis. Our tailored function, in its current form, makes it difficult to tell that this measurement constitutes a significant improvement over previously reported values. We describe our new analysis using a Gaussian fitting function in our revised Results section (Page 6, Lines 12–18).

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## Agreement

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**Revisions**

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**Revisions**

**Location**

# Disagreeing with reviewers

***Reviewer Comment:** In your analysis of the data you have chosen to use a somewhat obscure fitting function (regression). In my opinion, a simple Gaussian function would have sufficed. Moreover, the results would be more instructive and easier to compare to previous results.*

**Response:** Although a simple Gaussian fit would facilitate comparison with the results of other studies, our tailored function allows for the analysis of the data in terms of the Smith model [Smith et al., 1998]. We have now explained the use of this function and the Smith model in our revised Discussion section (Page 12, Lines 2–6).

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Evidence

Revisions

Location

# “Unfair” reviewer comments

*Reviewer comment: Currently, the authors' conclusion that this gene is involved in heart development is not completely validated by their in vitro analyses. They should do additional in vivo experiments using a genetic mouse model to show that heart development is regulated by this gene.*

## ***Reasons why reviewers might make these comments***

- ❖ **Current results are not appropriate for the impact factor of the journal**
- ❖ **Reviewer is being “unfair”**

# If rejected, what should you do?

## Option 1: New submission to the same journal

- ❖ Fully revise manuscript
- ❖ Prepare point-by-point responses
- ❖ Include the original manuscript ID number

## Option 2: New submission to a different journal

- ❖ Revise manuscript
- ❖ Reformat according to the author guidelines

# If accepted, what's next?

*Your goal is not only to be published,  
but also to be widely read/cited*

## ❖ Promote your work on social networks

- Twitter, LinkedIn, Research Gate

## ❖ Respond to post-publication comments

## ❖ Present your work at conferences

- Promote your publication
- Allows you to discuss your work personally with your peers
- Get feedback about your work and future directions

# Be an effective communicator

- ✓ **Write effectively**
- ✓ **Choose the best journal to reach your target audience**
- ✓ **Logically present your research in your manuscript**
- ✓ **Convey the significance of your work to journal editors**
- ✓ **Properly revise your manuscript after peer review**

# *Any questions?*

*Thank you!*

Daniel McGowan: [dmcgowan@edanzgroup.com](mailto:dmcgowan@edanzgroup.com)



[edanzediting.com/mexico2014](http://edanzediting.com/mexico2014)

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