Beyond the Data:
Telling your Story Effectively in a Scientific Paper

June 6, 2025

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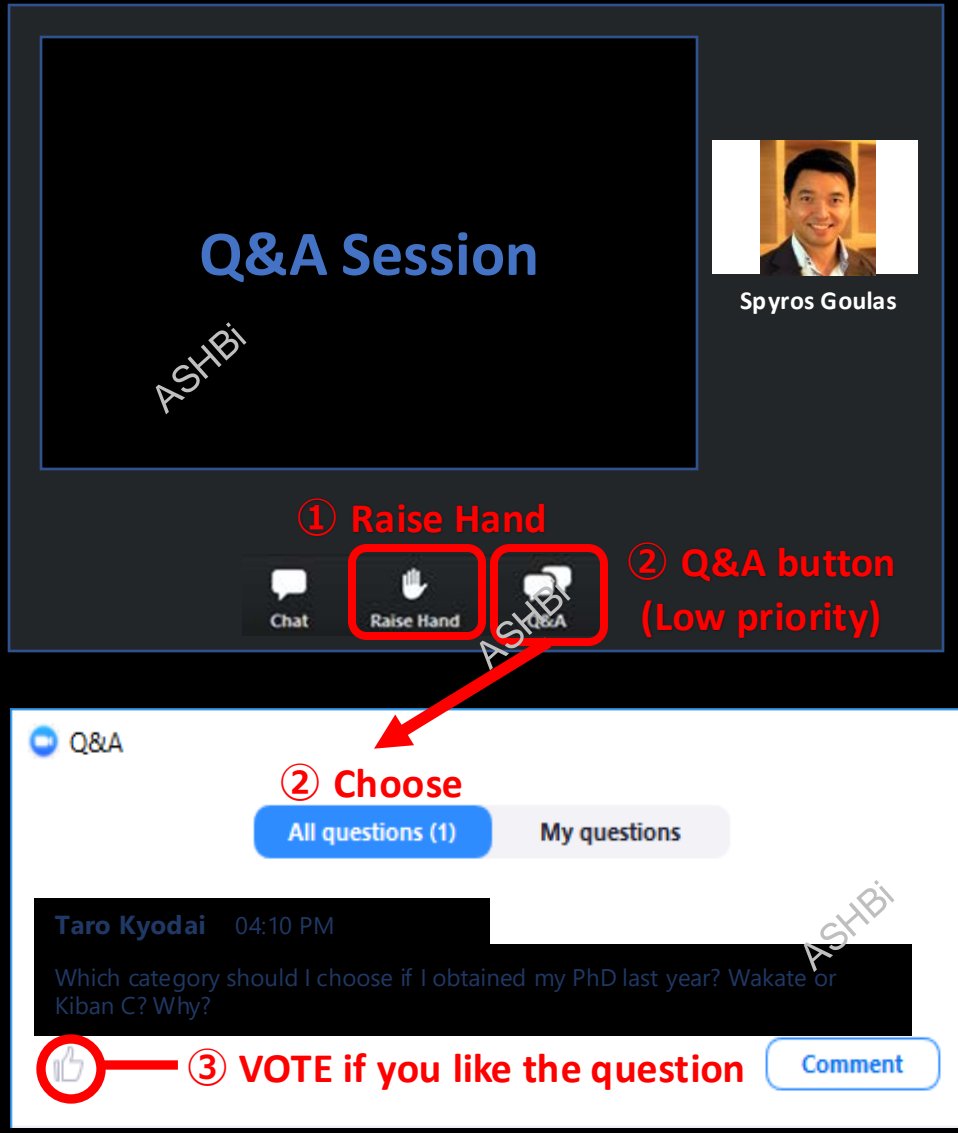
Ex-Researcher & Journal Editor

Requests to the Audience: Q&A

During the Q&A Session, we will take questions from the audience.

1. If you have a question, click "Raise Hands". Once your name is called, please "unmute" yourself to ask your question.
2. You can also ask questions using "Q&A button". You can write your questions during the talk.
3. You can also vote for questions already asked that you like by press the "Thumbs Up button" in Q&A.

[NOTE] We will prioritize oral questions from the audience. The questions in the "Q&A" box may not all be answered due to time constraints.

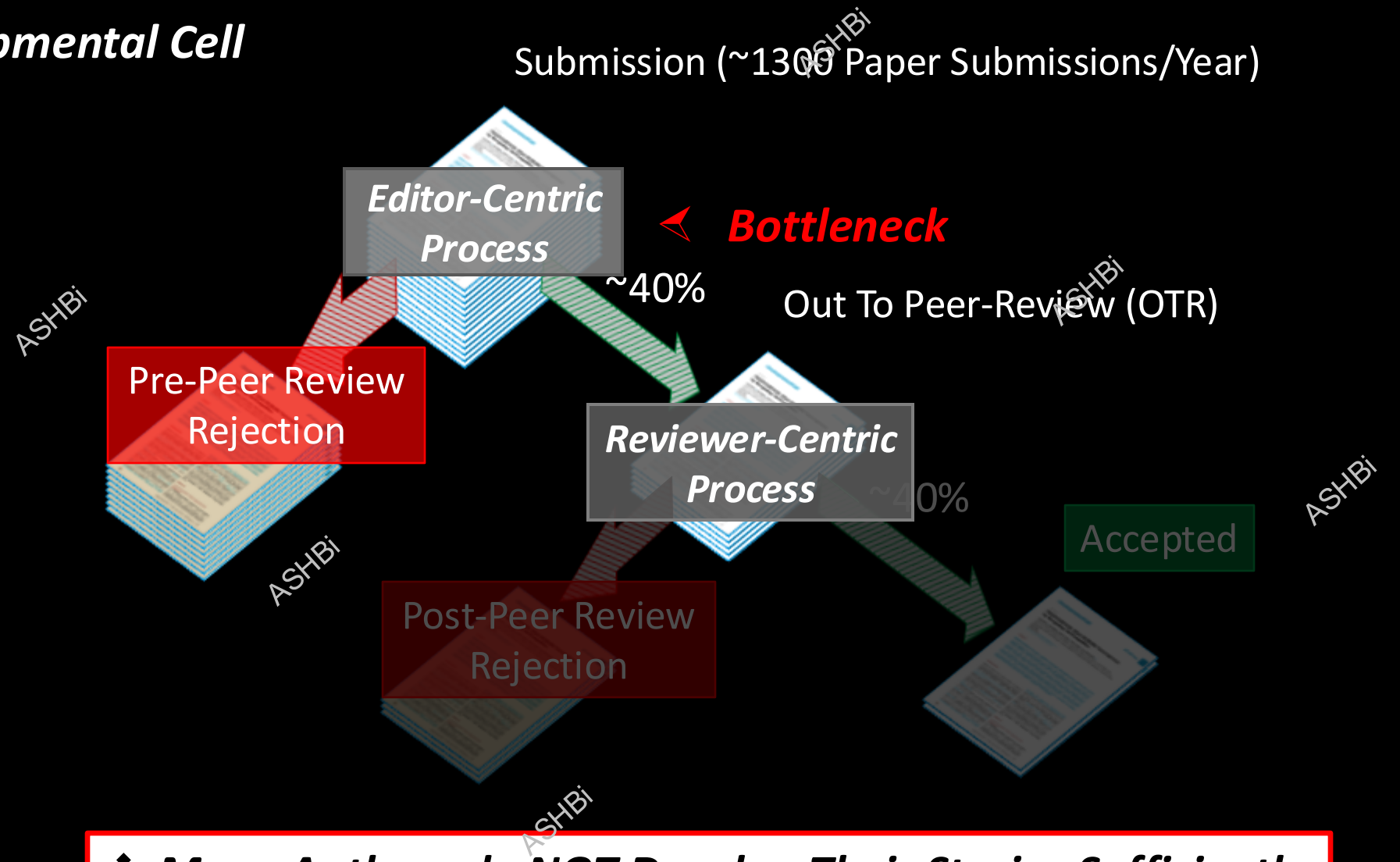


Who am I?

- 2006-2011, ***PhD in Molecular Biology*** (lab of **Dr. Juergen Knoblich**)
  Institute of Molecular Biotechnology Austria (IMBA)/University of Vienna
- 2012-2018, ***Post-Doc/Special-Appointed Assis. Prof.*** (lab of **Prof. Shigeo Ohno**)
  Yokohama City University
- 2018-2022, ***Associate Scientific Editor*** (handled **~1600 papers**) at ***Developmental Cell***
  (**Cell Press/Elsevier**)
- 2022- , ***Scientific Advisor***
  Institute of Advanced Studies for Human Biology (WPI-**ASHBi**)/Kyoto University
  and the Stellar Science Foundation (SS-F)

The Editorial Process from Submission to Acceptance

eg. Developmental Cell



❖ ***Many Authors do NOT Develop Their Stories Sufficiently***

Aim of Today's Talk

From a Former Editor's Perspective:

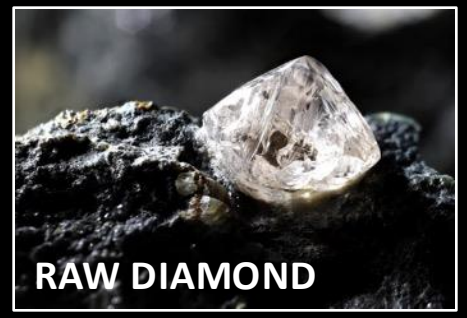
- *The Value and Function of Telling your Story in a Scientific Paper*
- *Strategies on How to Tell your Story more Effectively*

Ultimate Goal =*To Provide the Necessary Know-How to Publish You Paper Efficiently*

Today's Agenda

1) *What is Scientific Storytelling?*
2) *Why is Telling your Story Important?*
3) *How to Tell your Story Effectively*
   a. *The Basic Structure of a Scientific Story*
   b. *Example of a Paper with Good Storytelling*
4) *Practical Advice on How to Prepare your Paper*

Storytelling in a Paper is like Diamond Processing



Diamond Processing
eg. cutting, polishing

Storytelling

Storytelling

❖ ***Storytelling is <u>NOT</u> Magic***

A Good Story is a Path to Establishing New Understanding

Existing KNOWLEDGE → **Your QUESTION** → **Your HYPOTHESIS** → **Your DATA** → **Your INTERPRETATION** → **New KNOWLEDGE** → **New UNDERSTANDING**

Interpretation – *To Find the Meaning Behind the Raw Data*

Often Underdeveloped

Knowledge – *Facts Generated from Data Points*

Understanding – *Assimilation of Various Pieces of Knowledge to Acquire a Deeper Insight into Processes, How They Function and its Potential Impact on Society*

Editors *Scientists* *Public*

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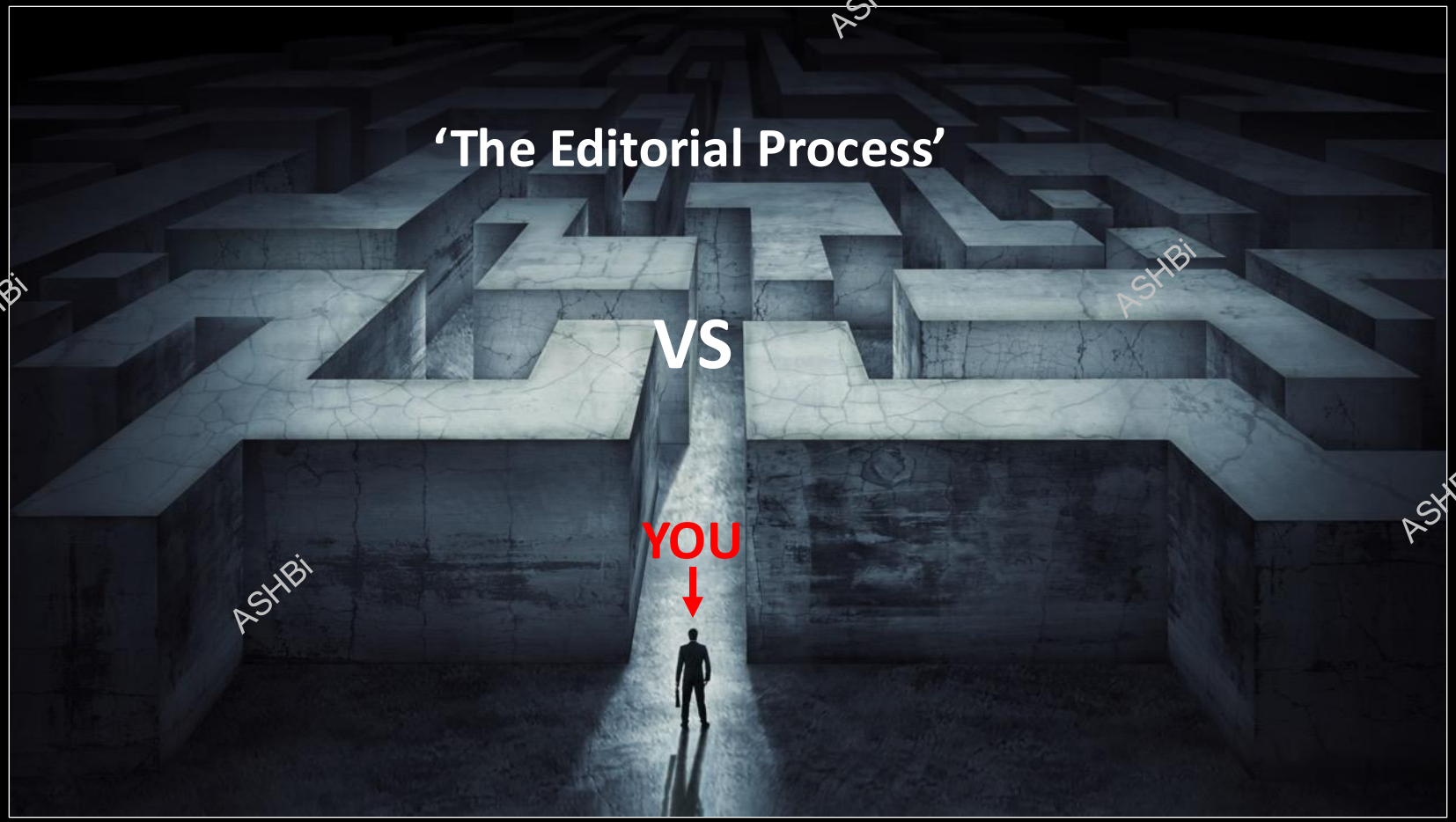
Author’s Perspective of the Editorial Process



What Happens during the Editorial Process?

Limited Time to Convince the Editor your Paper is Interesting

AUTHOR(S)

Paper Submission

Our paper is interesting because...

~ 20mins-1hr

HANDLING Editor

Editorial Evaluation

- *Read Paper*
- *Background/Relevant Literature*
- *Strengths/Weaknesses of Paper*

I also have another 20-30 papers I need to handle...

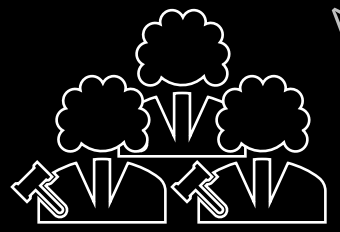
❖ ***Communicating your Paper Effectively is Critical during its Editorial Evaluation***

Editors Need to Convince Their Team to OTR your Paper

AUTHOR(S) **HANDLING Editor** **STEP 2: Editorial Team** **Peer Review**

Submission

Meeting



OTR

Our paper is interesting because…

~20mins-1hr

Their paper is interesting because…

~5-10mins.

What are the Criteria used to Evaluate Manuscripts?

Editorial Criterias used for Manuscript Evaluation

Evaluation Criteria

- Novelty/Originality
- Conceptual Advance
- Technical Advance
- Utility of Dataset
- Scientific Landscape
- Publication Landscape
- etc.

Other Considerations

- Experimental Design
- Reader Accessibility
- etc.

X-Factor

- Editorial Enthusiasm

- Does it ask a broadly interesting/important question?
- Does it significantly move the field forward?
- Will it be of interest beyond the immediate field?
- Does it change the way we think about a biological idea or process?

❖ ***Many Evaluation Critierias used for Manuscripts are Subjective***

Common Conceptual Reasons for Rejections Pre-Peer Review

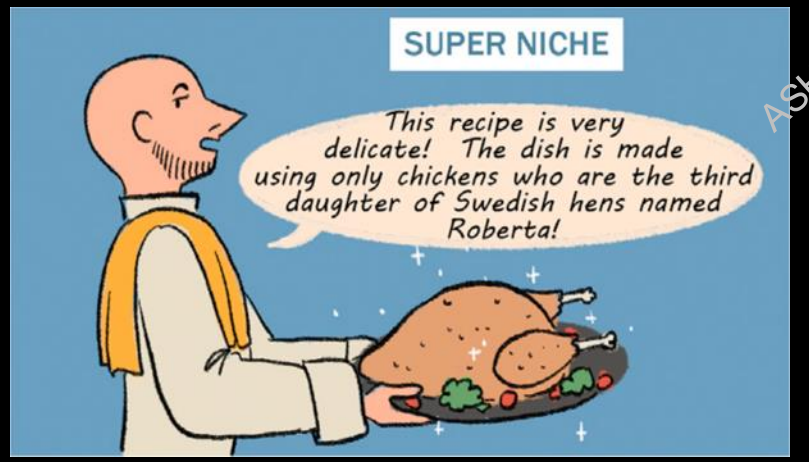
The 4 Rejection Archetypes, Via Cooking Metaphors

Chin & Crawford, 2020. Matter

eg. factor A replaced by B

eg. identifying additional factors/functions in process

eg. factor A and B are known but are combined



eg. appealing to a narrow audience

❖ ***Developing your Story can Change the Opinion & Value of your Research to an Editor***

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Orthodox Structure of a Story in Your Paper

Introduction

Methods

Results

Discussion

General Background & Broad Concepts/Questions

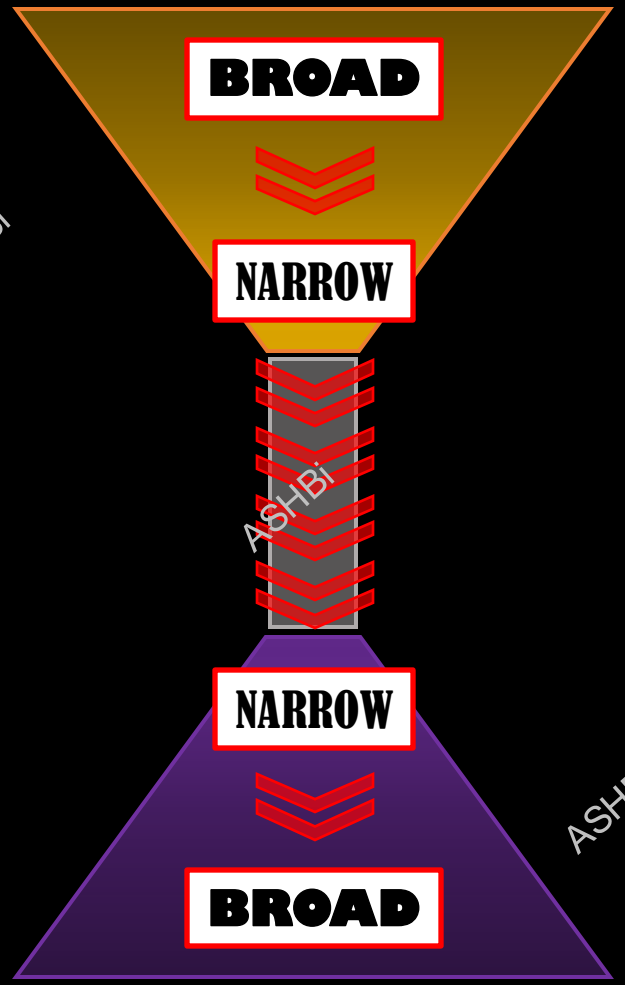
Specific Background & Your Specific Question

Your Methodology/Findings

Answering Your Question

Social Impact & Broader Implications

Hour Glass Structure of Paper



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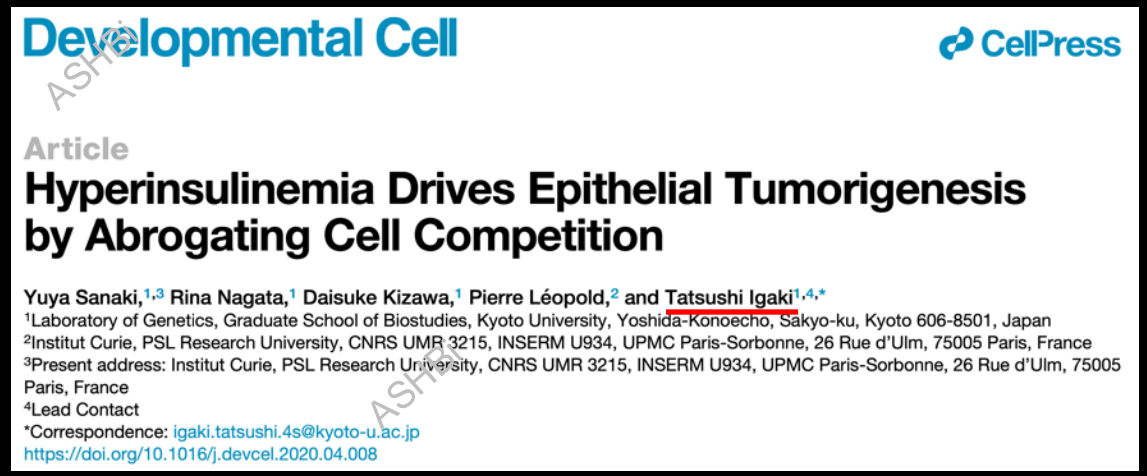
Example of a Scientific Paper with Good Storytelling

DOI:https://doi.org/10.1016/j.devcel.2020.04.008
Sanaki et al., 2020. Dev Cell



Developmental Cell

CellPress

Article

Hyperinsulinemia Drives Epithelial Tumorigenesis by Abrogating Cell Competition

Yuya Sanaki,[1,3] Rina Nagata,[1] Daisuke Kizawa,[1] Pierre Léopold,[2] and Tatsushi Igaki[1,4,*]
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https://doi.org/10.1016/j.devcel.2020.04.008

The Central QUESTION

How does Hyperinsulinemia Lead to Tumor Growth?

1) Identify a Factor needed for ***Tumor***-removing ***Cell Competition***
2) Loss of this Factor causes ***Hyperinsulinemia***
3) Increased Insulin prevents Tumor-removing Cell Competition
4) Changing Diet or using Anti-Diabetic Drug affects Tumor Growth

Key Definitions

Drosophila= Fruit Fly (ショウジョウバエ)
Hyperinsulinemia= The Overproduction of Insulin
Cell Competition (細胞競合)= Mechanism that Removes Abnormal 'Loser' Cells like Tumor Cells
Tumor (腫瘍)= Abnormal Cells with Potential to become Cancer

The ANSWER

Hyperinsulinemia Promotes Tumor Growth by Preventing Cell Competition

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Introduction: Framing your Question to Engage the Audience

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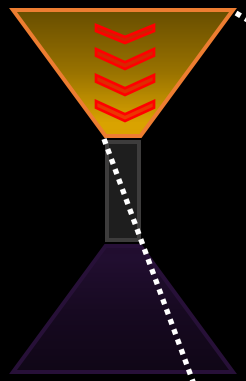
Sanaki et al., 2020. Dev Cell

PAPER STRUCTURE



Introducing Context/Problem of Broad Interest

- *Metabolic diseases*
- *Cancers*
- *Links between Metabolism and Cancer*

Advantages of System Used

The Central QUESTION

- *How does hyperinsulinemia promote tumor growth?*

INTRODUCTION

Metabolic diseases such as type 2 diabetes and obesity are often accompanied by hyperinsulinemia, which is characterized by high levels of circulating insulin. In epidemiology, hyperinsulinemia has been implicated in increased cancer incidence (Pollak, 2008; Giovannucci et al., 2010; Shi and Hu, 2014; Tsujimoto et al., 2017). For instance, the risk of liver, pancreas, endometrium, kidney, and bladder cancers increases 1.5- to 2-fold in people with hyperinsulinemia (Vigneri et al., 2009). Although previous studies in *Drosophila* and rodents unveiled some aspects of the mechanism by which hyperinsulinemia promotes tumor growth and malignancy (Hirabayashi et al., 2013; Xu et al., 2018), the underlying mechanisms are still largely unknown (Zhang et al., 2019).

Introduction: Heightening Curiosity & Anticipation

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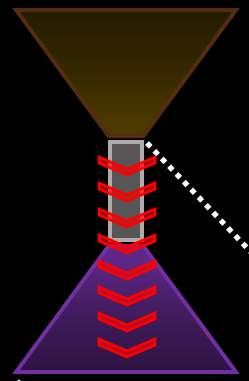

teros-Arias, 2015; Di Gregorio et al., 2016; Clavería and Torres, 2015; Baker, 2017; Madan et al., 2018; Nagata and Igaki, 2018). We have previously found multiple mechanisms that drive this cell elimination via cell-cell interaction between *scrib* and wild-type cells, which include Sas-PTP10D ligand-receptor interaction (Yamamoto et al., 2017), Slit-Robo2-Ena/VASP-mediated *scrib* cell extrusion (Vaughen and Igaki, 2016), and engulfment of *scrib* cells by wild-type cells (Ohsawa et al., 2011). Here, through a genetic screen in *Drosophila*, we find an unexpected new regulatory mechanism whereby hyperinsulinemia systemically abrogates tumor-suppressive cell competition and thus causes tumorigenesis in the epithelium. Our data could provide a mechanistic explanation for the epidemiological evidence that links hyperinsulinemia and cancer incidence, thus contributing to a better understanding of cancer biology *in vivo*.

Brief Description of Results

- *Heightens anticipation and curiosity of results*

Brief Description of Implications

- *Linking results broadly to disease and society*

Results: Using Active Subheadings & Linking Subsections

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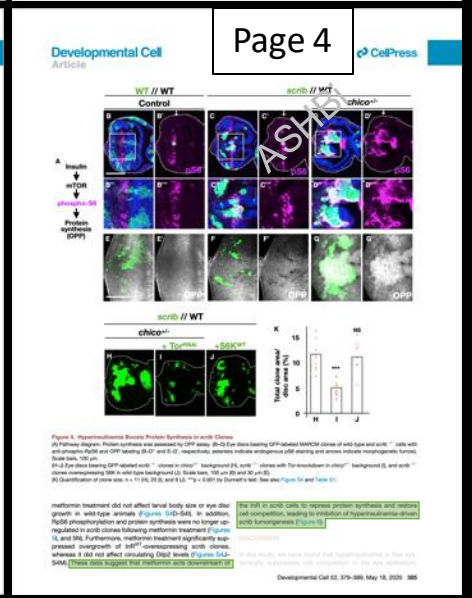

Downregulation of *chico* in IPCs Causes Hyperinsulinemia

We next investigated the consequences of *chico* downregulation in IPCs. As IPCs produce and secrete *Drosophila* insulin-like peptides (Dilps) (Brogiolo et al., 2001; Ikeya et al., 2002), we examined whether *chico* downregulation affects Dilps levels. Intriguingly, *chico* heterozygosity or IPCs-specific *chico* knockdown caused a significant increase in the expression of *dilp2* and *dilp5* mRNAs as well as anti-Dilp2 and anti-Dilp5 immunostainings without affecting IPCs' volume (Figures 2A, 2B, and S2A–S2E). These data indicate that downregulation of insulin signaling in IPCs causes upregulation of insulin peptide production probably via a feedback mechanism. To examine whether this leads to high levels of circulating insulin peptide, we measured circulating Dilp2 levels by a sandwich ELISA method using a modified Dilp2 fused with hemagglutinin (HA)-tag and FLAG-tag (aka. Dilp2-HF) (Park et al., 2014). Crucially, circulating Dilp2-HF levels was significantly increased in *chico* heterozygous animals at both early and late third instar larval stages (Figure 2C). Furthermore, analysis of insulin-PI3K signaling activity in the fat body, which would be elevated by circulating Dilps, by measuring the ratio of plasma membrane/cytosolic PH-GFP (GPH; Britton et al., 2002) signals revealed that insulin signaling activity was significantly increased in *chico* heterozygous larvae with or without *scrib* clones in the eye discs (Figures 2D–2I). Supporting this notion, circulating sugar levels in the larval hemolymph was significantly decreased in *chico* heterozygous animals at 96 h after egg deposition (AED) (Figure 2J). Together, these results indicate that downregulation of *chico* in IPCs causes hyperinsulinemia, an excess circulating insulin relative to blood sugar levels.

Active Subheadings

- *Allows reader to understand the essence of the sections with a glance*

Effective Linking Subsections

- *Allows reader to understand better your trail of thought and logic*

Subsection Summary Sentences

- *Re-enforces the message of each section*

Results: Logical Sequence of Data to Answer Your Question

INTRODUCTION

The Central QUESTION - *How does hyperinsulinemia promote tumor growth?* **(ie. what is the mechanism)**

RESULTS

WHAT factor regulates (the mechanism of) tumor growth?

1. *chico* Is Required for Tumor-Suppressive Cell Competition (*Fig. 1*)

WHERE does this factor function?

2. *chico* Is Required in IPCs to Eliminate *scrib* Cells in the Eye Discs (*Fig. 1*)

HOW does this factor (and mechanism) function?

3. Downregulation of *chico* in IPCs Causes Hyperinsulinemia (*Fig. 2*)
4. Hyperinsulinemia Abrogates *scrib* Cell Competition (*Fig. 2*)
5. Hyperinsulinemia Suppresses Cell Competition by Boosting Protein Synthesis in *scrib* Cell (*Fig. 3&4*)

WHAT are the SOCIAL & CLINICAL implications

6. Diet-Induced High Levels of Insulin Causes *scrib* Tumorigenesis (*Fig. 4&5*)
7. Metformin Suppresses Hyperinsulinemia-Induced *scrib* Tumorigenesis (*Fig. 5&6*)

DISCUSSION

The ANSWER - *Hyperinsulinemia promotes tumor growth by preventing cell competition*

Discussion: Don't Summarize, Answer the Question!!

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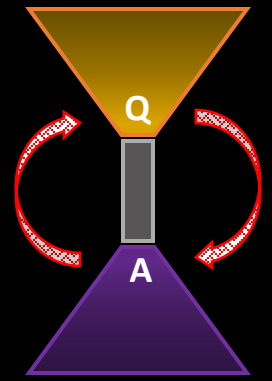
INTRODUCTION

Metabolic diseases such as type 2 diabetes and obesity are often accompanied by hyperinsulinemia, which is characterized by high levels of circulating insulin. In epidemiology, hyperinsulinemia has been implicated in increased cancer incidence (Pollak, 2008; Giovannucci et al., 2010; Shi and Hu, 2014; Tsujimoto et al., 2017). For instance, the risk of liver, pancreas, endometrium, kidney, and bladder cancers increases 1.5- to 2-fold in people with hyperinsulinemia (Vigneri et al., 2009). Although previous studies in *Drosophila* and rodents unveiled some aspects of the mechanism by which hyperinsulinemia promotes tumor growth and malignancy (Hirabayashi et al., 2013; Xu et al., 2018), the underlying mechanisms are still largely unknown (Zhang et al., 2019).

DISCUSSION

In this study, we have found that hyperinsulinemia in flies systemically suppresses cell competition in the eye epithelium, leading to tumorous overproliferation of polarity-deficient cells that are normally eliminated when surrounded by wild-type cells. It has been reported that high-sugar diet promotes tumor growth and metastasis of fly tumors with elevated Ras and Src signaling, providing a model of how abnormal physiology promotes tumor progression (Hirabayashi et al., 2013). In addition, studies in mice have shown that high-fat diet-induced obesity suppresses extrusion of oncogenic Ras^{V12}-expressing cells from mice intestine (Sasaki et al., 2018) and that endogenous hyperinsulinemia contributes to pancreatic ductal adenocarcinoma (Zhang et al, 2019). Thus, abnormal physiology, especially hyperinsulinemia,

PAPER STRUCTURE



100% Satisfied

Q: How does hyperinsulinemia promote tumor growth?

A: Hyperinsulinemia promotes tumor growth by avoiding cell competition

❖ ***Make Sure to ANSWER your QUESTION 100%***

Discussion: Going from Fact to Speculation

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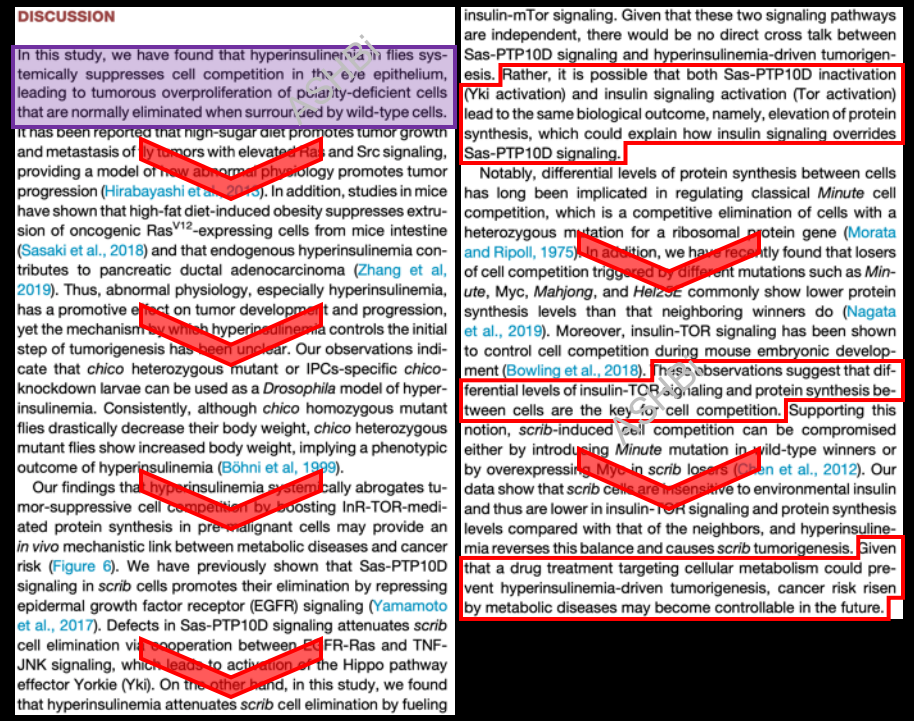

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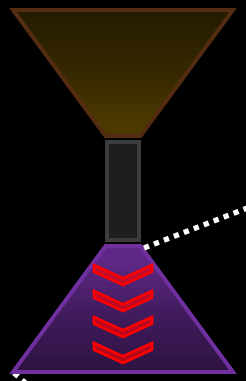
Our findings that hyperinsulinemia systemically abrogates tumor-suppressive cell competition by boosting InR-TOR-mediated protein synthesis in pre-malignant cells may provide an *in vivo* mechanistic link between metabolic diseases and cancer risk (Figure 6). We have previously shown that Sas-PTP10D signaling in *scrib* cells promotes their elimination by repressing epidermal growth factor receptor (EGFR) signaling (Yamamoto et al., 2017). Defects in Sas-PTP10D signaling attenuates *scrib* cell elimination via cooperation between EGFR-Ras and TNF-JNK signaling, which leads to activation of the Hippo pathway effector Yorkie (Yki). On the other hand, in this study, we found that hyperinsulinemia attenuates *scrib* cell elimination by fueling insulin-mTor signaling. Given that these two signaling pathways are independent, there would be no direct cross talk between Sas-PTP10D signaling and hyperinsulinemia-driven tumorigenesis. Rather, it is possible that both Sas-PTP10D inactivation (Yki activation) and insulin signaling activation (Tor activation) lead to the same biological outcome, namely, elevation of protein synthesis, which could explain how insulin signaling overrides Sas-PTP10D signaling.

Notably, differential levels of protein synthesis between cells has long been implicated in regulating classical *Minute* cell competition, which is a competitive elimination of cells with a heterozygous mutation for a ribosomal protein gene (Morata and Ripoll, 1975). In addition, we have recently found that losers of cell competition triggered by different mutations such as *Minute*, Myc, *Mahjong*, and *Hel25E* commonly show lower protein synthesis levels than that neighboring winners do (Nagata et al., 2019). Moreover, insulin-TOR signaling has been shown to control cell competition during mouse embryonic development (Bowling et al., 2018). These observations suggest that differential levels of insulin-TOR signaling and protein synthesis between cells are the key for cell competition. Supporting this notion, *scrib*-induced cell competition can be compromised either by introducing *Minute* mutation in wild-type winners or by overexpressing Myc in *scrib* losers (Claveria et al., 2012). Our data show that *scrib* cells are insensitive to environmental insulin and thus are lower in insulin-TOR signaling and protein synthesis levels compared with that of the neighbors, and hyperinsulinemia reverses this balance and causes *scrib* tumorigenesis. Given that a drug treatment targeting cellular metabolism could prevent hyperinsulinemia-driven tumorigenesis, cancer risk risen by metabolic diseases may become controllable in the future.

PAPER STRUCTURE



Answering the Central Question

STRONG Evidence ***FACT***

Supporting Evidence from Literature

Linking Back to Own Results

WEAK Evidence ***SPECULATION***

Broader Implications

❖ ***Answer your Question First & Describe Later the Implications of your Study***

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Good Storytelling Steers the Emotions of a Reader

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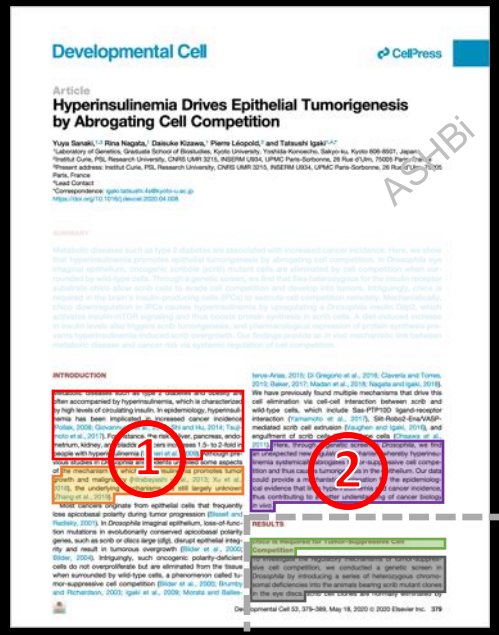
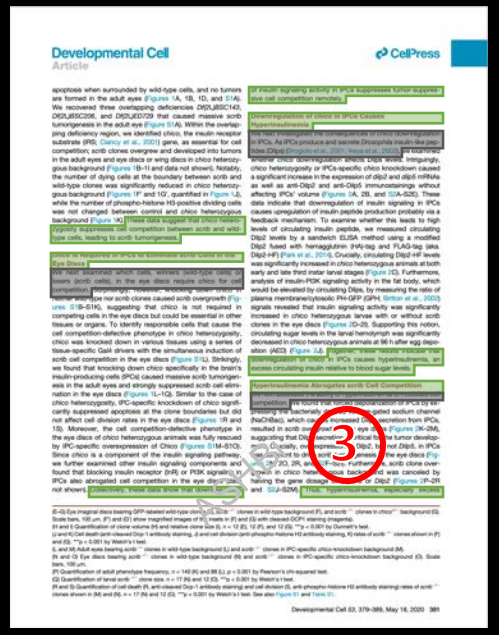
Sanaki et al., 2020. Dev Cell

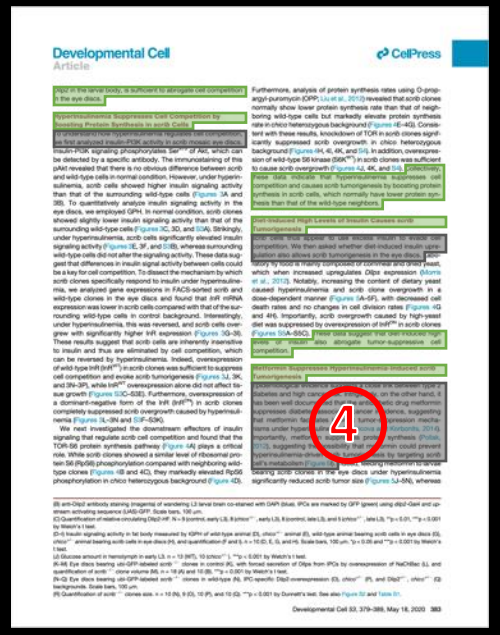
INTRODUCTION

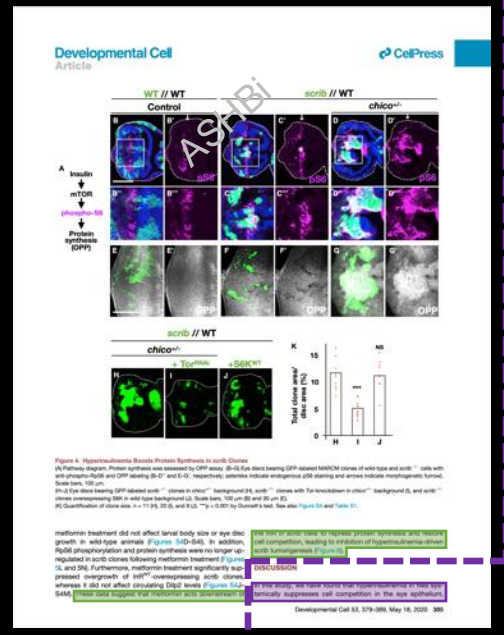
RESULTS

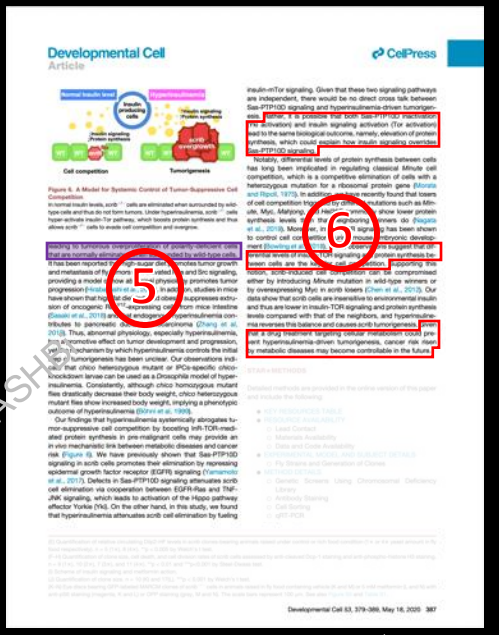
DISCUSSION











1. Engage Audience

2. Heighten Anticipation

3. Building Up to the CLIMAX

4. CLIMAX

5. Satisfaction

6. Deeper Appreciation

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    a. *The Basic Structure of a Scientific Story*
    b. *Example of a Paper with Good Storytelling*
        i. *Introduction*
        ii. *Results*
        iii. *Discussion/Conclusion*
        iv. *Impact of Good Storytelling to the Reader/Editor*
4) *Practical Advice on Preparing your Paper*

Abstract: NOT a Simple Summary but Rather a Small Story

https://www.nature.com/documents/nature-summary-paragraph.pdf

One or two sentences providing a **basic introduction** to the field, comprehensible to a scientist in any discipline.

Two to three sentences of **more detailed background**, comprehensible to scientists in related disciplines.

One sentence clearly stating the **general problem** being addressed by this particular study.

One sentence summarizing the main result (with the words "**here we show**" or their equivalent).

Two or three sentences explaining what the **main result** reveals in direct comparison to what was thought to be the case previously, or how the main result adds to previous knowledge.

One or two sentences to put the results into a more **general context**.

Two or three sentences to provide a **broader perspective**, readily comprehensible to a scientist in any discipline, may be included in the first paragraph if the editor considers that the accessibility of the paper is significantly enhanced by their inclusion. Under these circumstances, the length of the paragraph can be up to 300 words. (This example is 190 words without the final section, and 250 words with it).

During cell division, mitotic spindles are assembled by microtubule-based motor proteins[1,2]. T…on of spindles is essential for proper segreg…es, and requires plus-end-directed homotetrameric motor proteins of the widely conserved kinesin-5 (BimC) family[3]. Hypo…s for bipolar spindle formation include the 'push–pull mitotic muscle' model, in which kinesin-5 and opposing motor proteins a…ng microtubules[2,4,5]. However, the precise roles o…g this process are unknown. Here we show tha… kinesin-5 Eg5 drives the sliding of microtubules depe…g on their relative orientation. We found in controlled *in vitro* assays that Eg5 has the remarkable capability of simultaneously moving at ~20 nm s^{-1} towards the plus-ends of each of the two microtubules it crosslinks. For anti-parallel microtubules, this results in relative sliding at ~40 nm s^{-1}, comparable to spindle pole separation rates *in vivo*[6]. Furthermore, we found that Eg5 can tether microtubule plus-ends, suggesting an additional microtubule-binding mode for Eg5. Our results demonstrate how members of the kinesin-5 family are likely to function in mitosis, pushing apart interp…les as well as recruiting microtubules into bundles t…ntly polarized by relative sliding. We anticipate our assay to be a starting point for more sophisticated *in vitro* models of mitotic spindles. For example, the individual and combined action of multiple mitotic motors could be tested, including minus-end-directed motors opposing Eg5 motility. Furthermore, Eg5 inhibiti…f anti-cancer drug development, and a well-c…ive assay for motor function will be relevant for such developments.

BROAD

SPECIFIC

SPECIFIC

BROAD

❖ ***This is a Versatile Structure for Writing a Solid Abstract***

Abstracts: Current Trends in Abstract Structure

DOI:https://doi.org/10.1016/j.devcel.2020.04.008
Sanaki et al., 2020. Dev Cell

SUMMARY

Metabolic diseases such as type 2 diabetes are associated with increased cancer incidence. Here, we show that hyperinsulinemia promotes epithelial tumorigenesis by abrogating cell competition. In *Drosophila* eye imaginal epithelium, oncogenic *scribble* (*scrib*) mutant cells are eliminated by cell competition when surrounded by wild-type cells. Through a genetic screen, we find that flies heterozygous for the insulin receptor substrate *chico* allow *scrib* cells to evade cell competition and develop into tumors. Intriguingly, *chico* is required in the brain's insulin-producing cells (IPCs) to execute cell competition remotely. Mechanistically, *chico* downregulation in IPCs causes hyperinsulinemia by upregulating a *Drosophila* insulin Dilp2, which activates insulin-mTOR signaling and thus boosts protein synthesis in *scrib* cells. A diet-induced increase in insulin levels also triggers *scrib* tumorigenesis, and pharmacological repression of protein synthesis prevents hyperinsulinemia-induced *scrib* overgrowth. Our findings provide an *in vivo* mechanistic link between metabolic disease and cancer risk via systemic regulation of cell competition.

Engage Audience

A Situation of Broad Interest

The Answer

Background/Context

Summary of Results

Broader Implications

➢ *Current Trend uses **Active** rather than **Passive Voice***

❖ ***'Hook' the Editor in Immmediately to Grab their Interest for your Paper***

My Advice: How to Effectively Tell your Story

❖ *As Papers are written Retrospectively, it is **NOT NECESSARY** to write the Story based on the Original Logic of the Study*

1) Identifying **WHAT** is the Most Interesting/Unexpected Finding in your Paper
2) Identify **HOW** this Finding can have the **BROADEST** Impact to the Field or Society
3) Build your Story Around these Points
4) Retrospectively Establish your **'Unique'** Central Question
5) Fill in the Necessary Gaps/Information to be able to **Understand Sufficiently** the Story

➢ *One Suggestion is to First Write a Short Version of your Paper (eg. 1xA4 as in Science) before Writing your Actual Paper*

My Advice: Never Write According to the Layout of a Paper

❖ **BEFORE WRITING**: ***Always*** *Start by Assembling Figures*

Classic Layout of a Paper

The IMRaD Structure

1) (the **TITLE** and **ABSTRACT**)
2) the **INTRODUCTION**
3) the **METHODS**
4) the **RESULTS** and
5) the **DISCUSSION**

Write your Paper from:

1) the **RESULTS**
2) the **DISCUSSION/CONCLUSION**
3) the **INTRODUCTION**
4) the **ABSTRACT**
5) the **METHODS**
6) **the TITLE**

My Advice: Writing a Successful Scientific Story – The 3's

- ***Simple*** - a simple message is effective a memorable one (not to be confused with simplification)
- ***Solid*** - results should be concrete/reproducible (ie. be convincing)
- ***Surprising*** - surprising/unexpected findings make a more engaging story

❖ ***The 3S's will Help Construct a More Clear, Captivating and Memorable Story***

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Thank you for your Attention!

Other Affiliations

Upcoming Research Acceleration Program Event

“Visualizing your Research!
– Tips for creating the graphical abstract”

Date	Tuesday July 15th 2025
Time	16:00-17:30
Venue	Zoom
Language	Japanese (also accepting English Questions)
Eligibility	Researchers & Students

Register Here