ASHBi

RESEARCH ACCELERATION PROGRAM

An Ex-Cell Press Editor presents:

Telling your Story in a Scientific Paper

SPYROS GOULAS PhD

Scientific Advisor

Research Acceleration Unit

WPI-ASHBi (Kyoto University)

Who am I?

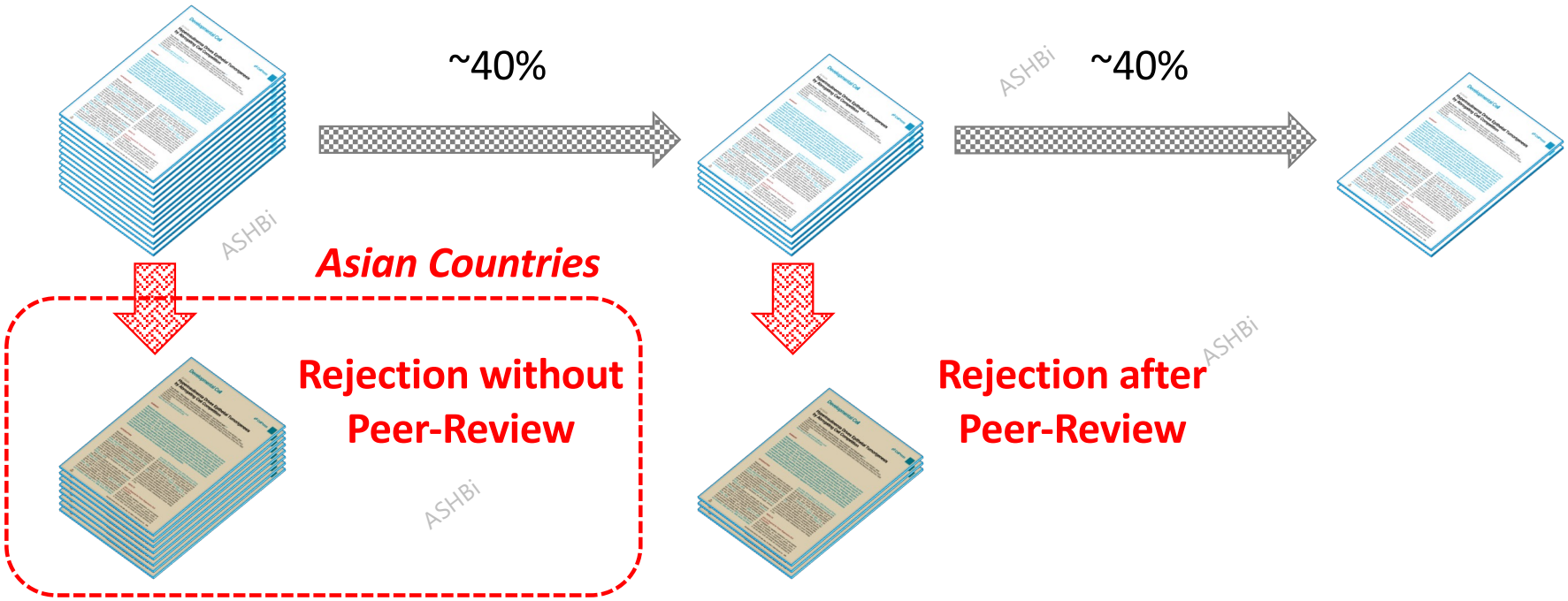
Spyros Goulas, PhD

- 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 Prof.*** (lab of **Prof. Shigeo Ohno**)
  Yokohama City University
- 2018-2022, ***Associate Scientific Editor*** (handled **~1600 papers**) at ***Developmental Cell***
  (IF=**12.27**; **Cell Press/Elsevier**)
- 2022- , ***Scientific Advisor***
  Institute of Advanced Studies for Human Biology (**ASHBi**), Kyoto University

Papers from Asia have a High Rate of Rejection Without Peer-Review

Developmental Cell

~1300 Paper Submissions/Year **Out To Peer-Review (OTR)** **Accepted (16.47%)**



⚠ ***From my Editorial Experience: Papers from Asia Often do not Develop Their Stories***

Aim of Today: How to Effectively Tell your Story in a Scientific Paper

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*

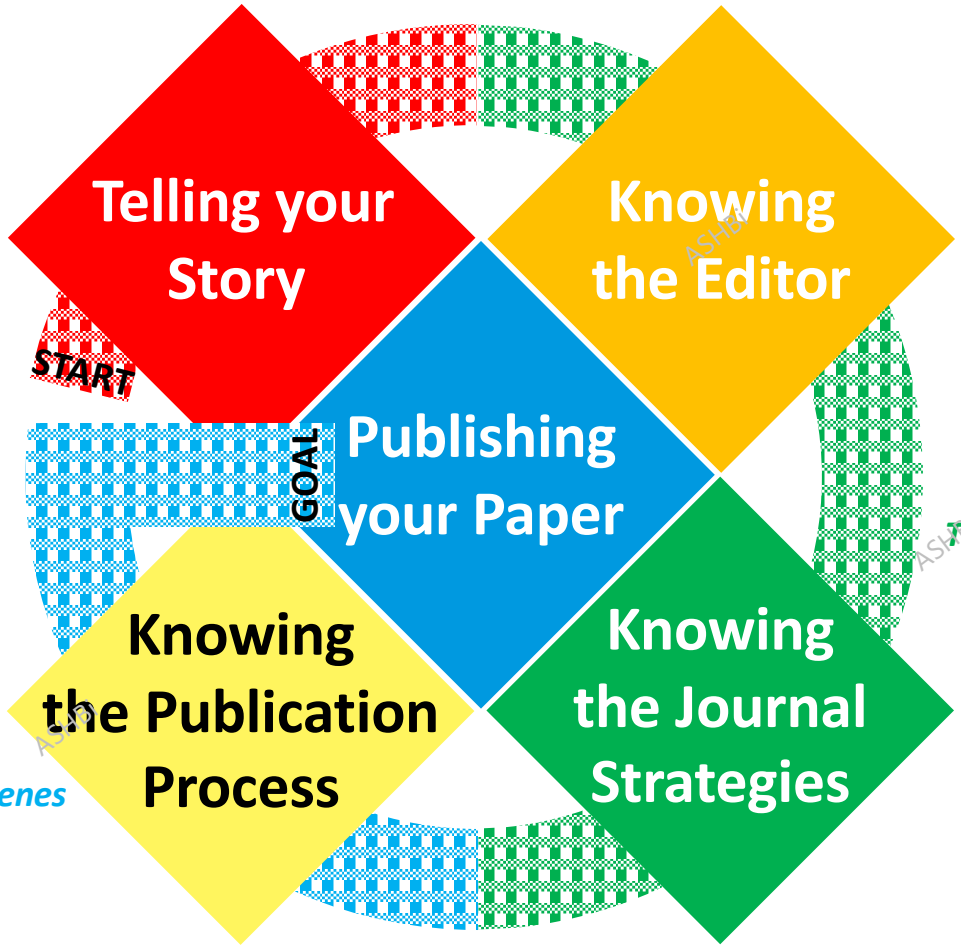
Ultimate Goal

To Provide you with the Necessary Toolbox to Publish your Work Efficiently

3-Part Seminar Series: Roadmap to Publishing Papers

Today's Seminar

PART I



PART II

The Editor – A Friend or Foe?

PART III

~What Happens Behind the Scenes of Publishing~

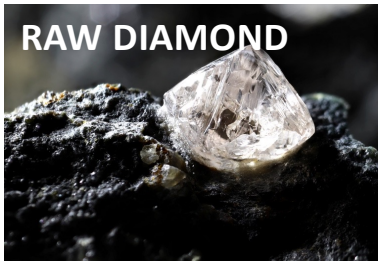
Today's Agenda

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

Today's Agenda

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

Scientific Storytelling is like *DIAMOND Processing*



PROCESSING

eg. cutting, polishing

RAW DATA → STORYTELLING → **SCIENCE PAPER**

Storytelling is NOT Magic, it can ONLY be as Good as your Actual Data

A Good Story is a Path to New Understanding

Interpretation – To find the Meaning Behind the Raw Data

Knowledge – Facts generated from data points

Understanding – Assimilation of various pieces of knowledge to acquire a deeper insight into processes and how they function

⚠ ***It is YOUR job to Explain the Deeper Insight Obtained from YOUR Data, it is NOT the Job of the Reader/Editor to EXTRACT This!!***

1) *What is Scientific Storytelling?*

2) ***Why is Telling your Story Important?***

3) *How to Tell your Story Effectively*
~The Basic Structure of a Scientific Story~

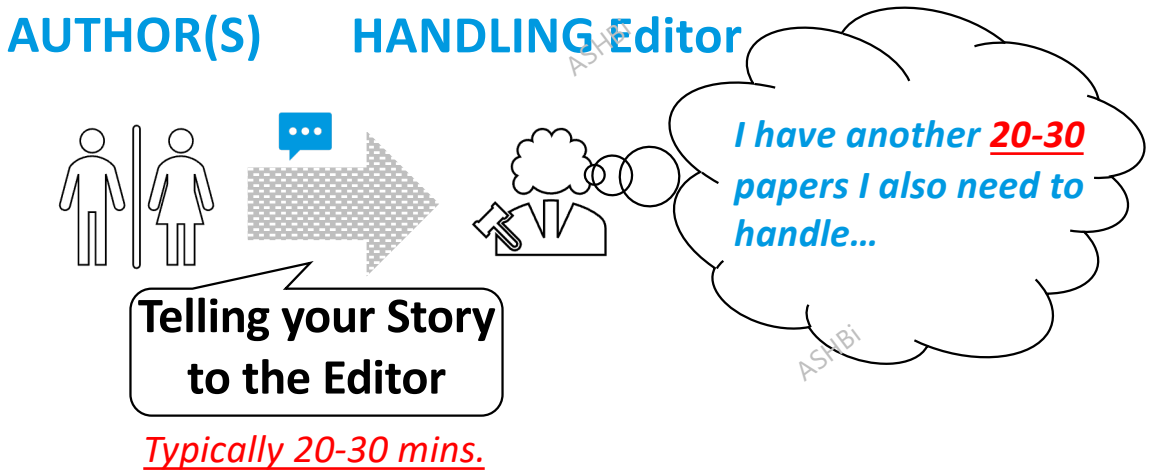
4) *Example of a Paper with Good Storytelling*

5) *Advice on How to Construct your Story Effectively*

ASHBi
WPI Kyoto University

…because you only get 20-30mins to Tell your Story to an Editor

…because you only get 20-30mins to Tell your Story to an Editor

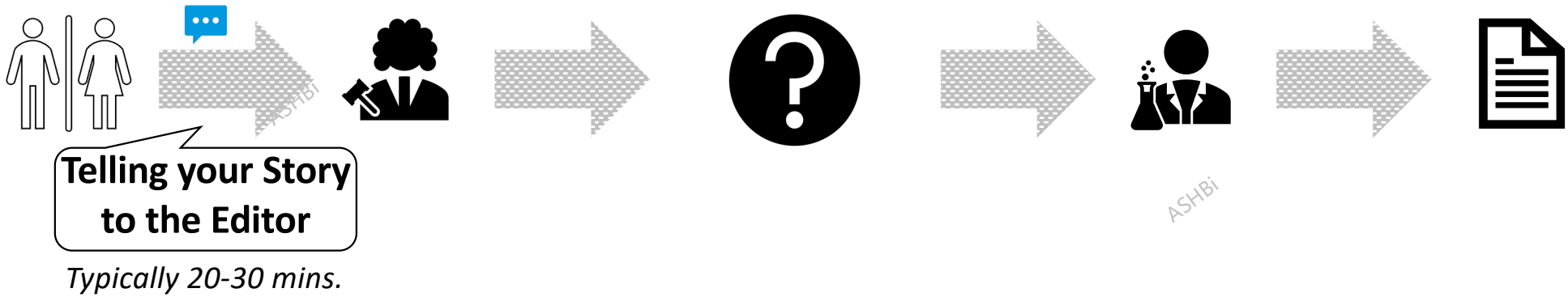


❖ *So Make Sure to Tell your Story Effectively to Convince the Editor your Paper is Interesting!!*

…because Your Storytelling Impacts How Editors Tell your Story to Their Team

What <u>ACTUALLY</u> Happens with your Paper…

AUTHOR(S) **STEP 1: HANDLING Editor** **Peer Review** **Publishing Paper**



Typically 20-30 mins.

…because Your Storytelling Impacts How Editors Tell your Story to Their Team

What <u>ACTUALLY</u> Happens with your Paper…

AUTHOR(S) | **STEP 1: HANDLING Editor** | **STEP 2: Editorial Team** | **Peer Review** | **Publishing Paper**

Telling your Story to the Editor

Typically 20-30 mins.

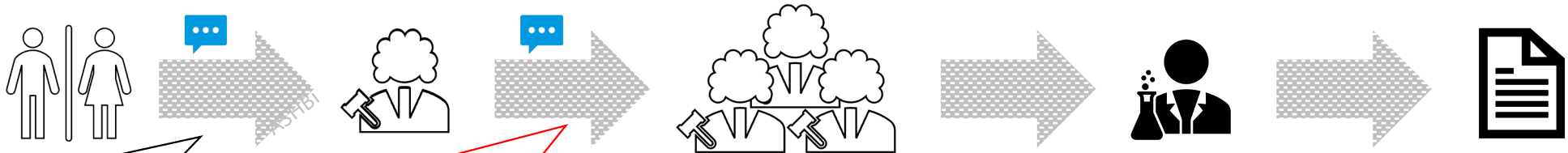
Handling Editor Conveying your Story to Their Team

<u>Typically 5-10 mins.</u>

…because Your Storytelling Impacts How Editors Tell your Story to Their Team

What <u>ACTUALLY</u> Happens with your Paper…

AUTHOR(S) | **STEP 1: HANDLING Editor** | **STEP 2: Editorial Team** | **Peer Review** | **Publishing Paper**



Telling your Story to the Editor

Typically 20-30 mins.

Handling Editor Conveying your Story to Their Team

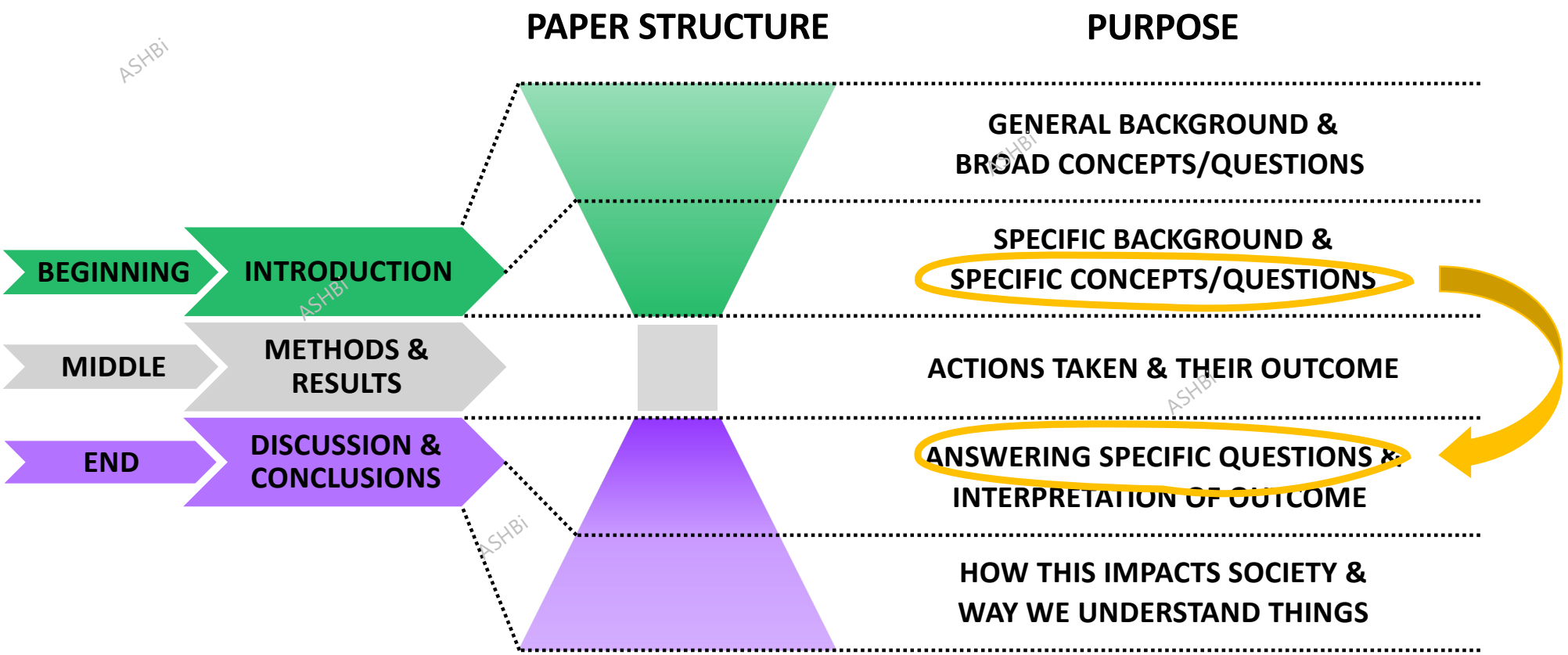
<u>Typically 5-10 mins.</u>

❖ ***Your Storytelling is also Important so that the Editor can Convince their Team it is Interesting***

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

ASHBi
WPI Kyoto University

Using the Hour-Glass Structure to Answer the Question in a Scientific Paper



1) *What is Scientific Storytelling?*
2) *Why is Telling your Story Important*
3) *How to Tell your Story Effectively*
   ~The Basic Structure of a Scientific Story~
4) ***Example of a Paper with Good Storytelling***
   - *I. INTRODUCTION*
   - *II. RESULTS*
   - *III. DISCUSSION*
   - *IV. Impact of Good Storytelling on a Reader/Editor*
5) *Advice on How to Construct your Story Effectively*

ASHBi
WPI Kyoto University

Example of a Scientific Paper with Good Storytelling

Developmental Cell

CellPress

DOI:https://doi.org/10.1016/j.devcel.2020.04.008

Sanaki et al., 2020. Dev Cell

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,*]

[1]Laboratory of Genetics, Graduate School of Biostudies, Kyoto University, Yoshida-Konoecho, Sakyo-ku, Kyoto 606-8501, Japan

[2]Institut Curie, PSL Research University, CNRS UMR 3215, INSERM U934, UPMC Paris-Sorbonne, 26 Rue d'Ulm, 75005 Paris, France

[3]Present address: Institut Curie, PSL Research University, CNRS UMR 3215, INSERM U934, UPMC Paris-Sorbonne, 26 Rue d'Ulm, 75005 Paris, France

[4]Lead Contact

*Correspondence: igaki.tatsushi.4s@kyoto-u.ac.jp

https://doi.org/10.1016/j.devcel.2020.04.008

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 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

The ANSWER

Hyperinsulinemia Promotes Tumor Growth by Avoiding Cell Competition

1) *What is Scientific Storytelling?*
2) *Why is Telling your Story Important*
3) *How to Tell your Story Effectively*
   ~The Basic Structure of a Scientific Story~
4) ***Example of a Paper with Good Storytelling***
   - ***I. INTRODUCTION***
   - ***II. RESULTS***
   - ***III. DISCUSSION***
   - *IV. Impact of Good Storytelling on a Reader/Editor*
5) *Advice on How to Construct your Story Effectively*

ASHBi
WPI Kyoto University

INTRODUCTION: Find a Context of Broad Relevance and Properly Set Up Your Central Question to Make a Story More Engaging

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

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).

PAPER STRUCTURE

Introducing Context/Problem of Broad Interest

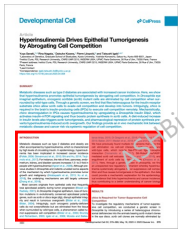
- *Metabolic diseases*
- *Cancers*
- *Links between Metabolism and Cancer*

Advantages of System being Used

The Central QUESTION

- *How does hyperinsulinemia promote tumor growth?*

INTRODUCTION: End it on a High NOTE by Heightening Anticipation and Curiosity

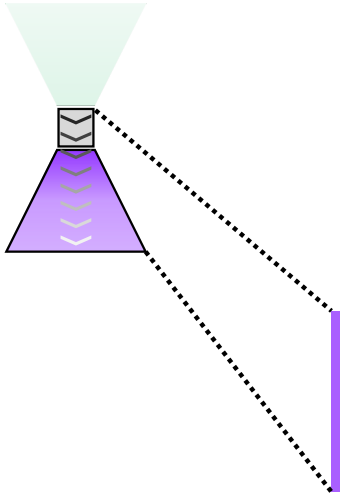

Developmental Cell

Hyperinsulinemia Drives Epithelial Tumorigenesis by Abrogating Cell Competition

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

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*.

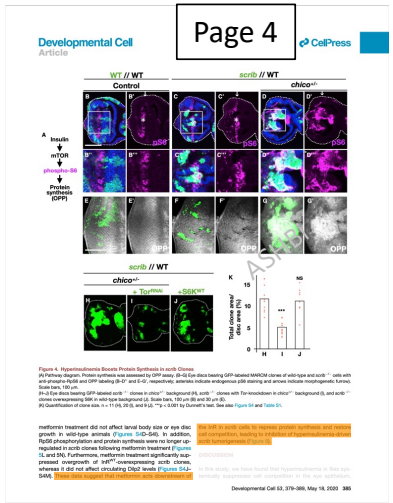
PAPER STRUCTURE



Brief Description of Results and Implications

- *Heightens anticipation and curiosity of results and their broader potential implications*

RESULTS: Use Active Subheadings and Linking Subsections to Make it More Reader- (and Editor-) Friendly



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

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: A Logical Data Sequence that Answers the Central Question

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

DISCUSSION **The ANSWER -** *Hyperinsulinemia promotes tumor growth by avoiding cell competition*

RESULTS: A Logical Data Sequence that Answers the Central Question

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

RESULTS

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

↓

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

↓

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*)

↓

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 avoiding cell competition*

RESULTS: A Logical Data Sequence that Answers the Central 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?

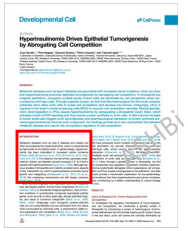
3. Downregulation of *chico* in IPCs Causes Hyperinsulinemia (*Fig. 2*)
4. Hyperinsulinemia Abrogates *scrib* Cell Competition (*Fig. 2*)
5. Hyperinsulinemia Suppresses Cell Competition by Boosting Protein Synthesis in *scrib* Cell (*Fig. 3&4*)

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 avoiding cell competition*

DISCUSSION: Don't Re-Summarize your Results, ANSWER the QUESTION!!



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

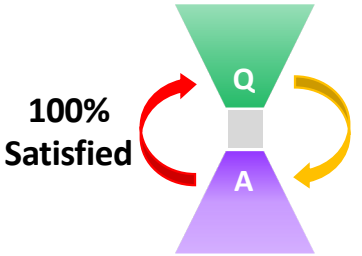
INTRODUCTION

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

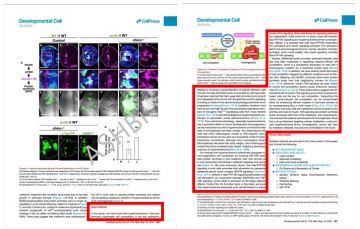


Q: How does hyperinsulinemia promote tumor growth?

A: Hyperinsulinemia promotes tumor growth by avoiding cell competition

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

DISCUSSION: Going from Fact to Speculation



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 flies 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, has a promotive effect on tumor development and progression, yet the mechanism by which hyperinsulinemia controls the initial step of tumorigenesis has been unclear. Our observations indicate that *chico* heterozygous mutant or IPCs-specific *chico*-knockdown larvae can be used as a *Drosophila* model of hyperinsulinemia. Consistently, although *chico* homozygous mutant flies drastically decrease their body weight, *chico* heterozygous mutant flies show increased body weight, implying a phenotypic outcome of hyperinsulinemia (Böhni et al., 1999).

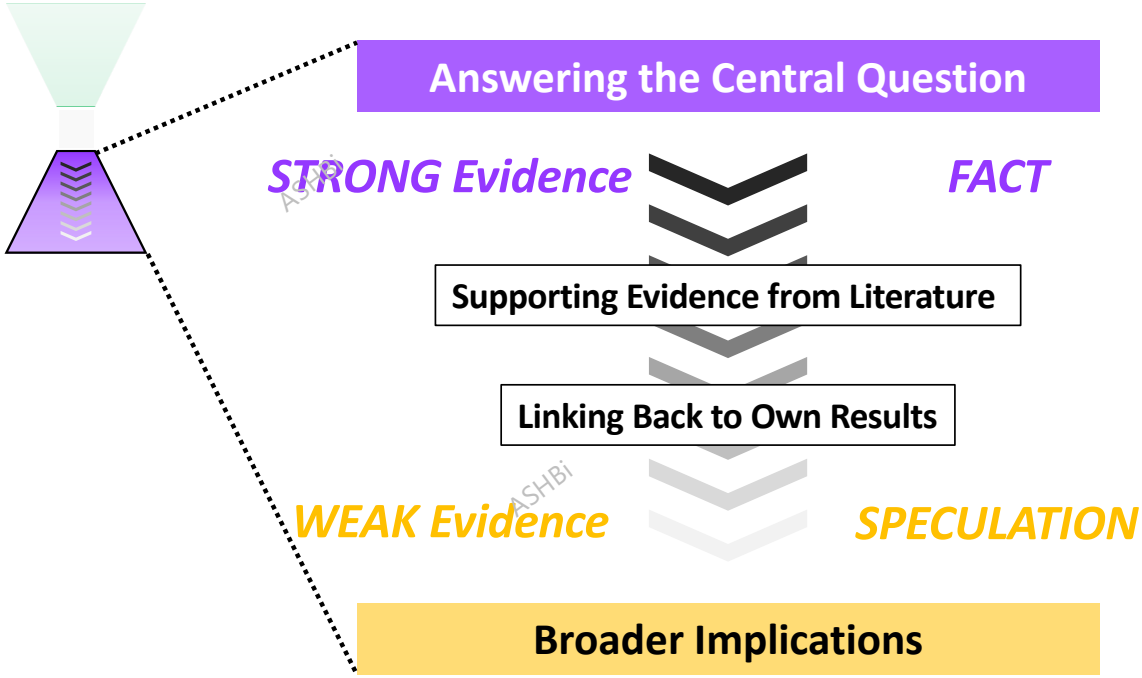
Our findings that hyperinsulinemia systemically abrogates tumor-suppressive cell competition by boosting InR-TOR-mediated protein synthesis in pre-malignant cells may provide an *in vivo* mechanistic link between metabolic diseases and cancer risk (Figure 6). We have previously shown that Sas-PTP10D 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 activation 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 could be a 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 26S proteasome commonly show lower protein synthesis levels than that of 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 (Chen 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 those of the neighbors, and hyperinsulinemia reverses this balance to promote *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

DOI:https://doi.org/10.1016/j.devcel.2020.04.008

Sanaki et al., 2020. Dev Cell



Answer your Question First and Then Finish off with the Broader Implications of your Study

1) *What is Scientific Storytelling?*
2) *Why is Telling your Story Important*
3) *How to Tell your Story Effectively*
   ~The Basic Structure of a Scientific Story~
4) ***Example of a Paper with Good Storytelling***
   - *I. INTRODUCTION*
   - *II. RESULTS*
   - *III. DISCUSSION*
   - ***IV. Impact of Good Storytelling on a Reader/Editor***
5) *Advice on How to Construct your Story Effectively*

ASHBi
WPI Kyoto University

Good Storytelling Steers the Emotions of a Reader/Editor

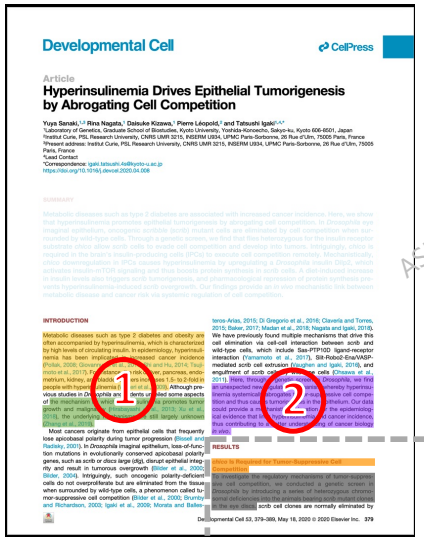
DOI:https://doi.org/10.1016/j.devcel.2020.04.008

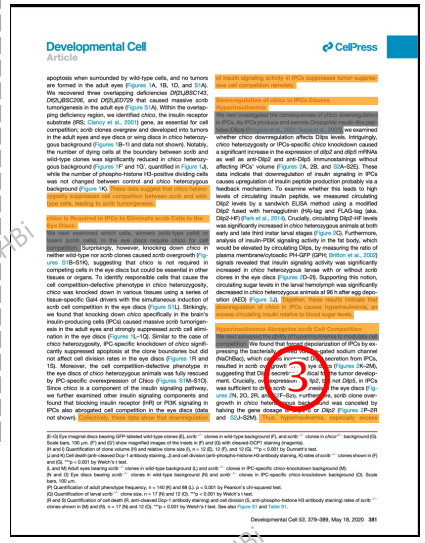
Sanaki et al., 2020. Dev Cell

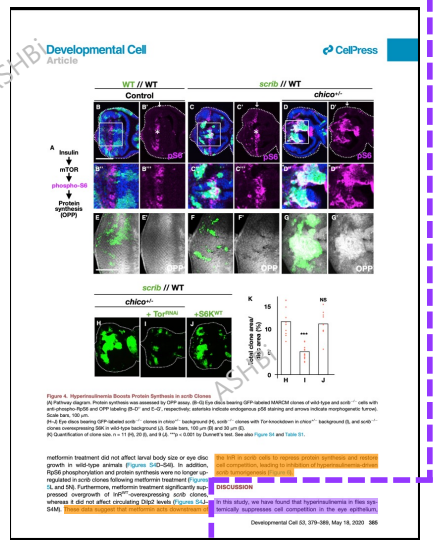
INTRODUCTION

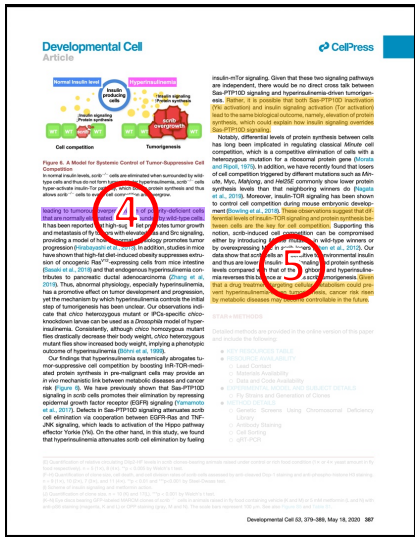
RESULTS

DISCUSSION









1. Engage Audience

2. Heighten Anticipation

3. Building Up to the CLIMAX

4. Satisfaction

5. Deeper Appreciation

1) *What is Scientific Storytelling?*
2) *Why is Telling your Story Important*
3) *How to Tell your Story Effectively*
   ~The Basic Structure of a Scientific Story~
4) *Example of a Paper with Good Storytelling*
   - *I. INTRODUCTION*
   - *II. RESULTS*
   - *III. DISCUSSION*
   - *IV. Impact of Good Storytelling on a Reader/Editor*
5) ***Advice on How to Construct your Story Effectively***

ASHBi
WPI Kyoto University

Advice on Abstracts: It is a Mini-Story so Leave a Strong First Impression


Developmental Cell

Hyperinsulinemia Drives Epithelial Tumorigenesis by Abrogating Cell Competition

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

Engage Audience

General Point of Broad Interest

The Answer

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.

Background/Context

Summary of Results

Broader Implications

❖ ***Make a Strong Impact Immediately to Grab the Attention of an Editor***

The 3S's to a Successful Scientific Story

- ***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

❖ ***Keeping the 3S's in Mind while Writing your Paper will Help you Construct a Story that is More Clear, Captivating and Memorable***

MY ADVICE on How to Effectively Tell your Story

- As Papers are written Retrospective, 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 **<u>BROADEST</u>** 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

How to Identify the Most Interesting Finding of your Paper?

Strategy for Identifying the Key Point(s) of your Paper

Strategy I

- **First** Write the Highlights of your Paper by Identifying **No More than 4 Key Points** (eg. as in Cell Press papers) of your Work and Pick the MOST IMPORTANT One to Build your Story Around

Strategy II

- **First** Write a Short Version of your Paper (eg. 1xA4 as in *Science*)

- **Reminder: It's ALWAYS Good ASK a Friend/Colleague for advice and WHAT they find is Interesting about your Work!!**

Acknowledgements

ASHBi Research Acceleration Unit

Tadashi Ogawa

Makoto Shida

Hiromi Inoue

Tomoki Shimizu

LiMe

Hiromi Sumita

ASHBi Administrative Office

Fumi Komori

Narumi Sano

Special Thanks to:

Tatsushi Igaki

Other Affiliations

Other Personal Affiliations

Upcoming Seminars from ASHBi Research Acceleration Unit

August 5th 2022

16:00-17:20

Register Here:

https://forms.gle/GxHT3XHWN4aJV4Df9