論文査読の問題点と新しい試み

東京大学　大学院医学系研究科・医学部

水島昇

COI Disclosure Information

Noboru Mizushima

eLife	Board of Reviewing Editor
Molecular Cell	Advisory Board
EMBO Reports	Advisory Board
Genes to Cells	Associate Editor
Cell Struct. Funct.	Associate Editor

専門分野
細胞生物学
生化学、分子生物学
基礎医学

査読の実際

現在の査読システムの問題点

査読の実際

現在の査読システムの問題点

査読ですべきこと

- 方法の正当性
- 結論の正当性
- 完成度
- 再現性
- 新規性
- 重要性
- 普遍性
- 倫理性

データの強さ

研究のインパクト

査読ですべきではないこと
しなくてもよいこと

- 興味本位の質問
- 結論には不要な実験の追加
- さらによくするためのおせっかい
- 採択の是非
- 自分の論文の引用の推奨
- スペルや文法の誤り

- 具体的な実験の提案？

How to Write a Peer Review（PLOS Peer Review Center）より

論文をどのように修正すべきかを正確に著者に伝えるべきではない。
彼らのために仕事をする必要はない。

Don't

- Recommend additional experiments or unnecessary elements that are out of scope for the study or for the journal criteria.
- Tell the authors exactly how to revise their manuscript—you don't need to do their work for them.
- Use the review to promote your own research or hypotheses.
- Focus on typos and grammar. If the manuscript needs significant editing for language and writing quality, just mention this in your comments.
- Submit your review without proofreading it and checking everything one more time.

https://plos.org/resource/how-to-write-a-peer-review/

×
データAから結論Bはサポートされない。

△
データAから結論Bはサポートされない。
そのために実験Cを行うべきである。

○
データAから結論Bはサポートされない。
なぜなら、結論Dもありうるからである。

結論をサポートするのにどうしても足りないこと以外は指摘しない。あるいは提案しても必須でない場合はそのことを明記する。

具体的な実験を提案する場合は、目的を明示して、実験の例として提案する。

査読の実際

現在の査読システムの問題点

現在の査読システムの問題点

著者

査読者

エディター

読者

ILLUSTRATION: DAVID PARKINS

HOW TO FIX PEER REVIEW

Journals and funders are trying to boost the effectiveness of systems under strain. **By David Adam**

Attached to the Very Large Telescope in Chile, the Multi Unit Spectroscopic Explorer (MUSE) allows researchers to probe the most distant galaxies. It's a popular instrument: for its next observing session, from October to April, scientists have applied for more than 3,000 hours of observation time. That's a problem. Even though it's dubbed a cosmic time machine, not even MUSE can squeeze 379 nights of work into just seven months.

The European Southern Observatory (ESO), which runs the Chile telescope, usually asks panels of experts to select the worthiest proposals. But as the number of requests has soared, so has the burden on the scientists asked to grade them.

"The load was simply unbearable," says

査読の現状

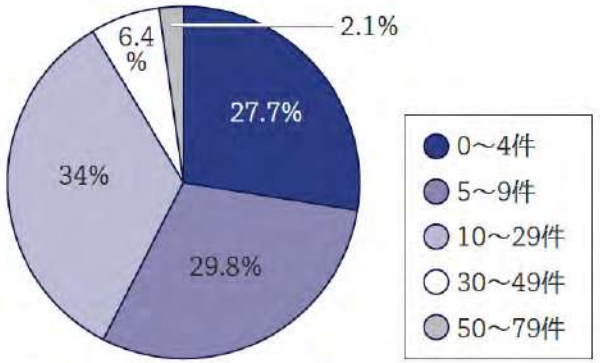


図1　査読者として1年間に行う論文査読件数（リビジョンも1つとカウント）

実験医学アンケート（2019年10月実施）より．回答者は査読経験者47名（以降同）．

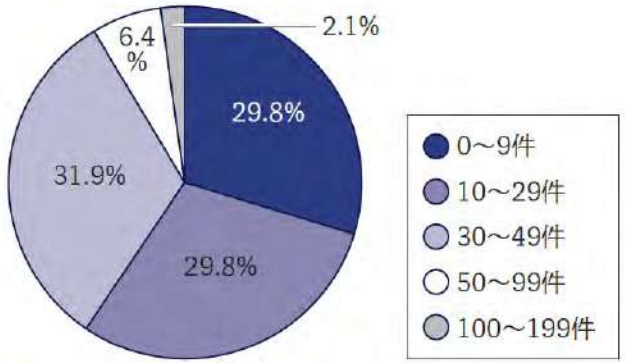


図2　1年間に受け取る査読依頼の件数

実験医学アンケートより．

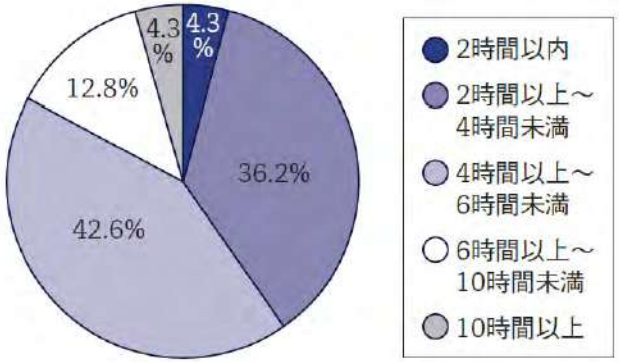


図3　1件の論文査読にかける平均時間

実験医学アンケートより．

水島 昇『科学を育む　査読の技法』羊土社、2021年、16頁より引用

査読の現状

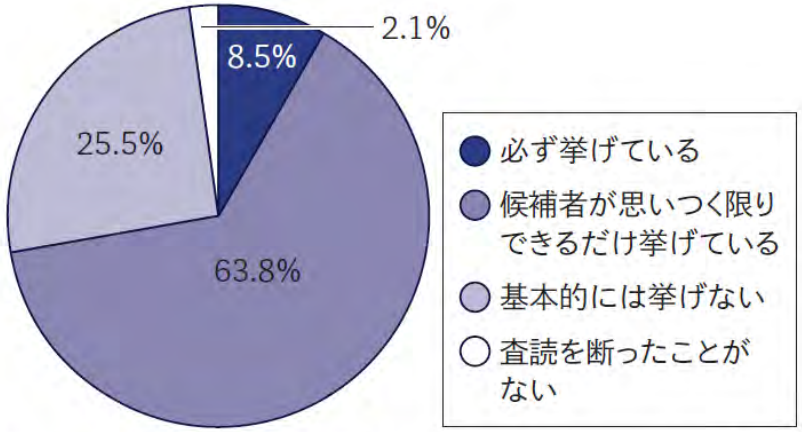


図4　査読を断る時に他の査読候補者名をあげるかどうか

実験医学アンケートより.

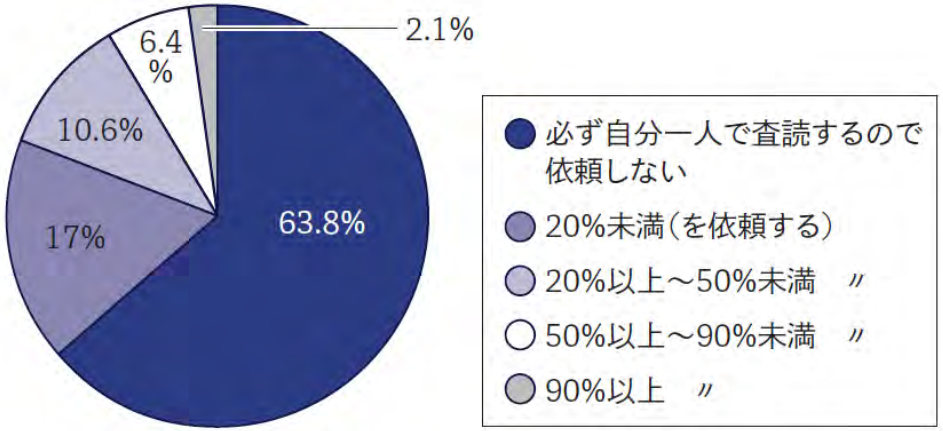
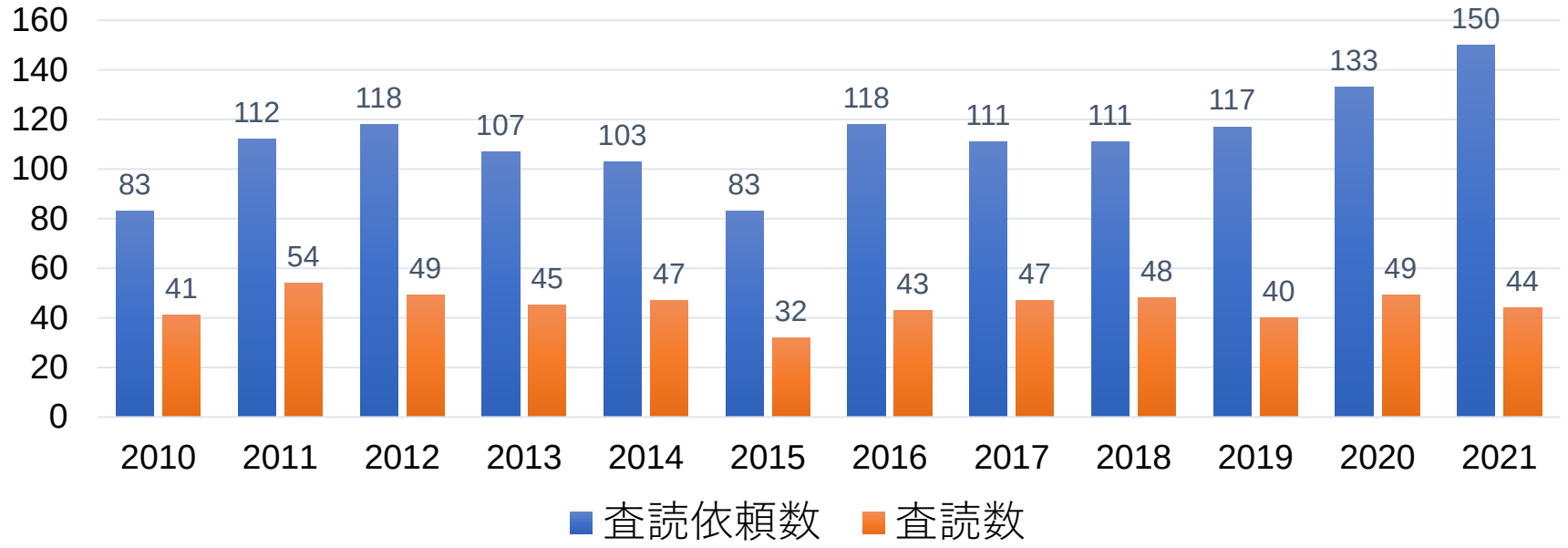


図2　研究室内のメンバーへの査読協力の依頼状況

実験医学アンケートより.

水島 昇『科学を育む　査読の技法』羊土社、2021年、19、37頁より引用

水島の査読実績


160
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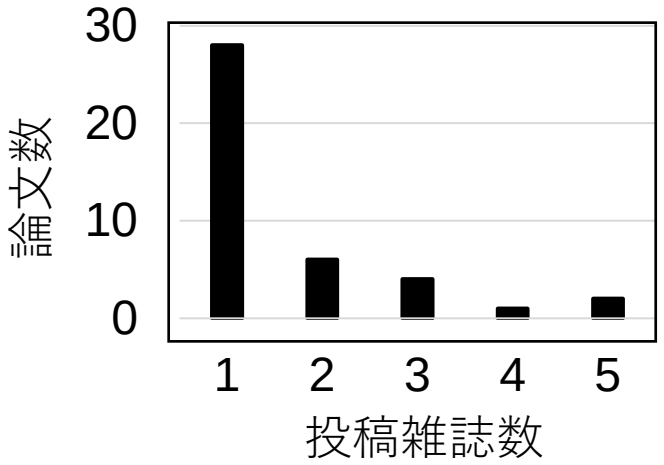
83
32
118
43
111
47
111
48
117
40
133
49
150
44
2010
2011
2012
2013
2014
2015
2016
2017
2018
2019
2020
2021
査読依頼数
査読数

1年で何件査読すべきか

- 査読誌に自分が責任著者として1年間に発表する論文の数をA
- その論文が最終的な雑誌にアクセプトされるまでに経由した雑誌の総数の平均値をB
- ひとつの雑誌あたりの平均査読者数をC
- その責任著者が1年間に消費している査読者数X
- X＝A×B×C
- 仮に、A＝3、B＝2、C＝3とすると、X＝18となる。
- これが査読すべき数



B値（平均）=1.61（査読された場合1.29）
（7割の論文が初回投稿誌に掲載）
（2004年～2018年）

現在の査読システムの問題点

- 査読者が研究に介入しすぎている
  - 査読者の役割の明確化（正しさと重要性の評価）
  - 研究指導者との混同（よけいなおせっかい）
  - 過剰な要求（Major Revisionは必要か？）

著者がもっとコントロール

- 少数の査読者がデータの公開を阻止している
  - 公的資金で行った研究の成果がなかなか公開されない

プレプリントサーバーの活用

- 少数の査読者による論文の格付け
  - 結局はインパクトファクターの問題か？

コミュニティーが長く評価を

- 査読の負担、査読者の枯渇

査読コメントの共有
Review Commonsなど

- 査読コメントの無駄死に

査読コメントも公開（アクセプトされたものだけ）

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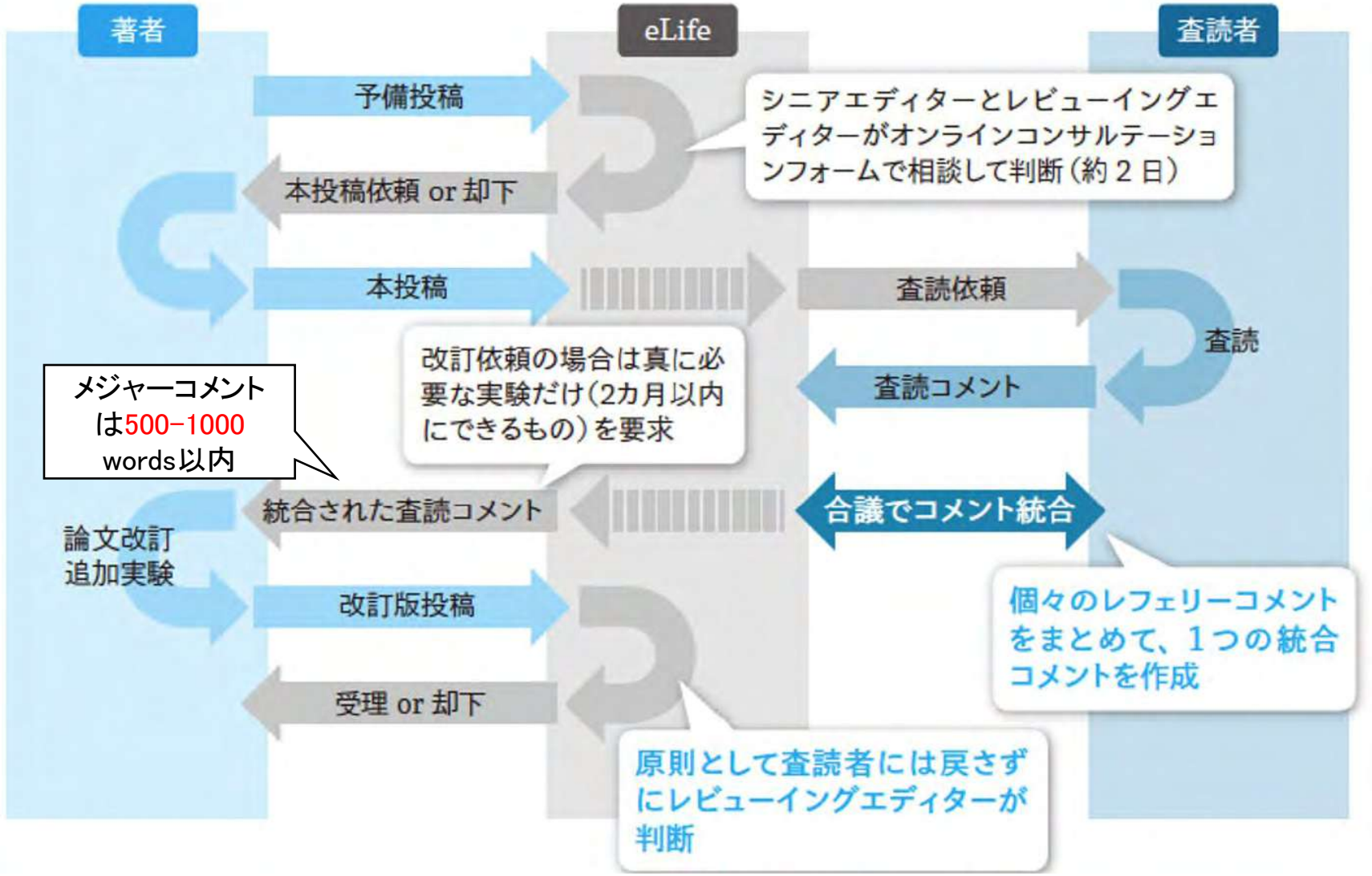
eLifeの当初の査読プロセス



水島 昇『科学を育む　査読の技法』羊土社、2021年、47頁より引用

Decision letter

Noboru Mizushima
Reviewing Editor; The University of Tokyo, Japan

David Ron
Senior Editor; University of Cambridge, United Kingdom

Noboru Mizushima
Reviewer; The University of Tokyo, Japan

Boyi Gan
Reviewer; The University of Texas MD Anderson Cancer Center, United States

これで必ずしも
全員ということで
はない

In the interests of transparency, eLife publishes the most substantive revision requests and the accompanying author responses.

Acceptance summary:

This paper shows that deletion of STK11 (also known as LKB1) in adult mice induces compensatory activation of autophagy to promote tissue homeostasis and animal survival. These results are contradictory to our expectation that STK11/LKB1 should inhibit autophagy as STK11/LKB1 is known to activate AMP-activated protein kinase (AMPK), a positive regulator of autophagy. Thus, this study provides important insights into the role of STK11/LKB1 in physiological settings in vivo, especially in terms of autophagy regulation.

https://elifesciences.org/articles/62377#sa1

Decision letter after peer review:

Thank you for submitting your article "Autophagy Compensates for Lkb1 Loss to Maintain Adult Mice Homeostasis and Survival" for consideration by *eLife*. Your article has been reviewed by three peer reviewers, including Noboru Mizushima as the Reviewing Editor and Reviewer #1, and the evaluation has been overseen by David Ron as the Senior Editor. The following individual involved in review of your submission has agreed to reveal their identity: Boyi Gan (Reviewer #2).

The reviewers have discussed the reviews with one another and the Reviewing Editor has drafted this decision to help you prepare a revised submission.

Summary:

This study shows that, if LKB1 is conditionally deleted in adult mice, autophagy is upregulated. This could be a compensatory mechanism because the lifespan of Lkb1 KO mice is shortened by additional KO of ATG7 (from 6 to 4 weeks). The authors further demonstrate that the intestinal barrier function is impaired after acute deletion of LKB1. This phenotype can also be partially rescued by autophagy through inhibition of p53 induction. Overall, this is an interesting and solid mouse genetic study, which significantly expands our understanding of LKB1 and autophagy in regulating tissue homeostasis and animal survival in vivo. One particular novel aspect of the study is that, while it is believed that LKB1 KO should inhibit autophagy by inactivating AMPK, this study showed convincingly that, in response to Lkb1 deficiency in vivo, autophagy is actually activated to promote tissue homeostasis and animal survival. This study, therefore, highlights the critical need to conduct rigorous in vivo genetic studies to study [illegible]

Revisions for this paper:

1) How Lkb1 deletion leads to autophagy activation in vivo remains unclear. The authors mentioned that, besides LKB1, other AMPK kinases, such as CaMKK2 and TAK can phosphorylate AMPK to activate autophagy. However, it seems unlikely that Lkb1 deletion would even increase AMPK phosphorylation in vivo (based on their observation that autophagy is increased upon Lkb1 deletion in vivo). In Figure 1B, authors need to check phosphor-AMPK and AMPK in various tissues of Lkb1 WT and KO mice.

2) Related to the above comment, one of the key messages in this study is that the Lkb1-AMPK-mTORC1 axis may not be critical in autophagy regulation. However, the activity of both AMPK and mTORC1 is not monitored in this study. The authors show that acute depletion of Lkb1 reduced the levels of amino acids and other major metabolites. Amino acid deficiency is expected to inactivate mTORC1 in an AMPK-independent manner, leading to autophagy induction. Therefore, it is important to determine whether mTORC1 is inhibited in Lkb1, ATG7, and double KO mice. If the activity of neither AMPK nor mTORC1 is changed, it is possible that the increased autophagy observed in various tissues of Lkb1 KO mice more likely reflects a systemic adaptive response to the metabolic collapse. Obviously, it will be challenging to formally test this hypothesis, but the authors should at least discuss this or other potential mechanisms which underlie increased autophagy phenotype in Lkb1 KO mice.

3) The authors conclude that autophagy is activated in Lkb1 KO mice solely based on the HCQ treatment experiments (Figure 1D). As this is one of the critical points in this study, autophagy induction should be confirmed by other methods, such as by checking phosphorylation of ULK1-S757 (PMID: 21258367) and ATG16L1-S278 (PMID: 31768061).

4) Despite the marked disruption of the intestinal barrier in the Lkb1-deficient mice (Figure 3F), a thorough study on epithelial cell death was missing. In addition to caspase-3 (Figure 3E), Tunel assay and other types of cell death should be evaluated. Also, the authors need to check whether p53 KO rescued the increased cleaved caspase-3 staining in triple KO mice.

どうしても必要な改訂だけを500-1000語で

https://elifesciences.org/articles/62377#sa1

Revisions expected in follow-up work:

While the elevation in autophagy extended the lifespan of Lkb1-KO mice, it failed to restore their homeostasis and survival. Also, although p53-deficiency improved the symptoms imposed by *Atg7*-deficiency, it did not ameliorate any defects caused by Lkb1-deficiency alone. Thus, it is important to understand the autophagy-independent pathway impaired by LKB1 loss and how it contributes to the dysfunctions caused by LKB1 deficiency. Although the authors could address these issues in the future, at least discuss these autophagy-independent roles of Lkb1 in more depth in this study.

将来やるとよいけれども、この論文では必要ないこと

minor commentsは公開されていない

[Editors' note: further revisions were suggested prior to acceptance, as described below.]

Thank you for resubmitting your work entitled "Autophagy Compensates for Lkb1 Loss to Maintain Adult Mice Homeostasis and Survival" for further consideration by *eLife*. Your revised article has been evaluated by David Ron as the Senior Editor and a Reviewing Editor.

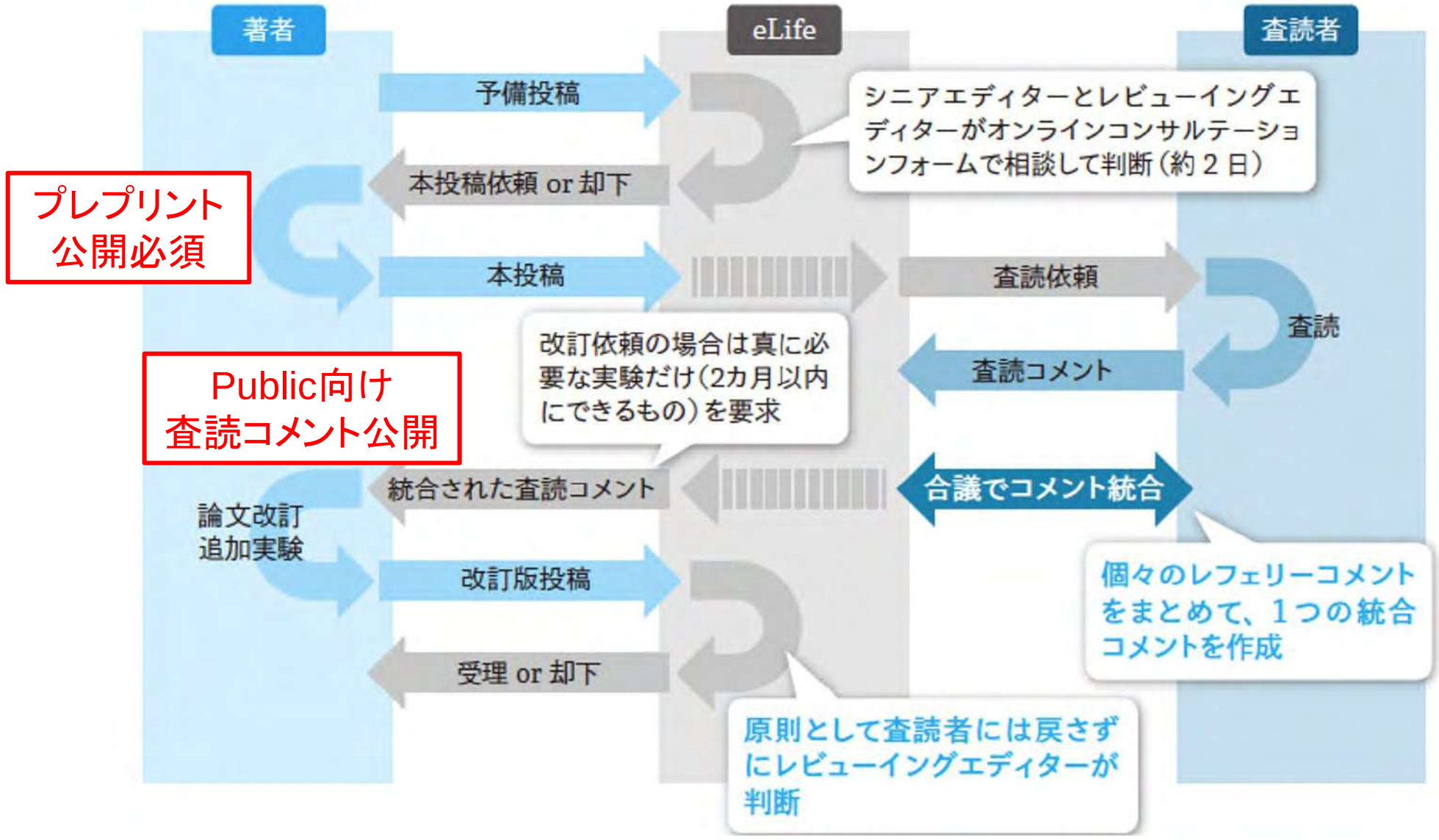
The manuscript has been improved but there are some remaining issues that need to be addressed before acceptance, as outlined below:

The authors monitor the level of ATG12-ATG5 as an indicator of autophagic activity. However, ATG12-ATG5 is not degraded by autophagy and is not generally considered as an autophagy indicator. To avoid confusing readers, the authors may consider removing the data in Figure 1G and the corresponding text.

改訂版に対する判定は査読者に回さずにエディターが迅速に判断

https://elifesciences.org/articles/62377#sa1

eLifeの当初の査読プロセス
2021年7月～



水島 昇『科学を育む　査読の技法』羊土社、2021年、47頁より改変

1. 評価の簡単なサマリー（公開）

2. Public review（公開）
論文の長所と短所、結論が支持されているかどうか

3. 著者向けの具体的なコメント（非公開）
・Essential Revision（合議で抽出）
・各査読者のコメント（参考）

Dear Dr Mizushima,

Thank you for submitting your article "A pulse-chasable reporter processing assay for mammalian autophagic flux with HaloTag" for consideration by eLife. Your article has been reviewed by 3 peer reviewers, and the evaluation has been overseen by a Reviewing Editor and Vivek Malhotra as the Senior Editor. The following individual involved in the review of your submission has agreed to reveal their identity: Sharon Tooze (Reviewer #3).

The reviewers have discussed their reviews with one another, and the Reviewing Editor has drafted this to help you prepare a revised submission.

Essential Revisions (for the authors):

1) Modify the text to address reviewer3's comment "However, I am not entirely convinced of the overall benefits of employing this system if there exists a battery of well-characterized stable cell lines with tandem fluorescence tagged ATG8s. The main benefit would come from tagging cargo which would make the assay independent of the ATG8s (such as the authors have done with Halo-ER markers)."

2) reviewer 1 "Does this assay can be used to measure autophagy influx in vivo? Authors may want to discuss this possibility in the discussion."

3) Line 188, Figure 2E: In order to convincingly conclude that there are no autolysosomes, can Fig 2e (especially the Baf treatment) be conducted using LAMP staining since the structures could represent autolysosomes that have lost their pH and therefore are not stained by lysotracker.

4) Figure 3G: There is a major band at approximately 37 kDa which also has in-gel fluorescence. Does this band represent free GFP or is it fluorescence from a cleavage product of the HaloTag construct? In addition, is the product localised to mitochondria or the cytosol (can be tested by crude mitochondrial isolation)? Western blotting with the GFP antibody would help clarify the identity of the species while gel fluorescence without TMR ligand could also be used. This is an important point to address because it could influence the interpretation of fluorescent signals observed via imaging.

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eLife's editorial process also produces an assessment by peers designed to be posted alongside a preprint for the benefit of readers.

Public Evaluation Summary:

This paper will be of interest to researchers in the autophagy field. It provides a useful tool to accurately measure autophagy flux, providing a useful alternative to the existing assay. The key claims of the manuscript are well supported by the data, and the approaches used are thoughtful and rigorous.

Reviewer #1 (Public Review):

In this manuscript, authors found Halo tag become resistant to lysosome degradation upon ligand binding, using this unique property, they developed a highly sensitive assay to monitor the autophagy flux. Measuring autophagy flux is one of the most important assays for studying autophagy, there are a few widely used assays to monitor the autophagy flux, such as p62 degradation, and LC3 processing, however, each of them has its own limitation, which is well known in the field. In this regard, this assay provides a simple, straight forward and sensitive assay for measuring autophagy flux, which I personally think is very likely it will be widely used by the autophagy community. This is a well-controlled, rigorous study and the manuscript is clearly written.

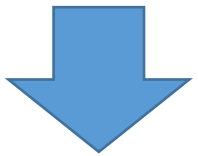
Reviewer #1 (Recommendations for the authors):

I only have a few minor suggestions.

評価と公開の順番をいれかえる

これまでの方法

査読者による評価



公開

これからの方法
Publish, Review, and Curate (PRC) model

公開（プレプリント）

査読者による評価

査読者コメント公開
（ジャーナルによる
公開論文の評価）

Implementing a “publish, then review” model of publishing. eLife. 2020;9, e64910.より
https://doi.org/10.7554/eLife.64910

評価と公開の順番をいれかえる

「原核生物と真核生物の中間に位置するアーキアの分離」

New Results

Isolation of an archaeon at the prokaryote-eukaryote interface

Hiroyuki Imachi, Masaru K. Nobu, Nozomi Nakahara, Yuki Morono, Miyuki Ogawara, Yoshihiro Takaki, Yoshinori Takano, Katsuyuki Uematsu, Tetsuro Ikuta, Motoo Ito, Yohei Matsui, Masayuki Miyazaki, Kazuyoshi Murata, Yumi Saito, Sanae Sakai, Chihong Song, Eiji Tasumi, Yuko Yamanaka, Takashi Yamaguchi, Yoichi Kamagata, Hideyuki Tamaki, Ken Takai

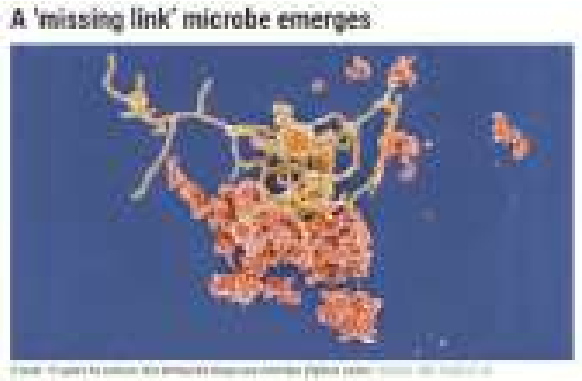
doi: https://doi.org/10.1101/726976

Posted: August 08, 2019

https://www.biorxiv.org/content/10.1101/726976v2

雑誌Scienceの"2019 BREAKTHROUGH OF THE YEAR"（9編）に選出！

https://vis.sciencemag.org/breakthrough2019/finalists/#Darkness-made-visible



Published: January 15, 2020

Article

Isolation of an archaeon at the prokaryote–eukaryote interface

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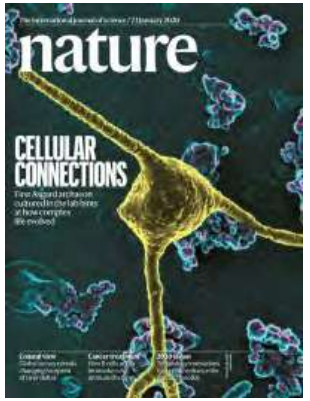
Published online: 15 January 2020
Open access

Hiroyuki Imachi[1,3]*, Masaru K. Nobu[2,3]*, Nozomi Nakahara[1,2,3], Yuki Morono[4], Miyuki Ogawara[1], Yoshihiro Takaki[5], Yoshinori Takano[6], Katsuyuki Uematsu[6], Tetsuro Ikuta[7], Motoo Ito[8], Yohei Matsui[9], Masayuki Miyazaki[1], Kazuyoshi Murata[9], Yumi Saito[1], Sanae Sakai[1], Chihong Song[10], Eiji Tasumi[1], Yuko Yamanaka[1], Takashi Yamaguchi[9], Yoichi Kamagata[5], Hideyuki Tamaki[2] & Ken Takai[1,10]



評価と公開の順番をいれかえる

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doi: https://doi.org/10.1101/726976

公開

Posted: August 08, 2019

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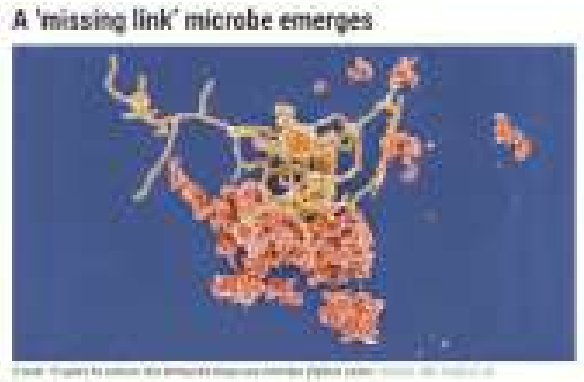

雑誌Scienceの"2019…GH OF THE YEAR"（9編）に選出！

https://vis.sciencema…lists/#Darkness-made-visible

評価

Published: January 15, 2020

Article

Isolation of an archaeon at the prokaryote–eukaryote interfa…

出版

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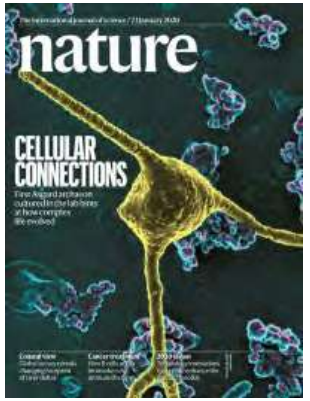


eLife

EDITORIAL

2023年1月31日から
査読した論文はすべて掲載する！
アクセプトもリジェクトもしない！

SCIENTIFIC PUBLISHING

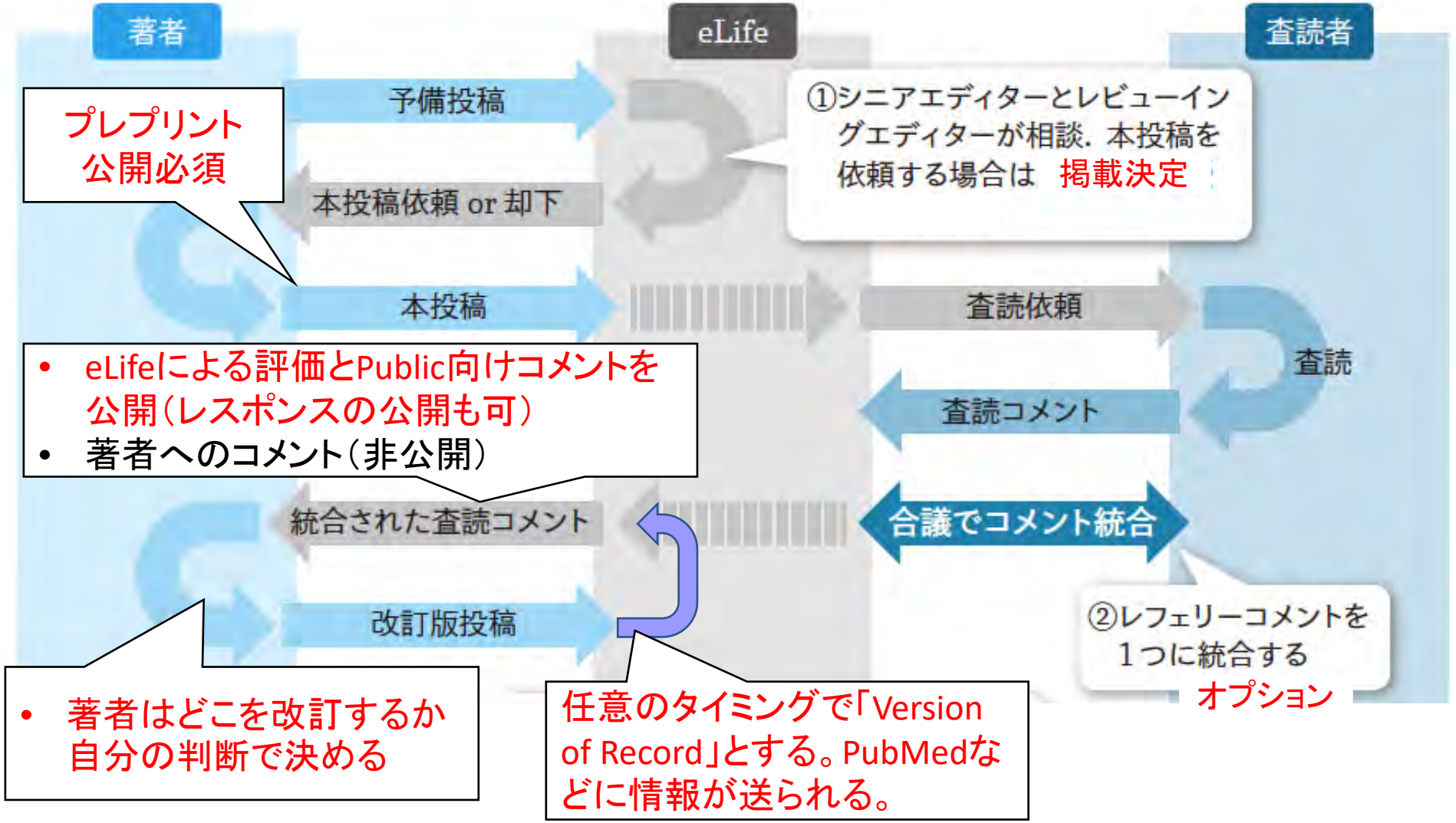
Peer review without gatekeeping

eLife is changing its editorial process to emphasize public reviews and assessments of preprints by eliminating accept/reject decisions after peer review.

MICHAEL B EISEN, ANNA AKHMANOVA, TIMOTHY E BEHRENS, JÖRN DIEDRICHSEN, DIANE M HARPER, MIHAELA D IORDANOVA, DETLEF WEIGEL AND MONE ZAIDI

Published 20 October 2022

2023年1月31日からのeLifeの新しいプロセス



評価を読者に委ねる

改訂を著者に委ねる

水島 昇『科学を育む　査読の技法』羊土社、2021年、49頁より改変

eLife Inside eLife

eLife's New Model: Changing the way you share your research

From next year, eLife is eliminating accept/reject decisions after peer review, instead focusing on public reviews and assessments of preprints.

https://elifesciences.org/inside-elife/54d63486/elife-s-new-model-changing-the-way-you-share-your-research

Syntaxin 17 recruitment to mature autophagosomes is temporally regulated by PI4P accumulation

Saori Shinoda, Yuji Sakai, Takahide Matsui, Masaaki Uematsu, Ikuko Koyama-Honda, Jun-ichi Sakamaki, Hayashi Yamamoto, Noboru Mizushima

Department of Biochemistry and Molecular Biology, Graduated School of Medicine, The University of Tokyo, Tokyo, Japan • Faculty of Life Sciences, Kyoto Sangyo University, Kyoto, Japan • Department of Biosystems Science, Institute for Life and Medical Sciences, Kyoto University, Kyoto, Japan ... show 3 more

https://doi.org/10.7554/eLife.92189.2

Abstract

During macroautophagy, cytoplasmic constituents are engulfed by autophagosomes. Lysosomes fuse with closed autophagosomes but not with unclosed intermediate structures. This is achieved in part by the late recruitment of the autophagosomal SNARE syntaxin 17 (STX17) to mature autophagosomes. However, how STX17 recognizes autophagosome maturation is not known. Here, we show that this temporally regulated recruitment of STX17 depends on the positively charged C-terminal region of STX17. Consistent with this finding, mature autophagosomes are more negatively charged compared with unclosed intermediate structures. This electrostatic maturation of autophagosomes is likely driven by the accumulation of phosphatidylinositol 4-phosphate (PI4P) in the autophagosomal membrane. Accordingly, dephosphorylation of autophagosomal PI4P prevents the association of STX17 to autophagosomes. Furthermore, molecular dynamics simulations support PI4P-dependent membrane insertion of the transmembrane helices of STX17. Based on these findings, we propose a model in which STX17 recruitment to mature autophagosomes is temporally regulated by a PI4P-driven change in the surface charge of autophagosomes.

eLife assessment

This paper addresses a **fundamental** issue in the field of autophagy: how is a protein responsible for autophagosome-lysosome fusion recruited to mature autophagosomes but not immature ones? The work succeeds in its ambition to provide a new conceptual advance. The evidence

Reviewed preprint version 2
April 10, 2024 (this version)

Reviewed preprint version 1
October 17, 2023 • Go to version

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September 12, 2023 • Go to preprint server

Sent for peer review
August 30, 2023

採択／却下のバイナリー評価ではなく、ニュアンスをもった評価へ

eLife assessment

This paper addresses a **fundamental** issue in the field of autophagy: how is a protein responsible for autophagosome-lysosome fusion recruited to mature autophagosomes but not immature ones? The work succeeds in its ambition to provide a new conceptual advance. The evidence supporting the conclusions is **convincing**, with fluorescence microscopy, biochemical assays, and molecular dynamics simulations. This work will be of broad interest to cell biologists and biochemists studying autophagy, and also those focusing on lipid/membrane biology.

https://doi.org/10.7554/eLife.92189.3.sa0

Read more about this assessment ▾

採択／却下のバイナリー評価ではなく、ニュアンスをもった評価へ

eLife assessment

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Significance of the findings: 発見の重要性

Fundamental: Findings that substantially advance our understanding of major research questions

Landmark | **Fundamental** | Important | Valuable | Useful

Strength of evidence: 証拠の強さ

Convincing: Appropriate and validated methodology in line with current state-of-the-art

Exceptional | Compelling | **Convincing** | Solid | Incomplete | Inadequate

発見の重要性

Describing the **significance of the findings**:

- **Landmark**: findings with profound implications that are expected to have widespread influence
- **Fundamental**: findings that substantially advance our understanding of major research questions
- **Important**: findings that have theoretical or practical implications beyond a single subfield
- **Valuable**: findings that have theoretical or practical implications for a subfield
- **Useful**: findings that have focused importance and scope

https://elifesciences.org/about/elife-assessments

証拠の強さ

Describing the **strength of evidence**:

- **Exceptional**: exemplary use of existing approaches that establish new standards for a field
- **Compelling**: evidence that features methods, data and analyses more rigorous than the current state of the art
- **Convincing**: appropriate and validated methodology in line with current state-of-the-art
- **Solid**: methods, data and analyses broadly support the claims with only minor weaknesses
- **Incomplete**: main claims are only partially supported
- **Inadequate**: methods, data and analyses do not support the primary claims

https://elifesciences.org/about/elife-assessments

Reviewer #1 (Public Review):

In this manuscript, the authors report a molecular mechanism for recruiting syntaxin 17 (Syn17) to the closed autophagosomes through the charge interaction between enriched PI4P and the C-terminal region of Syn17. How to precisely control the location and conformation of proteins is critical for maintaining autophagic flux. Particularly, the recruitment of Syn17 to autophagosomes remains unclear. In this paper, the author describes a simple lipid-protein interaction model beyond previous studies focusing on protein-protein interactions. This represents conceptual advances.

https://doi.org/10.7554/eLife.92189.2.sa2

Reviewer #2 (Public Review):

Summary:

Syntaxin17 (STX17) is a SNARE protein that is recruited to mature (i.e., closed) autophagosomes, but not to immature (i.e., unclosed) ones, and mediates the autophagosome-lysosome fusion. How STX17 recognizes the mature autophagosome is an unresolved interesting question in the autophagy field. Shinoda and colleagues set out to answer this question by focusing on the C-terminal domain of STX17 and found that PI4P is a strong candidate that causes the STX17 recruitment to the autophagosome.

Strengths:

The main findings are: 1) Rich positive charges in the C-terminal domain of STX17 are sufficient for the recruitment to the mature autophagosome; 2) Fluorescence charge sensors of different strengths suggest that autophagic membranes have negative charges and the charge increases as they mature; 3) Among a battery of fluorescence biosensors, only PI4P-binding biosensors distribute to the mature autophagosome; 4) STX17 bound to isolated autophagosomes is released by treatment with Sac1 phosphatase; 5) By dynamic molecular simulation, STX17 TM is shown to be inserted to a membrane containing PI4P but not to a membrane without it. These results indicate that PI4P is a strong candidate that STX17 binds to in the autophagosome.

Weaknesses:

• It was not answered whether PI4P is crucial for the STX17 recruitment in cells because manipulation of the PI4P content in autophagic membranes was not successful for unknown reasons.

Reviewed Preprint

Revised by authors after peer review.

About eLife's process

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August 30, 2023

Research Article
Cell Biology

Syntaxin 17 recruitment to mature autophagosomes is temporally regulated by PI4P accumulation

Saori Shinoda, Yuji Sakai, Takahide Matsui, Masaaki Uematsu, Ikuko Koyama-Honda, Jun-ichi Sakamaki, Hayashi Yamamoto, Noboru Mizushima

Department of Biochemistry and Molecular Biology, Graduated School of Medicine, The University of Tokyo, Japan; Department of Biosystems Science, Institute for Life and Medical Sciences, Kyoto University, Japan; Department of Molecular Oncology, Institute for Advanced Medical Sciences, Nippon Medical School, Japan

Jun 4, 2024 • https://doi.org/10.7554/eLife.92189.3

eLife assessment

This paper addresses a **fundamental** issue in the field of autophagy: how is a protein responsible for autophagosome-lysosome fusion recruited to mature autophagosomes but not immature ones? The work succeeds in its ambition to provide a new conceptual advance. The evidence supporting the conclusions is **convincing**, with fluorescence microscopy, biochemical assays, and molecular dynamics simulations. This work will be of broad interest to cell biologists and biochemists studying autophagy, and also those focusing on lipid/membrane biology.

https://doi.org/10.7554/eLife.92189.3.sa0

Version of Record
June 4, 2024
Read the peer reviews

Reviewed Preprint
v2 • April 10, 2024

Reviewed Preprint
v1 • October 17, 2023

Hide all versions

SUBMISSION TRENDS

After *eLife* did away with the conventional 'accept or reject' model for publishing papers in January 2023, submissions stayed relatively steady and were similar to pre-COVID levels. The journal saw a surge in papers received in 2020–21, attributed to a rush of research related to the pandemic.

https://www.nature.com/articles/d41586-024-04199-z

eLifeはインパクトファクターを失うことに

nature

nature › news › article

NEWS | 18 December 2024

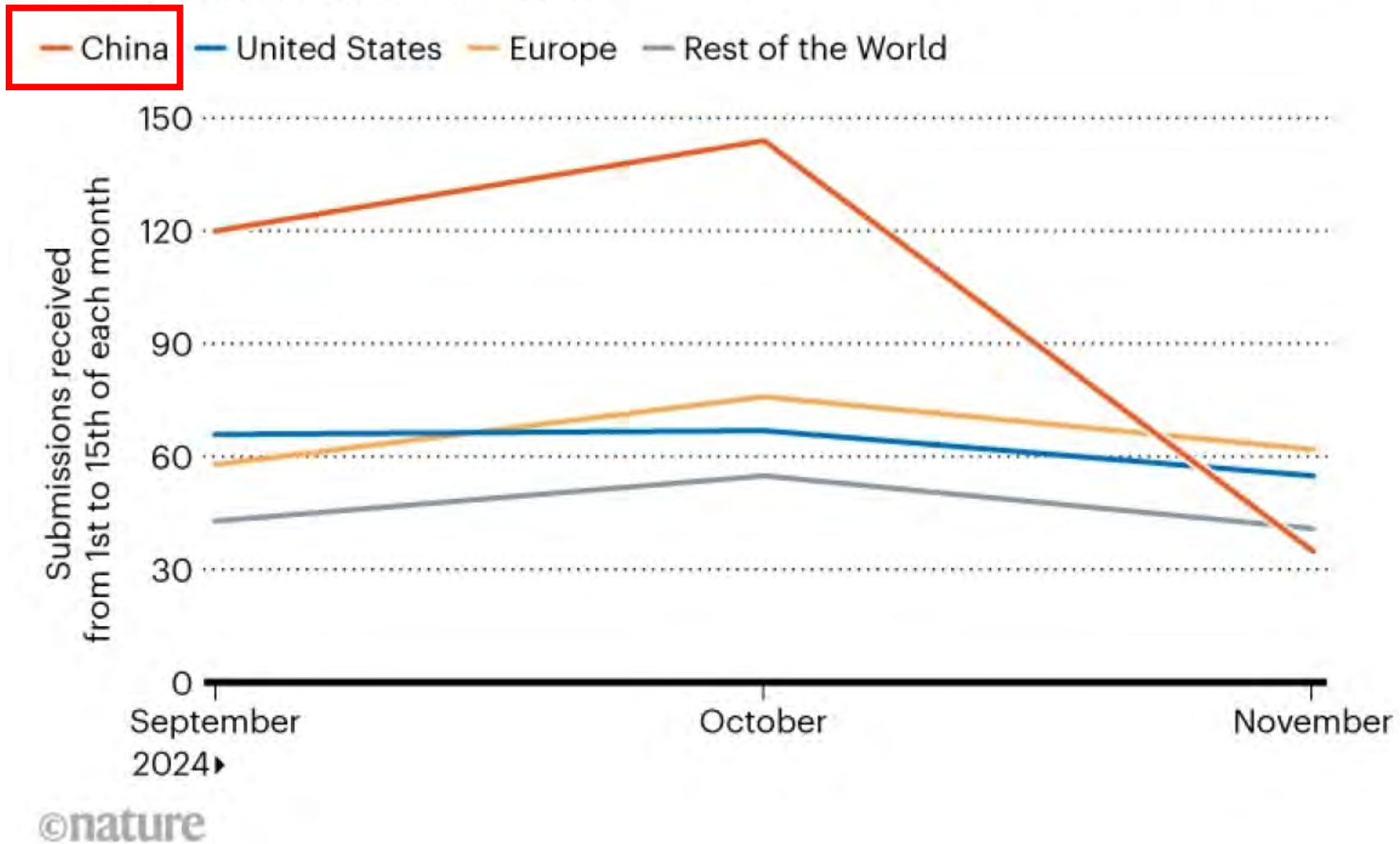
Pioneering journal *eLife* faces major test after loss of impact factor

The open-access title's bold publishing model has bought long-bubbling conflicts to the fore.

By Diana Kwon

IMPACT-FACTOR EFFECT

After analytics firm Clarivate announced in October that the Web of Science database would no longer index *eLife* papers, meaning that the journal would no longer receive an impact factor, there was a substantial fall in submissions from China, where there is a strong dependence on the metric.



https://www.nature.com/articles/d41586-024-04199-z

Inside eLife

Research organisations still consider eLife papers in funding and hiring decisions

We've just announced that more than 100 funders and institutions combined continue to consider eLife papers when evaluating research contributions – regardless of our Impact Factor loss last year.

80 views • May 8, 2025

PLOS BIOLOGY

PERSPECTIVE

A proposal for the future of scientific publishing in the life sciences

Bodo M. Stern*, Erin K. O'Shea*

Howard Hughes Medical Institute, Chevy Chase, Maryland, United States of America

* osheae@hhmi.org (EKO); sternb@hhmi.org (BMS)

"Publish first, curate second"

PLoS Biol 17: e3000116 (2019). https://doi.org/10.1371/journal.pbio.3000116

Publish, Review, Curate: eLife, F1000, BioPhysics Colab and more

Organisations are also bringing these elements together and offering end-to-end Publish, Review, Curate experiences for authors.

eLife's model is one example of a PRC-approach to publishing that still provides all the things authors need from a journal. Authors whose work is reviewed by eLife will then have their Reviewed Preprint published – a citable, DOId article that includes public reviews and an eLife Assessment that many funders accept for evaluation as part of research and researcher assessment. In 2023, BioPhysics Colab were the first group to adopt a version of eLife's approach to PRC and later this year MetaROR is set to launch a two-year pilot for a PRC model for metaresearch.

Data- and software-focused life science journal, Gigabyte, offers a "release then review" model of publishing. The Peer Community In family of journals focuses on recommendations and published evaluations of preprints. And as mentioned earlier, the F1000 and Open Research Europe (ORE) publishing models offer PRC without preprinting.

https://elifesciences.org/inside-elife/dc24a9cd/open-science-what-is-publish-review-curate?utm_source=newsletter&utm_medium=email&utm_campaign=eLife_News_Dec2024

プレプリント投稿を奨励するために

- スクープされることへの恐れ
  - 実はかなり稀
  - むしろプライオリティーの点では有利（scoop protection）
  - 永続的なDOIが付与される
- High-impact journalへ掲載できなくなる
  - いまはほぼすべてのトップジャーナルはOK
  - むしろよく多く引用されるようになるとの結果あり
- 研究助成機関や研究機関による認知度（プレスリリース対応も）
  - 改善する必要があるかもしれない。
- 査読前の論文を公表することの不安
  - 査読者のコメントと著者の回答も掲載されるのであまり心配ない

https://elifesciences.org/inside-elife/e5f8f1f7/what-we-have-learned-about-preprints?utm_source=newsletter&utm_medium=email&utm_campaign=eLife_News_Sep21より

現在の査読システムの問題点

- 査読者が研究に介入しすぎている
  - 査読者の役割の明確化（正しさと重要性の評価）
  - 研究指導者との混同（よけいなおせっかい）
  - 過剰な要求（Major Revisionは必要か？）

- 少数の査読者がデータの公開を阻止している
  - 公的資金で行った研究の成果がなかなか公開されない

- 少数の査読者による論文の格付け
  - 結局はインパクトファクターの問題か？

- 査読の負担、査読者の枯渇

査読コメントの共有
Review Commonsなど

- 査読コメントの無駄死に

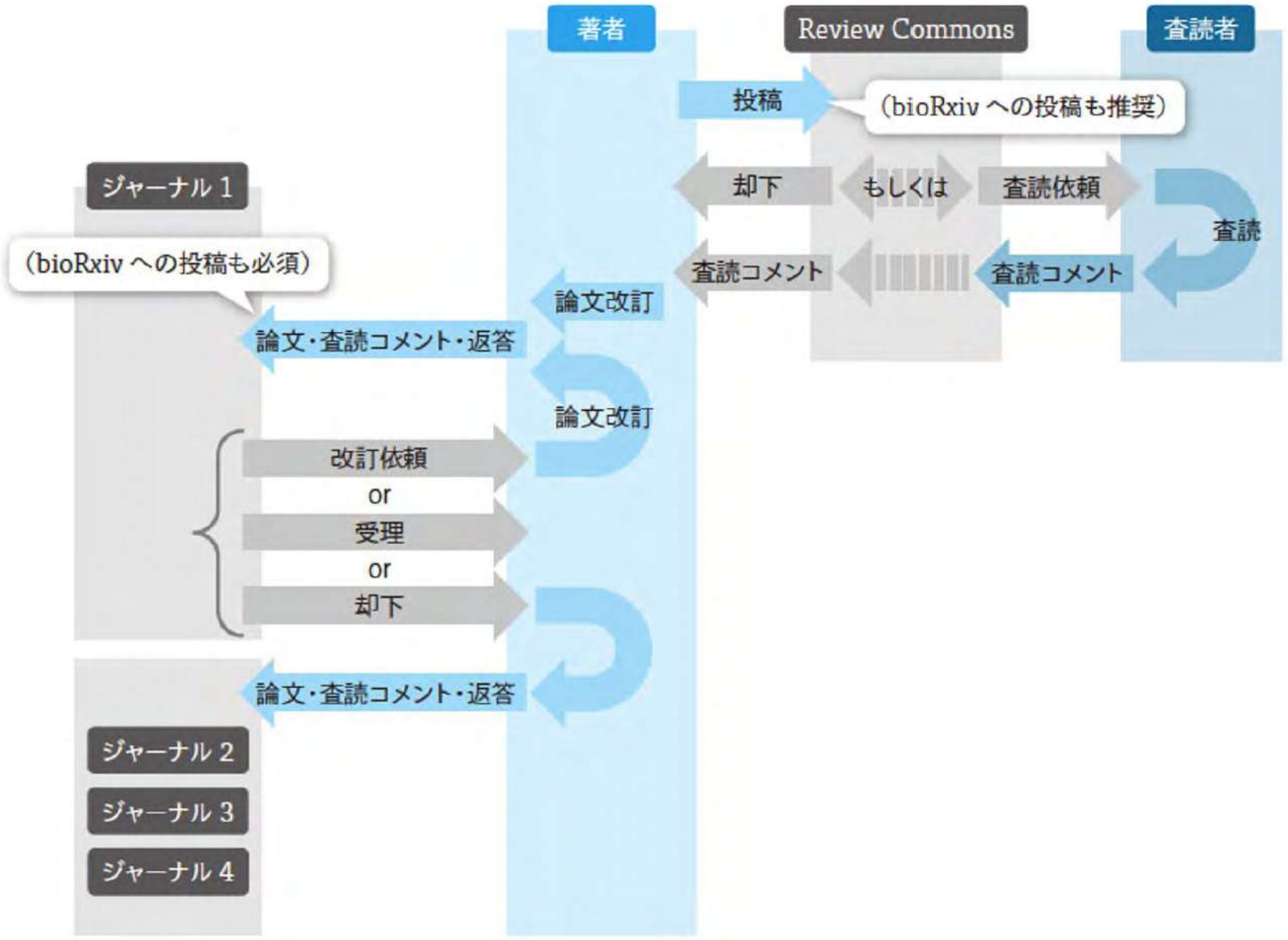
Review Commons

- 2019年12月立ち上げ
- 複数出版社の28誌が連携（2025年8月現在）
- 査読されてから投稿（4誌まで）
- 査読コメントはジャーナルで共有され、査読の負担が軽減
- 出版までの期間の短縮
- 査読者の枯渇を防ぐ

https://www.reviewcommons.org/

Review Commonsの査読プロセス

※その時点からスクーププロテクションの対象



水島 昇『科学を育む　査読の技法』羊土社、2021年、52頁より引用

若手育成

nature

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EDITORIAL | 13 May 2025

Nature project to encourage early-career researchers in peer review is working

Science stands to benefit from a project in which experienced academics and early-career researchers co-review studies.

査読におけるAIの使用

多くのジャーナルは制限（情報漏洩や正確性の懸念）

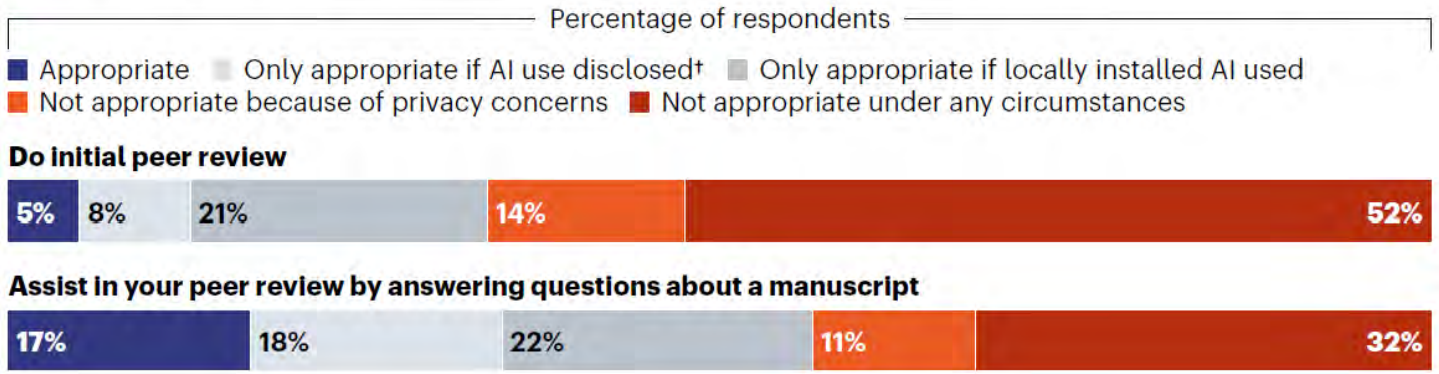
特に制限していないジャーナルも（例：eLife）

A *Nature* survey of 5,000 researchers finds contrasting views on when it's acceptable to involve AI in research papers and what needs to be disclosed.

By Diana Kwon

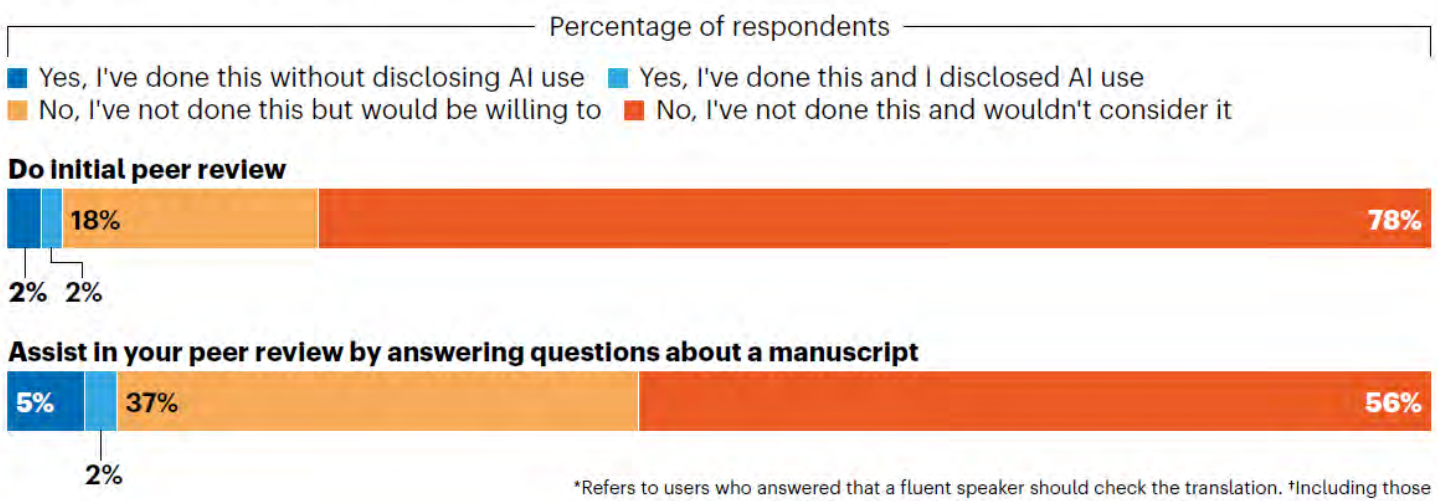
OPINIONS ON AI IN PEER REVIEW

Researchers felt less favourably about using AI to do an initial peer review of a paper, or to assist in peer review.



EXPERIENCES WITH AI IN PEER REVIEW

Very few researchers said they had used AI to help with peer review.



*Refers to users who answered that a fluent speaker should check the translation. †Including those who said prompts should be disclosed. Percentages might not add to 100% owing to rounding.

SCIENTISTS HIDE MESSAGES IN PAPERS TO GAME AI PEER REVIEW

Some preprints containing hidden instructions, visible only to machines, will be withdrawn.

By Elizabeth Gibney

who insert such hidden prompts into papers

白い文字や見えないくらい小さな文字で好意的な査読を促すようなプロンプトを忍ばせておく

査読者へのインセンティブ

- 金銭はほとんどない
- 著者名の公開
  - 論文ごと（Natureなど）
  - まとめて
- 優れた査読者の表彰（J Biochemなど）
- ジャーナルやデータベースの無料購読サービス
  - ジャーナルのオンライン無料購読
  - データベース無料アクセス（Scopusなど）
- 系列オープンアクセス誌への掲載料無料・半額サービス
- 査読証明書の発行
  - 公的プラットフォーム（Publons、ORCID）
  - ジャーナルや出版社によるもの

ジャーナルからの査読実績証明書

SPRINGER NATURE

Noboru Mizushima

The editors of Springer Nature Limited wish to thank you for serving as a reviewer for the Nature journals. Your thoughtful and critical comments are essential to the quality of the articles we publish. Your willingness to offer your time and expertise to the peer-review process is greatly appreciated.

Following is a record of your refereeing activity for the Nature journals. We hope you can use this record to demonstrate your contribution to the peer-review process and to the scientific community.

My Refereeing Activity Number of unique* papers reviewed for Nature journals (by calendar year).	
2022	4
2021	3
2020	4
2019	5
2018	3
2017	5
2016	3
2015	3
2014	6
2013	3
2012	1
2011	1
2010	2
2009	6
2008	1
All Years	50

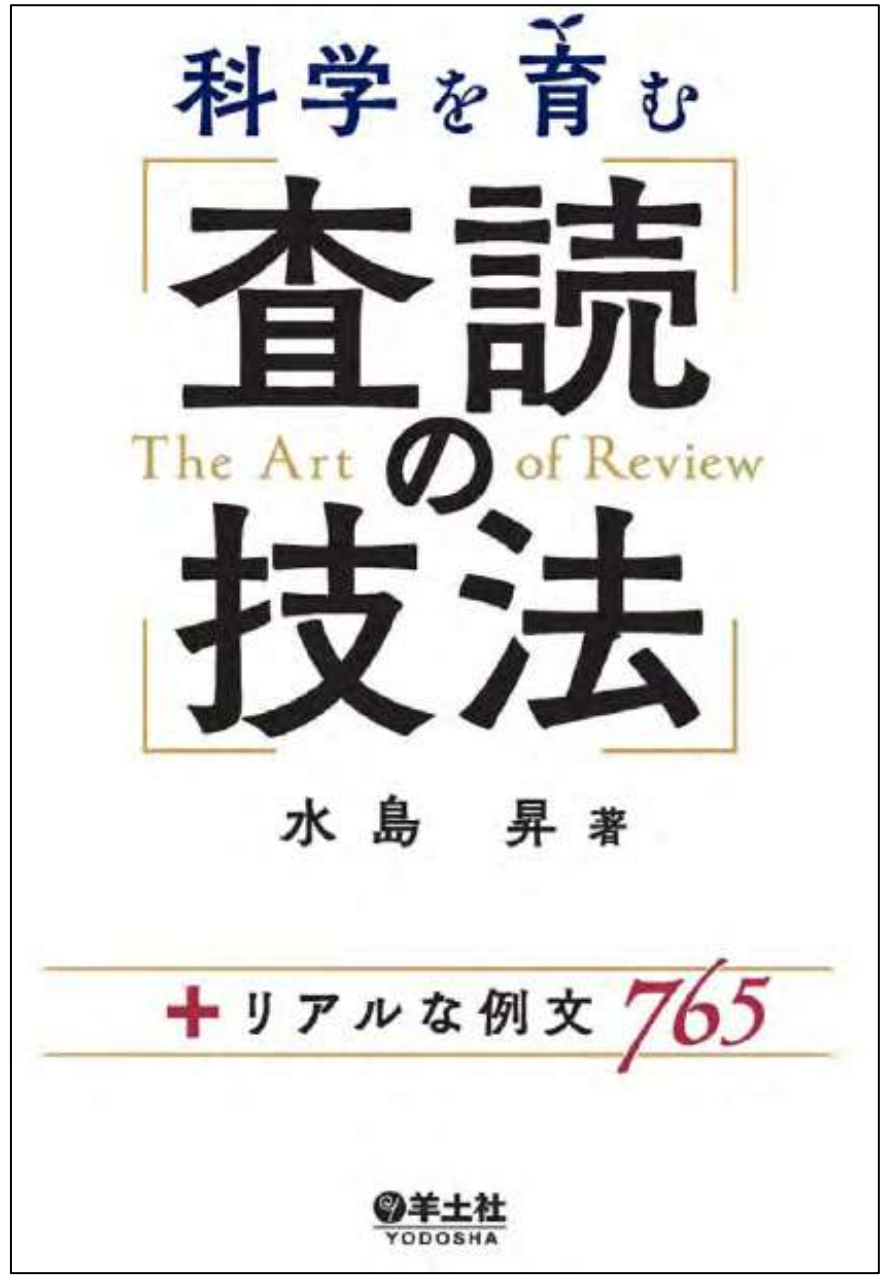
Generated on *2022/08/28 22:13:51 EST*

*Not counting revisions

査読の透明性

- 査読コメントの公開
  - 多くのジャーナルですでに行われている
  - 基本的にはアクセプトされたもの
  - プレプリントに対してもコメントできる
    (Transparent Review in Preprints（TRiP）)
- 査読者名の公開
  - 希望者のみ（Natureなど）
- 査読状況の完全公開
  - 査読コメントと査読者名（F1000 Research）

- ダブルブラインド



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