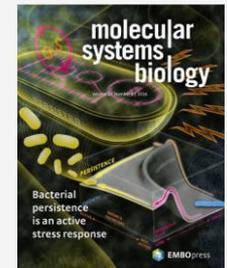
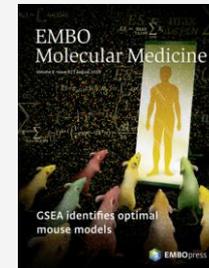
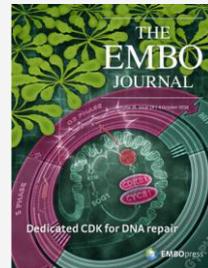


How can institutions help researchers?

Transparent Publishing, Preprints & Open Science: the EMBO Press paradigm

Bernd Pulverer
Chief Editor | *The EMBO Journal*
Head | Scientific Publications, EMBO



What can Journals do?

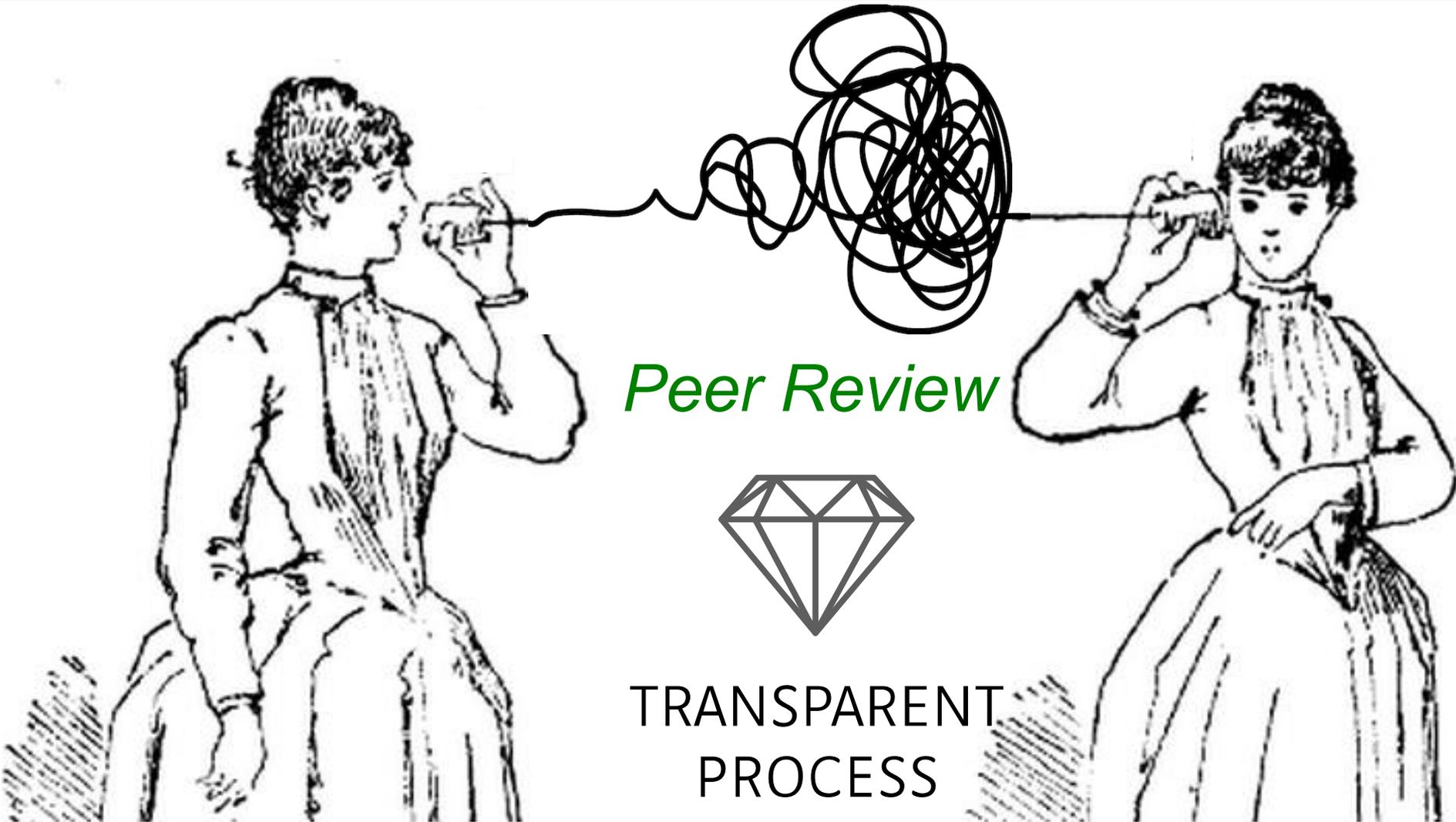
Research Integrity & Reproducibility

- Prepublication checks
- Optimized process
- Enhanced papers

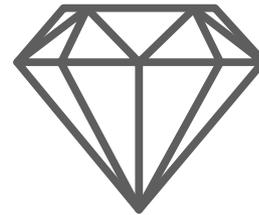
What can Journals do?

- **Efficient Process**
 - Single round revision
 - Manuscript transfers
- **Reproducible Science**
 - Open source data
 - Open references (*i4OS*)
 - Open methods/protocols; e-labbooks
 - Self-correction & Versioning
- **Enhanced Quality Control**
 - Prepublication Integrity checks
 - Data Curation
 - Technical Review
- **Discoverability**
 - Forward-Linking to confirmatory / refuting data
 - Data-directed Search (*SourceData*)
- **Community engagement**
 - Reforming Research Assessment (*DORA*)
 - Journal < > Institutional dialogue (*CLUE*)
 - Training

Journals

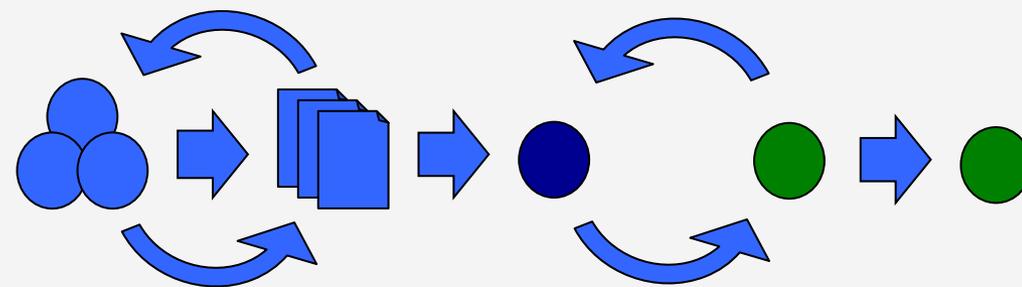


Peer Review



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- **Transparent Process:**
 - Open editorial process to authors and readers
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- **‘Scooping Protection’**



referees

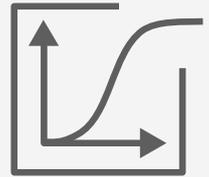
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author



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Data Transparency



SOURCE
DATA

Published data should be **accessible**,
reproducible and **re-usable** by others

Metabolic profiling of the human response to a glucose challenge
O Shalem et al

insulin, we hypothesized that insulin sensitivity could be reflected not only by changes in glucose but also by the OGTT response of multiple other metabolites. Because our initial

Table II Regression models relating fasting insulin to 2-h metabolite change in individuals with impaired glucose tolerance (FOS-IGT)

| Predictor (s) | R ² _{adj} | P-value | Prediction error ^a |
|--|-------------------------------|---------|-------------------------------|
| Δ ^b Leucine/isoleucine | 0.36 | 9E-4 | 6.65 |
| Δ Valine | 0.17 | 3E-2 | 7.74 |
| Δ Lactate | 0.16 | 3E-2 | 7.60 |
| Δ Glycochenodeoxycholic acid | 0.14 | 4E-2 | 7.86 |
| Δ Methionine | 0.14 | 4E-2 | 7.68 |
| Δ β-Hydroxybutyrate | 0.14 | 4E-2 | 7.90 |
| Δ Leucine/isoleucine + Δ glycerol ^c | 0.54 | 7E-5 | 5.66 |
| PLS ^d | 0.46 | 1E-4 | 6.89 |
| BMI | 0.33 | 1E-3 | 6.74 |

^aThe prediction error is expressed as the root mean square error of prediction (RMSEP), in micro-molar per milliliter insulin.
^bΔ denotes log of the 2-h fold change of metabolite levels.
^cA bivariate model consisting of the 2-h changes in leucine/isoleucine and in glycerol.
^dPartial least squares based on changes in the 18 validated metabolites.

studies were focused on normal, healthy individuals spanning a narrow range of fasting insulin levels, we performed a third analysis on a group of individuals with impaired glucose tolerance from the Framingham Offspring Study, FOS-IGT, who spanned a broader range of fasting insulin concentrations (Table II).

First, to systematically evaluate the relationship between individual metabolite excursions and fasting insulin, we performed linear regression of the fasting insulin concentration on each of the 18 2-h excursions. Out of the 18, 6 showed a statistically significant ($P < 0.05$) correlation with fasting insulin, and included the excursions in lactate, β-hydroxybutyrate, amino acids (leucine/isoleucine, valine, and methionine), and a bile acid (GCDC) (Table II). Taken together with the glycerol excursion, which scored ($P = 0.07$) slightly below the significance threshold, the response of four distinct insulin action markers correlated with fasting insulin (Figure 5A). Individuals with high fasting insulin exhibited a blunted excursion in all seven metabolites; they had a smaller change both in increasing metabolites (lactate and GCDC) and in decreasing metabolites (the other five). Notably, the glucose excursion was not correlated with fasting insulin ($P = 0.20$). These findings suggest that resistance to the action of insulin

‘The two vital components of the scientific endeavor – the idea and the evidence – are too frequently separated’

Science as an open enterprise, Royal Society, 2012

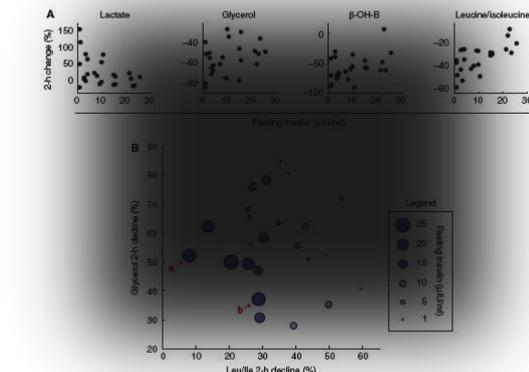
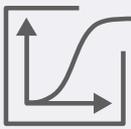


Figure 5 Correlation between fasting insulin and 2h metabolite changes in individuals with impaired glucose tolerance (FOS-IGT). (A) 2-h changes in markers of insulin actions are correlated with fasting insulin concentrations. Each dot corresponds to an individual. (B) A bivariate model explaining fasting insulin using the 2-h decline of Leu/Ile and glycerol. Each circle represents an individual, and the circle size is proportional to fasting insulin levels. *A representative individual exhibiting a blunted decline in Leu/Ile (resistant to proteolysis suppression). **A representative individual exhibiting a blunted decline in glycerol (resistant to lipolysis suppression).

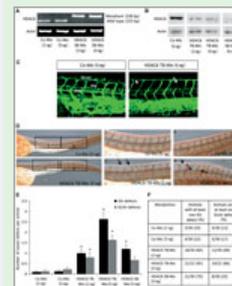


Source Data



SOURCE
DATA

Figure 2.



Silencing of HDAC6 impairs embryonic vessel formation in zebrafish. (A) Aberrant splicing of *Danio rerio* HDAC6 mRNA after HDAC6 splice-blocking Mo injection by PCR. Injection of the HDAC6 SB-Mo

generated at 24 h post fertilization of 338 bp, whereas the HDAC6 mRNA disappeared (253 bp), showing that the HDAC6 SB-Mo was effective. Whole-zebrafish embryos were analyzed 48 h after Mo injection and subjected to RT-PCR. HDAC6 mRNA expression serves as a loading control.

HDAC6 protein expression was analyzed in zebrafish embryo lysate at 48 h post fertilization. HDAC6 translation-blocking Mo was used as a control. Protein lysates were subjected to Western blot analysis with HDAC6-specific antibody and α -tubulin as a loading control. C-F phenotypic analysis of morphants 48 h post fertilization was performed using confocal fluorescence pictures of the anterior part of *tg(fli1:EGFP)* embryos. Injection of HDAC6 translation-blocking Mo was used as a control. Arrows indicate vessel defects. Quantification of vessel defects was performed in control Mo-treated zebrafish embryos and HDAC6 SB-Mo-treated embryos using anti-GFP antibody. Overview pictures and quantification of vessel defects are shown in Figure 2B.

Quantification of vessel defects was performed in control Mo-treated zebrafish embryos and HDAC6 SB-Mo-treated embryos using anti-GFP antibody. Overview pictures and quantification of vessel defects are shown in Figure 2B.

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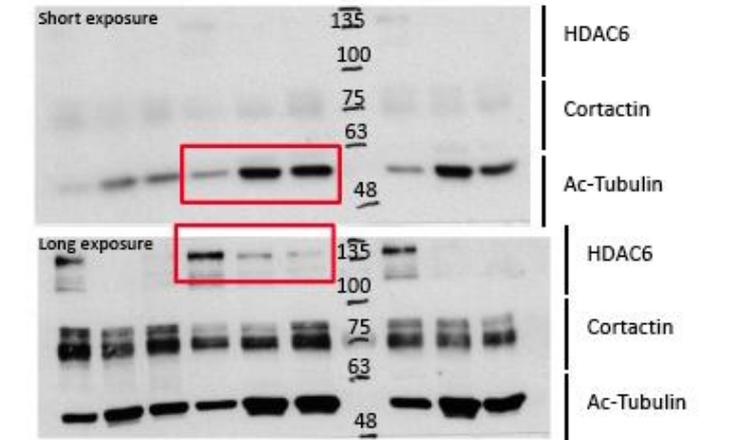
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- Minimally Processed Data
- Replicates



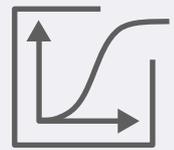
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| 5 | 0.395173883 | 0.389974466 | 0.555355249 |
| 15 | 0.690917146 | 1.236910363 | 1.632582883 |
| 30 | 0.394324884 | 0.72081196 | 1.488299981 |
| 60 | 0.38782972 | 0.38107614 | 0.428561181 |
| | 0.384442827 | 0.216360469 | 0.458929493 |

- Archive
- Transparency
- Replicates
- Reanalysis
- Reuse
- Discourage manipulation
- Figure Level Authorship

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Source Data



SOURCE DATA

- Archive
- Transparency
- Replicates
- Reanalysis
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- Discourage manipulation
- Figure Level Authorship

Figure 2.

Silencing of HDAC6 impairs embryonic vessel formation in zebrafish. (A) Aberrant splicing of *Danio rerio* HDAC6 mRNA after HDAC6 splice-blocking Mo injection by PCR. Injection of the HDAC6 SB-Mo generated at 24 h post fertilization a morphant signal of 338 bp, whereas the HDAC6 wt signal completely disappeared (253 bp), shown in the gel image. (B) Whole-zebrafish embryo 48 h after Mo injection and subsequent vessel formation. (C) Representative confocal fluorescence pictures of vessel in the anterior part of *tg(fli1:EGFP)* zebrafish embryos after injection of HDAC6 translational inhibitor. (D) Quantification of vessel defects in HDAC6 morphants compared to control Mo-treated zebrafish embryos. (E) Representative ISVs and DLAVs for HDAC6 morphants. Statistical significance is indicated by asterisks.

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‘I’m a great believer in seeing all the data – this is an important lever for transparency’
 Michael Farthing, founder COPE

Fig1b&d_raw.txt

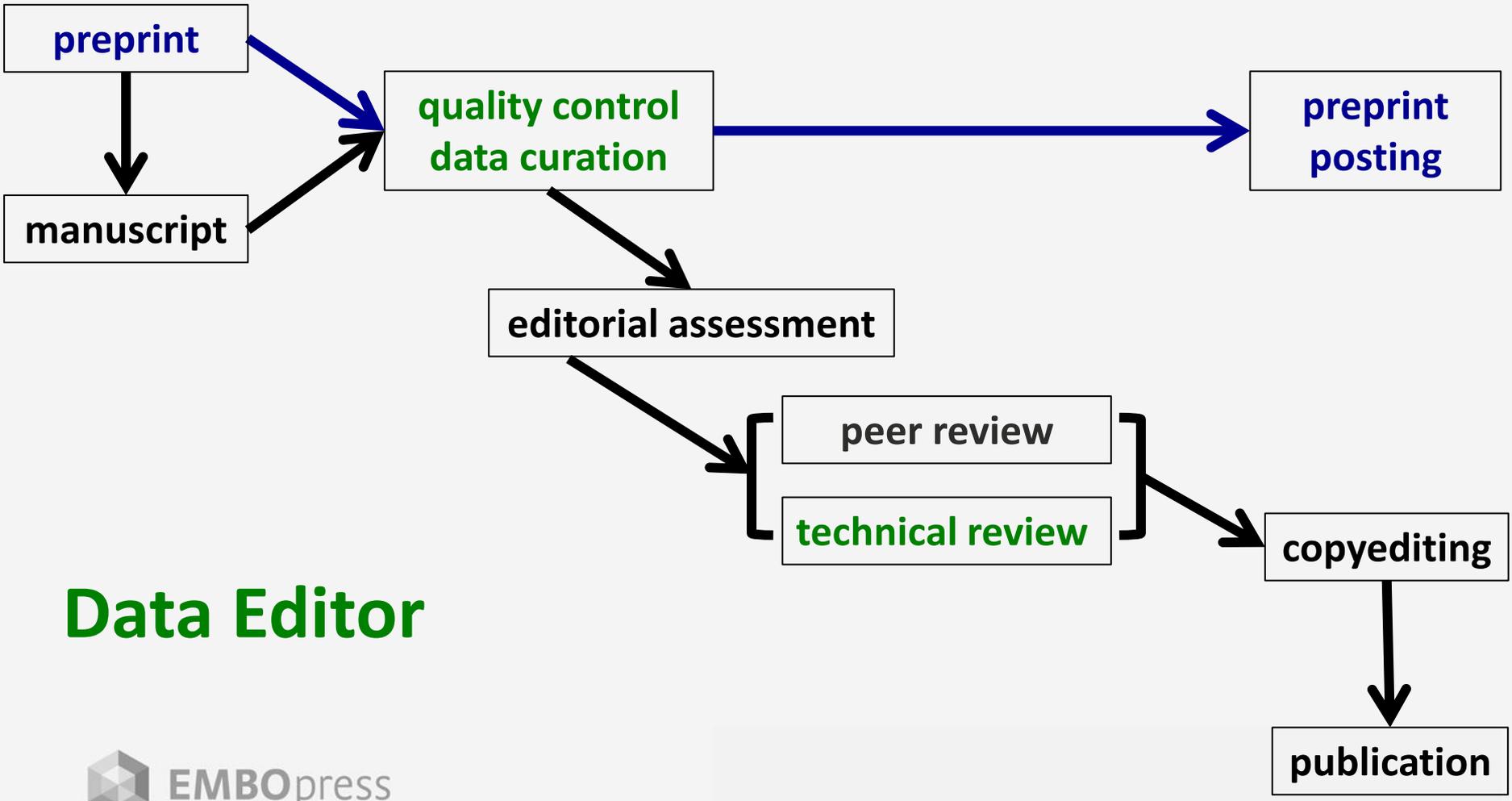
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|----|-----------------|--------------------|--------------------|--------------------|
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| | | p-Erk/t-Erk, 30 pM | p-Erk/t-Erk, 30 pM | p-Erk/t-Erk, 30 pM |
| | | PDGF, control | PDGF, control | PDGF, control |
| 2 | Time (min) | vector, Expt. 1 | vector, Expt. 2 | vector, Expt. 3 |
| 3 | 0 | 0.194672394 | 0.201524091 | 0.339116171 |
| 4 | 5 | 0.395173883 | 0.389974466 | 0.555355249 |
| 5 | 15 | 0.690917146 | 1.236910363 | 1.632582883 |
| 6 | 30 | 0.394324884 | 0.72081196 | 1.488299981 |
| 7 | 60 | 0.38782972 | 0.38107614 | 0.428561181 |
| 8 | 120 | 0.384442827 | 0.216360469 | 0.458929493 |
| 9 | | | | |
| 10 | | | | |



Peer Review, Quality Control & Data Curation

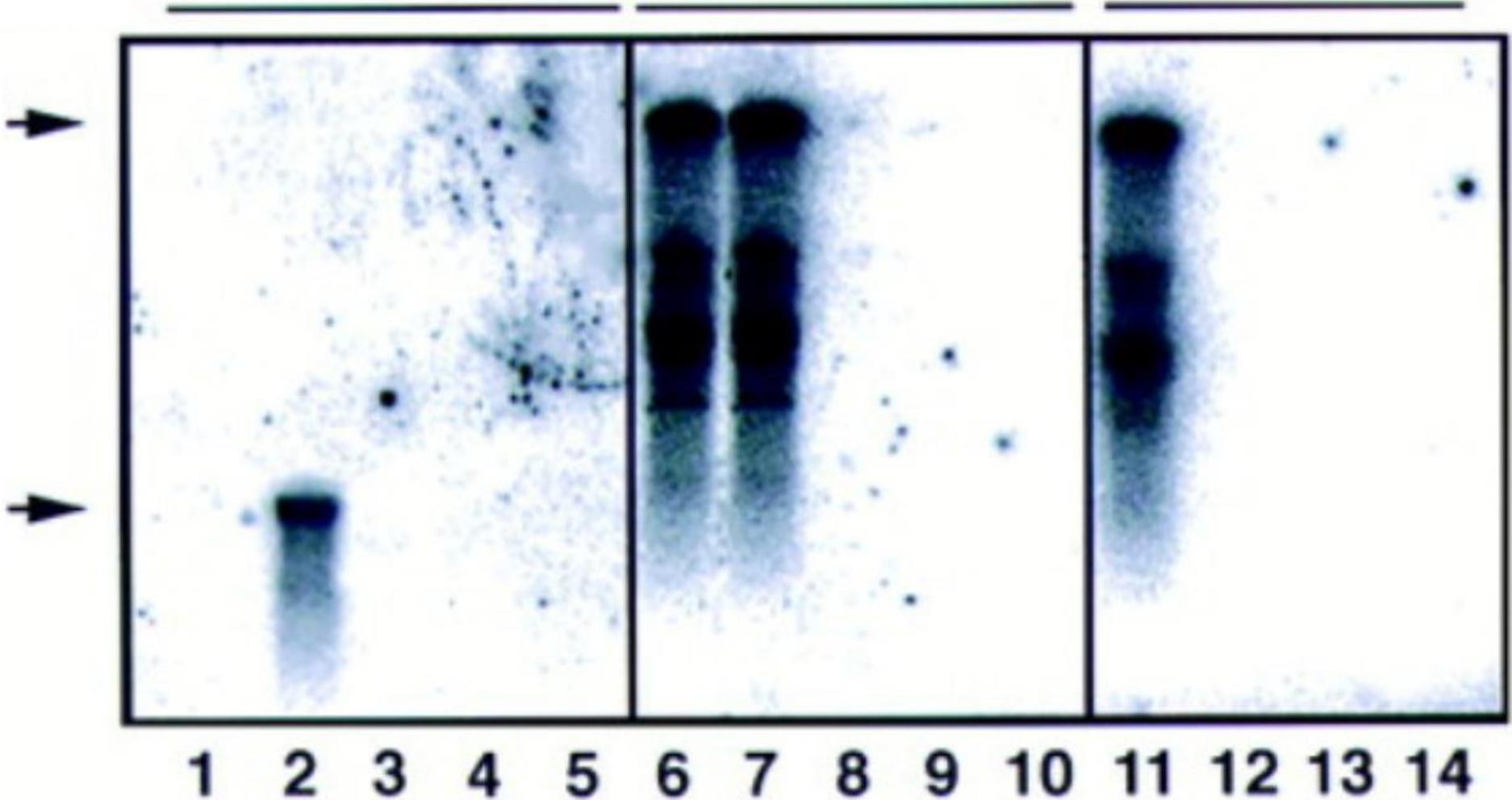
| What | Who |
|--|--|
| <ul style="list-style-type: none">• Editorial Preselection• Peer Review• Technical Review• Data Curation• Quality Control• Ethics (incl. referees) | <p>Scientific Editors Senior Investigators Postdocs Data Editors – Authoring tools Data Editors – Semi-automation Editors</p> |

Prepublication Quality Control @ Journals: the final checkpoint

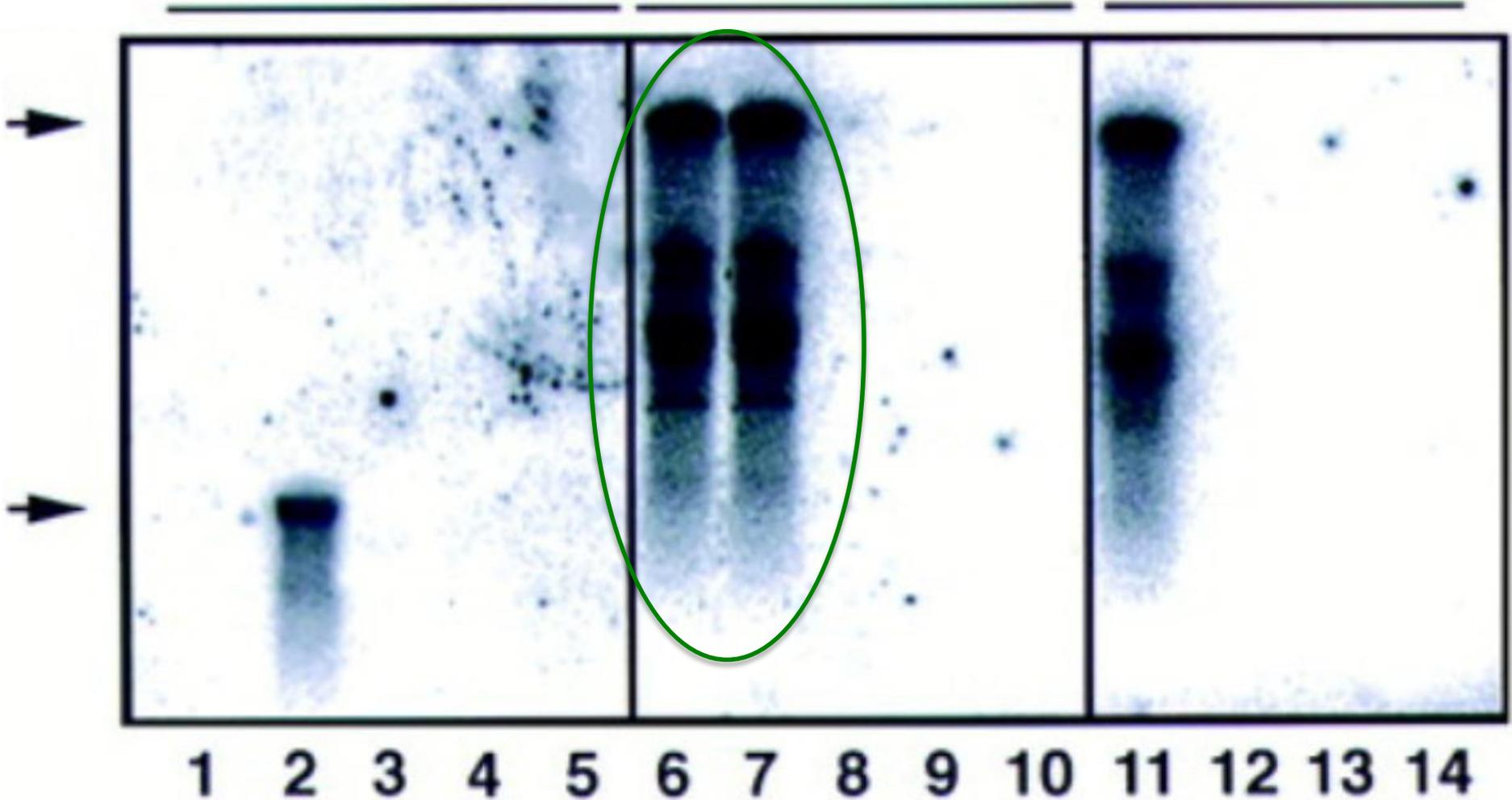


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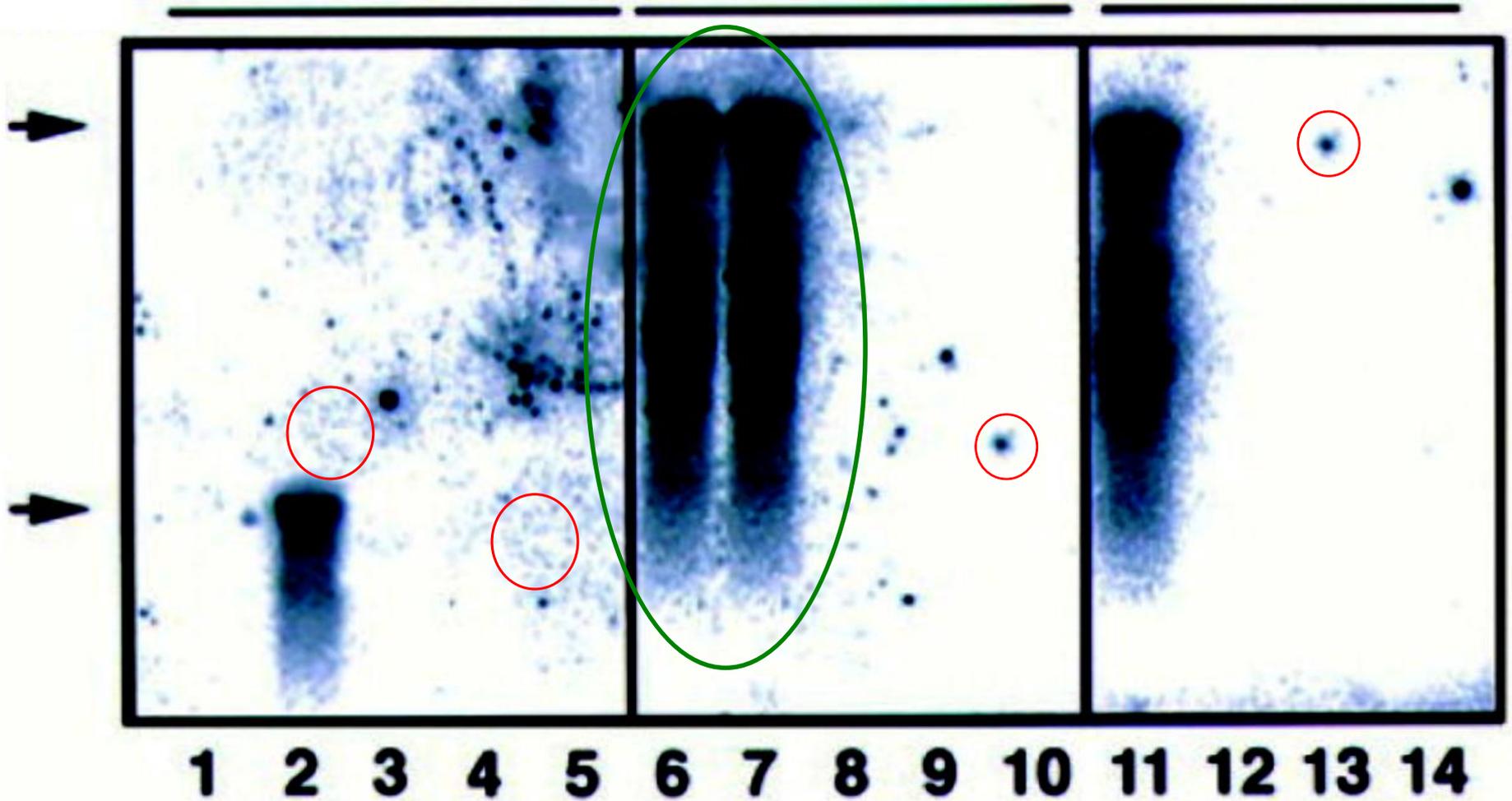
Beautification or Fraud?



Beautification or Fraud?



Fraud with intent



Standardization

Intra-Journal

| EMBO classification | Image Manipulation | Action | % |
|---------------------|--|----------------------------------|-------------|
| I | cosmetic & mistakes; source data & convincing author explanation | Revision No report | 12 |
| II | beautification & undeclared manipulation that changes conclusions; source data or new data | May allow revision May report | 8 |
| III | Undeclared manipulation with obfuscation & intent; no explanation; no source data | Reject and Report | < 0.5% |
| Total | | | 20.5 |

Standardization

- Intra-Journal
- **Cross-Journal**
- **Journal & Research Institution**

| Responsibilities | Res. Institution | Funder | Journal |
|------------------|------------------|--------|---------|
| Quality Control | Yes | Yes | Yes |
| Reporting | Yes | Yes | Yes |
| Sanctions | Yes | Yes | No |

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SourceData | Repositories

SourceData
Making data discoverable

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Systematic analysis of BRAF(V600E) melanomas reveals a role for JNK/c-Jun pathway in adaptive resistance to drug-induced apoptosis.

Fallahi-Sichani M et al. *Molecular systems biology* 2015

Figure 3

Source data
<https://www.ebi.ac.uk/biostudies/studies/S-EPMC4380931-SODA#panel3b>

Intervention
JNK-L...
vemur...

Figure 3-B

Vemurafenib and JNK-IN-8 combination

Assayed
WM115
WM15...
LOXIM...
COLO...

B, C Synergistic apoptosis induction in four cell lines (WM115, WM1552C, LOXIMV1, and COLO858) treated for 72 h with combinations of vemurafenib and JNK-IN-8. (B) Dose-response profiles for apoptosis induction with vemurafenib and JNK-IN-8 combination.

fluorescence microscopy

Figure 3-A Figure 3-B Figure 3-C Figure 3-D Figure 3-E Figure 3-F

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Systematic analysis of BRAF(V600E) melanomas reveals a role for JNK/c-Jun pathway in adaptive resistance to drug-induced apoptosis

Fallahi-Sichani M¹, Moerke NJ¹, Niepel M¹, Zhang T², Gray NS², Sorger PK³

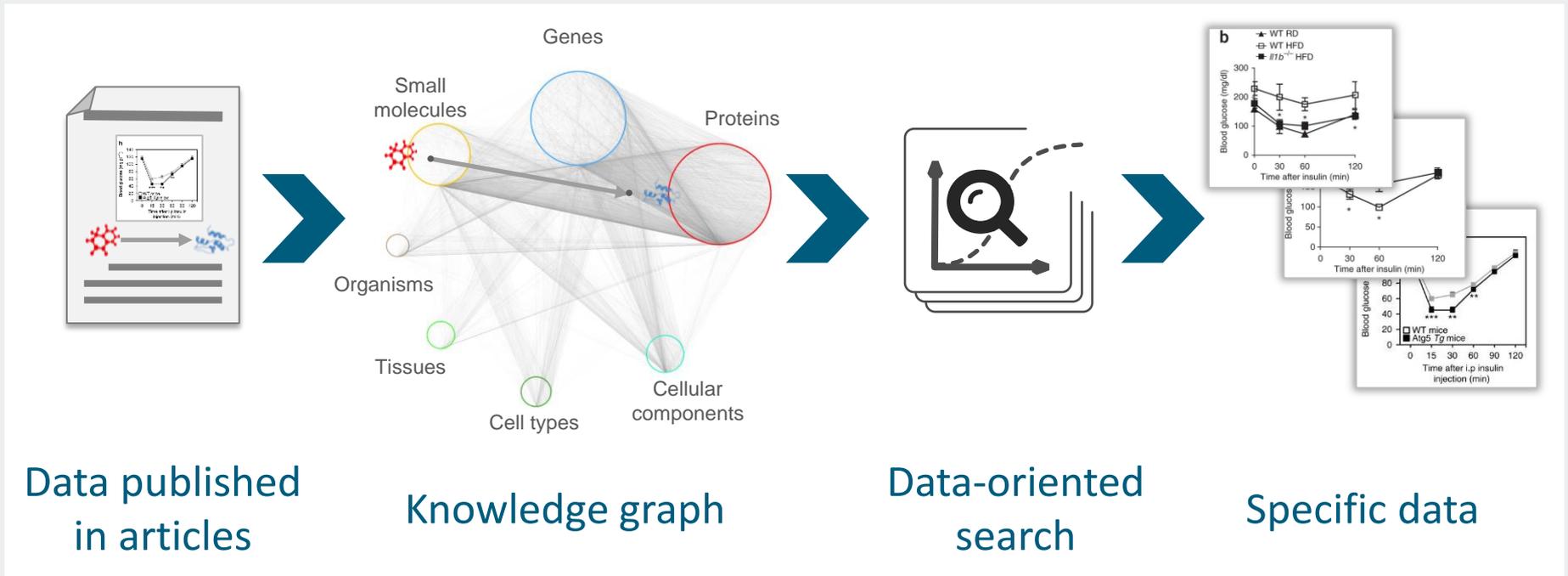
¹HMS LINCS Center, Department of Systems Biology, Harvard Medical School, Boston, MA, USA. ²Department of Cancer Biology, Dana-Farber Cancer Institute, Harvard Medical School, Boston, MA, USA. ³HMS LINCS Center, Department of Systems Biology, Harvard Medical School, Boston, MA, USA. peter_sorger@hms.harvard.edu.

Accession Number
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Enhanced Protocols

bio-protocol

B JoVE Biology

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Imaging the Intracellular Trafficking of APP with Photoactivatable GFP



Joshua H. K. Tam¹, Stephen H. Pasternak^{1,2}

¹Department of Physiology and Pharmacology, Robarts Research Institute, **Western University**,
²Department of Clinical Neurological Sciences, **Western University**

A Screenable *In Vivo* Assay for Mitochondrial Modulators Using Transgenic Bioluminescent *Caenorhabditis elegans*



Cristina Lagido¹, Debbie McLaggan¹, L. Anne Glover¹

¹Institute of Medical Sciences, **University of Aberdeen**

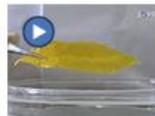
Use of Enzymatic Biosensors to Quantify Endogenous ATP or H₂O₂ in the Kidney



Oleg Palygin¹, Vladislav Levchenko¹, Louise C. Evans¹, Gregory Blass¹, Allen W. Cowley Jr.¹, Alexander Staruschenko¹

¹Department of Physiology, **Medical College of Wisconsin**

Relating Stomatal Conductance to Leaf Functional Traits



Wenzel Kröber¹, Isa Plath¹, Heike Heklau¹, Helge Bruehlheide^{1,2}

¹Institute of Biology / Geobotany and Botanical Garden, **Martin-Luther-University Halle-Wittenberg**, ²German Centre for Integrative Biodiversity Research

Beyond retractions:

Self correction

- Stanford METRICS workshop, 12-2016
- Versioning

| CURRENT OR PROPOSED CATEGORY NAMES |
|--|
| erratum |
| correction corrigendum |
| addendum/clarification |
| version/edition |
| partial retraction, retraction with replacement |
| refutation, matters arising |
| withdrawal |
| retired |
| cancelled |
| self-retraction |
| expression of concern |
| retraction |
| removal |

Fanelli D. et al.,

Research Assessment: Beyond high impact papers

- *High quality, important* data beyond JIF & journal name
- Other contributions: peer review, research support, training

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Declaration on Research Assessment



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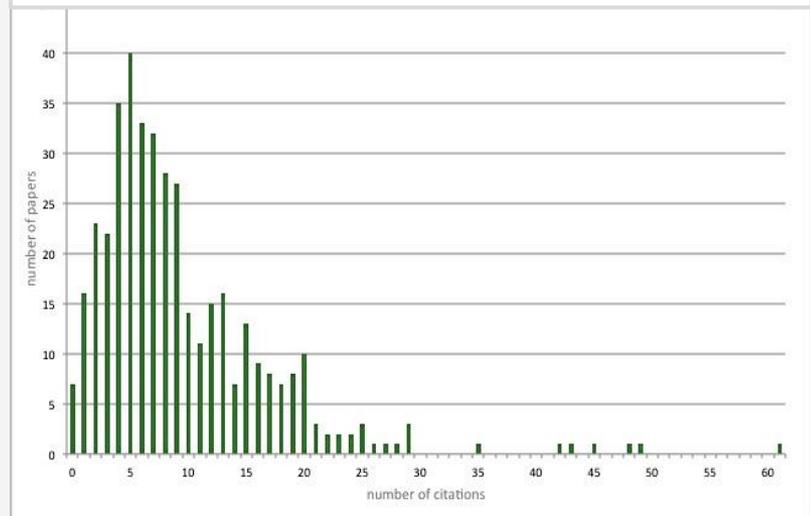
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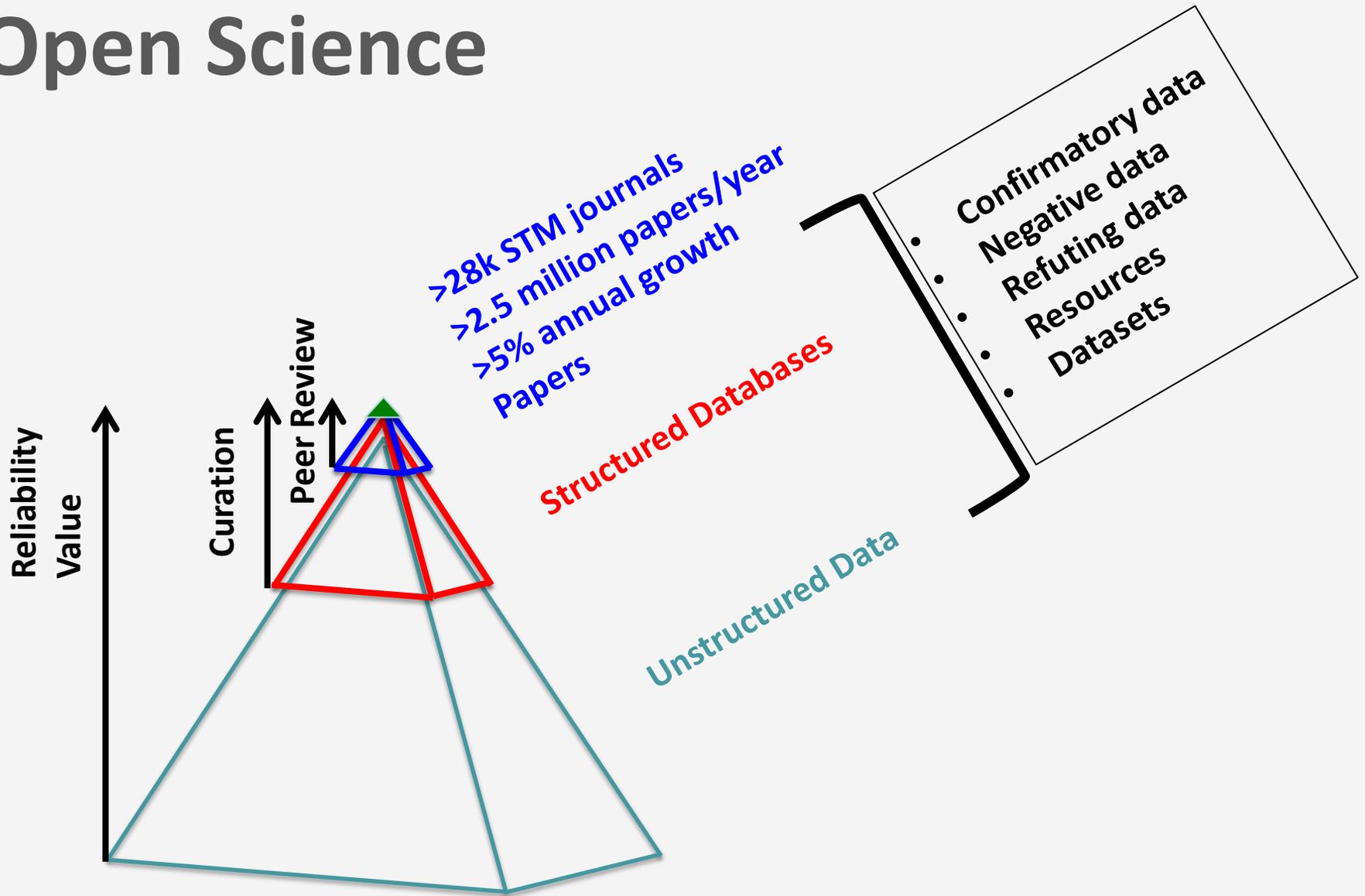
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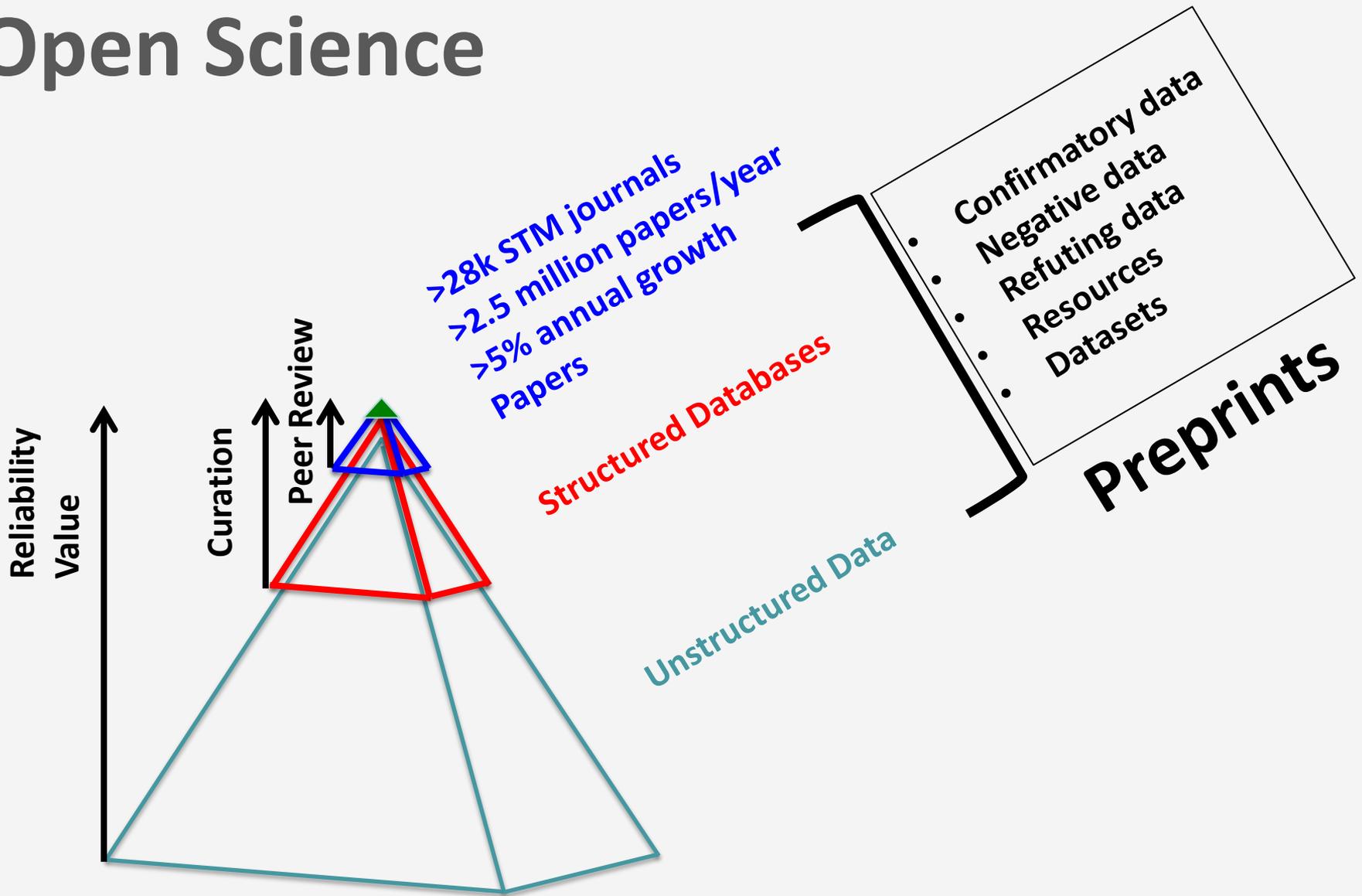
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Training (authors & referees)

EDITORIAL

nature
cell biology

Appreciating data: warts, wrinkles and all

In the glitzy world of Hollywood and Bollywood, each year sees the development of more extravagant digital special effects. Many productions have long since broken the constraints imposed by physics and biology and although the superhuman feats of modern

We hope these guidelines will aid the publication of more informative datasets. Importantly, we reemphasize that neither the referees nor the editors are the data-police (see also *Nature Cell Biology*, 8, 101 (2006)). Senior investigators and corresponding authors are responsible for assuring that data submitted for publication represents the experimental results accurately and fairly. We suggest that they are also responsible for ensuring that their students are educated in appropriate scientific conduct.

2006; doi:10.1038/ncb0306-203a



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Journals are not Data Police



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