

Automating Assessment of Scientific Reproducibility and Responsibility in the Health Sciences

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Reproducibility

Crisis

Billions spent on non-reproducible research.
Science is complicated. Trust in science is challenged.

Causes

Selective reporting, publication pressure, poor statistics,
unavailable methods/code/data, insufficient peer review

Solutions

More robust research design, understanding of statistics,
journals enforcing standards, **transparent reporting**

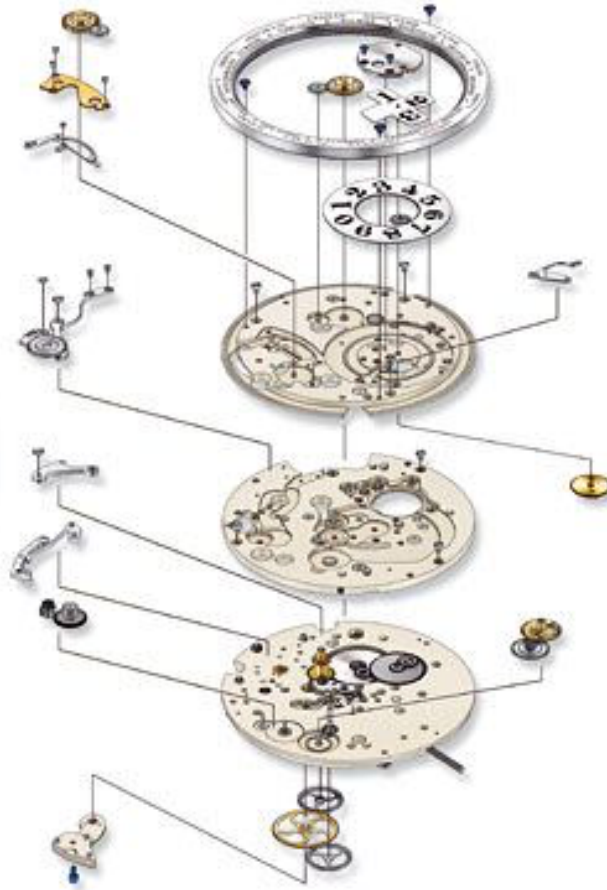
Publication



Methodological Workflow



Responsible Reporting





Guidelines for Reporting



400

different sets

Article Reviews



Peer review
can take
6 months
to over a year

Improving
scientific reproducibility
with
minimal burden
on scientists
and those evaluating research

Measuring Reproducibility and Scientific Impact

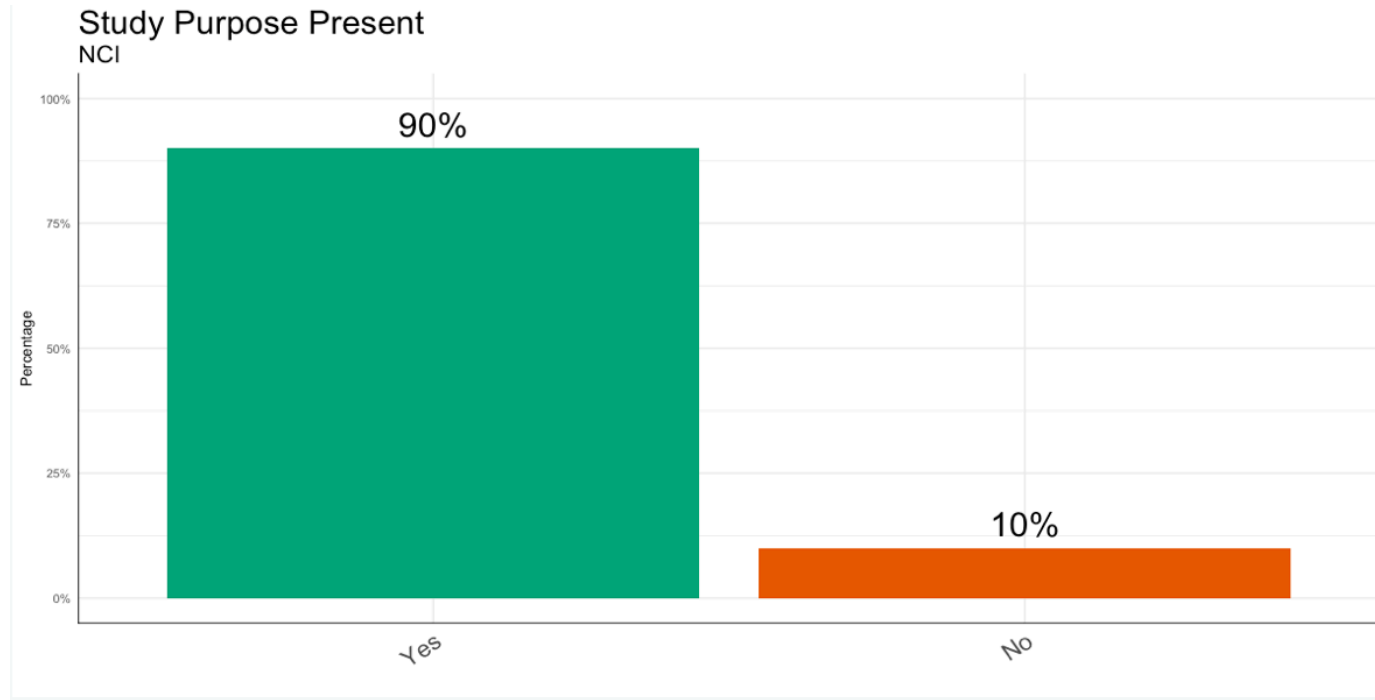
Detecting causes of non-reproducibility

Defining, measuring, and predicting the potential reproducibility of a single publication.

Monitoring impacts of non-reproducibility

Tracking the scientific gain and impact of a publication as moderated by the science's reproducibility.

Study Overview: NCI-funded* publications



*based on an analysis of 10 articles which have been funded by the NCI Moonshot project

Study Overview: Sufficient

Our aim was to demonstrate that identification of dMMR with clinically available assays has limitations, and we hypothesized that a multipronged approach is necessary to adequately stratify mCRPC patients who could potentially benefit from immunotherapy with immune checkpoint–blocking drugs.

Article Title: Immunogenomic analyses associate immunological alterations with mismatch repair defects in prostate cancer

Article DOI: [10.1172/jci121924](https://doi.org/10.1172/jci121924)

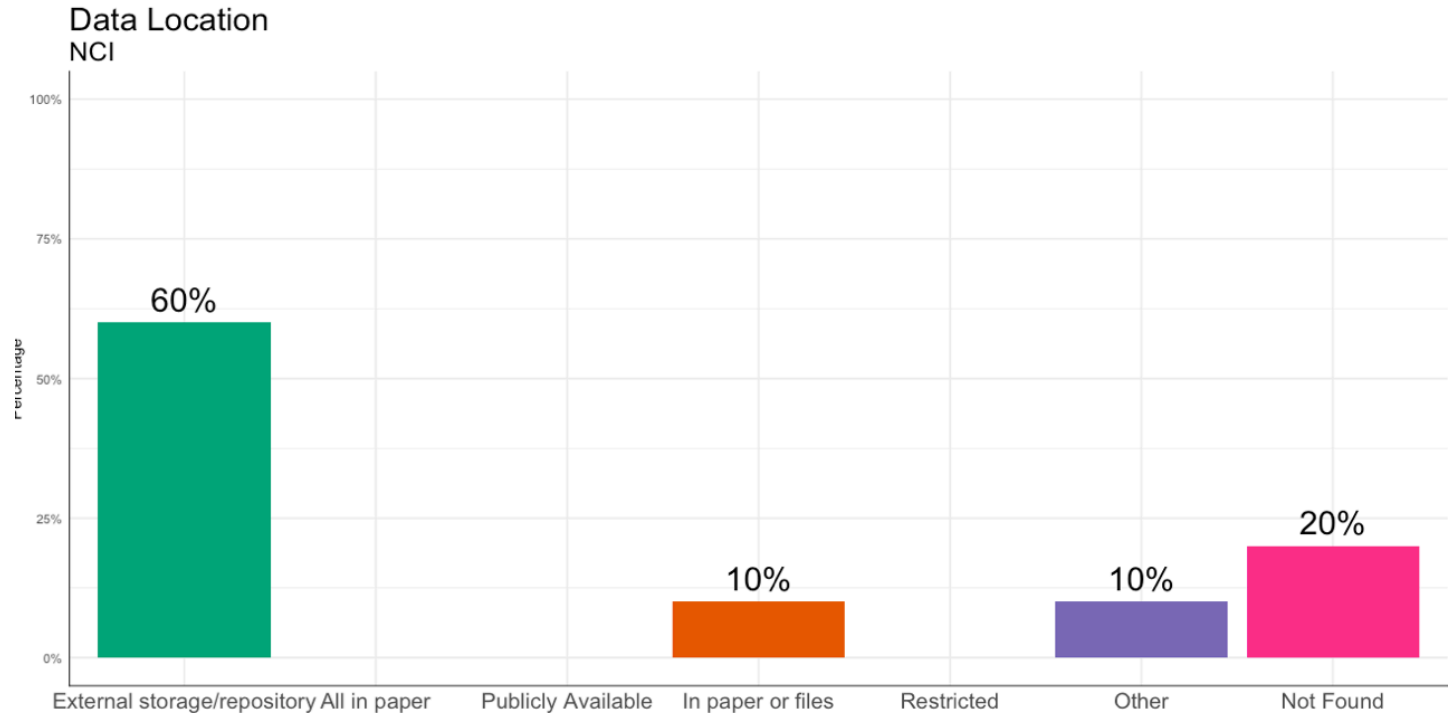
Study Overview: Needs Improvement

To this end, a number of bromodomain inhibitors, including JQ1 and I-BET, have been developed and have shown promising outcomes in early clinical trials. Although resistance to BET inhibitors has been documented in preclinical models^{13–15}, the molecular mechanisms underlying acquired resistance are largely unknown. Here we report that cullin-3SPOP earmarks BET proteins, including BRD2, BRD3 and BRD4, for ubiquitination-mediated degradation.

Article Title: Prostate cancer–associated SPOP mutations confer resistance to BET inhibitors through stabilization of BRD4

Article DOI: 10.1038/nm.4378

Data Sharing: NCI-funded* publications



*based on an analysis of 10 articles which have been funded by the NCI Moonshot project

Data Sharing: External Repository

The RNA-seq and ERRBS data generated during the current study are available through **Gene Expression Omnibus (GEO) accession number: GSE112830** with the following sub-series: <https://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE112786>, <https://www.ncbi.nlm.nih.gov/geo/query/acc.cgi?acc=GSE112829>. The whole exome sequencing data related to this study are available through **Sequence Read Archive (SRA) with accession number SRP138000**. The published human data are available through dbGap:phs000909.v.p1 (http://www.cbioportal.org/study?id=nepc_wcm_2016)6..

Article Title: Patient derived organoids to model rare prostate cancer phenotypes

Article DOI: 10.1038/s41467-018-04495-z

Data Sharing: In Paper or Files

*“...supplemental material available online with this article...
Detailed methods are described in the Supplemental
Methods.”*

Article Title: COP1-DET1-ETS axis regulates ERK transcriptome and sensitivity to MAPK inhibitors

Article DOI: [10.1172/jci94840](https://doi.org/10.1172/jci94840)

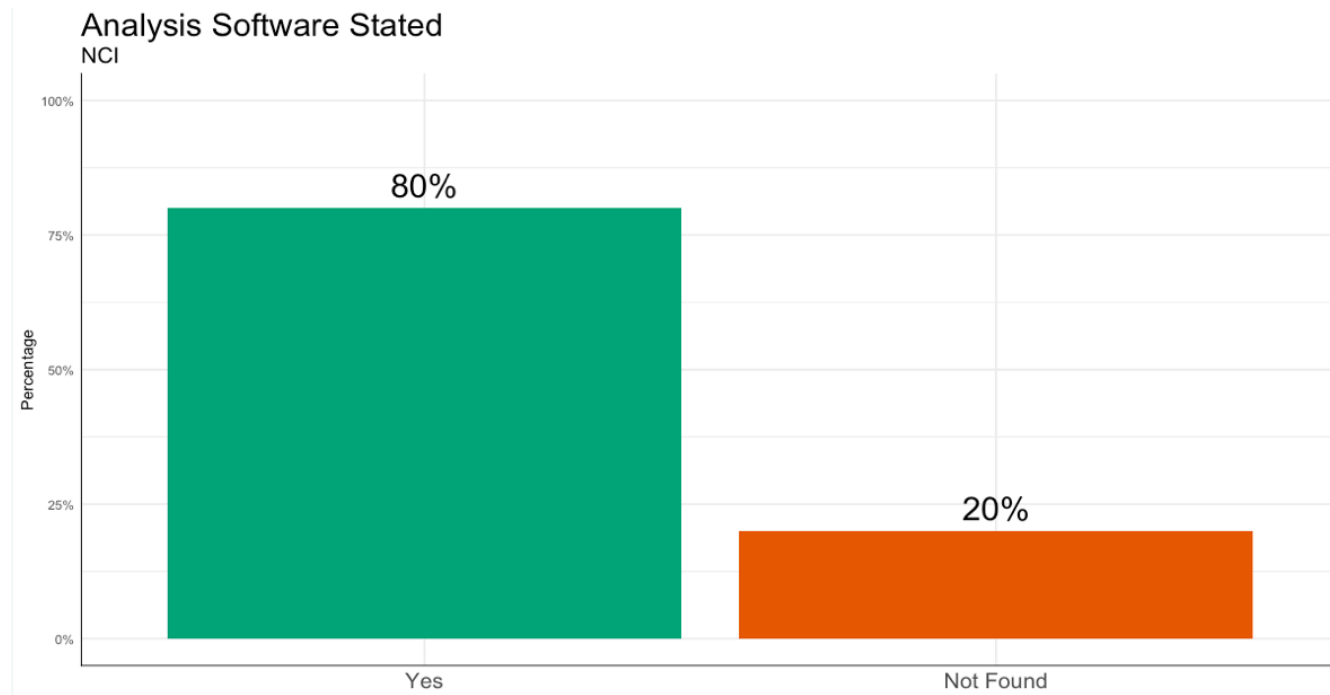
Data Sharing: Other

Uncropped images for immunoblots are available in Supplementary Figures 11–14. All other relevant data are available from the corresponding author upon reasonable request.

Article Title: Prostate cancer–associated SPOP mutations confer resistance to BET inhibitors through stabilization of BRD4

Article DOI: [10.1038/nm.4378](https://doi.org/10.1038/nm.4378)

Analyses: NCI-funded* publications



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Data Analyses: Sufficient

For Cox regression analyses, the primary aim was to determine whether tumor CNA burden is associated with cancer specific survival (CSS). First, we assessed whether there was an association between tumor CNA burden and CSS by utilizing a univariate Cox model, censoring patients who did not die at the date of their last follow-up and patients who died of other causes at their death date. Secondly, in order to assess whether there is information from tumor CNA burden over and above biopsy Gleason score, we utilized a multivariable Cox model, adjusting for biopsy Gleason sum categorized as ≤ 6 , 7, and ≥ 8 . Finally, to assess whether there is an association between tumor CNA burden and CSS after accounting for the preoperative predictors of CSS, we utilized a multivariable Cox model...

Article Title: Tumor copy number alteration burden is a pan-cancer prognostic factor associated with recurrence and death

Article DOI: [10.7554/elife.372941](https://doi.org/10.7554/elife.372941)

Note: The article provided both the how the data were collected as well as analyzed.

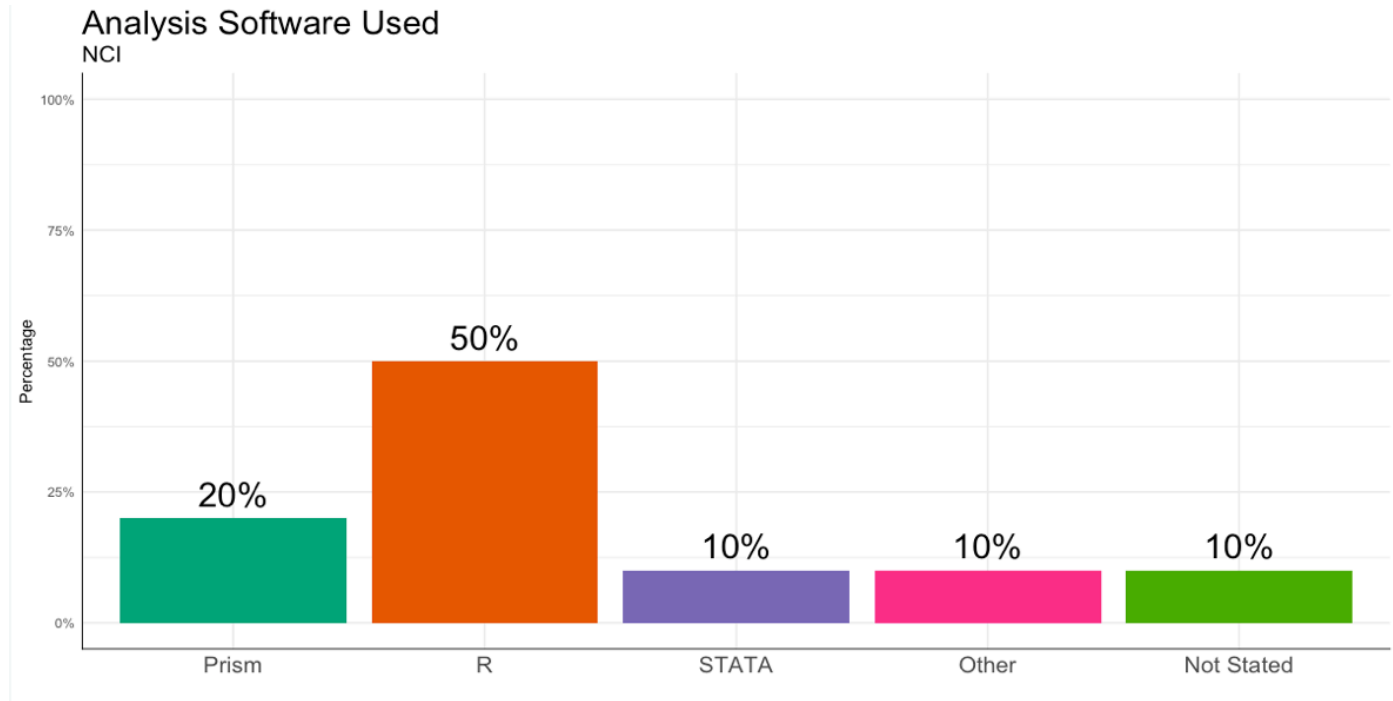
Data Analyses: Needs Improvement

All statistical comparisons between two groups were performed by GraphPad Prism software 6.0 using a two-tailed unpaired t-test. The variance between the statistically compared groups was similar.

Article Title: Aberrant Activation of a Gastrointestinal Transcriptional Circuit in Prostate Cancer Mediates Castration Resistance

Article DOI: [10.1016/j.ccell.2017.10.008](https://doi.org/10.1016/j.ccell.2017.10.008)

Software: NCI-funded* publications



*based on an analysis of 10 articles which have been funded by the NCI Moonshot project

Software: Sufficient

Graphs were generated by using **Graphpad Prism 5 project** (Graphpad Software Inc, CA, USA) or Microsoft Office Excel 2010... Differences between groups were compared by unpaired **t-tests or Wilcoxon rank sum test** with continuity correction by **R software version 2.15.0** (<http://www.r-project.org>).

Article Title: Dual inhibition of AKT-mTOR and AR signaling by targeting HDAC3 in PTEN- or SPOP-mutated prostate cancer

*based on an analysis of 10 articles which have been funded by the NCI Moonshot project
Article DOI: 10.15252/emmm.201708478

Code: Needs Improvement

No code was provided.

Article Title: Challenges in validating candidate therapeutic targets in cancer

Article DOI: 10.7554/elife.32402



Researchers

Rapidly check your pre-print and published manuscripts to improve the transparency of reporting your research



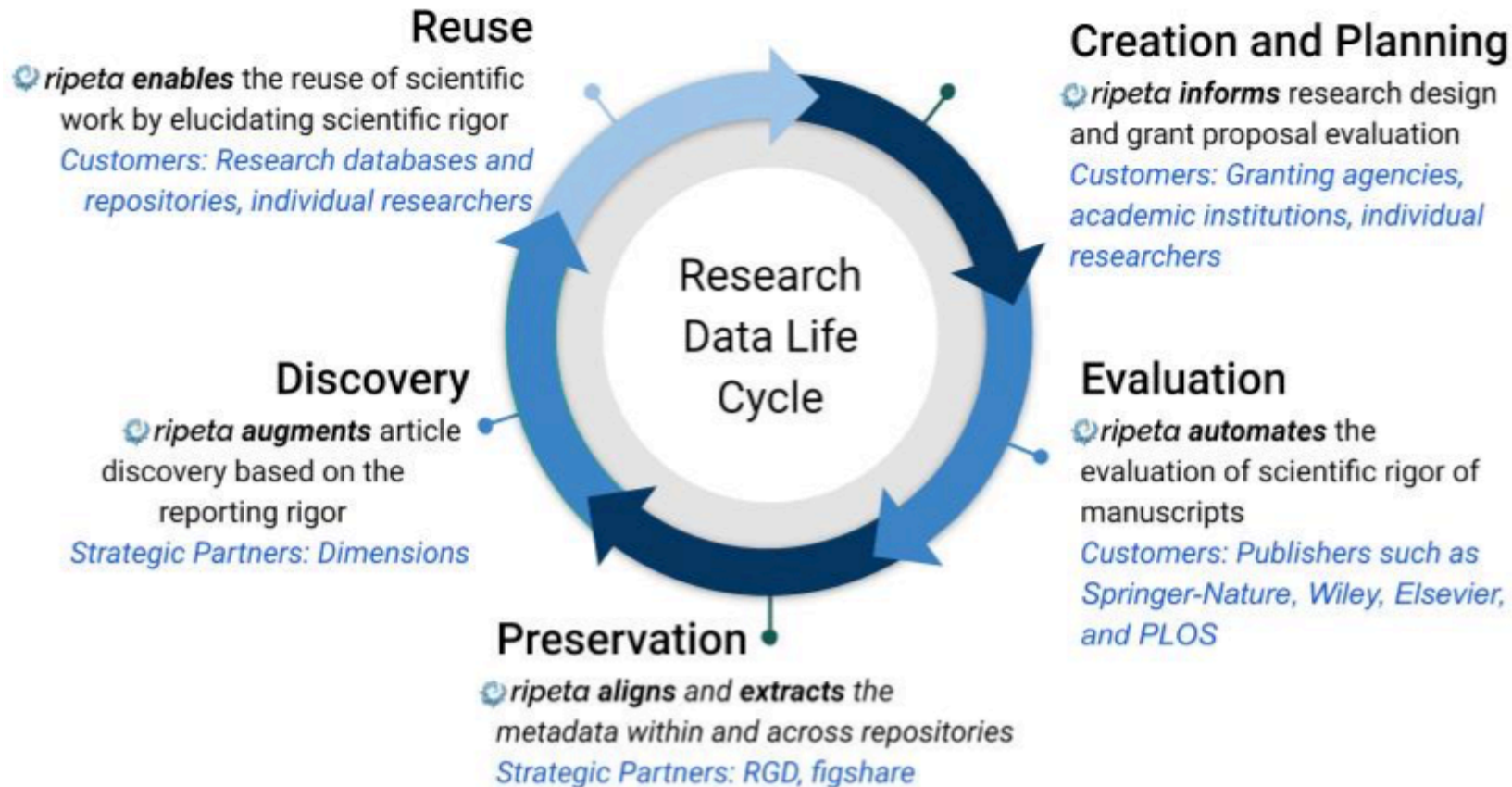
Funders

Evaluate your portfolio for robust scientific reporting by checking publications on your projects

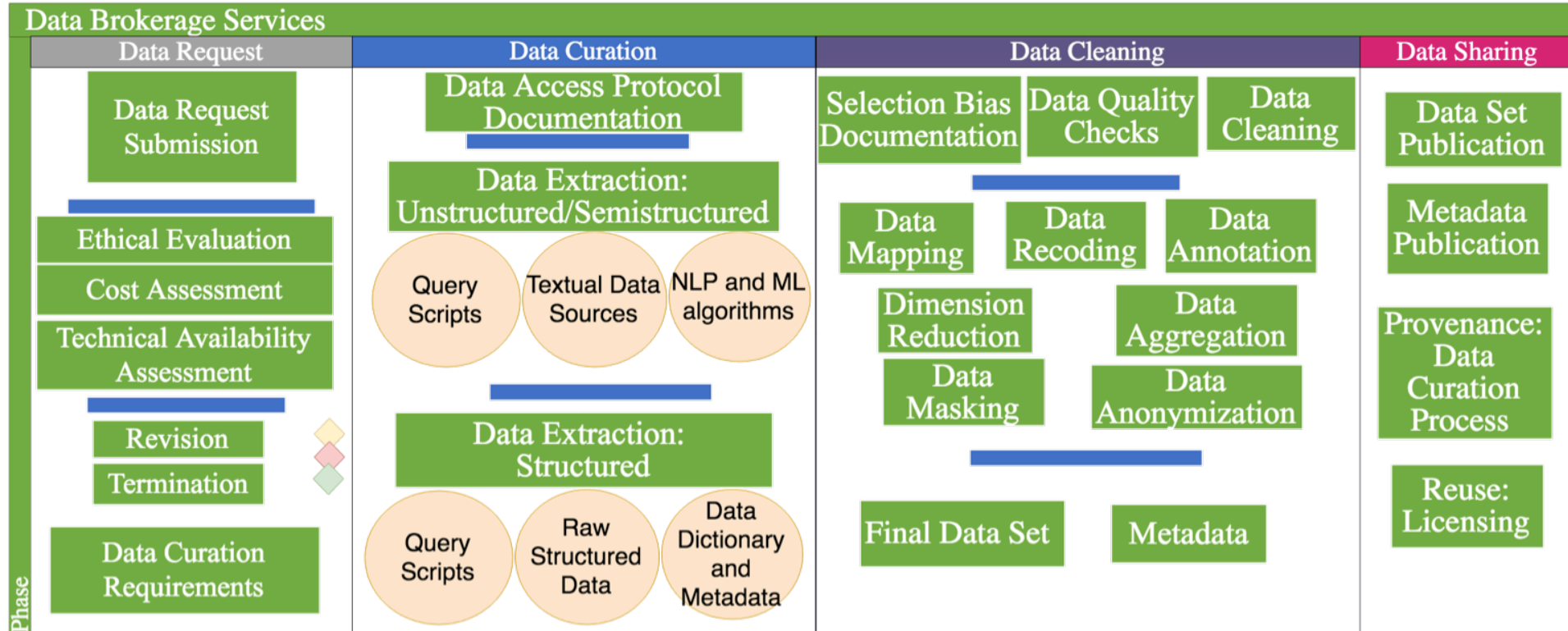


Publishers

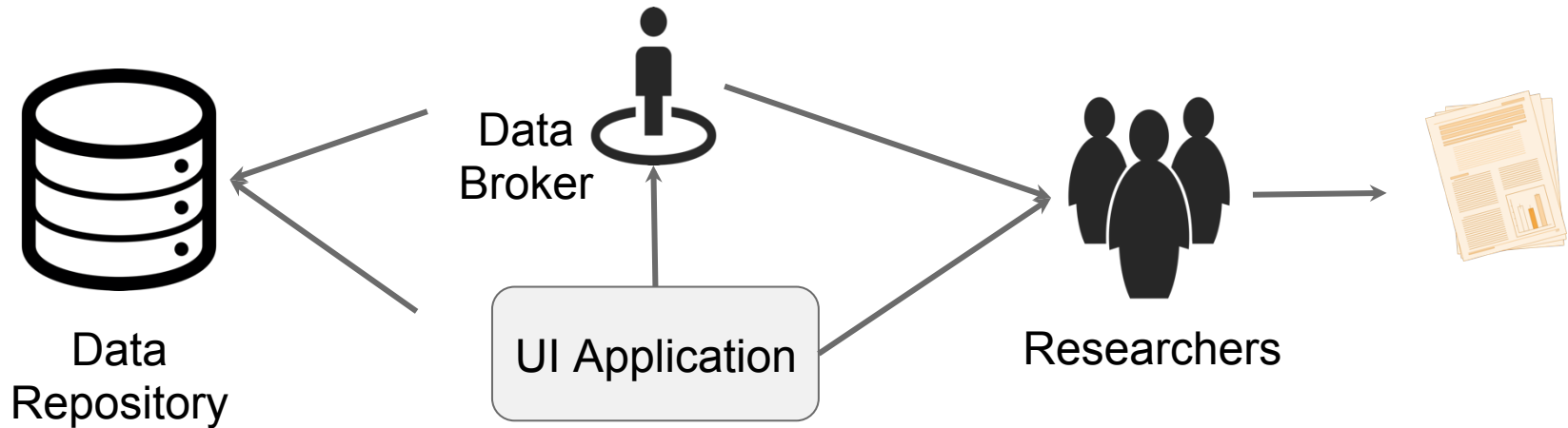
Improve the reproducibility of articles during the peer-review process with an automated tool that supports evidence-based science



Reproducible Health Data Workflows



Research Data as a Service



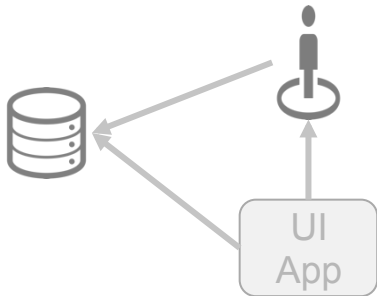
Publications as a Product



Researchers



Publications



Reproducible Scientific Workflows

“Reproducibility implies repetition and thus a requirement to also move back – to retrace one’s steps, question or change assumptions, and move forward again.”

Millman, K. J., & Perez, F. (2014). Developing Open-Source Scientific Practice (V. Stodden, F. Leisch, & R. D. Peng, Eds.). In Implementing Reproducible Research (CRC the R series, pp. 149-183). Boca Raton, FL: Taylor & Francis Group, LLC.

The Grand Why

“The construction of a
scientific heritage

where anyone can validate the work of
others and build upon it.”¹

Millman, K. J., & Perez, F. (2014). Developing Open-Source Scientific Practice (V. Stodden, F. Leisch, & R. D. Peng, Eds.). In Implementing Reproducible Research (CRC the R series, pp. 149-183). Boca Raton, FL: Taylor & Francis Group, LLC.

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