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Open Access: Achievements & Challenges

Christian Gutknecht, Main Library University of Zurich

Opendata.ch 2012 Conference
28.6.2012, Zurich

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guardian.co.uk, Friday 8 June 2012 18.58 BST
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Science
Open access scientific publishing · Peer review and scientific publishing

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http://www.guardian.co.uk/science/2012/jun/08/open-access-research-inevitable-nature-editor

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Alok Jha, science correspondent
guardian.co.uk, Tuesday 19 June 2012 07.00 BST
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Ian Sample, science correspondent
guardian.co.uk, Tuesday 24 April 2012 17.45 BST
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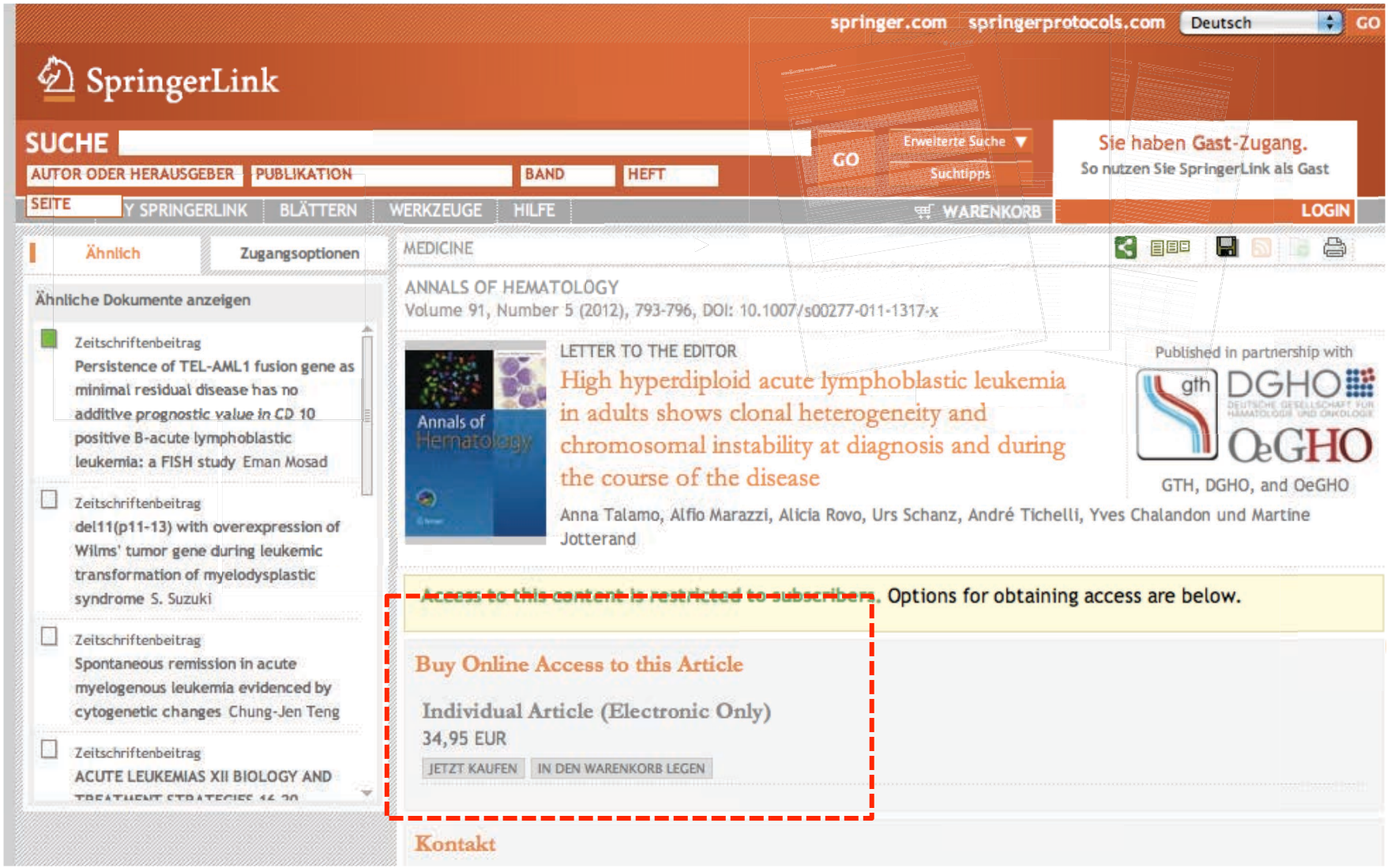
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Spontaneous remission in acute myelogenous leukemia evidenced by cytogenetic changes Chung-Jen Teng
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Volume 91, Number 5 (2012), 793-796, DOI: 10.1007/s00277-011-1317-x
Annals of Hematology
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High hyperdiploid acute lymphoblastic leukemia in adults shows clonal heterogeneity and chromosomal instability at diagnosis and during the course of the disease
Anna Talamo, Alfio Marazzi, Alicia Rovo, Urs Schanz, André Tichelli, Yves Chalandon und Martine Jotterand
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The Open Knowledge Foundation: Open Data Means Better Science

Article Metrics Related Content Comments: 0

Jennifer C. Molloy*

Department of Zoology, University of Oxford, Oxford, United Kingdom

Citation: Molloy JC (2011) The Open Knowledge Foundation: Open Data Means Better Science. PLoS Biol 9(12): e1001195. doi:10.1371/journal.pbio.1001195

Published: December 6, 2011

Funding: The author received no specific funding for this work.

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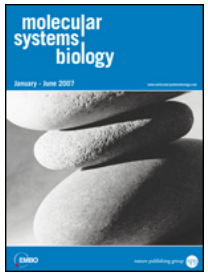

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> Open access contributions include **original scientific research results, raw data** and metadata, source materials, digital representations of pictorial and graphical materials and scholarly multimedia material.

http://oa.mpg.de/lang/de/berlin-prozess/berliner-erklarung/

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Guidelines Swiss National Science Foundation (since 2007)

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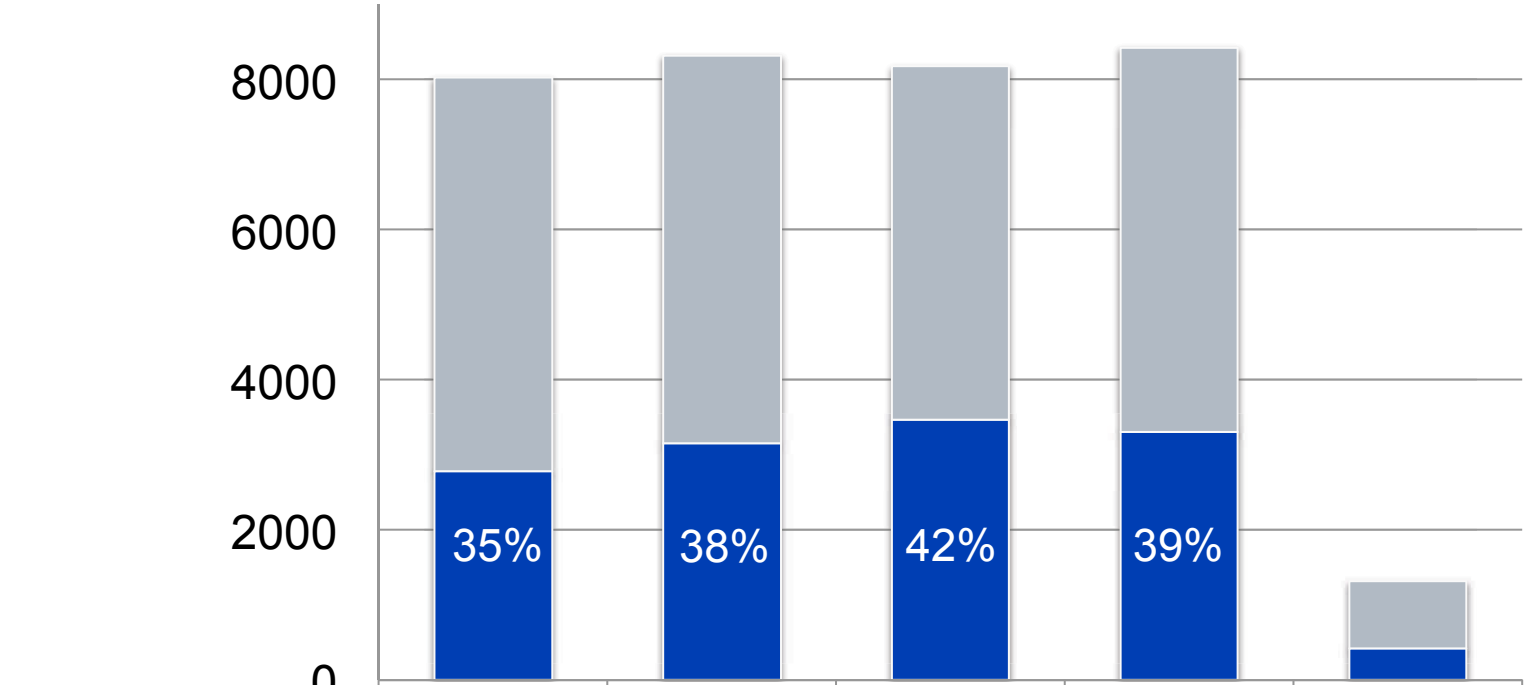

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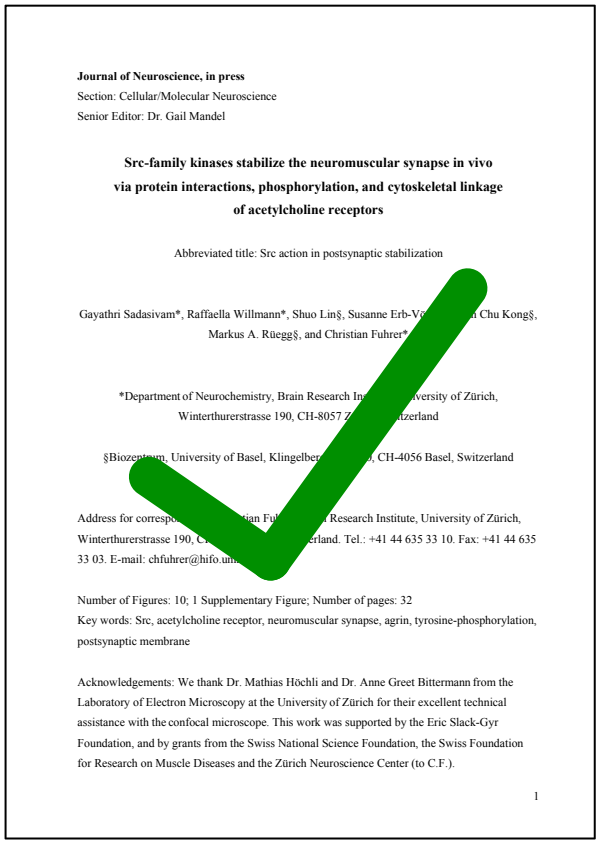

Journal of Neuroscience, in press
Section: Cellular/Molecular Neuroscience
Senior Editor: Dr. Gail Mandel

Src-family kinases stabilize the neuromuscular synapse in vivo via protein interactions, phosphorylation, and cytoskeletal linkage of acetylcholine receptors

Abbreviated title: Src action in postsynaptic stabilization

Gayathri Sadasivam*, Raffaella Willmann*, Shuo Lin§, Susanne Erb-Vö… Chu Kong§, Markus A. Rüegg§, and Christian Fuhrer*

*Department of Neurochemistry, Brain Research In… iversity of Zürich, Winterthurerstrasse 190, CH-8057 Z… tzerland

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Number of Figures: 10; 1 Supplementary Figure; Number of pages: 32
Key words: Src, acetylcholine receptor, neuromuscular synapse, agrin, tyrosine-phosphorylation, postsynaptic membrane

Acknowledgements: We thank Dr. Mathias Höchli and Dr. Anne Greet Bittermann from the Laboratory of Electron Microscopy at the University of Zürich for their excellent technical assistance with the confocal microscope. This work was supported by the Eric Slack-Gyr Foundation, and by grants from the Swiss National Science Foundation, the Swiss Foundation for Research on Muscle Diseases and the Zürich Neuroscience Center (to C.F.).

1

Accepted manuscript (Post-Print)

Cellular/Molecular

Src-Family Kinases Stabilize the Neuromuscular Synapse *In Vivo* via Protein Interactions, Phosphorylation, and Cytoskeletal Linkage of Acetylcholine Receptors

Gayathri Sadasivam,[1] Raffaella Willmann,[1] Shuo Lin,[2] Susanne Erb-Vögtli,[1] Xian Chu Kong,[2] Markus A. Rüegg,[2] and Christian Fuhrer[1]
[1]Department of Neurochemistry, Brain Research Institute, University of Zürich, CH-8057 Zürich, Switzerland, and [2]Biozentrum, University of Basel, CH-4056 Basel, Switzerland

Postnatal stabilization and maturation of the postsynaptic membrane are important for development and function of the neuromuscular junction (NMJ), but the underlying mechanisms remain poorly characterized. We examined the role of Src-family kinases (SFKs) *in vivo*. Electroporation of kinase-inactive Src constructs into soleus muscles of adult mice caused NMJ disassembly: acetylcholine receptor (AChR)-rich areas became fragmented; the topology of nerve terminal, AChRs, and synaptic nuclei was disturbed; and occasionally nerves started to sprout. Electroporation of kinase-overactive Src produced similar but milder effects. We studied the mechanism of SFK action using cultured *src*$^{-/-}$*fyn*$^{-/-}$ myotubes, focusing on clustering of postsynaptic proteins, their interaction with AChRs, and AChR phosphorylation. Rapsyn and the utrophin-glycoprotein complex were recruited normally into AChR-containing clusters by agrin in *src*$^{-/-}$*fyn*$^{-/-}$ myotubes. But after agrin withdrawal, clusters of these proteins disappeared rapidly in parallel with AChRs, revealing that SFKs are of general importance in postsynaptic stability. At the same time, AChR interaction with rapsyn and dystrobrevin and AChR phosphorylation decreased after agrin withdrawal from mutant myotubes. Unexpectedly, levels of rapsyn protein were increased in *src*$^{-/-}$*fyn*$^{-/-}$ myotubes, whereas rapsyn–cytoskeleton interactions were unaffected. The overall cytoskeletal link of AChRs was weak but still strengthened by agrin in mutant cells, consistent with the normal formation but decreased stability of AChR clusters. These data show that correctly balanced activity of SFKs is critical in maintaining adult NMJs *in vivo*. SFKs hold the postsynaptic apparatus together through stabilization of AChR–rapsyn interaction and AChR phosphorylation. In addition, SFKs control rapsyn levels and AChR-cytoskeletal linkage.

Key words: Src; acetylcholine receptor; neuromuscular synapse; agrin; tyrosine phosphorylation; postsynaptic membrane

Introduction

Neuromuscular junctions (NMJs) develop in a series of steps in which the postsynaptic membrane first forms by concentrating acetylcholine receptors (AChRs) and associated proteins in a flat topology. Postnatally, NMJs mature, and AChRs get arranged at the crests of postjunctional folds. Concomitantly, all but one axon withdrew, paralleled by destabilization of adjacent AChRs (Sanes and Lichtman, 2001). Maturation and stabilization of AChR clusters ensure proper synaptic development, which forms the basis for nerve-evoked muscle contractibility.

Much is known about the molecular pathways that first form NMJs. Neural agrin, by activating the muscle-specific kinase (MuSK), is crucial by triggering downstream cascades (for re-

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This work was supported by the Eric Slack-Gyr Foundation and by grants from the Swiss National Science Foundation, the Swiss Foundation for Research on Muscle Diseases, and the Zürich Neuroscience Center (C.F.). We thank Drs. Mathias Höchli and Anne Greet Bittermann (Laboratory of Electron Microscopy, University of Zürich) for their excellent technical assistance with the confocal microscope.
Correspondence should be addressed to Christian Fuhrer, Brain Research Institute, University of Zürich, Winterthurerstrasse 190, CH-8057 Zürich, Switzerland. E-mail: chfuhrer@hifo.unizh.ch.
DOI:10.1523/JNEUROSCI.2103-05.2005
 0270-6474/05/2510479-15$15.00/0

…ow, see Bezakova and Ruegg, 2003; Luo et al., 2003). Central in this is rapsyn, the main AChR-anchoring protein mediating clustering (Gautam et al., 1995). Rapsyn increasingly binds to AChRs in response to agrin (Moransard et al., 2003), mediates agrin-induced phosphorylation of the AChR β and δ subunits (Mittaud et al., 2001), and links the receptor to β-dystroglycan, a component of the postsynaptic utrophin-glycoprotein complex (UGC) (Cartaud et al., 1998; Bartoli et al., 2001). In clustering, AChRs become immobilized and less detergent extractable, both in agrin-treated myotubes (Prives et al., 1982; Stya and Axelrod, 1983; Podleski and Salpeter, 1988) and developing NMJs (Dennis, 1981; Slater, 1982). The players in this cytoskeletal link remain uncertain. Agrin-induced phosphorylation of AChR β is involved (Borges and Ferns, 2001) and can occur through Abl- and Src-family kinases (SFKs) (Finn et al., 2003; Mittaud et al., 2004).

Much less is known about the mechanisms that mature NMJs and stabilize AChR clusters postnatally. Although MuSK is required (Kong et al., 2004), some of these pathways may not be essential in initial NMJ formation (Willmann and Fuhrer, 2002), as illustrated by mice lacking utrophin and dystrophin or the UGC components α-dystrobrevin or dystroglycan (Grady et al.,

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Image: Charles Le Brun, 1760

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Assumed conflicts with publishers and editors

> Ich fürchte schlicht Komplikationen mit den jeweiligen Herausgebern, die ich ja nicht gefragt habe. Ein gutes Verhältnis zu denen ist mir aber wichtig, und das möchte ich nicht aufs Spiel setzen.
>
> Seminarleiter, April 2012

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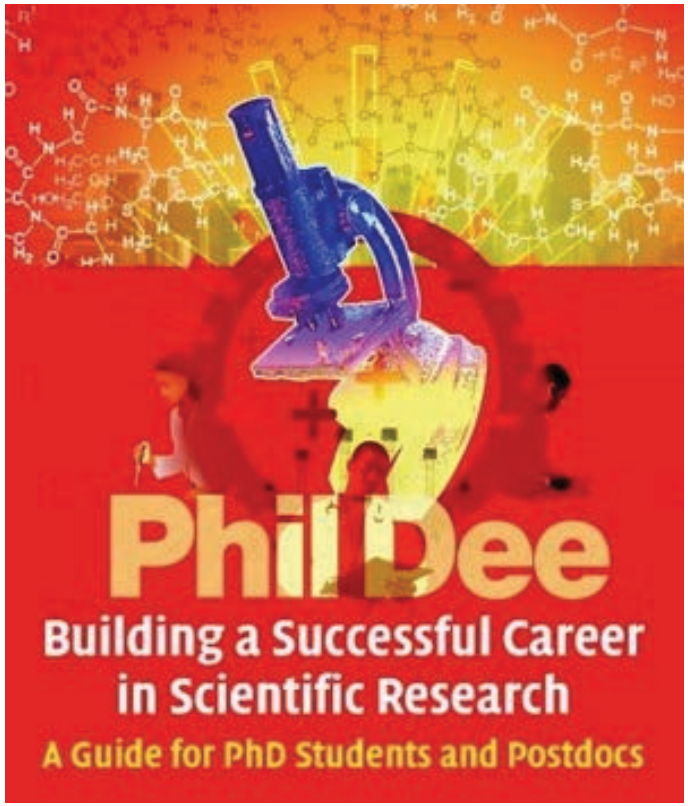

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No interest & Ignorance of guidelines

> Danke für Ihre freundliche Frage. Ich verzichte auf die Präsentation in ZORA.
> Danke und mit den besten Grüssen
>
> Professorin, März 2012

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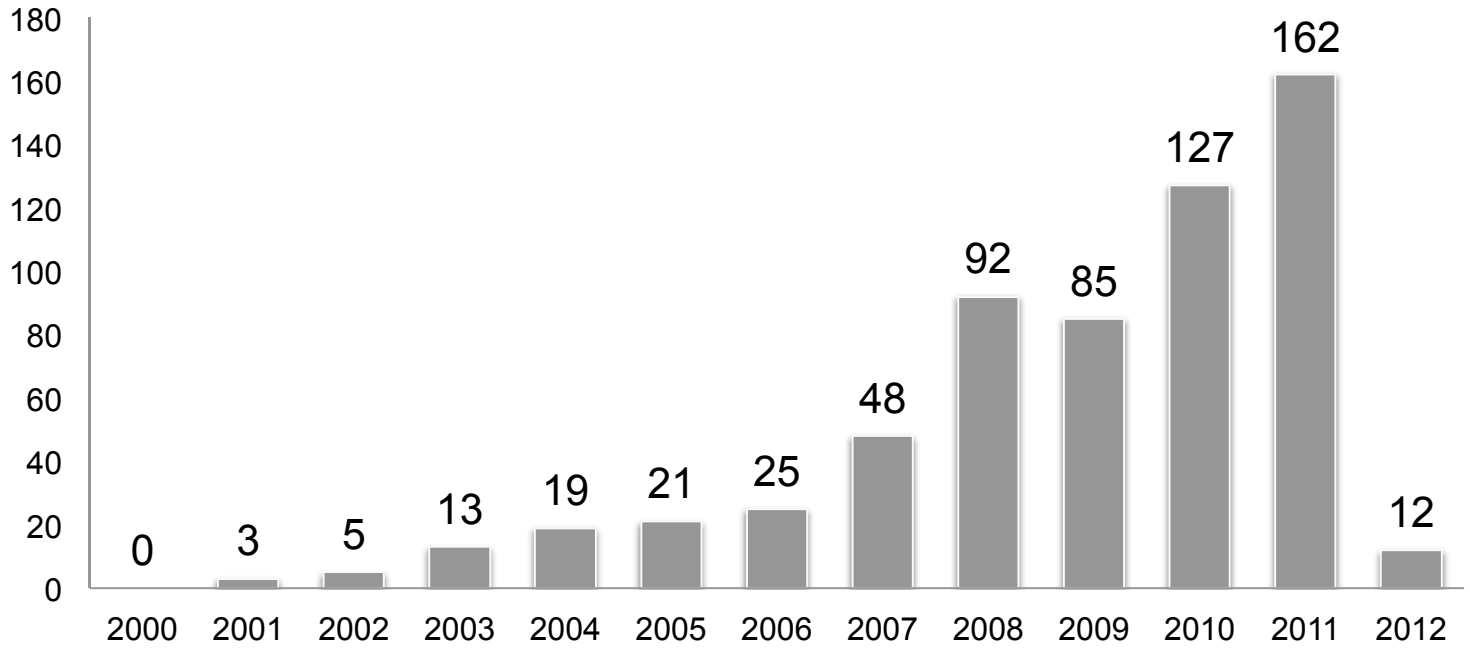

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Year 2011

- Open Access funding: 162‘000 CHF

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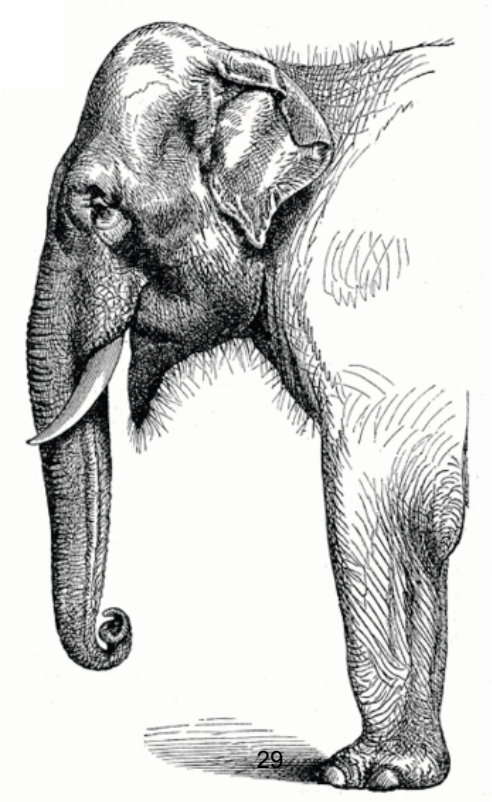

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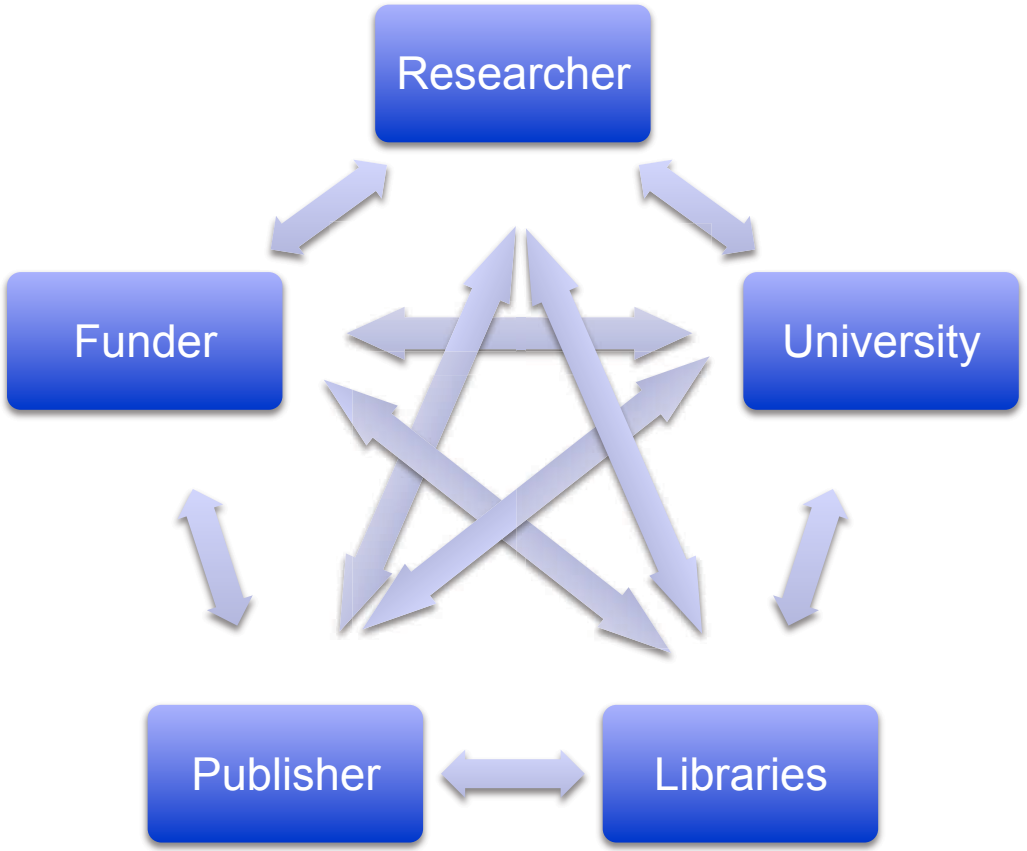
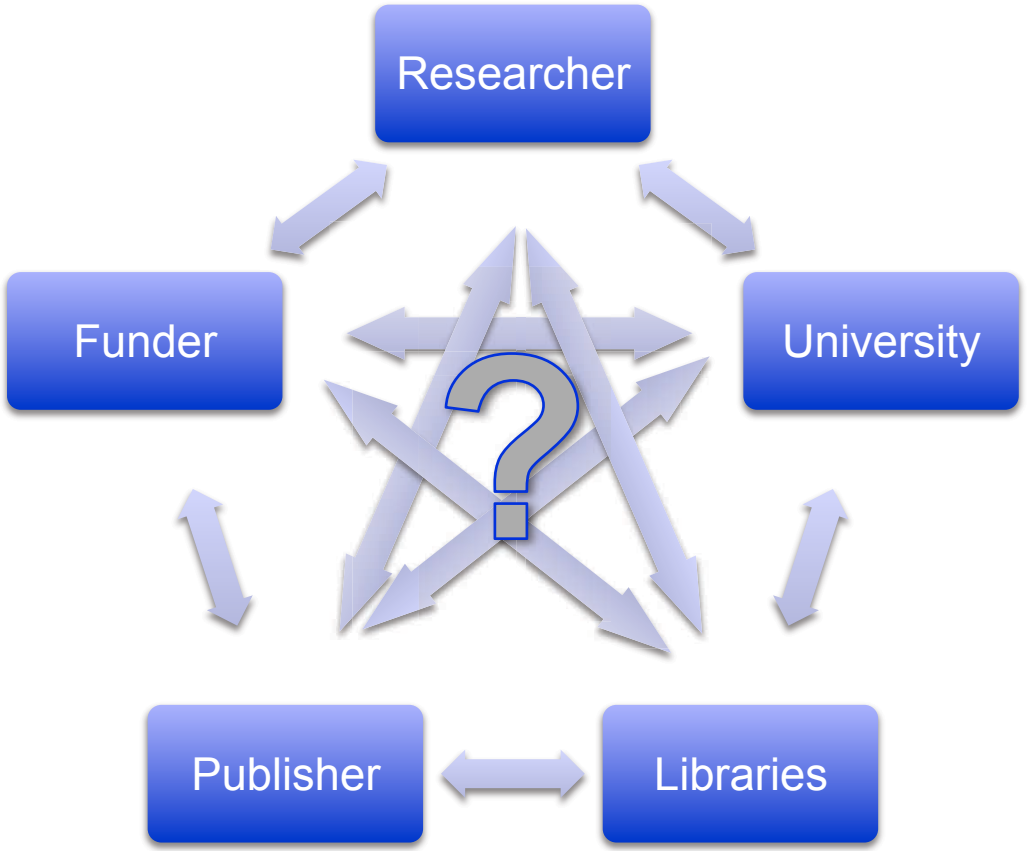
Challenge: Who can do what?



Who is doing the coordination?



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Main Library University of Zurich is on of 41 project partners in the EU-Project:

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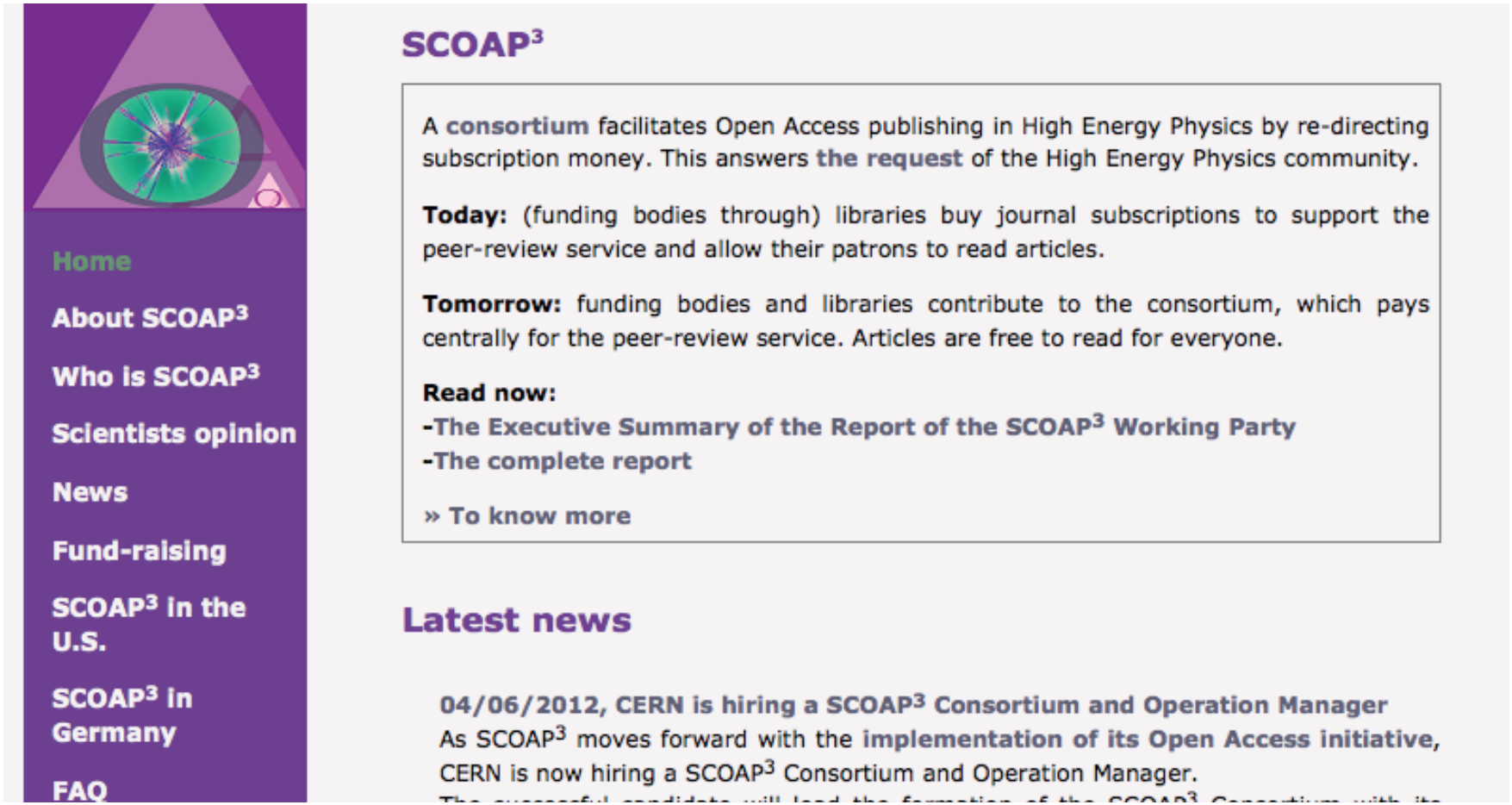
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As SCOAP³ moves forward with the implementation of its Open Access initiative, CERN is now hiring a SCOAP³ Consortium and Operation Manager.

Summary

- Open Access is a proven and solid business model
- It works for top journals
- Open Access is growing slowly, but constantly
- 100% Open Access is not expected to cost less, but there is added value.

- … Open Access to research data is an upcoming topic

BACKUP-SLIDES

Open Access Infrastructure for Research in Europe

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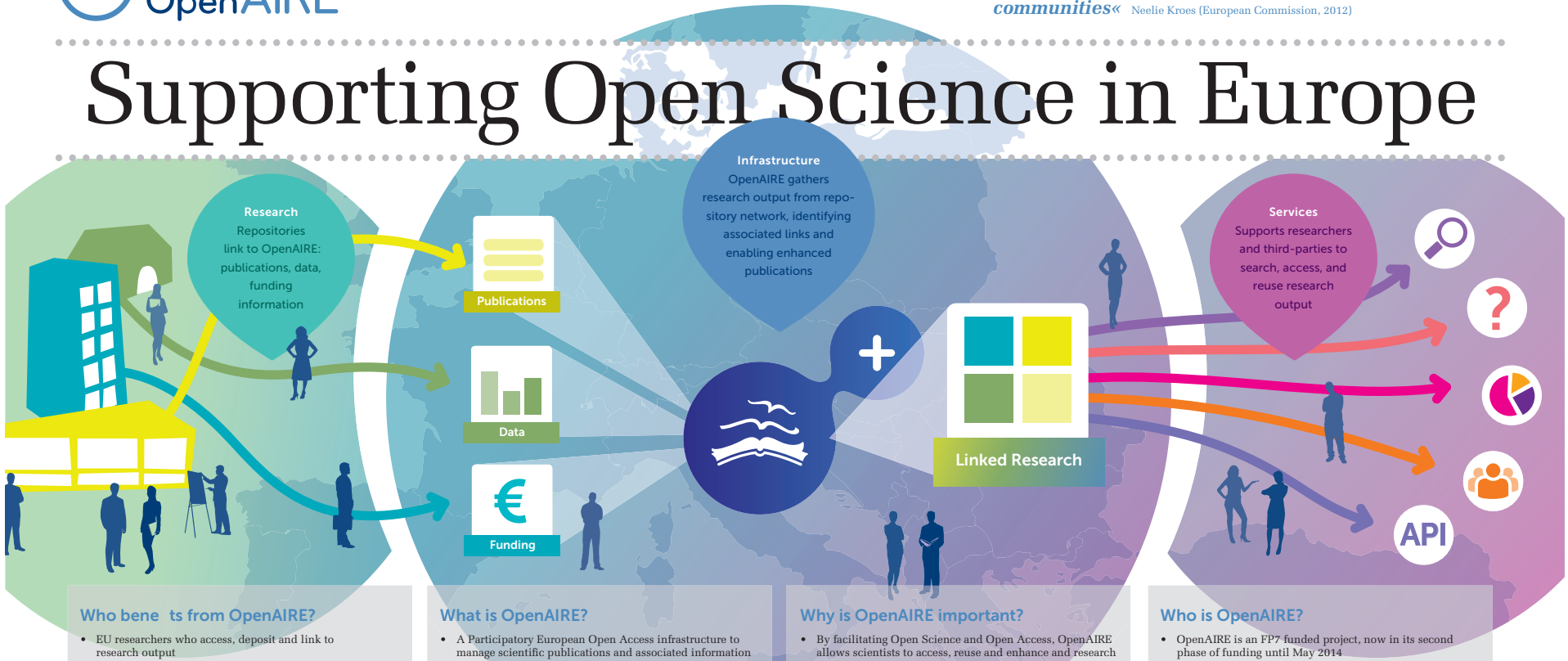
»Modern science needs the free flow of knowledge…in an e-infrastructure that is open across national borders, disciplines and scientific communities« Neelie Kroes (European Commission, 2012)

Supporting Open Science in Europe



Who bene ts from OpenAIRE?

- EU researchers who access, deposit and link to research output
- National Open Access initiatives
- Repository managers
- Policy makers and funders who monitor funded work
- Publishers who wish to raise visibility of output
- Potential data providers who want to explore linking up their research

What is OpenAIRE?

- A Participatory European Open Access infrastructure to manage scientific publications and associated information via repository networks
- Harvests and indexes FP7 Open Access publications
- Harvests subsets of related data, and other contextual information, cross-linking them to demonstrate *Enhanced Publications*
- The OpenAIRE portal provides a suite of services
  - deposit and access
  - guidelines and a helpdesk
- OpenAIRE runs a series of workshops, and produces reports on Open Access issues

Why is OpenAIRE important?

- By facilitating Open Science and Open Access, OpenAIRE allows scientists to access, reuse and enhance and research output
- OpenAIRE provides a cross-discipline support service for European Scientists
- Tools such as publication usage statistics
- OpenAIRE is based on
  - versatile technology and innovative research
  - European outreach effort which advocates Open Access

Who is OpenAIRE?

- OpenAIRE is an FP7 funded project, now in its second phase of funding until May 2014
- 41 project partners include 3 scientific communities: EBI, DANS and BADC
- Collaboration with EuroCRIS, EUDAT, DataCite, COAR, LIBER, SPARC Europe
- **Contact**
  Project Coordinator:
  Mike Hatzopoulos, mike@di.uoa.gr
- **www.openaire.eu**

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SEVENTH FRAMEWORK PROGRAMME

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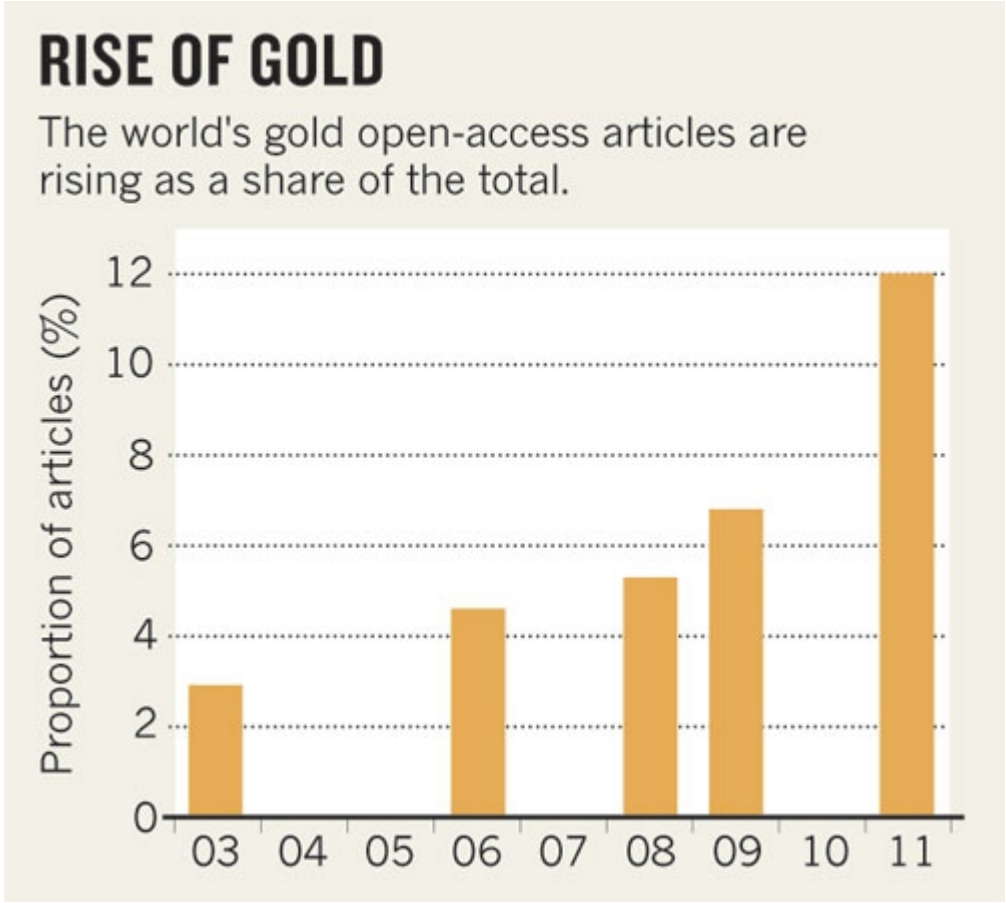
Coordination of Libraries?

> A possible explanation is that to do something about the situation requires coordinated action. Even if one library refuses to subscribe to Elsevier journals, plenty of others will feel that they can't refuse, and Elsevier won't mind too much. But if all libraries were prepared to club together and negotiate jointly, doing a kind of reverse bundling — accept this deal or none of us will subscribe to any of your journals — then Elsevier's profits (which are huge, by the way) would be genuinely threatened. However, it seems unlikely that any such massive coordination between libraries will ever take place.
>
> Timothy Gowers, Mathematician, Cambridge University

http://gowers.wordpress.com/2012/01/21/elsevier-my-part-in-its-downfall/



Van Noorden, Richard (2012) Nature 486, 302–303, http://dx.doi.org/10.1038/486302a

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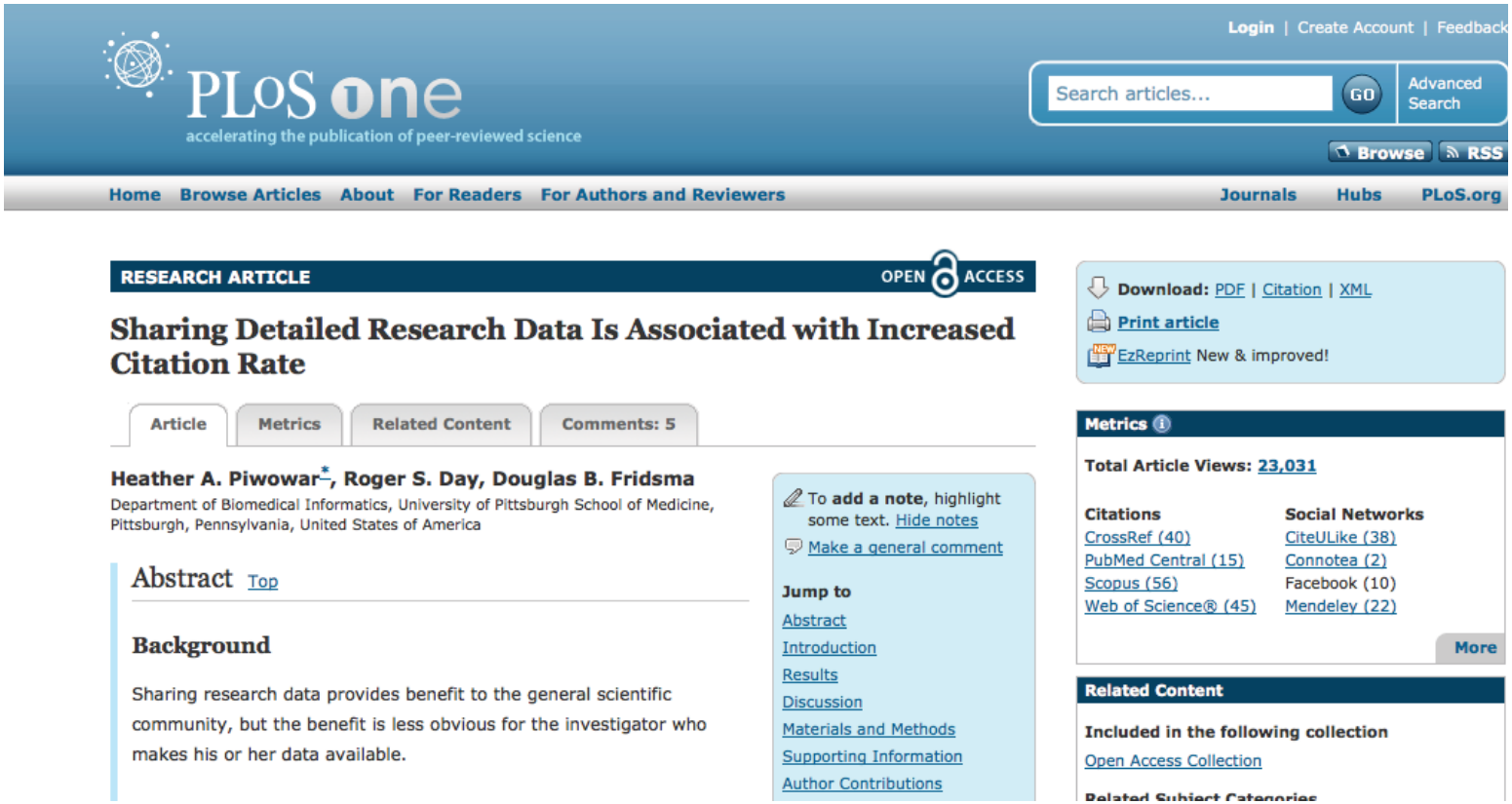

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