Is there an API for that?

What's new in Editorial Online Systems
STM E-Production Seminar 2012

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eJournalPress

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Peer Review
and
Production
Tracking
Systems for
Innovators

Integration

- Variety has become the modus operandi
- Publishers expect innovative integration between online editorial systems and other initiatives, tools, and platforms
- Common implementation motivation

Is there an API for that?

- Application Programming Interface (API)
- Specification used by software companies/components to communicate
- What's included varies based on the systems which will interact and the data that should be communicated

Some Recent Integrations

- FundRef
- ORCID
- Data Harmony

FundRef

- FundRef is a collaborative pilot project of scholarly publishers and funding agencies, facilitated by CrossRef, to provide a standard way of reporting funding sources for published scholarly research.[1]

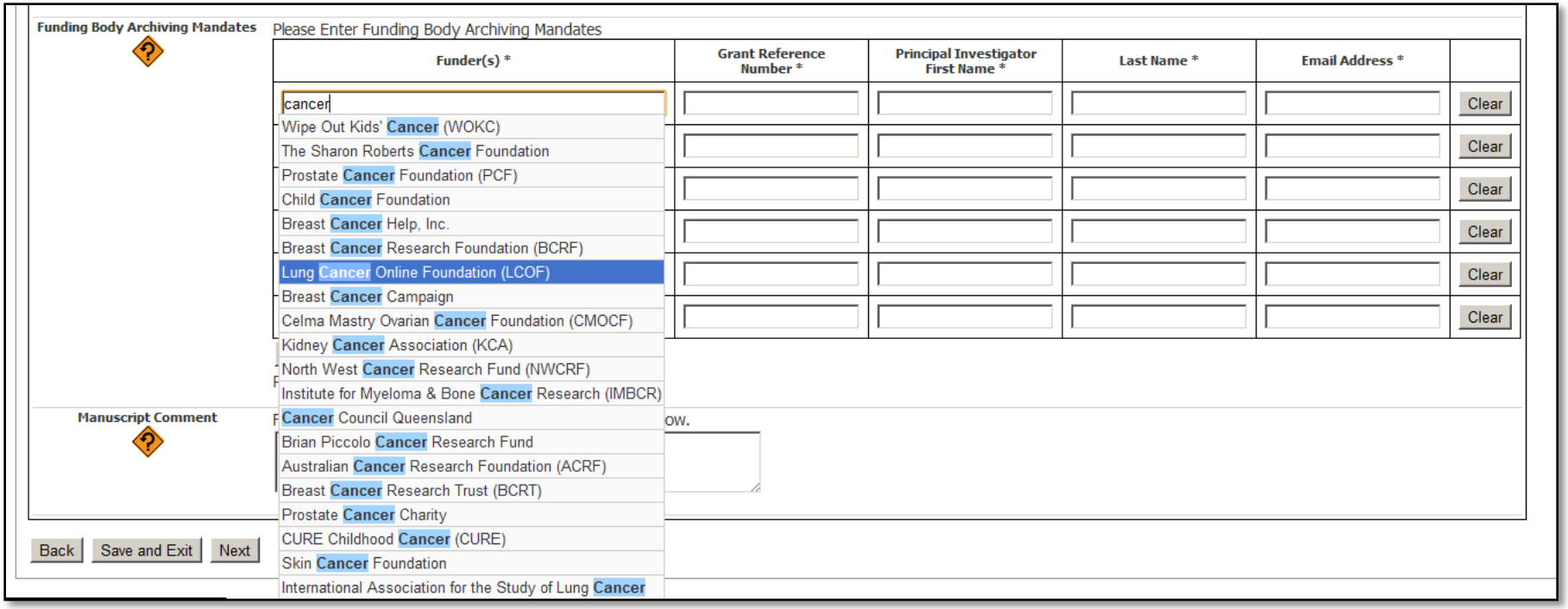
FundRef Submission Question

Funding Body Archiving Mandates

Please Enter Funding Body Archiving Mandates

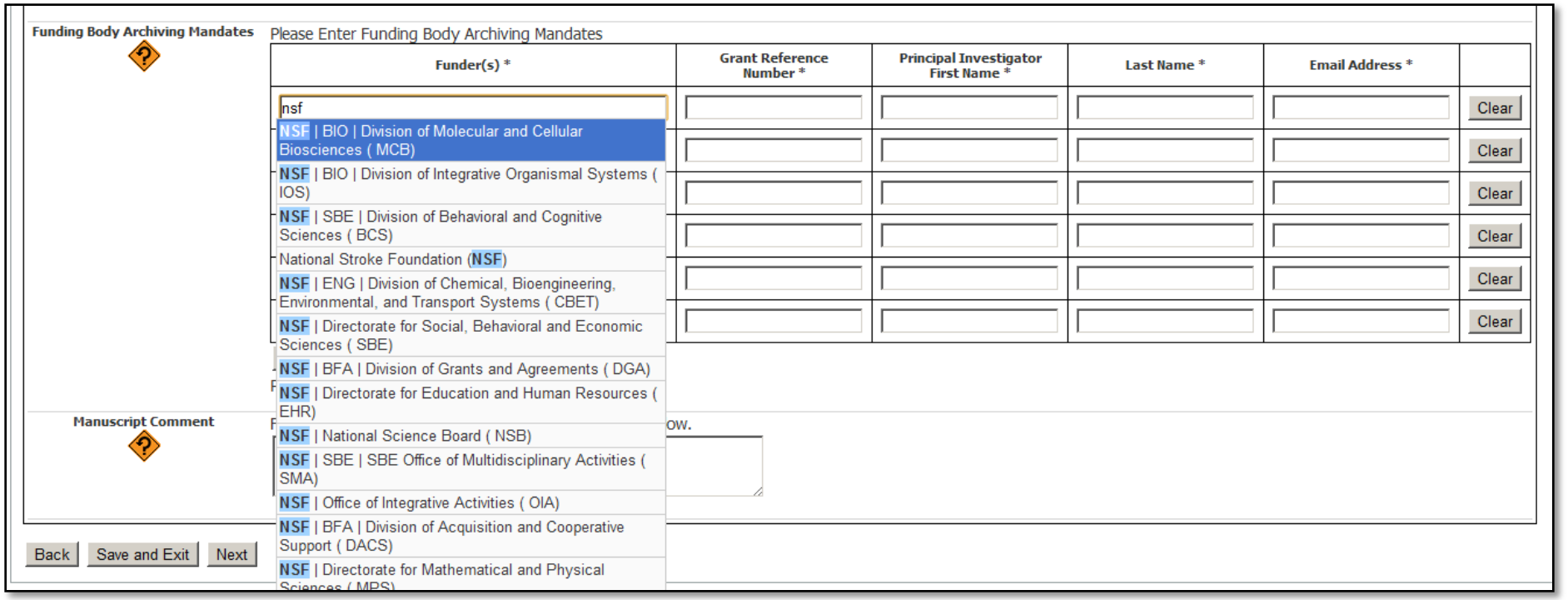
Funder(s) *	Grant Reference Number *	Principal Investigator First Name *	Last Name *	Email Address *	
					Clear
					Clear
					Clear
					Clear
					Clear
					Clear

Auto-completion



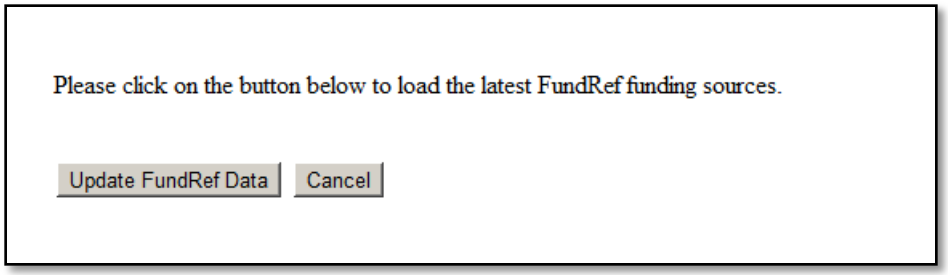
Hierarchical Data / Parent Organizations

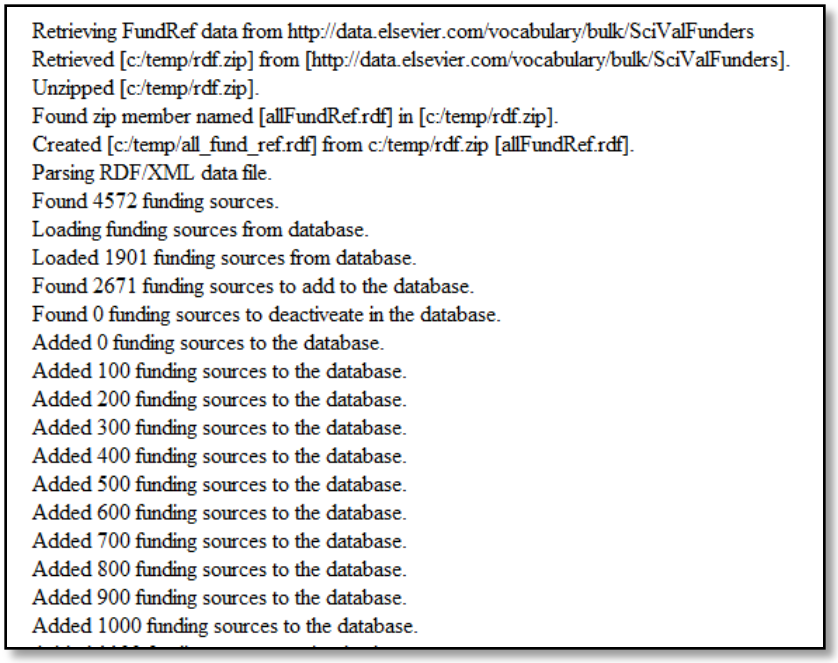
Parent Organization's “Short Name” pre-pended



Configuration Screens

- Journal Staff FundRef configuration screen
  - Allows for download of latest FundRef XML file on demand
  - Local database updated to reflect FundRef XML file changes

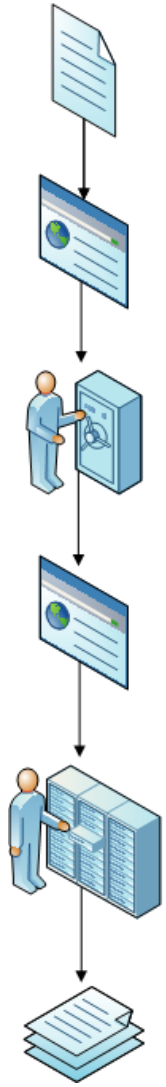




ORCID

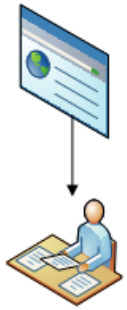
ORCID is an open, non-profit, community-based effort to create and maintain a registry of unique researcher identifiers and a transparent method of linking research activities and outputs to these identifiers.[2]

Scenario 1 - Corresponding author does NOT know ORCID's



- Co-author receives an e-mail with encrypted link
- Co-author directed to EJPress screen
  - instructional text
  - link to ORCID (OAUTH2)
- End-user logs into ORCID
  - registers if new
- ORCID provides back to EJPress ORCID ID of end-user
- EJPress stores ORCID
- Export
  - ORCID part of Author XML fields

Scenario 2 - Corresponding author knows ORCID's



- Corresponding author enters co-author ORCID's
- EJPress system queries ORCID by ID
  - gets name / association
  - maybe gets e-mail
  - might or might not be enough to verify ORCID
- Full verification (no trust) requires Scenario #1

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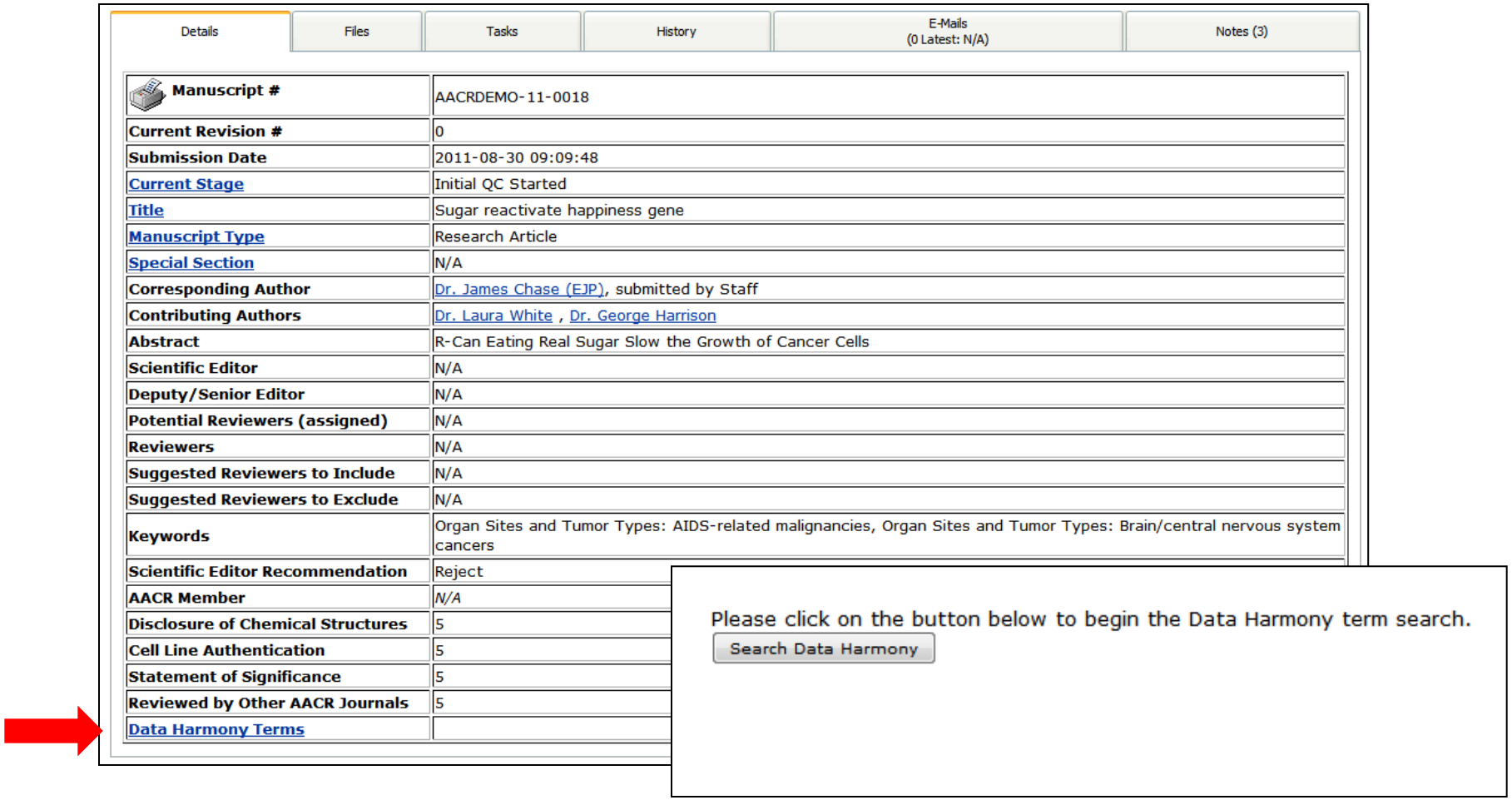
Data Harmony

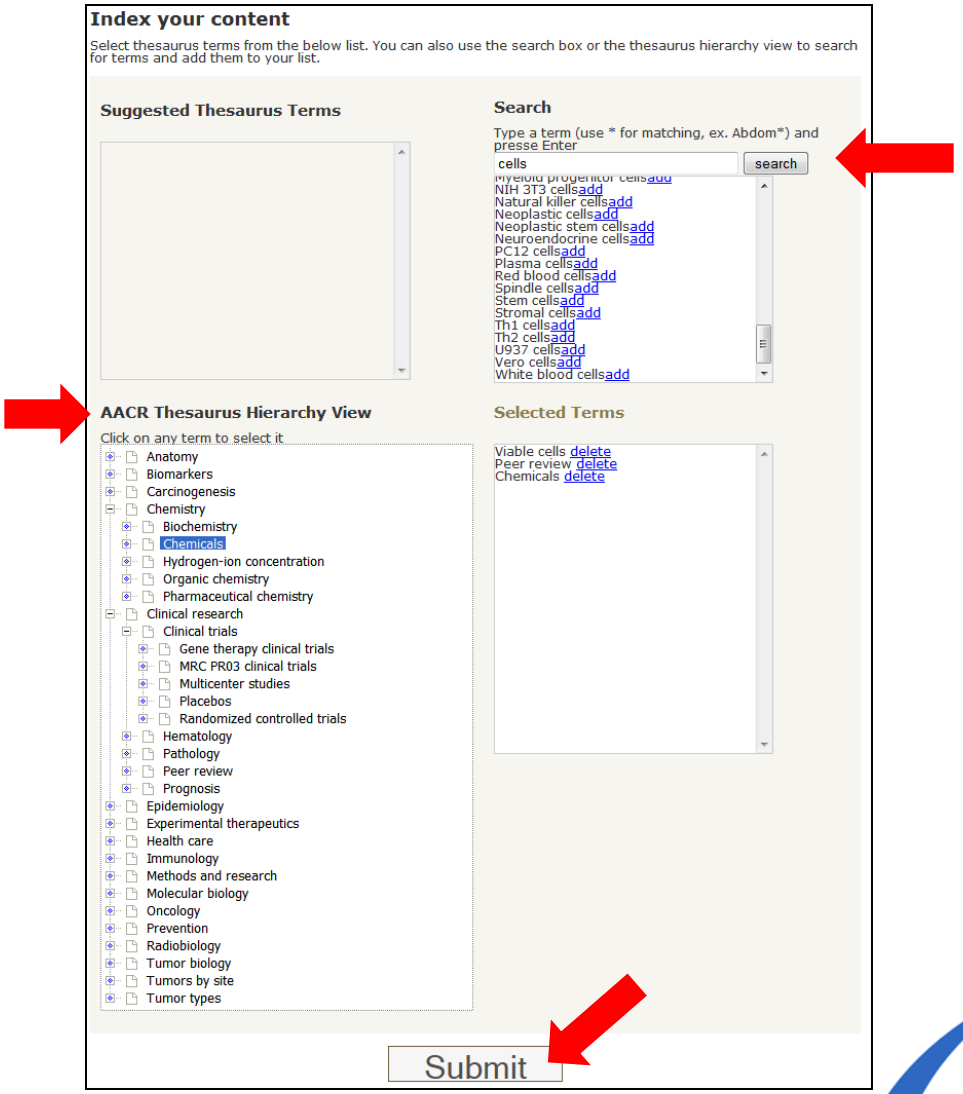
- "… provides knowledge management solutions to organize your information resources, based on a well-built and systematically applied taxonomy or thesaurus."[3]

Data Harmony Integration

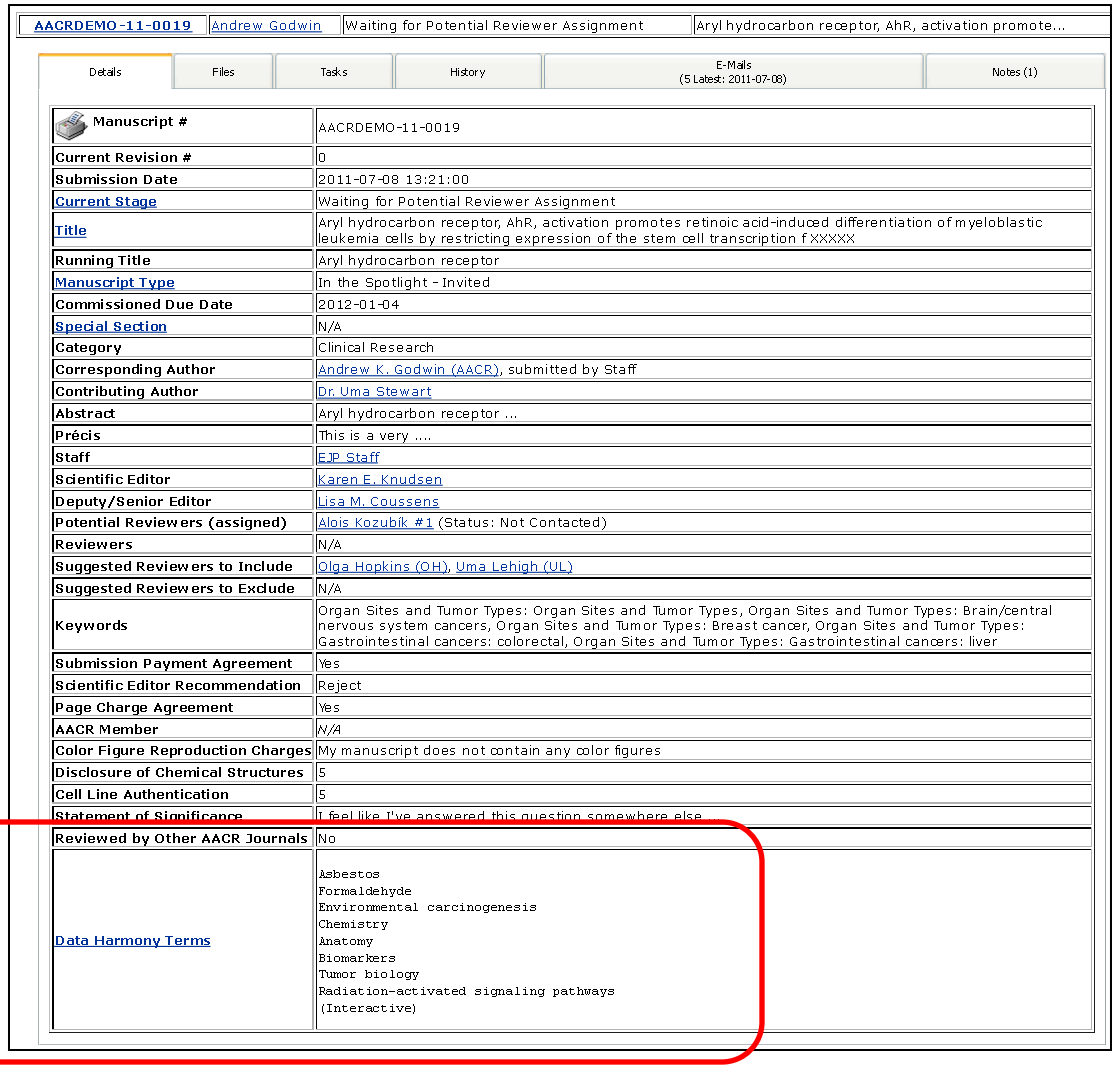


Data Harmony

- Searching:
  - Type terms
  - Choose from selection list
- Submit selected terms



Data Harmony Interactive Results



AACRDEMO-11-0019	Andrew Godwin	Waiting for Potential Reviewer Assignment	Aryl hydrocarbon receptor, AhR, activation promote...

Details | Files | Tasks | History | E-Mails (5 Latest: 2011-07-08) | Notes (1)

Manuscript #	AACRDEMO-11-0019
Current Revision #	0
Submission Date	2011-07-08 13:21:00
Current Stage	Waiting for Potential Reviewer Assignment
Title	Aryl hydrocarbon receptor, AhR, activation promotes retinoic acid-induced differentiation of myeloblastic leukemia cells by restricting expression of the stem cell transcription f XXXXX
Running Title	Aryl hydrocarbon receptor
Manuscript Type	In the Spotlight - Invited
Commissioned Due Date	2012-01-04
Special Section	N/A
Category	Clinical Research
Corresponding Author	Andrew K. Godwin (AACR), submitted by Staff
Contributing Author	Dr. Uma Stewart
Abstract	Aryl hydrocarbon receptor ...
Précis	This is a very ....
Staff	EJP Staff
Scientific Editor	Karen E. Knudsen
Deputy/Senior Editor	Lisa M. Coussens
Potential Reviewers (assigned)	Alois Kozubik #1 (Status: Not Contacted)
Reviewers	N/A
Suggested Reviewers to Include	Olga Hopkins (OH), Uma Lehigh (UL)
Suggested Reviewers to Exclude	N/A
Keywords	Organ Sites and Tumor Types: Organ Sites and Tumor Types, Organ Sites and Tumor Types: Brain/central nervous system cancers, Organ Sites and Tumor Types: Breast cancer, Organ Sites and Tumor Types: Gastrointestinal cancers: colorectal, Organ Sites and Tumor Types: Gastrointestinal cancers: liver
Submission Payment Agreement	Yes
Scientific Editor Recommendation	Reject
Page Charge Agreement	Yes
AACR Member	N/A
Color Figure Reproduction Charges	My manuscript does not contain any color figures
Disclosure of Chemical Structures	5
Cell Line Authentication	5
Statement of Significance	I feel like I've answered this question somewhere else ...
Reviewed by Other AACR Journals	No
Data Harmony Terms	Asbestos Formaldehyde Environmental carcinogenesis Chemistry Anatomy Biomarkers Tumor biology Radiation-activated signaling pathways (Interactive)

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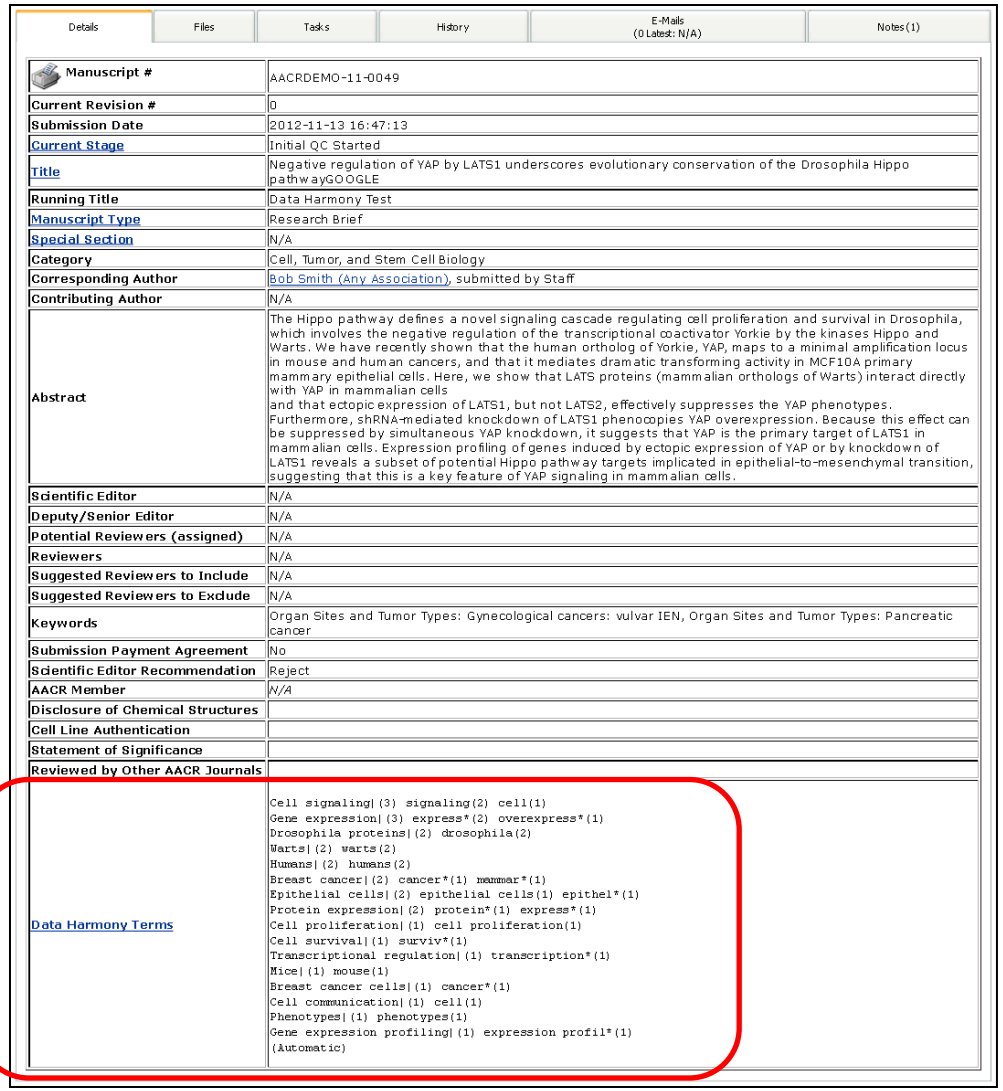
Data Harmony Automatic Results



Details | Files | Tasks | History | E-Mails (0 Latest: N/A) | Notes (1)

Manuscript #	AACRDEMO-11-0049
Current Revision #	0
Submission Date	2012-11-13 16:47:13
Current Stage	Initial QC Started
Title	Negative regulation of YAP by LATS1 underscores evolutionary conservation of the Drosophila Hippo pathwayGOOGLE
Running Title	Data Harmony Test
Manuscript Type	Research Brief
Special Section	N/A
Category	Cell, Tumor, and Stem Cell Biology
Corresponding Author	Bob Smith (Any Association), submitted by Staff
Contributing Author	N/A
Abstract	The Hippo pathway defines a novel signaling cascade regulating cell proliferation and survival in Drosophila, which involves the negative regulation of the transcriptional coactivator Yorkie by the kinases Hippo and Warts. We have recently shown that the human ortholog of Yorkie, YAP, maps to a minimal amplification locus in mouse and human cancers, and that it mediates dramatic transforming activity in MCF10A primary mammary epithelial cells. Here, we show that LATS proteins (mammalian orthologs of Warts) interact directly with YAP in mammalian cells and that ectopic expression of LATS1, but not LATS2, effectively suppresses the YAP phenotypes. Furthermore, shRNA-mediated knockdown of LATS1 phenocopies YAP overexpression. Because this effect can be suppressed by simultaneous YAP knockdown, it suggests that YAP is the primary target of LATS1 in mammalian cells. Expression profiling of genes induced by ectopic expression of YAP or by knockdown of LATS1 reveals a subset of potential Hippo pathway targets implicated in epithelial-to-mesenchymal transition, suggesting that this is a key feature of YAP signaling in mammalian cells.
Scientific Editor	N/A
Deputy/Senior Editor	N/A
Potential Reviewers (assigned)	N/A
Reviewers	N/A
Suggested Reviewers to Include	N/A
Suggested Reviewers to Exclude	N/A
Keywords	Organ Sites and Tumor Types: Gynecological cancers: vulvar IEN, Organ Sites and Tumor Types: Pancreatic cancer
Submission Payment Agreement	No
Scientific Editor Recommendation	Reject
AACR Member	N/A
Disclosure of Chemical Structures	
Cell Line Authentication	
Statement of Significance	
Reviewed by Other AACR Journals	
Data Harmony Terms	Cell signaling|(3) signaling(2) cell(1) Gene expression|(3) express*(2) overexpress*(1) Drosophila proteins|(2) drosophila(2) Warts|(2) warts(2) Humans|(2) humans(2) Breast cancer|(2) cancer*(1) mammar*(1) Epithelial cells|(2) epithelial cells(1) epithel*(1) Protein expression|(2) protein*(1) express*(1) Cell proliferation|(1) cell proliferation(1) Cell survival|(1) surviv*(1) Transcriptional regulation|(1) transcription*(1) Mice|(1) mouse(1) Breast cancer cells|(1) cancer*(1) Cell communication|(1) cell(1) Phenotypes|(1) phenotypes(1) Gene expression profiling|(1) expression profil*(1) (Automatic)

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Common Motivation

Q: Why did eJournalPress integrate with each of these systems?

A: Because our customers' business required it.

Information Resources

1. FundRef
   http://www.crossref.org/fundref/index.html
2. ORCID
   http://about.orcid.org/about/what-is-orcid
3. Data Harmony
   http://www.dataharmony.com/

Thank You For Your Time!

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