

SMAFIRA Web tool for searching the scientific literature for alternatives

10.04.2025, FC3R

Mariana Neves, Daniel Butzke

Agenda

Introduction

Demo

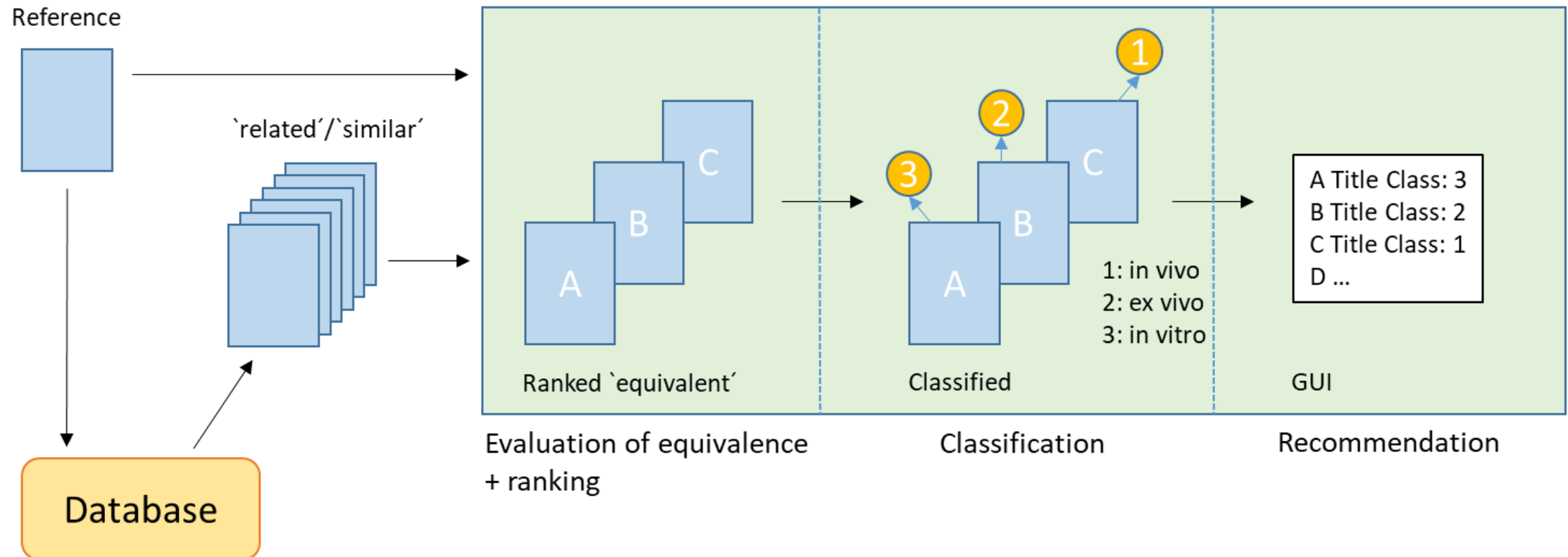
Methods

Current work


What is SMAFIRA?

- **A free online tool to screen the literature database PubMed© for candidate alternative methods**
- **Employs Artificial Intelligence and human feedback**
- **No login, no sensitive information, no search terms required**
- **No judgement on the suitability of retrieved methods as alternatives (reserved to human experts)**

Basic Workflow of SMAFIRA



Reference article



Advanced

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> [PLoS One](#). 2011 Apr 6;6(4):e18568. doi: 10.1371/journal.pone.0018568 🔗.

Dopaminergic neuronal loss, reduced neurite complexity and autophagic abnormalities in transgenic mice expressing G2019S mutant LRRK2

David Ramonet ¹, João Paulo L Daher, Brian M Lin, Klodjan Stafa, Jaekwang Kim, Rebecca Banerjee, Marie Westerlund, Olga Pletnikova, Liliane Glauser, Lichuan Yang, Ying Liu, Deborah A Swing, M Flint Beal, Juan C Troncoso, J Michael McCaffery, Nancy A Jenkins, Neal G Copeland, Dagmar Galter, Bobby Thomas, Michael K Lee, Ted M Dawson, Valina L Dawson, Darren J Moore

Affiliations + expand

PMID: 21494637 🔗 PMCID: [PMC3071839](#) 🔗 DOI: [10.1371/journal.pone.0018568](#) 🔗

Abstract

Mutations in the leucine-rich repeat kinase 2 (LRRK2) gene cause late-onset, autosomal dominant familial Parkinson's disease (PD) and also contribute to idiopathic PD. LRRK2 mutations represent the most common cause of PD with clinical and neurochemical features that are largely indistinguishable from idiopathic disease. Currently, transgenic mice expressing wild-type or disease-causing mutants of LRRK2 have failed to produce overt neurodegeneration, although abnormalities in nigrostriatal

SMAFIRA Demo

<https://smafira.bf3r.de>

SMAFIRA is a free online tool that is intended to support biomedical researchers and animal welfare officers in their efforts to screen the literature database PubMed® for candidate alternative methods to animal experiments. SMAFIRA DOES NOT judge on the suitability of retrieved methods as alternatives. This judgement is reserved to human experts. Anyway, SMAFIRA decreases the time needed to search the literature for candidate alternatives. All you need is a PubMed® identifier (PMID).



Quick Tutorial



Search for a [PubMed®](#) identifier that describes an animal experiment with the research goal of your interest. Insert the PMID in the field. By clicking on the "Search for PUBMED similar articles" button, SMAFIRA will first let you confirm if it is the correct reference article.

Please enter a PMID identifier of an in vivo publication:

Search for PUBMED similar articles

Input of a PMID, confirmation of the reference article


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SMAFIRA helps you to search in [PubMed®](#) for alternative methods for an animal experiment. All you need is a PubMed® identifier (PMID).

Please enter a PMID identifier of an in vivo publication:

21494637

Search for PUBMED similar articles

Reference article: [21494637](#): **Dopaminergic neuronal loss, reduced neurite complexity and autophagic abnormalities in transgenic mice expressing G2019S mutant LRRK2.** 

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Filters for the used experimental models

Vertebrate (other than human)

☐ in vivo

☐ tissue/biopsy

☐ primary cell

☒ immortal cell

Invertebrate

☐ all

Human

☐ all

Virtual Model

☒ all

Others

☒ all

PMID	SMAFIRA Rank	PubMed® Rank	My Rank	Title	Labels
1. 25174890	[1]	[1]	?	Conditional expression of Parkinson's disease-related R1441C LRRK2 in midbrain dopaminergic neurons of mice causes nuclear abnormalities without neurodegeneration.	
2. 21698001	[2]	[7]	?	Temporal expression of mutant LRRK2 in adult rats impairs dopamine reuptake.	
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4. 20382224	[4]	[10]	?	LRRK2-mediated neurodegeneration and dysfunction of dopaminergic neurons in a Caenorhabditis elegans model of Parkinson's disease.	
5. 20130188	[5]	[11]	?	Enhanced striatal dopamine transmission and motor performance with LRRK2 overexpression in mice is eliminated by familial Parkinson's disease mutation G2019S.	
9. 26123485	[9]	[2]	?	Selective expression of Parkinson's disease-related Leucine-rich repeat kinase 2 G2019S missense mutation in midbrain dopaminergic neurons impairs dopamine release and dopaminergic gene expression.	
10. 26363496	[10]	[27]	?	(R1441C) LRRK2 induces the degeneration of SN dopaminergic neurons and alters the expression of genes regulating neuronal survival in a transgenic mouse model.	

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



























☐ all 

Virtual Model

☒ all 

Others

☒ all 

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Vertebrate (other than human)

☐ in vivo

☐ tissue/biopsy

☐ primary cell

☒ immortal cell

☐ all

☐ all


☒ all

☒ all

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
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Invertebrate

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Human





















☐ all 

Virtual Model

☒ all 


Others

☒ all 

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Filters for the used experimental models

Vertebrate (other than human)


☐ in vivo 

☒ tissue/biopsy 


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Human





































☐ all 

Virtual Model


☐ all 

Others

☐ all 

PMID	SMAFIRA Rank	PubMed® Rank	My Rank	Title	Labels
1. 19667187 	[21]	[45]		R1441C mutation in LRRK2 impairs dopaminergic neurotransmission in mice.	   
2. 29038245 	[28]	[22]		Mitochondrial Calcium Dysregulation Contributes to Dendrite Degeneration Mediated by PD/LBD-Associated LRRK2 Mutants.	 
3. 28973664 	[42]	[53]		LRRK2 G2019S-induced mitochondrial DNA damage is LRRK2 kinase dependent and inhibition restores mtDNA integrity in Parkinson's disease.	   
4. 23231918 	[46]	[38]		Mutant LRRK2 elicits calcium imbalance and depletion of dendritic mitochondria in neurons.	   
5. 30954703 	[49]	[66]		An integrated transcriptomics and proteomics analysis reveals functional endocytic dysregulation caused by mutations in LRRK2.	 
6. 29855356 	[80]	[93]		LRRK2 activity does not dramatically alter α-synuclein pathology in primary neurons.	 
7. 22639965 	[88]	[94]		Leucine-rich repeat kinase 2 disturbs mitochondrial dynamics via Dynamin-like protein.	   

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


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- ☐ invertebrate (all) 
- ☐ human (all) 
- ☐ virtual model (all) 
- ☐ others (all) 

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Dopaminergic neuronal loss, reduced neurite complexity and autophagic abnormalities in transgenic mice expressing G2019S mutant LRRK2.

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 Click on a label to assign it to the article. Unclick it to remove it from the article.

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Classification of experimental models

GoldHamster corpus

- 1,600 annotated abstracts

	Total	No. Docs	Labels
1 label	862	213	"in vivo"
		156	"invertebrates"
		136	"human"
		97	"others"
		92	"organs"
		85	"in silico"
		30	"immortal cell lines"
		28	"primary cell lines"
		25	none
2 labels	31	6	"in silico+in vivo"
		5	"in vivo+primary cell lines"
		4	"in silico+invertebrates", or "immortal cell lines+in vivo", or "in vivo+organs"
		2	"organs+primary cell lines", or "in silico+primary cell lines", or "human+invertebrates", or "in silico+others"
3 labels	6	1	"human+in vivo+primary cell lines", or "in vivo+invertebrates", or "immortal cell lines+primary cell lines", or "in vivo+others", or "in silico+organs", or "immortal cell lines+organs"

Labels	Kappa	Level Of agreement
invertebrates	0.82	almost perfect
in vivo	0.78	substantial
in silico	0.72	substantial
human	0.63	substantial
organs	0.62	substantial
immortal cell lines	0.49	moderate
primary cell lines	0.45	moderate
others	0.42	moderate
overall	0.62	substantial


Automatic classification of experimental models in biomedical literature to support searching for alternative methods to animal experiments

Research | Open access | Published: 01 September 2023

Volume 14, article number 13, (2023) | Cite this article



Journal of Biomedical Semantics



Mariana Neves , Antonina Klippert, Fanny Knöspel, Juliane Rudeck, Ailine Stolz, Zsofia Ban, Markus Becker, Kai Diederich, Barbara Grune, Pia Kahnau, Nils Ohnesorge, Johannes Pucher, Gilbert Schönfelder, Bettina Bert & Daniel Butzke

Performance of the model

- Transformers pre-trained on PubMedBERT (BiomedBERT) and fine-tuned on the GoldHamster corpus

Labels	BioBERT	PubMedBERT	SciBERT	BLUE-BERT
invertebrates	0.89	0.95	0.95	0.95
in_vivo	0.88	0.88	0.89	0.83
human	0.82	0.86	0.82	0.73
organs	0.80	0.82	0.82	0.71
primary_cells	0.67	0.75	0.73	0.67
immortal_cell_lines	0.91	0.83	0.80	0.89
in_silico	0.67	0.75	0.75	0.86
others	0.70	0.78	0.67	0.76
All (average)	0.79	0.83	0.80	0.80

 Hugging Face

 microsoft/ **BiomedNLP-BiomedBERT-base-uncased-abstract-fulltext** 

Current work

“biomed-approach” corpus

- > 3000 annotated abstracts
- 23 disease categories (MeSH terms)
- Additional labels: human immortal cell lines, human primary cells, human organs/tissues, clinical studies
- Promising preliminary results

SMAFIRA Shared task



- **Call for participants!**
- **Annotation for selected reference articles (top 20)**
- **Training/evaluation set for re-ranking**
- **Manual (SMAFIRA Tool)**
- **Automatic (systems)**



The screenshot shows the SMAFIRA Shared Task website. The header is blue with the title "SMAFIRA Shared Task" and the subtitle "Assessing the similarity of the research goal". Below the header is a yellow bar with a "View On GitHub" button and a GitHub logo. The main content area has a light beige background. On the left is a sidebar with a list of links: Important dates, Data and annotation tool, Available data, Annotation tool, Test data, Annotation tasks, Manual annotation task, Automatic annotation task, Teams and participants, Release of the annotations (highlighted), Evaluation, Annotation guidelines, Registration, Contact, Organizers, and References. The main content area contains two paragraphs. The first paragraph starts with "Please join our discussion forum for announcements, questions, etc." and discusses strict legislation regarding animal testing and the 3R principles. The second paragraph starts with "The SMAFIRA project aims at supporting researchers for finding alternative methods to animal experiments" and describes the SMAFIRA Web tool, which takes a PubMed identifier (PMID) as input and retrieves up to 200 similar articles. It also mentions the re-ranking task and the three possible values for similarity: similar, uncertain, or not similar.

SMAFIRA Shared task

https://smafira-bf3r.github.io/smafira-st/



Infections	Neoplasms	Nervous System Diseases	Cardiovascular Diseases	Immune System Diseases
36159784	34233949	35709748	33635944	34503569
36577999	33320838	37084732	37010266	36179018
32485164	36311701	37339207	37380648	37079985
37071015	37429473	37749256	37268711	37256935
31689515	35623658	37126714	35917178	37168850

SMAFIRASearchFAQAboutData Protection DeclarationImprintContact

Back to list of results

Next

Are the two articles similar? Click on one of the options.

clear

Click on a label to assign it to the article. Unclick it to remove it from the article.

☐ in vivo

☐ tissue/biopsy

☐ primary cell

☐ immortal cell

☐ invertebrate (all)

☐ human (all)

☐ virtual model (all)

☐ others (all)

☒ in vivo

☐ tissue/biopsy

☐ primary cell

☐ immortal cell

☐ invertebrate (all)

☐ human (all)

☐ virtual model (all)

☐ others (all)

Reference article: 21494637

Dopaminergic neuronal loss, reduced neurite complexity and autophagic abnormalities in transgenic mice expressing G2019S mutant LRRK2.

Mutations in the leucine-rich repeat kinase 2 (LRRK2) gene cause late-onset, autosomal dominant familial Parkinson's disease (PD) and also contribute to idiopathic PD. LRRK2 mutations represent the most common cause of PD with clinical and neurochemical features that are largely indistinguishable from idiopathic disease. Currently, transgenic mice expressing wild-type or disease-causing mutants of LRRK2 have failed to produce overt neurodegeneration, although abnormalities in nigrostriatal dopaminergic neurotransmission have been observed. Here, we describe the development and characterization of transgenic mice expressing human

1. Similar Article: 25174890

Conditional expression of Parkinson's disease-related R1441C LRRK2 in midbrain dopaminergic neurons of mice causes nuclear abnormalities without neurodegeneration.

Mutations in the leucine-rich repeat kinase 2 (LRRK2) gene cause late-onset, autosomal dominant Parkinson's disease (PD). The clinical and neurochemical features of LRRK2-linked PD are similar to idiopathic disease although neuropathology is somewhat heterogeneous. Dominant mutations in LRRK2 precipitate neurodegeneration through a toxic gain-of-function mechanism which can be modelled in transgenic mice overexpressing human LRRK2 variants. A number of LRRK2 transgenic mouse models have been developed that display abnormalities in dopaminergic neurotransmission and alterations in tau

SMAFIRASearchFAQAboutData Protection DeclarationImprintContact

149 similar articles found for 21494637

Save the URL of your session: /artresults/?session=E4yHw1cjAjPpMFA

Filters for the used experimental models

Vertebrate (other than human)

☐ in vivo

☐ tissue/biopsy

☐ primary cell

☒ immortal cell

Invertebrate

☐ all

Human

☐ all

Virtual Model

☒ all

Others

☒ all

PMID	SMAFIRA Rank	PubMed® Rank	My Rank	Title	Labels
1. 25174890	[1]	[1]	?	Conditional expression of Parkinson's disease-related R1441C LRRK2 in midbrain dopaminergic neurons of mice causes nuclear abnormalities without neurodegeneration.	
2. 21698001	[2]	[7]	?	Temporal expression of mutant LRRK2 in adult rats impairs dopamine reuptake.	
3. 25174649	[3]	[3]	?	Behavioral, neurochemical, and pathologic alterations in bacterial artificial chromosome transgenic G2019S leucine-rich repeated kinase 2 rats.	
4. 20382224	[4]	[10]	?	LRRK2-mediated neurodegeneration and dysfunction of dopaminergic neurons in a Caenorhabditis elegans model of Parkinson's disease.	
5. 20130188	[5]	[11]	?	Enhanced striatal dopamine transmission and motor performance with LRRK2 overexpression in mice is eliminated by familial Parkinson's disease mutation G2019S.	

SMAFIRA Shared task



- **Publications**

- BioNLP Workshop (63rd Annual Meeting of the Association for Computational Linguistics , Vienna, Aug 1st 2025)
- Joint paper in a journal (end of 2025)

- Contact: mariana.lara-neves@bfr.bund.de



BIONLP 2025 and Shared Tasks @ ACL 2025

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