

Künstliche und menschliche Intelligenz bei der Literaturrecherche nach Alternativmethoden

Version April 2026

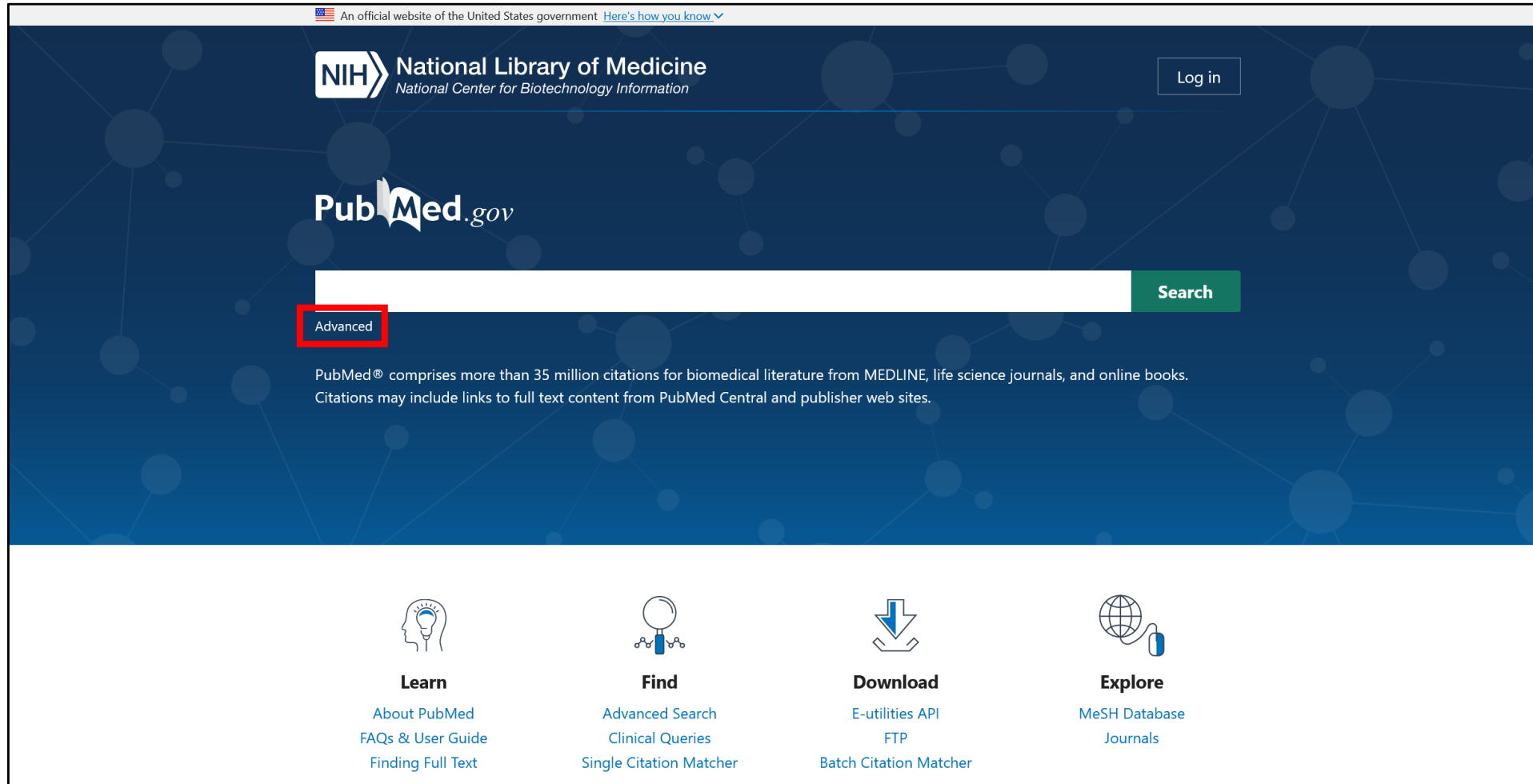
FG 94 – Tierschutz, Wissenstransfer und Nationaler Ausschuss

Abt. 9 – Deutsches Zentrum zum Schutz von Versuchstieren und experimentelle Toxikologie

Was steht auf dem Programm?

1. Einführung: **PubMed** - 'Advanced Search Builder'
2. Beispiele für Recherchen basierend auf ...
 - den klassischen Suchstrings (lexikalische Suche)
 - Klassifikationen (PubMed MeSH-Terme)
 - der „semantischen Ähnlichkeit“ von Abstracts und Suchabfrage (PubMed Similar Articles, **LitSense²**, **SMAFIRA**)
 - Retrieval Augmented Generation (**Consensus**)

PubMed als Suchoberfläche für MEDLINE, <https://pubmed.ncbi.nlm.nih.gov/>



Advanced Search Builder

The screenshot displays the PubMed Advanced Search Builder interface. At the top, a blue header contains the NIH logo, the text "National Library of Medicine National Center for Biotechnology Information", and a "Log in" button. Below the header, the page title "PubMed Advanced Search Builder" is visible. The main content area includes a "User Guide" link, a "Query box" for entering search terms, and a "History and Search Details" section. Annotations in German boxes with arrows point to specific features: "Metadaten-bezeichner" points to the "All Fields" dropdown; "Eingabefeld für Suchbegriffe" points to the "Enter a search term" input field; "Anleitung zur Recherche" points to the "User Guide" link; "Boolsche Operatoren" points to the "ADD" button; "Sofort-Suche oder Strategieentwicklung" points to the "Search" button; "Protokollierung der Recherche" points to the "History and Search Details" section; and "Anzeige des zusammengesetzten Suchstrings" points to the "Query box".

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PubMed Advanced Search Builder

Eingabefeld für Suchbegriffe

PubMed.gov
User Guide

Anleitung zur Recherche

Metadaten-bezeichner

Add terms to the query box

All Fields

Enter a search term

ADD

Show Index

Boolsche Operatoren

Sofort-Suche oder Strategieentwicklung

Search

Query box

Enter / edit your search query here

Protokollierung der Recherche

History and Search Details

Your history is currently empty! As you use PubMed your recent searches will appear here.

Anzeige des zusammengesetzten Suchstrings

Recherchen basierend auf Suchstrings (lexikalisch)

Recherchen basierend auf Suchstrings

- Verwenden Kombinationen von freien Suchtermen, Booleschen Operatoren (AND, OR, NOT) und Metadatenbezeichnern (z.B. [Title])
- Suchstrings können sehr umfangreich werden
- Geeignet für systematische Recherchen
- Umfang der Trefferliste variiert mit der Spezifität des Suchstrings (z.B. AND versus OR Trefferlisten)

Freie Suchbegriffe

- Suchbegriffe sind die „Grundbausteine“ einer Recherche
- Sie sind oft nicht eindeutig:

Homonyme: z.B. Laster, Tau, Kiefer, etc.

Synonyme: z.B. Aspirin, Acetylsalicylsäure, ASS, etc.

Syntax

- Boolesche Operatoren AND, NOT → Koinzidenz
OR → Synonyme, Schreibweise
- Anführungszeichen „...“ → zusammengesetzte Begriffe, z.B. „animal use alternatives“
- Klammern (...) → worauf beziehen sich die Operatoren, z.B.
(mice OR rats) AND parkinson → 25.740 Resultate
mice OR (rats AND parkinson) → 1.910.484 Resultate
[...] → Metadatenfeldbezeichner, z.B. [Author]

Beispiel: PubMed-Filter für `COVID-19 treatment`

("COVID-19" OR "COVID19" OR "COVID-19"[MeSH Terms] OR "COVID-19 Vaccines" OR "COVID-19 Vaccines"[MeSH Terms] OR "COVID-19 serotherapy" OR "COVID-19 serotherapy"[MeSH Terms] OR "COVID-19 Nucleic Acid Testing" OR "covid-19 nucleic acid testing"[MeSH Terms] OR "COVID-19 Serological Testing" OR "covid-19 serological testing"[MeSH Terms] OR "COVID-19 Testing" OR "covid-19 testing"[MeSH Terms] OR "SARS-CoV-2" OR "SARSCoV2" OR "SARSCoV-2" OR "SARS-CoV2" OR "sars-cov-2"[MeSH Terms] OR "Severe Acute Respiratory Syndrome Coronavirus 2" OR "2019 NCOV" OR ("coronavirus"[MeSH Terms] OR "coronavirus" OR "COV" OR "NCOV") AND 2019/11/01[PDAT] : 3000/12/31[PDAT])) AND ("therapeutics"[MeSH Terms] OR "therapeutics"[All Fields] OR "treatments"[All Fields] OR "therapy"[MeSH Subheading] OR "therapy"[All Fields] OR "treatment"[All Fields] OR "treatment s"[All Fields] OR "treat*"[All Fields] OR ("clinical trial"[Publication Type] OR "clinical trials as topic"[MeSH Terms] OR "clinical trials"[All Fields]) OR ("clinical trial"[Publication Type] OR "clinical trials as topic"[MeSH Terms] OR "clinical trial"[All Fields]) OR ("randomized controlled trial"[Publication Type] OR "randomized controlled trials as topic"[MeSH Terms] OR "randomized controlled trial"[All Fields] OR "randomised controlled trial"[All Fields]) OR ("randomized controlled trial"[Publication Type] OR "randomized controlled trials as topic"[MeSH Terms] OR "randomized controlled trials"[All Fields] OR "randomised controlled trials"[All Fields]) OR ("therapeutics"[MeSH Terms] OR "therapeutics"[All Fields] OR "therapies"[All Fields] OR "therapy"[MeSH Subheading] OR "therapy"[All Fields] OR "therapy s"[All Fields] OR "therapys"[All Fields]) OR ("therapeutical"[All Fields] OR "therapeutically"[All Fields] OR "therapeuticals"[All Fields] OR "therapeutics"[MeSH Terms] OR "therapeutics"[All Fields] OR "therapeutic"[All Fields]))

Advanced Search Builder: hilft bei der Syntax

 An official website of the United States government [Here's how you know](#) ✓

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Title

mice

×

ADD

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Search

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Add terms to the query box

Title

Enter a search term

×

AND

[Show Index](#)

Query box

mice[Title]

×

Search

History and Search Details

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Add terms to the query box

Title ▾ rats ×

OR ▾

Add with AND

Add with OR

Add with NOT

Query box

mice[Title] ×

History and Search Details

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Add terms to the query box

Title

Enter a search term

×

OR

▼

[Show Index](#)

Query box

(mice[Title]) OR (rats[Title])

×

Search

▼

History and Search Details

Your history is currently empty! As you use PubMed your recent searches will appear here.

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Add terms to the query box

Title

parkinson

×

AND

Query box

(mice[Title]) OR (rats[Title])

×

Add with AND

Add with OR

Add with NOT

History and Search Details

Your history is currently empty! As you use PubMed your recent searches will appear here.

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Add terms to the query box

Title Enter a search term X

AND

Show Index

Query box

((mice[Title]) OR (rats[Title])) AND (parkinson[Title]) X

Search

((mice[Title]) OR (rats[Title])) AND (parkinson[Title])

History and Search Details

Your history is currently empty! As you use PubMed your recent searches will appear here.

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Suche im Titel ergibt Trefferlisten mittleren Umfangs und hoher Relevanz

870 Resultate

X

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Sorted by: Best match

Display options

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870 results

Page 1 of 87

RESULTS BY YEAR

TEXT AVAILABILITY

☐ Abstract

☐ Free full text

☐ Full text

ARTICLE ATTRIBUTE

☐ Associated data

ARTICLE TYPE

☐ Books and Documents

☐ Clinical Trial

☐ Meta-Analysis

☐ Randomized Controlled Trial

☐

1

α-Synuclein BAC transgenic mice exhibit RBD-like behaviour and hyposmia: a prodromal Parkinson's disease model.

Cite

Taguchi T, Ikuno M, Hondo M, Parajuli LK, Taguchi K, Ueda J, Sawamura M, Okuda S, Nakanishi E, Hara J, Uemura N, Hatanaka Y, Ayaki T, Matsuzawa S, Tanaka M, El-Agnaf OMA, Koike M, Yanagisawa M, Uemura MT, Yamakado H, Takahashi R.

Brain. 2020 Jan 1;143(1):249-265. doi: 10.1093/brain/awz380.

PMID: 31816026

☐

2

Pathological α-synuclein transmission initiates Parkinson-like neurodegeneration in nontransgenic mice.

Cite

Luk KC, Kehm V, Carroll J, Zhang B, O'Brien P, Trojanowski JQ, Lee VM.

Science. 2012 Nov 16;338(6109):949-53. doi: 10.1126/science.1227157.

PMID: 23161999

Free PMC article.

☐

3

Neuroprotection of Fasting Mimicking Diet on MPTP-Induced Parkinson's Disease Mice via Gut Microbiota and Metabolites.

Cite

Zhou ZL, Jia XB, Sun MF, Zhu YL, Qiao CM, Zhang BP, Zhao LP, Yang Q, Cui C, Chen X, Shen YQ.

Neurotherapeutics. 2019 Jul;16(3):741-760. doi: 10.1007/s13311-019-00719-2.

PMID: 30815845

Free PMC article.

☐

4

Intestinal infection triggers Parkinson's disease-like symptoms in Pink1^{-/-} mice.

Cite

Matheoud D, Cannon T, Voisin A, Penttinen AM, Ramet L, Fahmy AM, Ducrot C, Laplante A, Bourque MJ, Zhu L, Cayrol R, Le Campion A, McBride HM, Gruenheid S, Trudeau LE, Desjardins M.

Nature. 2019 Jul;571(7766):565-569. doi: 10.1038/s41586-019-1405-y. Epub 2019 Jul 17.

PMID: 31316206

Alternativmethodenbeispiel

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Add terms to the query box

Title

Enter a search term

×

AND

Show Index

Query box

("in vitro"[Title]) AND (parkinson[Title])

×

Search

("in vitro"[Title]) AND (parkinson[Title])

History and Search Details

Download

Delete

Search	Actions	Details	Query	Results	Time
#1	...	>	Search: ((mice[Title]) OR (rats[Title])) AND (parkinson[Title])	870	04:42:59

Showing 1 to 1 of 1 entries

Alternativmethodenbeispiel

277 Resultate

PubMed.gov

("in vitro"[Title]) AND (parkinson[Title])

×

Search

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Sorted by: Best match

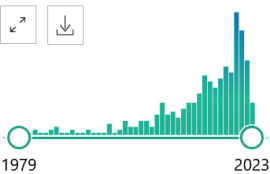
Display options ⚙

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RESULTS BY YEAR

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↓



1979 2023

TEXT AVAILABILITY

☐ Abstract

☐ Free full text

☐ Full text

ARTICLE ATTRIBUTE

☐ Associated data

ARTICLE TYPE

☐ Books and Documents

☐ Clinical Trial

☐ Meta-Analysis

☐ Randomized Controlled Trial

277 results

Page 1 of 28

☐ 1

In vivo, in vitro and pharmacologic models of Parkinson's disease.

Cite

Salari S, Bagheri M.

Physiol Res. 2019 Mar 6;68(1):17-24. doi: 10.33549/physiolres.933895. Epub 2018 Oct 23.

PMID: 30433804

Free article.

Review.

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☐ 2

SH-SY5Y human neuroblastoma cell line: in vitro cell model of dopaminergic neurons in Parkinson's disease.

Cite

Xie HR, Hu LS, Li GY.

Chin Med J (Engl). 2010 Apr 20;123(8):1086-92.

PMID: 20497720

Review.

Share

☐ 3

Neuroprotective effects of Danshensu on rotenone-induced Parkinson's disease models in vitro and in vivo.

Cite

Wang T, Li C, Han B, Wang Z, Meng X, Zhang L, He J, Fu F.

BMC Complement Med Ther. 2020 Jan 23;20(1):20. doi: 10.1186/s12906-019-2738-7.

PMID: 32020857

Free PMC article.

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☐ 4

Andrographolide suppresses NLRP3 inflammasome activation in microglia through induction of parkin-mediated mitophagy in in-vitro and in-vivo models of Parkinson disease.

Cite

Ahmed S, Kwatra M, Ranjan Panda S, Murty USN, Naidu VGM.

Brain Behav Immun. 2021 Jan;91:142-158. doi: 10.1016/j.bbi.2020.09.017. Epub 2020 Sep 22.

PMID: 32971182

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User Guide

Add terms to the query box

All Fields

Enter a search term

ADD

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Query box

Enter / edit your search query here

Search

History and Search Details

("mice"[Title] OR "rats"[Title]) AND "parkinson"[Title]

Search	Actions	Details	Query	Results	Time
#2	...	>	Search: ("in vitro"[Title]) AND (parkinson[Title])	277	04:58:50
#1	...	▼	Search: ((mice[Title] OR rats[Title])) AND (parkinson[Title]) ("mice"[Title] OR "rats"[Title]) AND "parkinson"[Title]	870	04:42:59

Showing 1 to 2 of 2 entries

Anführungszeichen um 'Query Translation' auszuschalten

Query Translation: Beispiel-Recherche über alle Metadatenfelder

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Add terms to the query box

All Fields Enter a search term AND Show Index

Query box

((mice) OR (rats)) AND (parkinson) Search

History and Search Details
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Query Translation: PubMed fügt Suchbegriffe hinzu

History and Search Details					 Download  Delete	
Search	Actions	Details	Query	Results	Time	
#1	...	▼	Search: ((mice) OR (rats)) AND (parkinson) ("mice"[MeSH Terms] OR "mice"[All Fields] OR ("rats"[MeSH Terms] OR "rats"[All Fields])) AND ("parkinson disease"[MeSH Terms] OR ("parkinson"[All Fields] AND "disease"[All Fields]) OR "parkinson disease"[All Fields] OR "parkinsons"[All Fields] OR "parkinson"[All Fields] OR "parkinson s"[All Fields] OR "parkinsonian disorders"[MeSH Terms] OR ("parkinsonian"[All Fields] AND "disorders"[All Fields]) OR "parkinsonian disorders"[All Fields] OR "parkinsonism"[All Fields] OR "parkinsonisms"[All Fields] OR "parkinsons s"[All Fields]) Translations mice: "mice"[MeSH Terms] OR "mice"[All Fields] rats: "rats"[MeSH Terms] OR "rats"[All Fields] parkinson: "parkinson disease"[MeSH Terms] OR ("parkinson"[All Fields] AND "disease"[All Fields]) OR "parkinson disease"[All Fields] OR "parkinsons"[All Fields] OR "parkinson"[All Fields] OR "parkinson's"[All Fields] OR "parkinsonian disorders"[MeSH Terms] OR ("parkinsonian"[All Fields] AND "disorders"[All Fields]) OR "parkinsonian disorders"[All Fields] OR "parkinsonism"[All Fields] OR "parkinsonisms"[All Fields] OR "parkinsons's"[All Fields]	25,765	09:55:54	

Showing 1 to 1 of 1 entries

Klassifikationsrecherchen auf der Basis von MeSH (Medical Subject Headings)

Klassifikationsrecherchen auf Basis von MeSH

- Verwenden vorgegebene, definierte Schlagwörter (Deskriptoren) aus einem kontrollierten Vokabular (Thesaurus)
- Kenntnis der relevanten Schlagwörter ist erforderlich
- Geeignet für systematische Recherchen
- Erzeugen Trefferlisten mittleren Umfangs (Beispiel: `animal use alternatives[MeSH]' → 4.520 Treffer im April 2026)

MeSH-Terme: Aufruf eines Dokuments in PubMed

> [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](#)
[Free PMC article](#)

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 dopaminergic neurotransmission have been observed. Here, we describe the development and characterization of transgenic mice expressing human LRRK2 bearing the familial PD mutations, R1441C and G2019S. Our study demonstrates that expression of G2019S mutant LRRK2 induces the degeneration of nigrostriatal pathway dopaminergic neurons in an age-dependent manner. In addition, we observe autophagic and mitochondrial abnormalities in the brains of aged G2019S LRRK2 mice and markedly reduced neurite complexity of cultured dopaminergic neurons. These new LRRK2 transgenic mice will provide important tools for understanding the mechanism(s) through which familial mutations precipitate neuronal degeneration and PD.

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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](#)
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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 dopaminergic neurotransmission have been observed. Here, we describe the development and characterization of transgenic mice expressing human LRRK2 bearing the familial PD mutations, R1441C and G2019S. Our study demonstrates that expression of G2019S mutant LRRK2 induces the degeneration of nigrostriatal pathway dopaminergic neurons in an age-dependent manner. In addition, we observe autophagic and mitochondrial abnormalities in the brains of aged G2019S LRRK2 mice and markedly reduced neurite complexity of cultured dopaminergic neurons. These new LRRK2 transgenic mice will provide important tools for understanding the mechanism(s) through which familial mutations precipitate neuronal degeneration and PD.

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References

Publication types

MeSH terms

Substances

Related information

Grant support

LinkOut - more resources

Liste der zugeordneten MeSH-Terme

Publication types

- > Research Support, N.I.H., Extramural
- > Research Support, Non-U.S. Gov't

MeSH terms

- > Amino Acid Substitution / genetics*
- > Animals
- > Autophagy*
- > Behavior, Animal
- > Chromatography, High Pressure Liquid
- > Dopamine / metabolism*
- > Humans
- > Leucine-Rich Repeat Serine-Threonine Protein Kinase-2
- > Mesencephalon / metabolism
- > Mesencephalon / pathology
- > Mesencephalon / ultrastructure
- > Mice
- > Mice, Transgenic
- > Motor Activity
- > Mutant Proteins / metabolism*
- > Neurites / pathology*
- > Neurites / ultrastructure
- > Organ Culture Techniques
- > Protein Serine-Threonine Kinases / metabolism*
- > Protein Transport

Substances

- > Mutant Proteins
- > LRRK2 protein, human
- > Leucine-Rich Repeat Serine-Threonine Protein Kinase-2
- > Protein Serine-Threonine Kinases
- > Dopamine

MeSH-Terme spiegeln den Inhalt des Volltextes wieder (bis 2022)

[Review](#) > [Glia](#). 2013 Jan;61(1):3-9. doi: 10.1002/glia.22385. Epub 2012 Sep 17.

Animal models for studying microglia: the first, the popular, and the new

Dirk Sieger¹, Francesca Peri

Affiliations + expand
PMID: 22987329 DOI: [10.1002/glia.22385](#)

Abstract

Microglia, the resident phagocytes of brain, have been intensively studied since their discovery in the 1920s. There is no doubt that the possibility of culturing microglia in vitro has advanced enormously our understanding of these cells. However, as we know today, that microglia react to even small changes in the brain, it is crucial to also study these cells by preserving as much as possible their natural environment. Nowadays, advances in imaging technologies and transgenic cell labeling methods allow the direct observation of cells at work. These in vivo approaches have already changed our view on microglia by showing that these cells are active even in the healthy adult brain. As today, there is upcoming evidence that microglia can directly influence neuronal activity, understanding their roles and, in particular, their interactions with neurons is of great importance. The aim of this review is to illustrate three animal models that are currently used for microglial research and to discuss their characteristics and advantages by presenting recent achievements in microglial research. In our view the availability of different systems for studying microglia will lead to a more comprehensive understanding of their functions.

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MeSH terms

- > [Animals](#)
- > [Humans](#)
- > [Leeches](#)
- > [Mice](#)
- > [Microglia / classification](#)
- > [Microglia / physiology*](#)
- > [Models, Animal*](#)
- > [Species Specificity](#)
- > [Zebrafish](#)

Cited by

Beispiel `Organ Culture Techniques`[MeSH]

Publication types

- > Research Support, N.I.H., Extramural
- > Research Support, Non-U.S. Gov't

MeSH terms

- > Amino Acid Substitution / genetics*
- > Animals
- > Autophagy*
- > Behavior, Animal
- > Chromatography, High Pressure Liquid
- > Dopamine / metabolism*
- > Humans
- > Leucine-Rich Repeat Serine-Threonine Protein Kinase-2
- > Mesencephalon / metabolism
- > Mesencephalon / pathology
- > Mesencephalon / ultrastructure
- > Mice
- > Mice, Transgenic
- > Motor Activity
- > Mutant Proteins / metabolism*
- > Neurites / pathology*
- > Neurites / ultrastructure
- > Organ Culture Techniques

- > Protein Serine-Threonine Kinases / metabolism*
- > Protein Serine-Threonine Kinases
- > Dopamine

ACTIONS

- Search in PubMed
- Search in MeSH
- Add to Search

Findet alle Zitationen in PubMed,
die entsprechend „vermesht“ sind

Beispiel 'Organ Culture Techniques'[MeSH] → Klassifikationsrecherche

26.521 Resultate

PubMed.gov

"Organ Culture Techniques"[MeSH] Search

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26,521 results

RESULTS BY YEAR

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TEXT AVAILABILITY

☐ Abstract

☐ Free full text

☐ Full text

ARTICLE ATTRIBUTE

☐ Associated data

ARTICLE TYPE

☐ Books and Documents

☐ Clinical Trial

☐ Meta-Analysis

☐ Randomized Controlled Trial

☐ Review

☐ Systematic Review

1 ☐ **Transcriptional Profiling Provides New Insights into Organ Culture-Induced Changes in Human Donor Corneas.**

Cite Wolf J, Kammrath Betancor P, Maier P, Heinzelmann SU, Jiang J, Lange C, Reinhard T, Schlunck G, Lapp T. Int J Mol Sci. 2022 Nov 22;23(23):14507. doi: 10.3390/ijms232314507.

Share PMID: 36498835 Free PMC article.

2 ☐ **Fetal Thymic Organ Culture and Negative Selection.**

Cite Teixeira E, Daniels MA. Methods Mol Biol. 2023;2580:293-302. doi: 10.1007/978-1-0716-2740-2_18.

Share PMID: 36374465

3 ☐ **A Simplified Approach to Evaluating T Cell Development by Flow Cytometry.**

Cite Lee JYM, Love PE. Methods Mol Biol. 2023;2580:131-149. doi: 10.1007/978-1-0716-2740-2_7.

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4 ☐ **Cisplatin Induces Apoptosis in Mouse Neonatal Testes Organ Culture.**

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5 ☐ **Protocol to Study West Nile Virus Infection in Brain Slices In Vitro.**

Cite Vig PJS. Methods Mol Biol. 2023;2585:97-103. doi: 10.1007/978-1-0716-2760-0_10.

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Beispiel 'Organ Culture Techniques'[MeSH]

Publication types

- > Research Support, N.I.H., Extramural
- > Research Support, Non-U.S. Gov't

MeSH terms

- > Amino Acid Substitution / genetics*
- > Animals
- > Autophagy*
- > Behavior, Animal
- > Chromatography, High Pressure Liquid
- > Dopamine / metabolism*
- > Humans
- > Leucine-Rich Repeat Serine-Threonine Protein Kinase-2
- > Mesencephalon / metabolism
- > Mesencephalon / pathology
- > Mesencephalon / ultrastructure
- > Mice
- > Mice, Transgenic
- > Motor Activity
- > Mutant Proteins / metabolism*
- > Neurites / pathology*
- > Neurites / ultrastructure
- > Organ Culture Techniques
- > Protein Serine-Threonine Kinases / metabolism*

ACTIONS

- Search in PubMed
- Search in MeSH
- Add to Search

Zeigt Details zum gewählten MeSH-Term

Beispiel 'Organ Culture Techniques'[MeSH] → MeSH Database

The screenshot shows the MeSH database interface for the term 'Organ Culture Techniques'. The interface includes a search bar at the top with the term entered and a 'Search' button. Below the search bar, there are links for 'Create alert', 'Limits', and 'Advanced'. The main content area is divided into several sections:

- Organ Culture Techniques**: A definition of the term, its history, and PubMed search builder options.
- Subheadings**: A list of subheadings with checkboxes for selection, including classification, economics, ethics, history, instrumentation, methods, standards, statistics and numerical data, trends, and veterinary.
- Tree Number(s)**: E05.481.500.484
- MeSH Unique ID**: D009924
- Entry Terms**: A list of synonyms for the term.
- Previous Indexing**: A list of previous indexing terms.
- See Also**: A list of related terms.
- All MeSH Categories**: A hierarchical list of categories leading to the term.

Annotations on the left side of the screenshot point to specific sections:

- Erklärung** points to the definition of 'Organ Culture Techniques'.
- Möglichkeiten um weiter einzuschränken** points to the 'Subheadings' section.
- Synonyme** points to the 'Entry Terms' section.
- Hierarchie** points to the 'All MeSH Categories' section.

On the right side of the screenshot, there are additional sections:

- PubMed Search Builder**: A section for building PubMed search queries.
- Related information**: A section for related information, including PubMed, PubMed - Major Topic, Clinical Queries, and NLM MeSH Browser.
- Recent Activity**: A section for recent activity.

Beispiel `Organ Culture Techniques`[MeSH] → Advanced Search Builder

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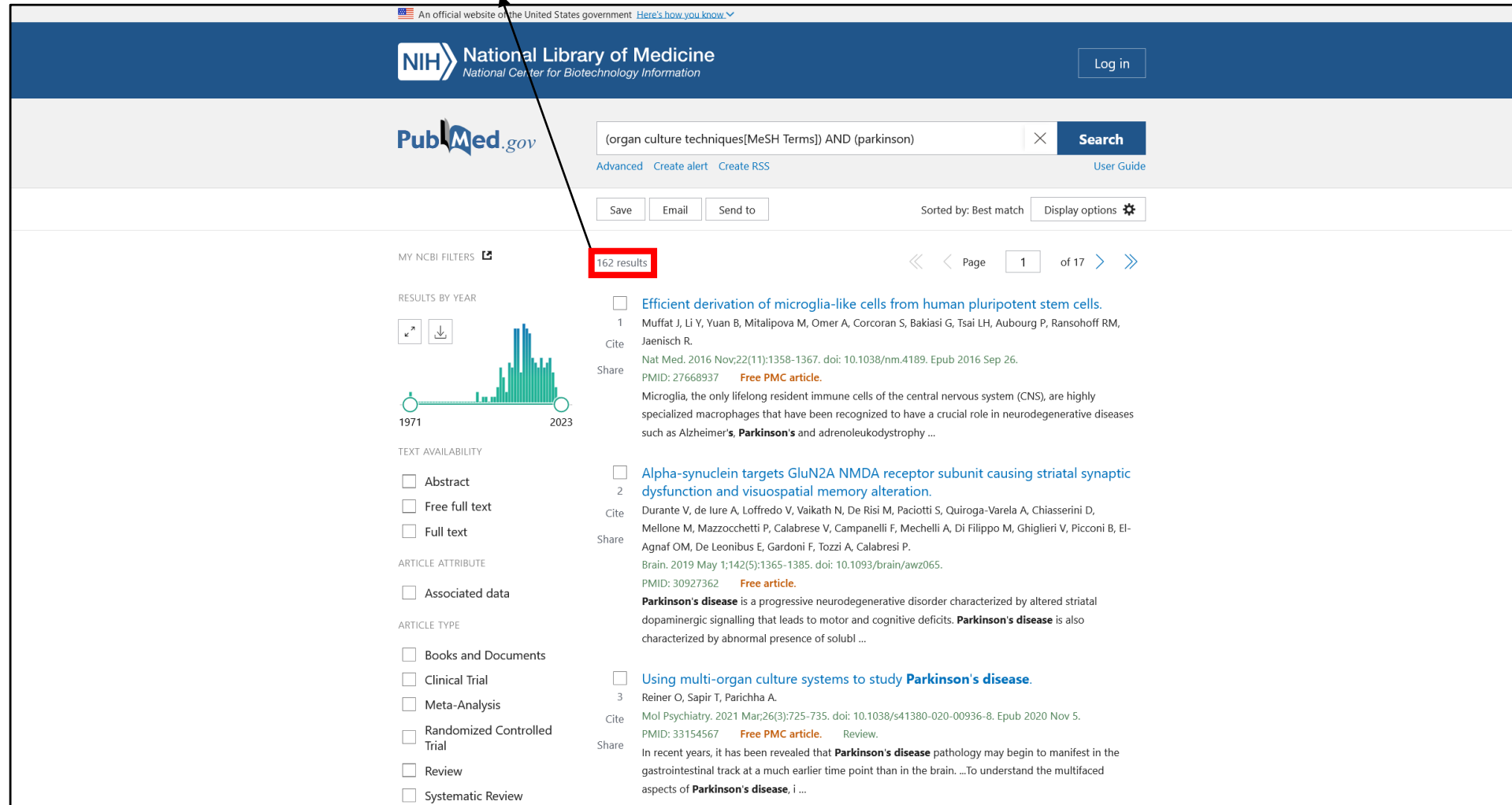
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☐ Using multi-organ culture systems to study Parkinson's disease.

3

Reiner O, Sapir T, Parichha A.

Cite

Mol Psychiatry. 2021 Mar;26(3):725-735. doi: 10.1038/s41380-020-00936-8. Epub 2020 Nov 5. PMID: 33154567 [Free PMC article.](#) [Review.](#)

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In recent years, it has been revealed that **Parkinson's disease** pathology may begin to manifest in the gastrointestinal track at a much earlier time point than in the brain. ...To understand the multifaced aspects of **Parkinson's disease**, i ...

☐ Biotechnological production of bacosides from cell and organ cultures of Bacopa monnieri.

4

Murthy HN.

Cite

Appl Microbiol Biotechnol. 2022 Mar;106(5-6):1799-1811. doi: 10.1007/s00253-022-11834-0. Epub 2022 Feb 24. PMID: 35201388 [Review.](#)

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(BM), also known as 'Brahmi' or 'Water Hyssop', has been utilized as a brain tonic, memory enhancer, sensory organ revitalizer, cardioprotective, anti-anxiety, antidepressant and anticonvulsant agent in the Indian system of medicine Ayurveda for centuries. BM is beneficial in the tree ...

☐ Organotypic brain slice cultures: A review.

5

Humpel C.

Cite

Neuroscience. 2015 Oct 1;305:86-98. doi: 10.1016/j.neuroscience.2015.07.086. Epub 2015 Aug 5. PMID: 26254240 [Free PMC article.](#) [Review.](#)

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This review summarizes (1) the historical development of organotypic brain slices focusing on the membrane technology, (2) methodological aspects regarding culturing procedures, age of donors or media, (3) whether the cholinergic neurons serve as a model of neurodegeneration in A ...

☐ Human iPS Cell-Derived Patient Tissues and 3D Cell Culture Part 2: Spheroids, Organoids, and Disease Modeling.

6

Eglen RM, Reisine T.

Cite

SLAS Technol. 2019 Feb;24(1):18-27. doi: 10.1177/2472630318803275. PMID: 30798678 [Review.](#)

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Given the specific degeneration seen in discrete neuronal circuitry in Alzheimer's **disease** (AD) and **Parkinson's disease** (PD), iPSC culture systems are proving to be a major advance. In the present review, the second part of a two-part review, w ...

☐ rAAV-based brain slice culture models of Alzheimer's and Parkinson's disease inclusion pathologies.

7

Croft CL, Cruz PE, Ryu DH, Ceballos-Diaz C, Strang KH, Woody BM, Lin WL, Deture M, Rodriguez-Lebrón E, Dickson DW, Chakrabarty P, Levites Y, Giasson BI, Golde TE.

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J Exp Med. 2019 Mar 4;216(3):539-555. doi: 10.1084/jem.20182184. Epub 2019 Feb 15. PMID: 30770411 [Free PMC article.](#)

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Using three-dimensional mouse brain slice cultures (BSCs), we have developed a paradigm that rapidly and robustly recapitulates mature neurofibrillary inclusion and Lewy body formation found in Alzheimer's and **Parkinson's disease**, respectively. This wa ...

☐ Limitations of cellular models in Parkinson's disease research.

8

Falkenburger BH, Schulz JB.

Cite

J Neural Transm Suppl. 2006;(70):261-8. doi: 10.1007/978-3-211-45295-0_40. PMID: 17017539 [Review.](#)

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Cell cultures for **Parkinson's disease** research have the advantage of virtually unlimited access, they allow rapid screening for **disease** pathogenesis and drug candidates, and they restrict the necessary number of animal experiments. ...In most cases, ne ...

Using multi-organ culture systems to study Parkinson's disease.

Organotypic brain slice cultures: A review.

Human iPS Cell-Derived Patient Tissues and 3D Cell Culture Part 2: Spheroids, Organoids, and Disease Modeling.

rAAV-based brain slice culture models of Alzheimer's and Parkinson's disease inclusion pathologies.

Limitations of cellular models in Parkinson's disease research.

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Beispiele für 3R-relevante MeSH-Terme

- **Animal Use Alternatives**, Animal Testing Alternatives
- **In Vitro Techniques**, Cell Culture Techniques, Organ Culture Techniques, Tissue Culture Techniques
- **Invertebrates**
- **Computer simulation**

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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

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

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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 dopaminergic neurotransmission have been observed. Here, we describe the development and characterization of transgenic mice expressing human LRRK2 bearing the familial PD mutations, R1441C and G2019S. Our study demonstrates that expression of G2019S mutant LRRK2 induces the degeneration of nigrostriatal pathway dopaminergic neurons in an age-dependent manner. In addition, we observe autophagic and mitochondrial abnormalities in the brains of aged G2019S LRRK2 mice and markedly reduced neurite complexity of cultured dopaminergic neurons. These new LRRK2 transgenic mice will provide important tools for understanding the mechanism(s) through which familial mutations precipitate neuronal degeneration and PD.

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Cite Ramonet D, Daher JP, Lin BM, Stafa K, Kim J, Banerjee R, Westerlund M, Pletnikova O, Glauser L, Yang L, Liu Y, Swing DA, Beal MF, Troncoso JC, McCaffery JM, Jenkins NA, Copeland NG, Galter D, Thomas B, Lee MK, Dawson TM, Dawson VL, Moore DJ.

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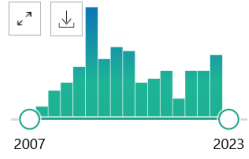
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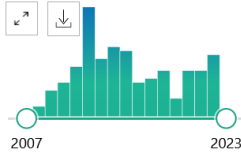
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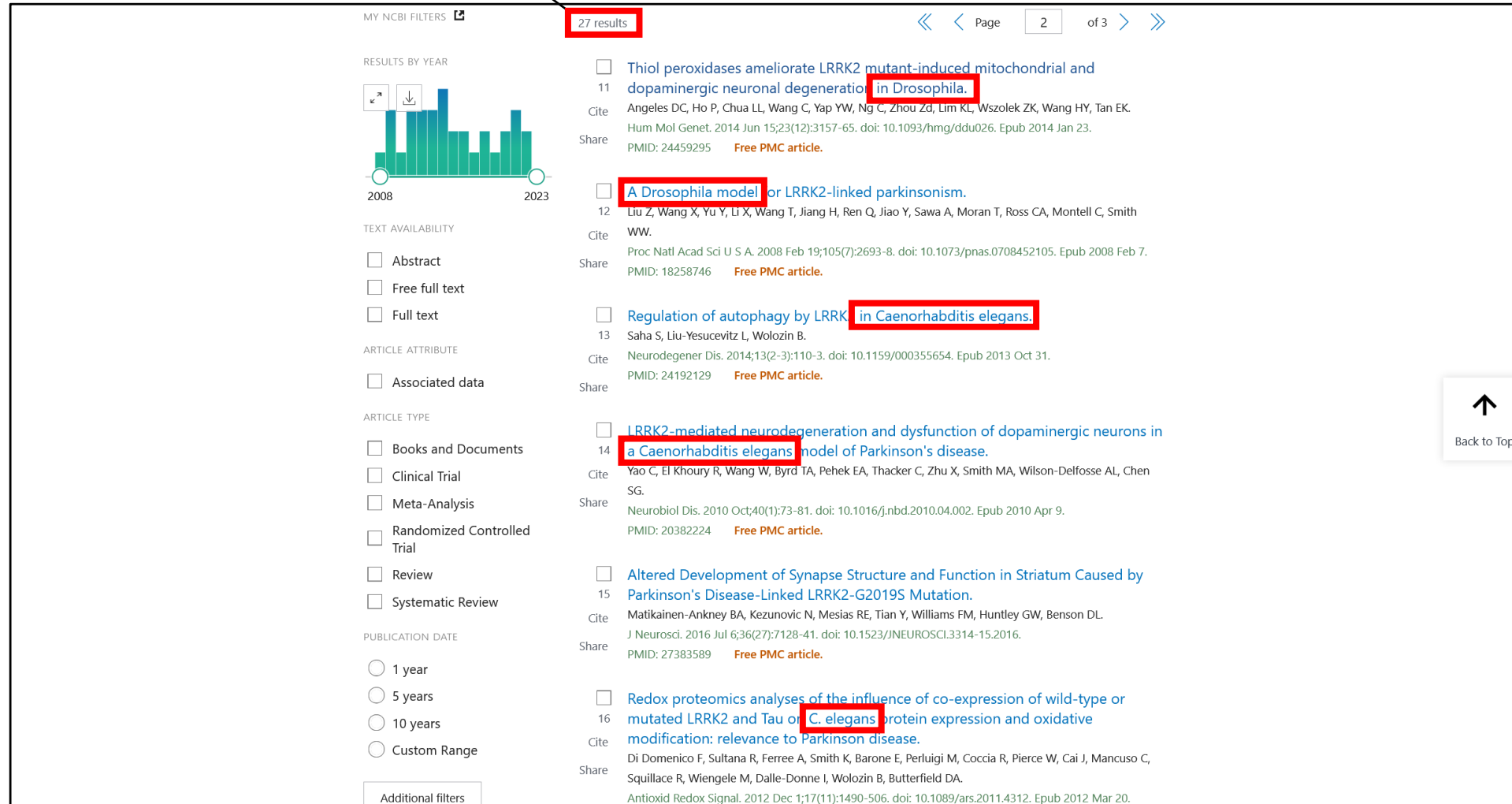
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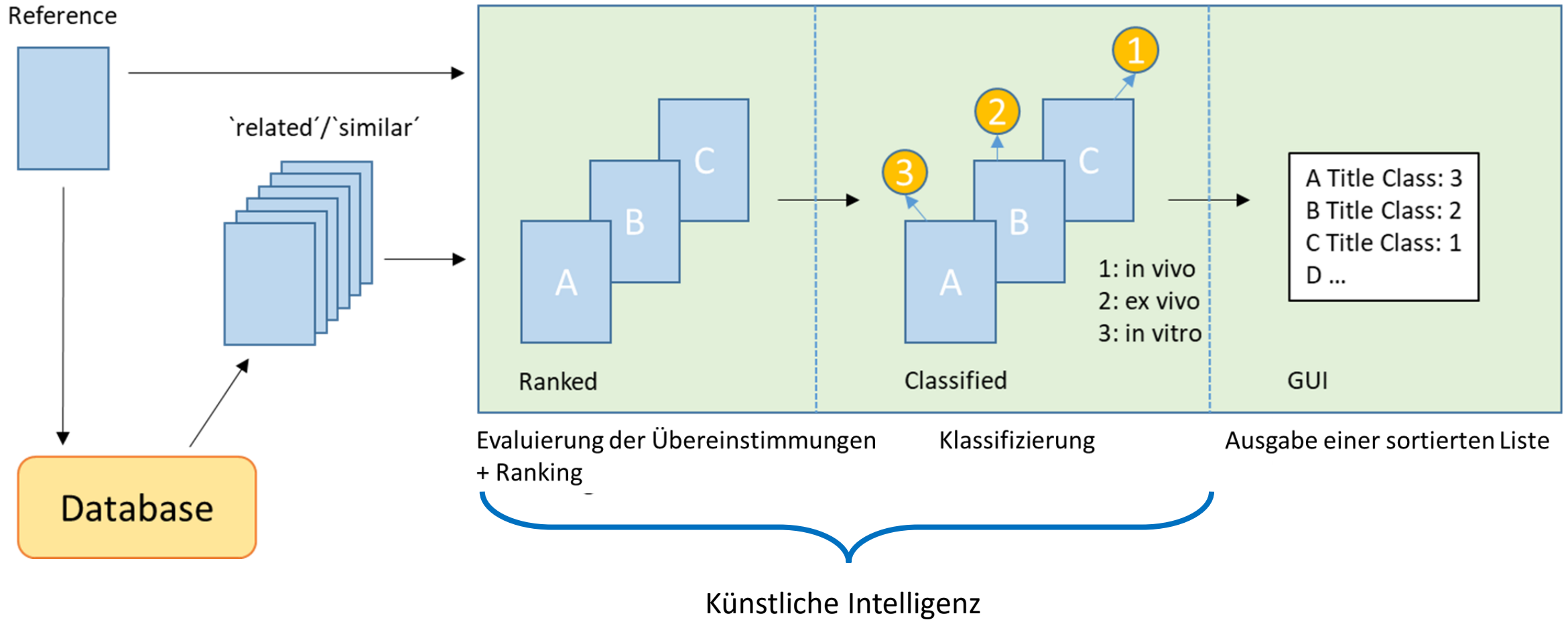
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Filters for the used experimental models

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.	18258746		[2]	[33]	?	A Drosophila model for LRRK2-linked parkinsonism.	
3.	21698001		[3]	[7]	?	Temporal expression of mutant LRRK2 in adult rats impairs dopamine reuptake.	
4.	29386392		[4]	[17]	?	Robust kinase- and age-dependent dopaminergic and norepinephrine neurodegeneration in LRRK2 G2019S transgenic mice.	
5.	20382224		[5]	[10]	?	LRRK2-mediated neurodegeneration and dysfunction of dopaminergic neurons in a Caenorhabditis	
10.	25836420		[10]	[14]	?	Progressive dopaminergic alterations and mitochondrial abnormalities in LRRK2 G2019S knock-in mice.	

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Invertebrate

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Invertebrate

☐ all

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☐ all

Virtual Model

☒ all

Others

☒ all

	PMID		SMAFIRA Rank	PubMed® Rank	My Rank	Title	Labels
1.	19667187		[16]	[44]		R1441C mutation in LRRK2 impairs dopaminergic neurotransmission in mice.	
2.	30954703		[47]	[61]		An integrated transcriptomics and proteomics analysis reveals functional endocytic dysregulation caused by mutations in LRRK2.	
3.	28973664		[53]	[53]		LRRK2 G2019S-induced mitochondrial DNA damage is LRRK2 kinase dependent and inhibition restores mtDNA integrity in Parkinson's disease.	
4.	29038245		[59]	[22]		Mitochondrial Calcium Dysregulation Contributes to Dendrite Degeneration Mediated by PD/LBD-Associated LRRK2 Mutants.	
5.	29855356		[61]	[93]		LRRK2 activity does not dramatically alter α -synuclein pathology in primary neurons.	
10.	38785531		[119]	[142]		Expression of G2019S LRRK2 in Rat Primary Astrocytes Mediates Neurotoxicity and Alters the Dopamine Synthesis Pathway in N27 Cells via Astrocytic Proinflammatory Cytokines and Neurotrophic Factors.	

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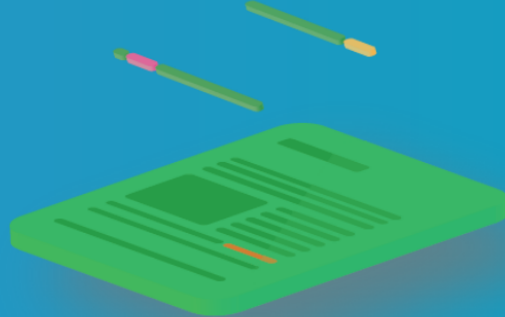
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LitSense²  Tutorial

Search for sentences or longer passages in 38+ million biomedical publications in PubMed and PMC.

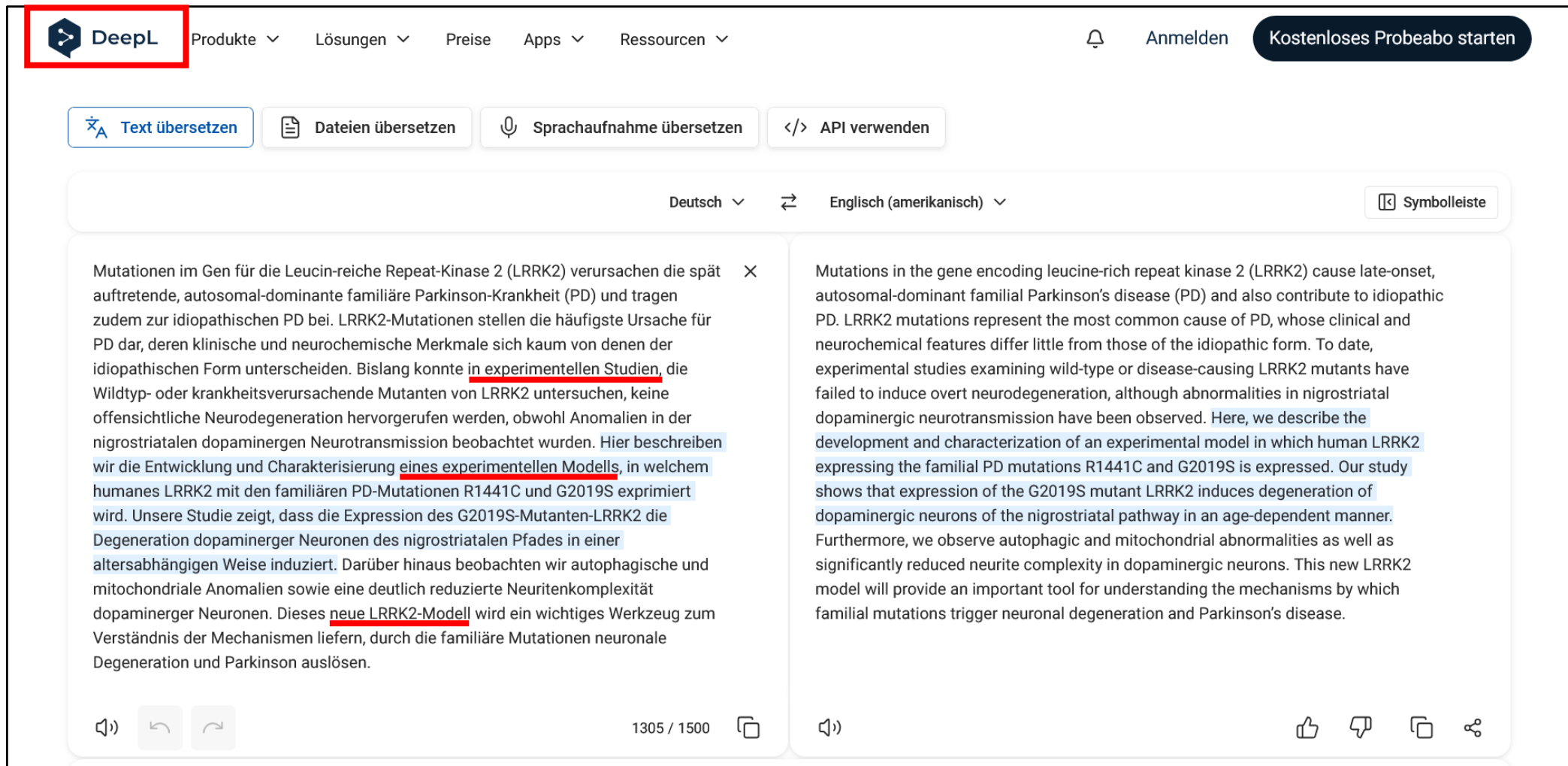
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bzw.
280 Millionen Textpassagen

Beispiel: deutsche Zusammenfassung, welche das Modell 'abstrahiert'



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Text übersetzen Dateien übersetzen Sprachaufnahme übersetzen API verwenden

Deutsch ↕ Englisch (amerikanisch) Symbolleiste

Mutationen im Gen für die Leucin-reiche Repeat-Kinase 2 (LRRK2) verursachen die spät auftretende, autosomal-dominante familiäre Parkinson-Krankheit (PD) und tragen zudem zur idiopathischen PD bei. LRRK2-Mutationen stellen die häufigste Ursache für PD dar, deren klinische und neurochemische Merkmale sich kaum von denen der idiopathischen Form unterscheiden. Bislang konnte in experimentellen Studien, die Wildtyp- oder krankheitsverursachende Mutanten von LRRK2 untersuchen, keine offensichtliche Neurodegeneration hervorgerufen werden, obwohl Anomalien in der nigrostriatalen dopaminergen Neurotransmission beobachtet wurden. Hier beschreiben wir die Entwicklung und Charakterisierung eines experimentellen Modells, in welchem humanes LRRK2 mit den familiären PD-Mutationen R1441C und G2019S exprimiert wird. Unsere Studie zeigt, dass die Expression des G2019S-Mutanten-LRRK2 die Degeneration dopaminergener Neuronen des nigrostriatalen Pfades in einer altersabhängigen Weise induziert. Darüber hinaus beobachten wir autophagische und mitochondriale Anomalien sowie eine deutlich reduzierte Neuritenkomplexität dopaminergener Neuronen. Dieses neue LRRK2-Modell wird ein wichtiges Werkzeug zum Verständnis der Mechanismen liefern, durch die familiäre Mutationen neuronale Degeneration und Parkinson auslösen.

Mutations in the gene encoding leucine-rich repeat kinase 2 (LRRK2) 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, whose clinical and neurochemical features differ little from those of the idiopathic form. To date, experimental studies examining wild-type or disease-causing LRRK2 mutants have failed to induce overt neurodegeneration, although abnormalities in nigrostriatal dopaminergic neurotransmission have been observed. Here, we describe the development and characterization of an experimental model in which human LRRK2 expressing the familial PD mutations R1441C and G2019S is expressed. Our study shows that expression of the G2019S mutant LRRK2 induces degeneration of dopaminergic neurons of the nigrostriatal pathway in an age-dependent manner. Furthermore, we observe autophagic and mitochondrial abnormalities as well as significantly reduced neurite complexity in dopaminergic neurons. This new LRRK2 model will provide an important tool for understanding the mechanisms by which familial mutations trigger neuronal degeneration and Parkinson's disease.

Ergebnis: sehr viele Treffer, wenig Filtermöglichkeiten

LitSense² Passages Mutations in the gene encoding leucine-rich repeat kinase 2 (LRRK2) cause late-onset, autosomal-dominant familial NIH NLM Tutorial

Showing passages 1 to 10. Page 1 of 3004

SECTIONS

- ☒ TITLE
- ☒ ABSTRACT
- ☐ TITLE AND ABSTRACT
- ☐ INTRODUCTION
- ☐ METHODS
- ☐ RESULTS
- ☐ DISCUSSION
- ☐ CONCLUSION

PUBLICATION DATE

- ☐ LAST YEAR
- ☐ LAST 3 YEARS
- ☐ LAST 5 YEARS

☒ BIOCONCEPTS

☒ GENE ☒ DISEASE

☒ CHEMICAL ☒ MUTATION

☒ SPECIES ☒ CELLLINE

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PMID21494637 • PMC3071839 2011

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

Ramonet D, Daher JP ... Moore DJ • PLoS One

TITLE AND ABSTRACT

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 LRRK2 bearing the familial PD mutations, R1441C and G2019S. Our study demonstrates that expression of G2019S mutant LRRK2 induces the degeneration of nigrostriatal pathway dopaminergic neurons in an age-dependent manner. In addition, we observe autophagic and mitochondrial abnormalities in the brains of aged G2019S LRRK2 mice and markedly reduced neurite complexity of cultured dopaminergic neurons. These new LRRK2 transgenic mice will provide important tools for understanding the mechanism(s) through which familial mutations precipitate neuronal degeneration and PD.

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PMID35229999 • PMC8879627 2022

Animal models in the study of Alzheimer's disease and Parkinson's disease: A historical perspective.

Banerjee R, Rai A ... Tare M • Animal Model Exp Med

Alternativmethoden auf den ersten Seiten

LitSense² Passages Mutations in the gene encoding leucine-rich repeat kinase 2 (LRRK2) cause late-onset, autosomal-dominant NIH NLM Tutorial

Showing passages 21 to 30. < Page **3** of 3004 >

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- ☐ TITLE AND ABSTRACT
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- ☐ DISCUSSION
- ☐ CONCLUSION

PUBLICATION DATE

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- ☐ LAST 5 YEARS

BIOCONCEPTS

- ☒ GENE
- ☒ DISEASE
- ☒ CHEMICAL
- ☒ MUTATION
- ☒ SPECIES
- ☒ CELLLINE

PMID18258746 2008

A Drosophila model for LRRK2-linked parkinsonism.

Liu Z, Wang X ... Smith WW • Proc Natl Acad Sci U S A

TITLE AND ABSTRACT

A Drosophila model for LRRK2-linked parkinsonism. Mutations in the leucine-rich repeat kinase (LRRK2) gene cause late-onset autosomal dominant Parkinson's disease (PD) with pleiomorphic pathology. Previously, we and others found that expression of mutant LRRK2 causes neuronal degeneration in cell culture. Here we used the GAL4/UAS system to generate transgenic Drosophila expressing either wild-type human LRRK2 or LRRK2-G2019S, the most common mutation associated with PD. Expression of either wild-type human LRRK2 or LRRK2-G2019S in the photoreceptor cells caused retinal degeneration. Expression of LRRK2 or LRRK2-G2019S in neurons produced adult-onset selective loss of dopaminergic neurons, locomotor dysfunction, and early mortality. Expression of mutant G2019S-LRRK2 caused a more severe parkinsonism-like phenotype than expression of equivalent levels of wild-type LRRK2. Treatment with L-DOPA improved mutant LRRK2-induced locomotor impairment but did not prevent the loss of tyrosine hydroxylase-positive neurons. To our knowledge, this is the first in vivo "gain-of-function" model which recapitulates several key features of LRRK2-linked human parkinsonism. These flies may provide a useful model for studying LRRK2-linked pathogenesis and for future therapeutic screens for PD intervention.

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PMID34539790 • PMC8448601 2021

Brain Organoids: Studying Human Brain Development and Diseases in a Dish.

Xu J, Wen Z • Stem Cells Int

PMID30898538 • PMC6751032 2020

Using induced pluripotent stem cell neuronal models to study neurodegenerative diseases.

Zhang X, Hu D ... Qi X • Biochim Biophys Acta Mol Basis Dis

PMID39859333 • PMC11766061 2025

Two- and Three-Dimensional In Vitro Models of Parkinson's and Alzheimer's Diseases: State-of-the-Art and Applications.

Solana-Manrique C, Sánchez-Pérez AM ... Muñoz-Descalzo S • Int J Mol Sci

‘Passage’-Suche mit diesem Abstract wiederholen (iterative Suche)

Recherchen mit Prompts und Retrieval Augmented Generation (RAG)

Prompts & RAG

- Man kann Fragen in natürlicher Sprache stellen
- Man bekommt Antworten in natürlicher Sprache
- Für die Recherche muss man RAG-Systeme verwenden, diese liefern auch noch die Literatur-Zitate, auf denen die Antwort beruht

Bei einigen Versionen:

- Sehr eingeschränkte Liste mit Literatur-Zitaten (n =10 - 20)
- Teilweise kein bzw. eingeschränkt transparenter Recherche-/Auswahlprozess
- Variabilität der Antworten

consensus

<https://consensus.app/>

**Retrieval Augmented Generation,
Consensus Meter**

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General

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Design

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☐ SYSTEMATIC REVIEW

☐ RCT

☐ NON-RCT

☐ OBSERVATIONAL STUDY

☐ LITERATURE REVIEW

☐ CASE REPORT

☐ ANIMAL STUDY

☐ IN VITRO STUDY

Sample size

At least 1

Duration

At least 0

Day

Controlled studies only

Human studies only

Fields of study

23 fields of study

Consensus: Antwort + „Consensus Meter“

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Gibt es eine in vitro Methode, mit welcher der Versuchszweck erreicht werden kann, der in der wissenschaftlichen Publikation mit dem Titel "Dopaminergic neuronal loss, reduced neurite complexity and autophagic..."

Pro · 2 steps · 20 sources

Ja, es gibt mehrere etablierte in-vitro-Modelle, die den in der Mausstudie beschriebenen Versuchszweck (dopaminergen Neuronenverlust, reduzierte Neuritkomplexität, Autophagiedefekte bei LRRK2-G2019S) nachbilden können.

Can in vitro models reproduce dopaminergic loss, reduced neurite complexity and autophagy abnormalities caused by LRRK2-G2019S?

14

Yes

100%

Possibly

0%

Mixed

0%

No

0%

All details

FIGURE 1

Forschungsübereinstimmung zu LRRK2-G2019S in vitro Modellen

Ask a follow up...

Pro Deep Corpus

Filter

References / Gibt es eine in vitro Methode, mit welcher...

Results

1

Disease-specific phenotypes in dopamine neurons from human iPS-based models of genetic and sporadic Parkinson's disease

KEY TAKEAWAY · Induced pluripotent stem cells from patients with idiopathic Parkinson's disease and familial Parkinson's disease show morphological alterations and autophagic compromise, allowing for investigation of disease-related pathogenesis.

IN VITRO STUDY HIGHLY CITED

2012 · 559 citations · A. Sánchez-Danes et al.

2

3D Cultures of Parkinson's Disease-Specific Dopaminergic Neurons for High Content Phenotyping and Drug Testing

KEY TAKEAWAY · 3D microfluidics optimizes Parkinson's disease-specific neurons, revealing robust endophenotypes and supporting personalized drug development.

IN VITRO STUDY HIGHLY CITED

2018 · 120 citations · S. Bolognin et al.

3

In Vitro CRISPR/Cas9-Directed Gene Editing to Model LRRK2 G2019S Parkinson's Disease in Common Marmosets

KEY TAKEAWAY · CRISPR/Cas9-directed gene editing successfully models LRRK2 G2019S Parkinson's disease in common marmosets, demonstrating the feasibility of creating a novel genetic-based model for PD.

IN VITRO STUDY RIGOROUS JOURNAL

72

BfR

Consensus: Antwort

Relevante in-vitro-Ansätze

1. hiPSC-abgeleitete dopaminerge Neurone (2D)

- Patientenspezifische iPSCs mit LRRK2-G2019S liefern dopaminerge Neurone mit:
 - **reduzierter Neuritenzahl/-Arborisierung** 1  5 16  18
 - **Autophagiedysfunktion / Autophagievakuolen** 1  3  5 6 7 12
 - **erhöhtem Zelltod/Apoptose**  2  3 6  18
- Diese Phänotypen entsprechen eng den Befunden im G2019S-Transgen-Mausmodell (Neuritenverkürzung, Autophagieveränderungen, dopaminergem Verlust)  17

2. 3D-Kulturen und Organoide

- 3D-Mikrofluidik-Kulturen aus LRRK2-G2019S-Neuronen zeigen **ausgeprägte dopaminerge Degeneration, verringerte Verzweigung, mitochondriale Defekte**, ohne externe Stressoren  2
- 3D-Seiden-Gerüste und Midbrain-Organoide aus LRRK2-G2019S-hiPSCs bilden **dichte dopaminerge Netzwerke**, mit **α -Synuclein-Pathologie, metabolischen Veränderungen und Neuronendegeneration** nach  2 4  8 11

3. Einfachere Zelllinien-Modelle

- Differenzierte SH-SY5Y-Zellen mit überexprimiertem G2019S-LRRK2: **Neuritenverkürzung + massive Autophagievakuolen**; Autophagiemodulation (LC3/Atg7-Knockdown, Rapamycin) beeinflusst den Phänotyp direkt 7
- LUHMES-Zellen mit G2019S-Überexpression: dopaminerge-ähnliche Neurone mit **mitochondrialen und lysosomalen Veränderungen** und **erhöhter dopaminergem Zellverlust**  20

Consensus: Antwort

Beispielhafte Übersicht			
Aspekt	In-vitro-Modell erfüllt?	Typische Systeme	Quellen
Dopaminergem Zellverlust	Ja	hiPSC-DAN, 3D, LUHMES	1 ☒ 2 ☒ 3 ☒ 6 ☒ 18 ☒ 20
Reduzierte Neuritenkomplexität	Ja	hiPSC-DAN, SH-SY5Y	1 ☒ 3 ☒ 5 ☒ 7 ☒ 16 ☒ 18
Autophagische Abnormalitäten	Ja	hiPSC-DAN, SH-SY5Y, Neuronen mit G2019S	1 ☒ 3 ☒ 5 ☒ 7 ☒ 12 ☒ 17

FIGURE 2 Vergleich zentraler Mausphänotypen mit in vitro Modellen

Schlussfolgerung

Mehrere gut charakterisierte in-vitro-Systeme – insbesondere human-iPSC-abgeleitete dopaminerge Neurone (2D und 3D) und SH-SY5Y/LUHMES-Modelle mit G2019S-LRRK2 – reproduzieren dopaminergen Verlust, verminderte Neuritenkomplexität und Autophagiedefekte und können somit den in der genannten Mauspublikation beschriebenen Versuchszweck erreichen.

Consensus Deep: Details zu Literaturrecherche

2. Methoden

Für diese Übersicht wurden über 170 Millionen wissenschaftliche Publikationen aus Datenbanken wie Semantic Scholar und PubMed durchsucht. Insgesamt wurden 971 relevante Arbeiten identifiziert, davon 269 nach Duplikatprüfung gescreent. Nach weiteren Relevanzfiltern blieben 235 Arbeiten übrig; die 50 relevantesten wurden für diese Analyse ausgewählt.

Search Strategy

[All details >](#)

FIGURE 2 Flussdiagramm des Such- und Auswahlprozesses für relevante Literatur.

Es wurden acht Suchgruppen mit insgesamt zwanzig gezielten Suchanfragen verwendet, um alle Aspekte von in vitro Modellen für LRRK2-G2019S-assoziierte Pathologien abzudecken.

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LRRK2 In vitro Methoden Alternativen 1

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2. Methoden

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Search Strategy

Hide details

Identification
N = 971

Screening
N = 269

Eligibility
N = 235

Included
N = 50

Identified 958 papers that matched 20 Consensus searches
+ 958 identified

Removed papers with missing abstracts
Removed duplicates
- 702 removed

Removed papers with low semantic relevance to each search
- 34 removed

Selected the to highest quality p after final rank
- 185 removed

FIGURE 2

Flussdiagramm des Such- und Auswahlprozesses für relevante Literatur.

Es wurden acht Suchgruppen mit insgesamt zwanzig gezielten Suchanfragen verwendet, um alle Aspekte von in vitro Modellen für LRRK2-G2019S-assoziierte Pathologien abzudecken.

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Ask a follow up...

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Deep Search

Gibt es eine in vitro Methode, mit welcher der Versuchszweck erreicht werden kann, der in der...

971
Identified

269
Screened

235
Eligible

50
Included

Initial survey & search strategy

in vitro methods for modeling dopaminergic neuronal loss, neurite complexity, autophagic abnormalities, G2019S mutant LRRK2

NON-RCT IN VITRO

Conducting an initial survey of the research based on your query. I'll use the findings to come up with a detailed search strategy for this literature review.

Ich werde zunächst die Publikation und deren Versuchszweck analysieren, dann gezielt nach in vitro Modellen suchen, die dopaminerge Neuronen und LRRK2-Mutationen abbilden, um Methoden und Limitationen systematisch zu erfassen.

1 search 1 filter · 41 new eligible papers

Clarify experimental aims

3 searches 1 filter · 57 new eligible papers

Survey in vitro models

3 searches 1 filter · 40 new eligible papers

Rephrase for technical synonyms

3 searches 1 filter · 18 new eligible papers

Contrast with in vivo findings

3 searches 1 filter · 25 new eligible papers

Background on LRRK2 models

3 searches 1 filter · 24 new eligible papers

Explore adjacent mechanisms

3 searches 1 filter · 17 new eligible papers

Consensus 101: Best Practice...

Daniel · 49m

Explore citation graph

Consensus **Deep**: Evidence Strength

Claim	Evidence Strength	Reasoning	Papers
iPSC-basierte Modelle können alle drei Zielpheänotypen zuverlässig abbilden	 Strong	Mehrere unabhängige Studien zeigen konsistente Ergebnisse bei Zellverlust/ Neuritendefekten/ Autophagiestörung	 2  3  4  5  6
CRISPR/Cas9-editierte isogene Linien minimieren Hintergrundeffekte	 Strong	Vergleich zwischen mutierten/korrigierten Linien zeigt spezifische Effekte der Mutation	 1  4
Autophagiedefekte sind direkt messbar mittels LC3/p62/EM	 Strong	Standardisierte Marker & Bildgebung liefern reproduzierbare Resultate	 6  7
3D-Kulturen/Organoide bieten physiologischere Bedingungen	 Moderate	Bessere Nachbildung zellulärer Interaktionen; noch limitiert durch technische Herausforderungen	 3  31
SH-SY5Y-Zellmodelle sind weniger physiologisch als iPSC-Neurone	 Moderate	Tumorlinien unterscheiden sich teils stark von primären/ menschlichen Zellen	 7
Langzeitfolgen & komplexe Interaktionen bleiben schwer abbildbar	 Moderate	Organoide/Co-Kulturen adressieren dies teilweise; vollständige In-vivo-Nachbildung bleibt schwierig	 31

FIGURE Zentrale Aussagen & Evidenzstärke aus den analysierten Arbeiten.

Andere AI Tools:

- Undermind, undermind.ai**
- Elicit, elicit.com**
- SciSpace, scispace.com/de**

Bundesinstitut für Risikobewertung
bfr.bund.de

BfR | Risiken erkennen –
Gesundheit schützen

Verbraucherschutz zum Mitnehmen

BfR2GO – das Wissenschaftsmagazin des BfR

bfr.bund.de/de/wissenschaftsmagazin_bfr2go.html

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 social.bund.de/@bfr

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Die Leistungsfähigkeit der Recherchestrategie messen

Trefferlisten können sehr umfangreich sein → wie einschränken?

1.875 Resultate

PubMed.gov

(parkinson) AND (in vitro techniques[MeSH Terms])

Search

Advanced Create alert Create RSS User Guide

Save Email Send to Sorted by: Best match Display options

MY NCBI FILTERS

RESULTS BY YEAR

1958 2023

TEXT AVAILABILITY

☐ Abstract

☐ Free full text

☐ Full text

ARTICLE ATTRIBUTE

☐ Associated data

ARTICLE TYPE

☐ Books and Documents

☐ Clinical Trial

☐ Meta-Analysis

☐ Randomized Controlled Trial

1,875 results

1 ☐ Reversing a model of **Parkinson's disease** with in situ converted nigral neurons.

Cite Qian H, Kang X, Hu J, Zhang D, Liang Z, Meng F, Zhang X, Xue Y, Maimon R, Dowdy SF, Devaraj NK, Zhou Z, Mobley WC, Cleveland DW, Fu XD.

Share Nature. 2020 Jun;582(7813):550-556. doi: 10.1038/s41586-020-2388-4. Epub 2020 Jun 24. PMID: 32581380 [Free PMC article.](#)

2 ☐ Selective targeting of the TLR2/MyD88/NF-κB pathway reduces α-synuclein spreading in vitro and in vivo.

Cite Dutta D, Jana M, Majumder M, Mondal S, Roy A, Pahan K.

Share Nat Commun. 2021 Sep 10;12(1):5382. doi: 10.1038/s41467-021-25767-1. PMID: 34508096 [Free PMC article.](#)

3 ☐ Crosstalk between astrocytes and microglia results in increased degradation of α-synuclein and amyloid-β aggregates.

Cite Rostami J, Mothes T, Kolahdouzan M, Eriksson O, Moslem M, Bergström J, Ingelsson M, O'Callaghan P, Healy LM, Falk A, Erlandsson A.

Share J Neuroinflammation. 2021 Jun 3;18(1):124. doi: 10.1186/s12974-021-02158-3. PMID: 34082772 [Free PMC article.](#)

4 ☐ LRRK2 modifies α-syn pathology and spread in mouse models and human neurons.

Cite Bieri G, Brahic M, Bousset L, Couthouis J, Kramer NJ, Ma R, Nakayama L, Monbureau M, Defensor E, Schüle B, Shamlou M, Melki R, Gitler AD.

Share Acta Neuropathol. 2019 Jun;137(6):961-980. doi: 10.1007/s00401-019-01995-0. Epub 2019 Mar 29. PMID: 30927072 [Free PMC article.](#)

Einschränken durch weitere Suchbegriffe, Beispiel: spezifischer Endpunkt

295 Resultate

AND (neurodegeneration)

PubMed.gov

parkinson AND (neurodegeneration) AND (in vitro techniques[MeSH Terms])

Search

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Save Email Send to Sorted by: Best match Display options

MY NCBI FILTERS

295 results

RESULTS BY YEAR

1986 2023

TEXT AVAILABILITY

☐ Abstract

☐ Free full text

☐ Full text

ARTICLE ATTRIBUTE

☐ Associated data

ARTICLE TYPE

☐ Books and Documents

☐ Clinical Trial

☐ Meta-Analysis

☐ Randomized Controlled Trial

1 ☐ Reversing a model of **Parkinson's disease** with in situ converted nigral neurons.

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Cite Nature. 2020 Jun;582(7813):550-556. doi: 10.1038/s41586-020-2388-4. Epub 2020 Jun 24.

Share PMID: 32581380 Free PMC article.

2 ☐ Cellular models of alpha-synuclein toxicity and aggregation.

Delenclos M, Burgess JD, Lamprokostopoulou A, Outeiro TF, Vekrellis K, McLean PJ.

Cite J Neurochem. 2019 Sep;150(5):566-576. doi: 10.1111/jnc.14806. Epub 2019 Jul 30.

Share PMID: 31265132 Free PMC article. Review.

3 ☐ Patient-Specific iPSC-Derived Astrocytes Contribute to Non-Cell-Autonomous **Neurodegeneration in Parkinson's Disease**.

di Domenico A, Carola G, Calatayud C, Pons-Espinal M, Muñoz JP, Richaud-Patin Y, Fernandez-Carasa I, Gut M, Faella A, Parameswaran J, Soriano J, Ferrer I, Tolosa E, Zorzano A, Cuervo AM, Raya A, Consiglio A.

Cite Stem Cell Reports. 2019 Feb 12;12(2):213-229. doi: 10.1016/j.stemcr.2018.12.011. Epub 2019 Jan 10.

Share PMID: 30639209 Free PMC article.

4 ☐ Modelling neurodegenerative **disease** using brain organoids.

Wray S.

Cite Semin Cell Dev Biol. 2021 Mar;111:60-66. doi: 10.1016/j.semcdb.2020.05.012. Epub 2020 Jun 5.

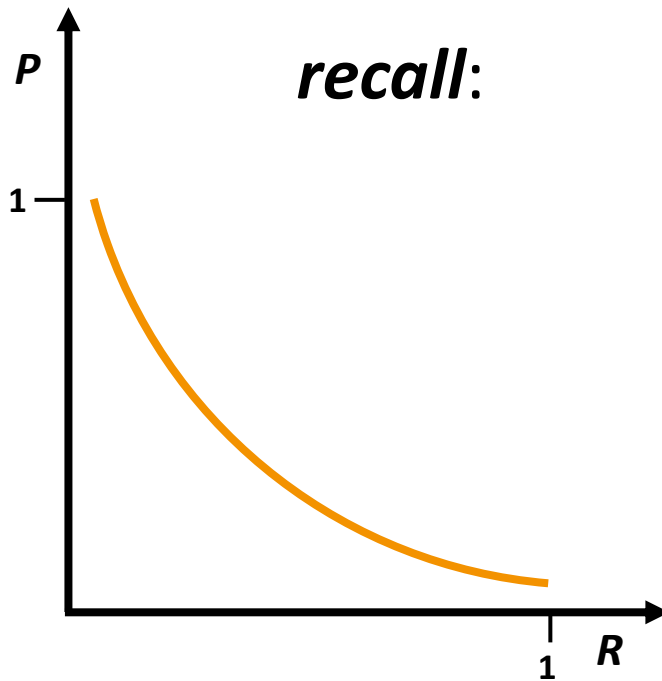
Share PMID: 32513498 Review.

ACHTUNG!
→ recall!

Die Kenngrößen 'precision' und 'recall'

precision: Anteil der für die Fragestellung relevanten Dokumente in der Trefferliste

recall: Zahl der gefundenen relevanten Dokumente im Verhältnis zur Gesamtzahl der real existierenden relevanten Dokumente



Recherchieren mit Positivkontrollen

Recherchieren mit Positivkontrollen (d.h. Relevanz ist bekannt)

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[User Guide](#)

Add terms to the query box

All Fields Enter a search term

OR

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Search

Add to History

PMIDs der Positivkontrollen

(21248115) OR (26253900) OR (21494637) OR (26744332)

History and Search Details

Your history is currently empty! As you use PubMed your recent searches will appear here.

> J Neurosci. 2011 Jan 19;31

A rat model of induced by the G2019S mutati

Julien Dusonchet¹, Olexiy K
Darren J Moore, Bernard L S

Affiliations + expand

PMID: 21248115 PMCID: P

PMID = PubMed Identifier

Recherchieren mit Positivkontrollen, Strategieentwicklung



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Enter a search term

ADD

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Query box

Enter / edit your search query here

Search

History and Search Details

[Download](#)
[Delete](#)

Search	Actions	Details	Query	Results	Time
#1	...	>	Search: (21248115) OR (26253900) OR (21494637) OR (26744332)	4	01:52:11

Showing 1 to 1 of 1 entries

4 Resultate

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Query box

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Search

Suche mit dem Begriff „parkinson“

History and Search Details

Download

Delete

Search	Actions	Details	Query	Results	Time
#2	...	>	Search: parkinson	162,517	01:56:23
#1	...	>	Search: (21248115) OR (26253900) OR (21494637) OR (26744332)	4	01:52:11

Showing 1 to 2 of 2 entries

162.517 Resultate

Einsatz von History and Search Details

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Enter a search term

ADD

Show Index

Query box

Enter / edit your search query here

Search

History and Search

Search	Actions
#2	...
#1	...

Showing 1 to 2 of 2 entries

Actions

Details

Query

...

Add query

...

Delete

...

Create alert

Download

Delete

Results	Time
162,517	01:56:23
4	01:52:11

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All Fields

Query box

(21248115) OR (26744332)

ADD

Show Index

Search

History and Search

Search	Actions
#2	...
#1	...

Showing 1 to 2 of 2 entries

to 2 of 2

Download Delete

Results	Time
162,517	01:56:23
(26744332)	4 01:52:11

Actions Details Query

...

Add with AND

Add with OR

Add with NOT

Delete

Create alert

Recherchieren mit Positivkontrollen: Abschätzen des recalls

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All Fields

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Query box

Enter / edit your search query here

Search

History and Search Details

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Search	Actions	Details	Query	Results	Time
#3	...	>	Search: (parkinson) AND ((21248115) OR (26253900) OR (21494637) OR (26744332))	4	02:14:49
#2	...	>	Search: parkinson	162,517	01:56:23
#1	...	>	Search: (21248115) OR (26253900) OR (21494637) OR (26744332)	4	01:52:11

Showing 1 to 3 of 3 entries

Kombination aus #1 und #2

Finde ich meine Positivkontrollen wieder?

4 Resultate

recall dieser Suchstrategie ist 100%

Recherchieren mit Positivkontrollen, Strategieentwicklung



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Enter a search term

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Query box

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Search

Einschränkung mit dem Begriff „neurodegeneration“

History and Search Details

Download
Delete

Search	Actions	Details	Query	Results	Time
#4	...	>	Search: (parkinson) AND (neurodegeneration)	14,776	09:08:02
#3	...	>	Search: (parkinson) AND ((21248115) OR (26253900) OR (21494637) OR (26744332))	4	09:06:40
#2	...	>	Search: parkinson	162,596	09:04:34
#1	...	>	Search: (21248115) OR (26253900) OR (21494637) OR (26744332)	4	09:04:03

Showing 1 to 4 of 4 entries

14.776 Resultate

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Query box

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History and Search Details

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Search	Actions	Details	Query	Results	Time
#5	...	>	Search: ((parkinson) AND (neurodegeneration)) AND ((21248115) OR (26253900) OR (21494637) OR (26744332))	3	09:14:35
#4	...	>	Search: (parkinson) AND (neurodegeneration)	14,776	09:08:02
#3	...	>	Search: (parkinson) AND ((21248115) OR (26253900) OR (21494637) OR (26744332))	4	09:06:40
#2	...	>	Search: parkinson	162,596	09:04:34
#1	...	>	Search: (21248115) OR (26253900) OR (21494637) OR (26744332)	4	09:04:03

Showing 1 to 5 of 5 entries

Finde ich meine Positivkontrollen wieder?

3 Resultate

recall dieser Suchstrategie ist nur 75%

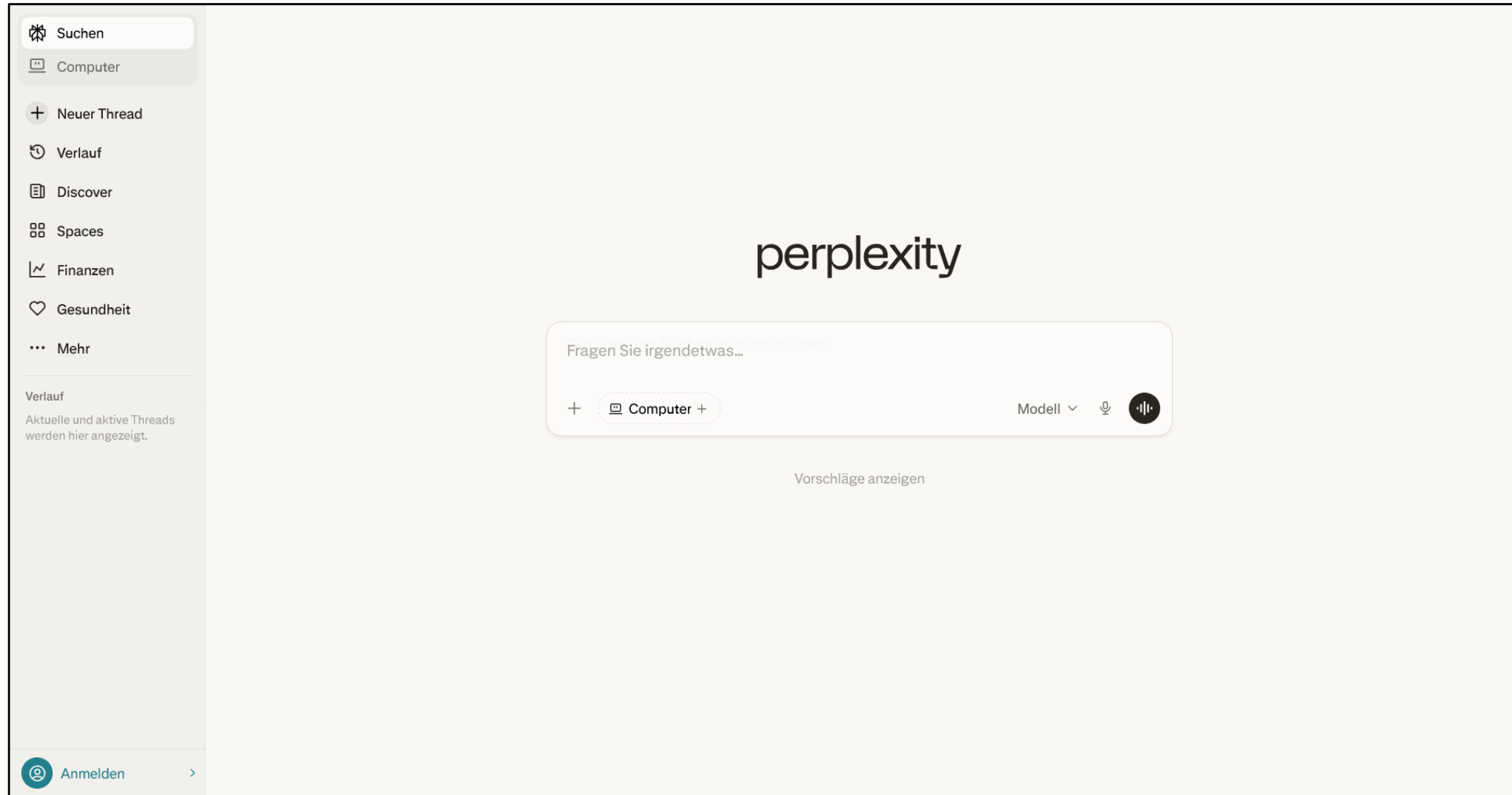
Mögliche Verbesserung durch Synonyme, z.B. „neuronal loss“

perplexity

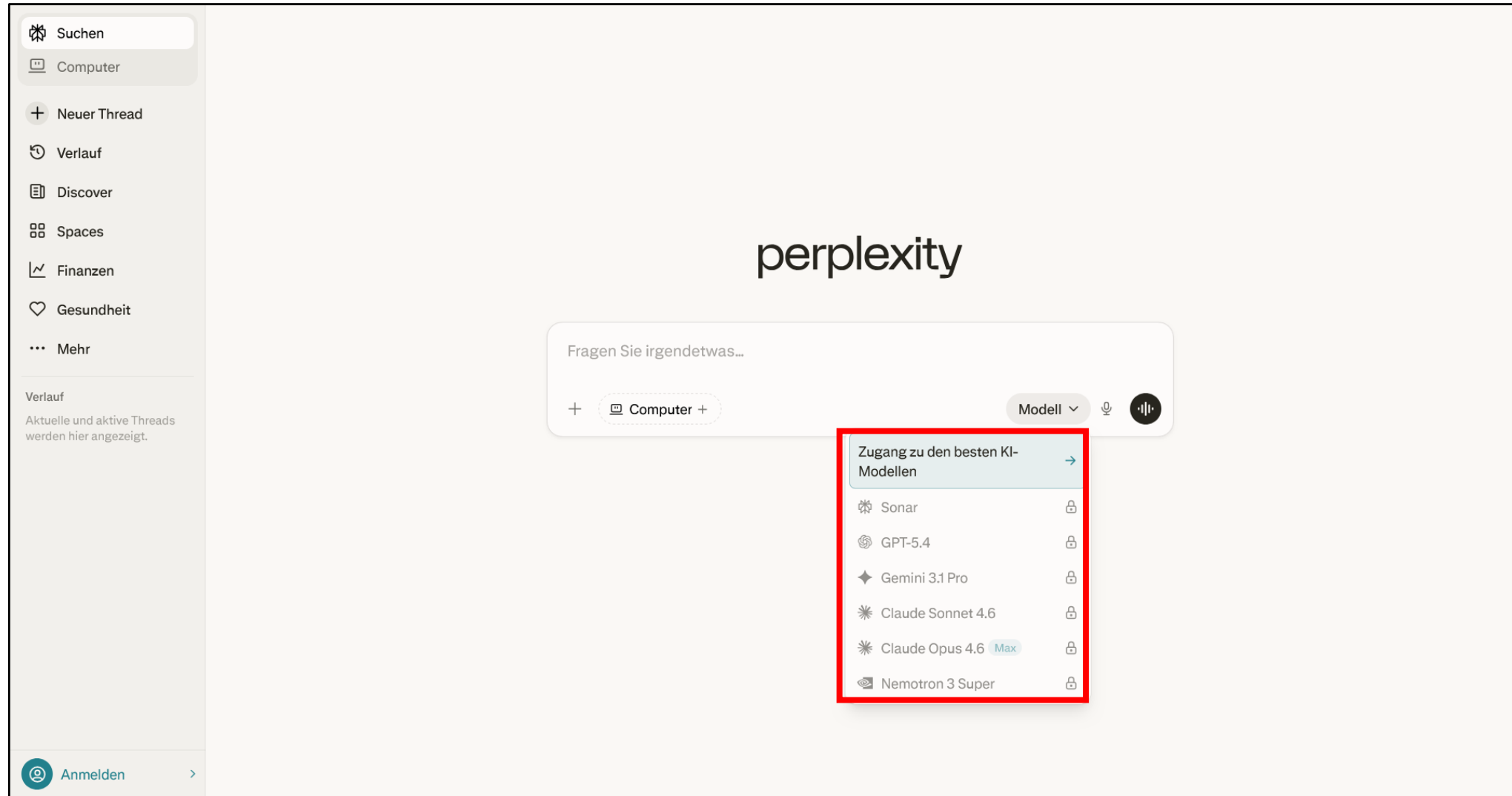
<https://www.perplexity.ai/>

Retrieval Augmented Generation

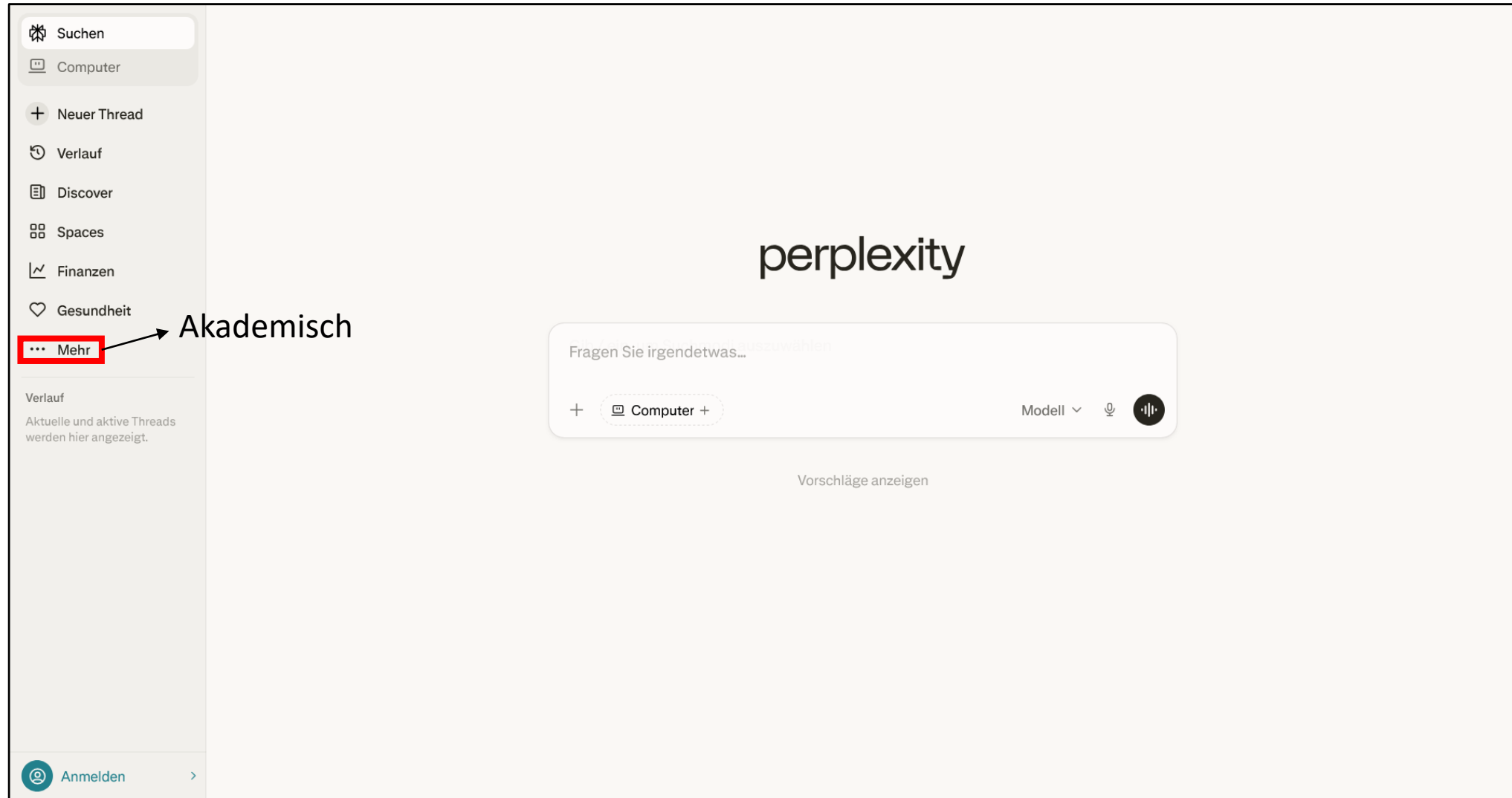
Perplexity:



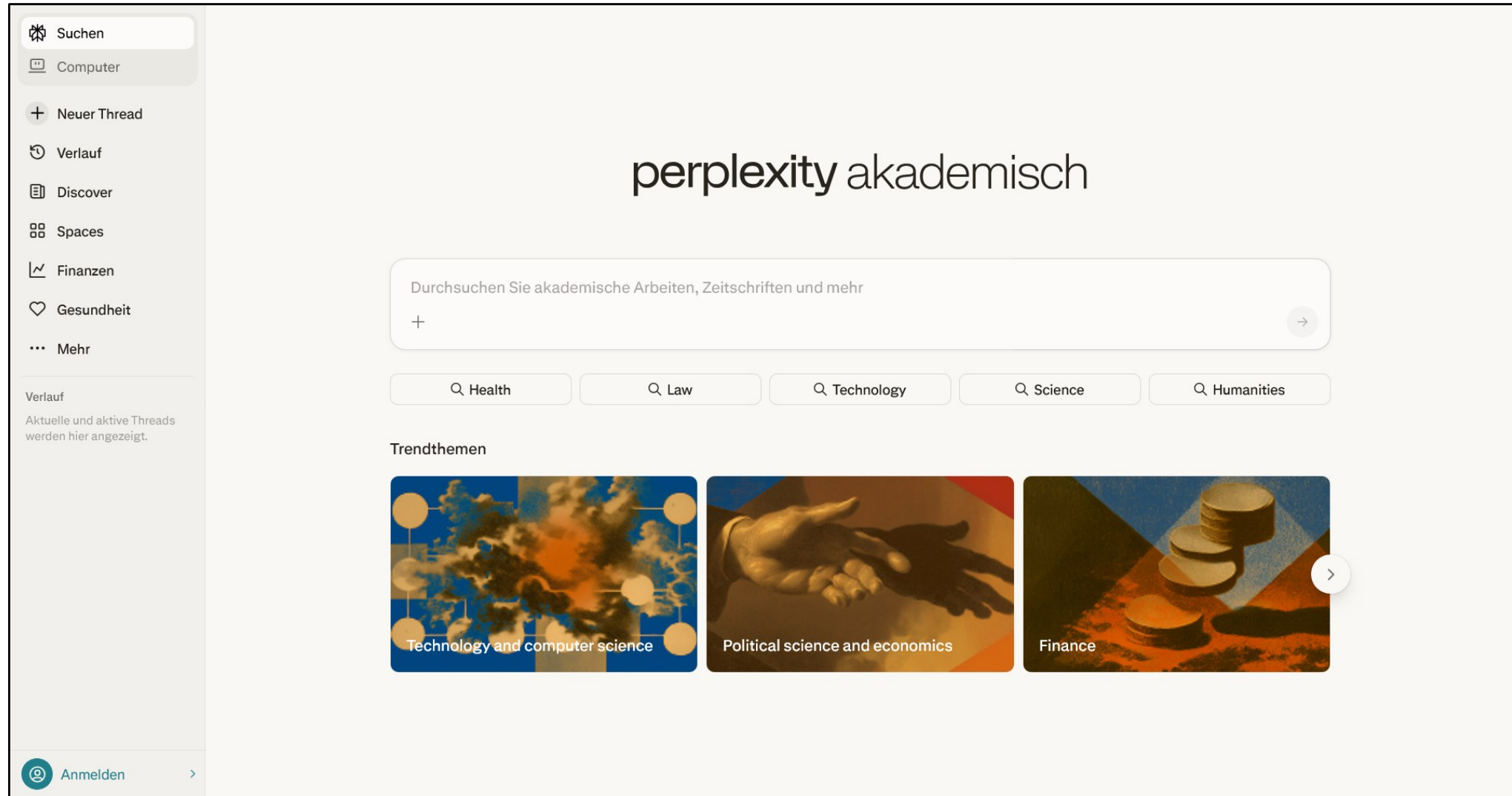
Perplexity: Mit Anmeldung Zugang zu verschiedenen KI-Modellen



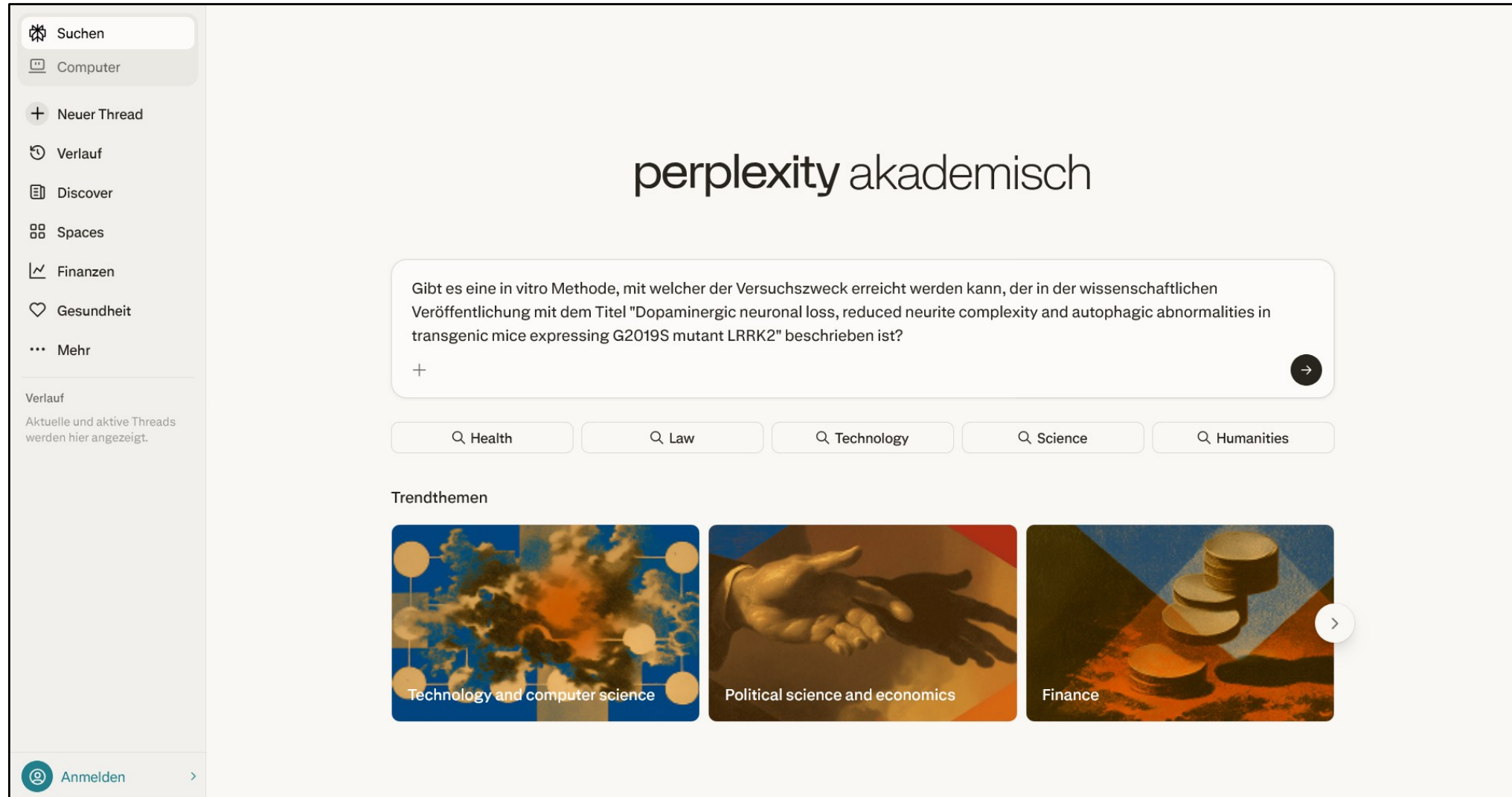
Perplexity: ohne Anmeldung nutzen (1 Frage/Antwort)



Perplexity: Einschränkung auf akademische Arbeiten



Perplexity: Frage



Perplexity: Antwort

Gibt es eine in vitro Methode, mit welcher der Versuchszweck erreicht werden kann, der in der wissenschaftlichen Veröffentlichung mit dem Titel "Dopaminergic neuronal loss, reduced neurite complexity and autophagic abnormalities in transgenic mice expressing G2019S mutant LRRK2" beschrieben ist?

Ja — teilweise. Für die in der Arbeit beschriebenen Effekte gibt es **in vitro**-Modelle, die den Versuchszweck weitgehend abbilden können, vor allem **primäre midbrain-/mesencephale Dopamin-Neuronenkulturen** und **patienten- bzw. geneditierte iPSC-abgeleitete dopaminerge Neuronen** mit der LRRK2-G2019S-Mutation. Diese Systeme werden genutzt, um **reduzierte Neuritenkomplexität, autophagische/mitochondriale Auffälligkeiten** und **zellulären Stress oder Zelltod** unter kontrollierten Bedingungen zu untersuchen.

[pmc.ncbi.nlm.nih +1](#)

Was in vitro gut nachbildbar ist

Die Publikationslage zeigt, dass G2019S-LRRK2 in Zellkulturen typische pathologische Merkmale verursacht, darunter **verkürzte und vereinfachte Neuriten, erhöhte ROS/ mitochondriale Dysfunktion und Autophagie-Veränderungen**. Auch die in der Mausarbeit beobachtete **verminderte neuritische Komplexität** lässt sich in primären Kulturen direkt messen, z. B. mit Morphometrie oder Sholl-Analyse. [pmc.ncbi.nlm.nih +2](#)

Was in vitro nur eingeschränkt ersetzt

Der spezifische **dopaminerge Neuronenverlust im nigrostriatalen System** aus der Mausstudie ist ein **Gewebe- und Netzwerkphänomen** und wird durch einfache Monokulturen nicht vollständig reproduziert. Für diesen Teil sind eher **komplexere Systeme** wie **3D-Midbrain-Organotide** oder Ko-Kulturen sinnvoll, weil sie Zell-Zell-Interaktionen und Reifung besser abbilden. [pmc.ncbi.nlm.nih +1](#)

Praktische Antwort

Wenn dein Ziel ist, die **pathogenen Zellphänotypen der Veröffentlichung experimentell zu erfassen**, dann ist die beste in vitro-Entsprechung meist:

- **iPSC-abgeleitete dopaminerge Neuronen mit LRRK2 G2019S**
- oder **primäre mesenzepale Dopamin-Neuronenkulturen**, in denen man Neuritenlänge, Verzweigung, Autophagie und Mitochondrienparameter misst. [pmc.ncbi.nlm.nih +1](#)

Wenn du möchtest, kann ich dir als Nächstes eine **konkrete in vitro-Versuchsstrategie** skizzieren, die die Endpunkte der Mausstudie möglichst eng nachbildet.

🔗 ⬇️ 📄 ↺ 🗣️ 10 Quellen 👍 🗨️ ...

Nachfrage stellen

+

Modell ▾ 🎤 ⬆️

Perplexity: Quellen

Suchen

Computer

Neuer Thread

Verlauf

Discover

Spaces

Finanzen

Gesundheit

Mehr

Verlauf

Aktuelle und aktive Threads werden hier angezeigt.

Anmelden

Antwort

Links

Bilder

Teilen

10 Quellen

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Nachfrage stellen

Modell

10 Quellen

Quellen für Gibt es eine in vitro Methode, mit welcher der Versuchszweck erreicht werden kann, der in der wissenschaftlichen...

pmc.ncbi.nlm.nih

In Vitro Modeling of Leucine-Rich Repeat Kinase 2 G2019S ...

Leucine-rich repeat kinase 2 (LRRK2) G2019S (glycine to serine) is the most common mutation associated with sporadic and familial Parkinson's disease (PD) with 80% penetrance by age 70. This mutation is found worldwide, with up to 40% of individuals ...

pmc.ncbi.nlm.nih

Dopaminergic Neuronal Loss, Reduced Neurite Complexity and ...

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 ...

pmc.ncbi.nlm.nih

The Parkinson's disease-associated LRRK2-G2019S variant ... - PMC

A growing body of evidence implicates inflammation as a key hallmark in the pathophysiology of Parkinson's disease (PD), with microglia playing a central role in mediating neuroinflammatory signaling in the brain. However, the molecular mechanisms ...

pmc.ncbi.nlm.nih

Dopaminergic neuron loss in mice due to increased levels of wild ...

Understanding the intricate pathogenic mechanisms behind Parkinson's disease (PD) and its multifactorial nature presents a significant challenge in disease modeling. To address this, we explore genetic models that better capture the disease's ...

impress

Targeting the LRRK2 G2019S Mutation Found in Parkinson's Disease

Background: Parkinson's disease (PD) is a progressive neurodegenerative disorder with which the leucine-Rich repeat

Perplexity: weitere Schritte

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[10 Quellen](#)

Nachfrage stellen

+

Modell ▾

🔊

↑