MDC MAX-DELBRÜCK-CENTRUM FÜR MOLEKULARE MEDIZIN BERLIN-BUCH IN DER HELMHOLTZ-GEMEINSCHAFT e.V.

# Applications of Text and Data Mining of biomedical databases

Miguel Andrade
Computational Biology & Data Mining group
Max Delbrück Center for Molecular Medicine
miguel.andrade@mdc-berlin.de

Gene structures

Gene expression

Protein sequences

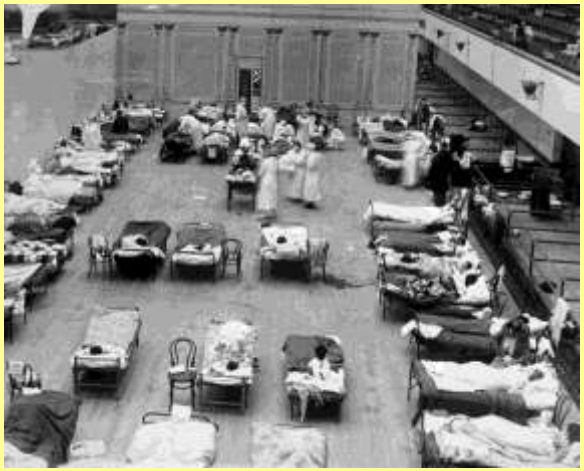
Protein databases

Literature databases

AGCTGGTACGAAGATGTCTCGCA
MLVPIEKAEVPRYILKTEFRKAILTS
in a phosphorylation dependent
0010010010001001010111011

Biological predictions

Human disease



# Molecular Biology databases

Protein and nucleotide sequences (UniProt, Entrez),
Protein domains (PFAM, SMART),
Structures (PDB),
Diseases (OMIM),
Gene expression (GEO),
Bibliography (records, MEDLINE)
(full text, PubMed Central)

# Molecular Biology databases

## Bibliography (records, MEDLINE)
## (full text, PubMed Central)

Compressed PubMed in XML: **17GB**

23M items (exhaustive back to 1966, oldest from 1809)

PubMed Central open access subset **26GB** of raw XML files (text only), compressed 8GB.

2.6M items

# Molecular Biology databases

## Bibliography (records, MEDLINE)
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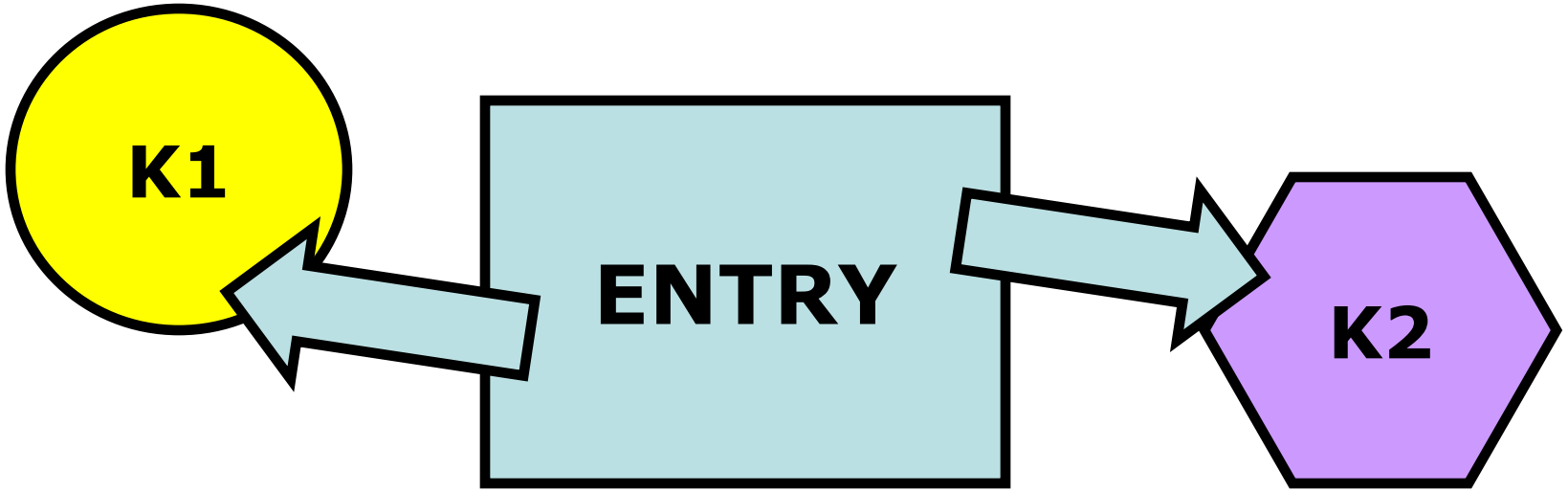
PubMed Central open access subset **26GB** of raw XML files (text only), compressed 8GB.

2.6M items 1 Human Genome **320GB**

# Mapping systems



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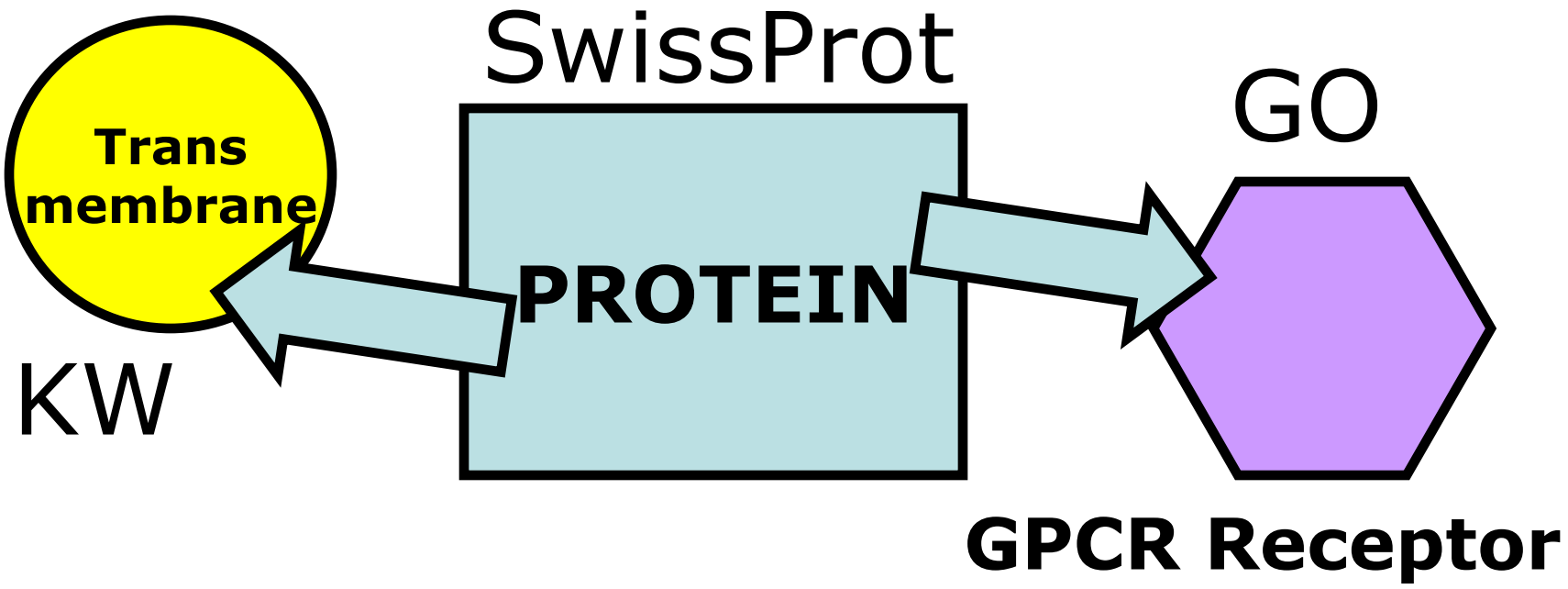

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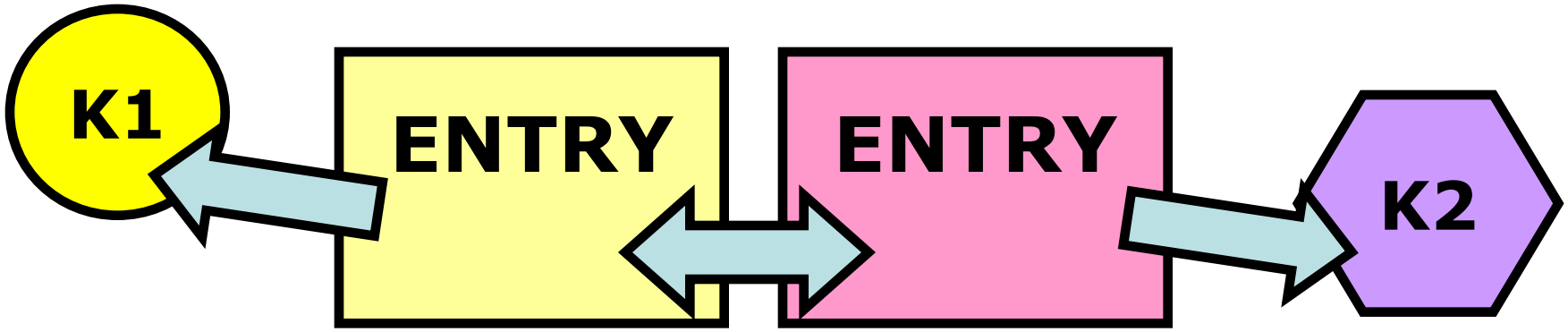

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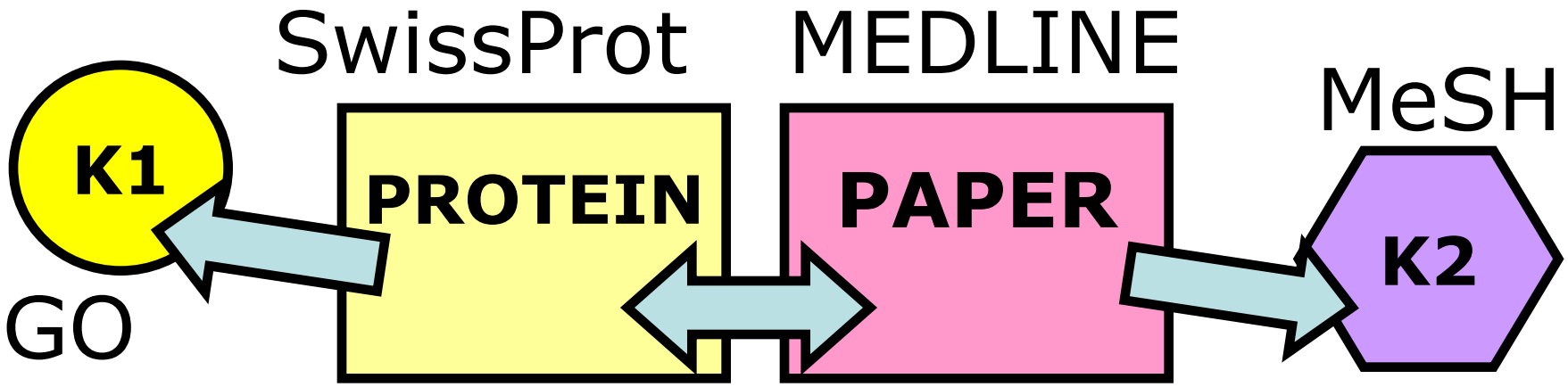

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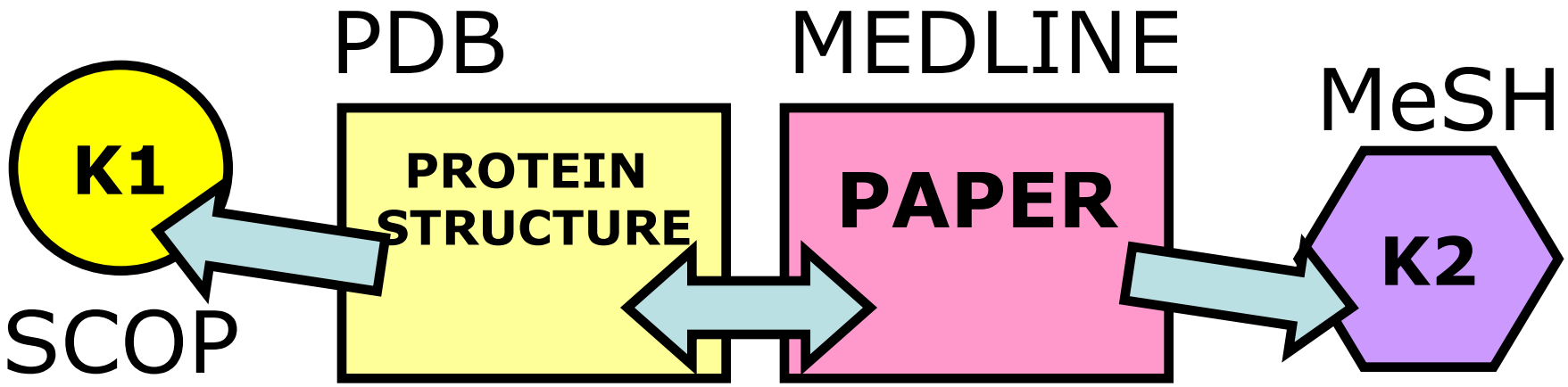
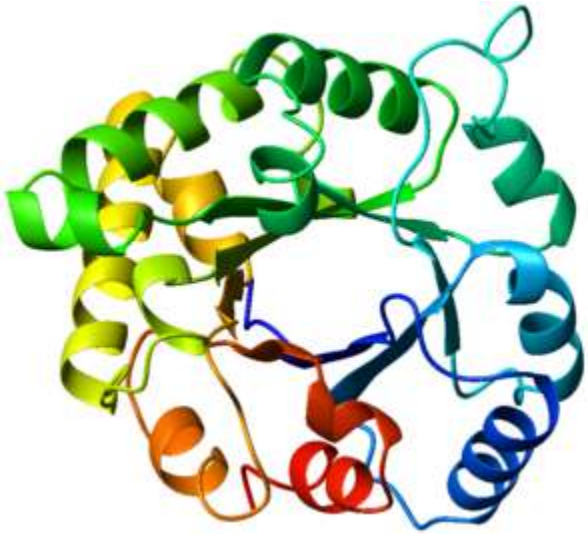



TIM barrel



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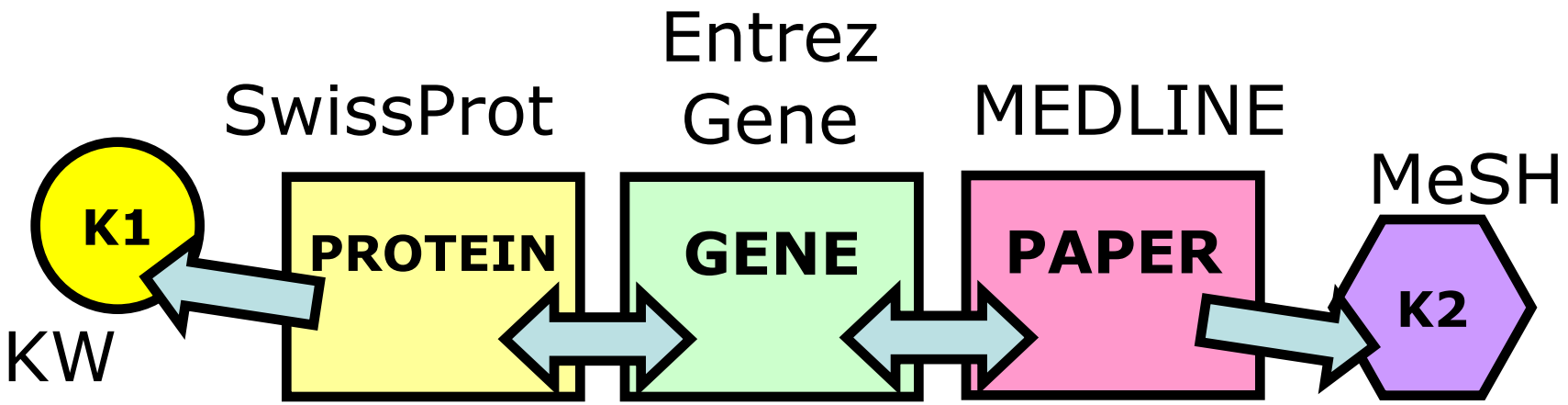
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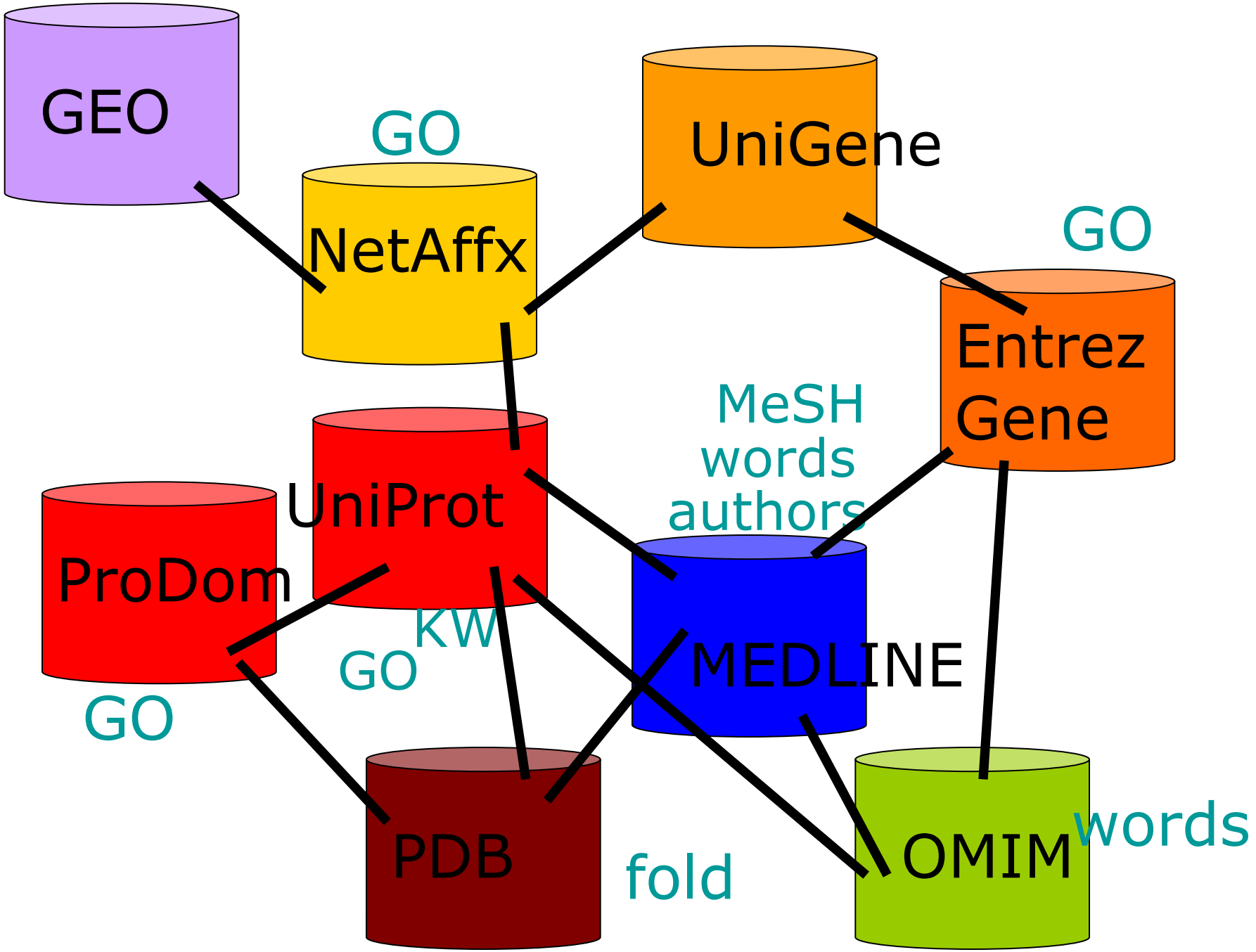
PROTEIN
GENE
PAPER
K2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NetAffx
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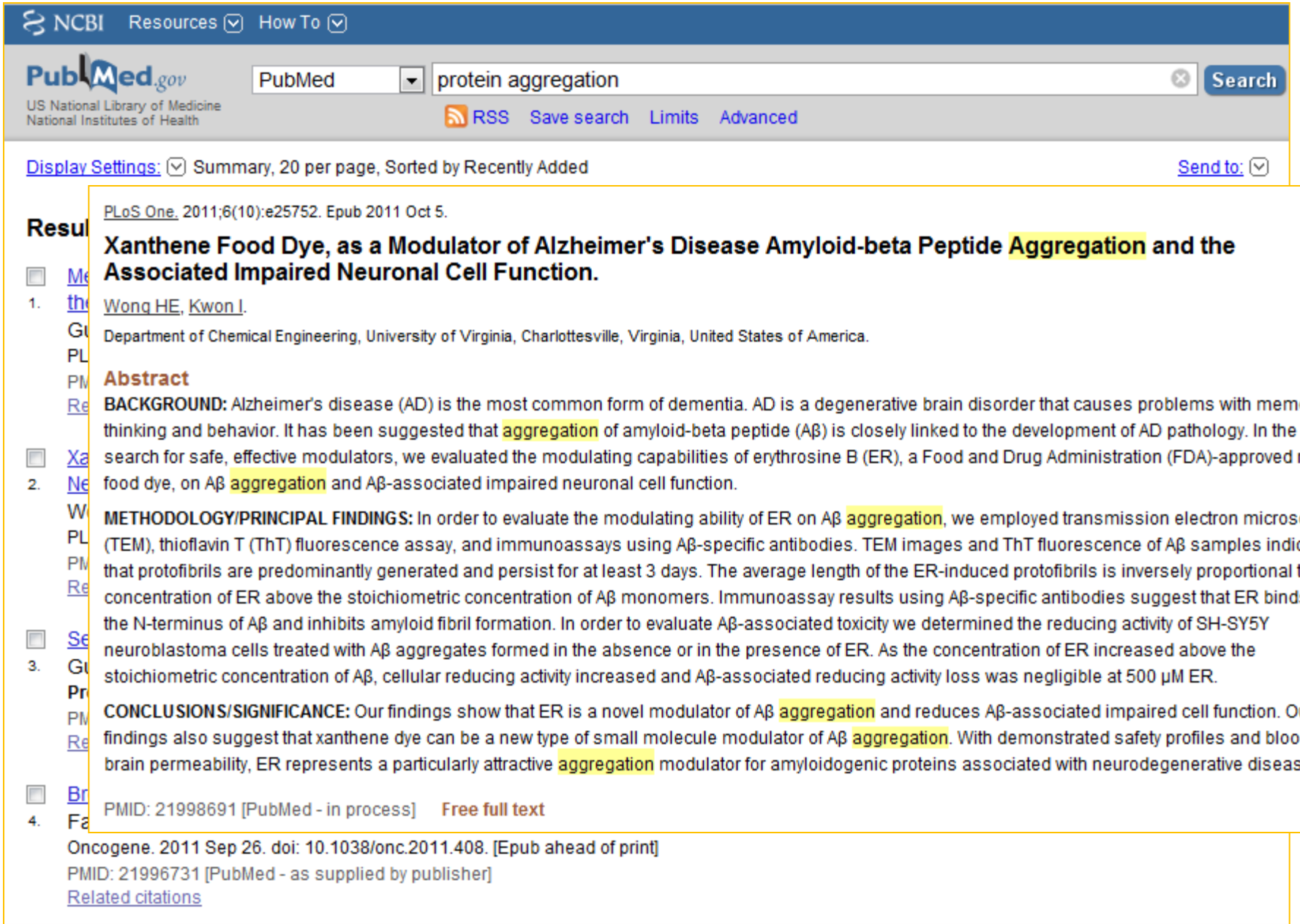
# PubMed


NCBI Resources How To
PubMed.gov
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PubMed
protein aggregation
Search
RSS Save search Limits Advanced
Display Settings: Summary, 20 per page, Sorted by Recently Added
Send to:
PLoS One. 2011;6(10):e25752. Epub 2011 Oct 5.
Xanthene Food Dye, as a Modulator of Alzheimer's Disease Amyloid-beta Peptide Aggregation and the Associated Impaired Neuronal Cell Function.
Wong HE, Kwon I.
Department of Chemical Engineering, University of Virginia, Charlottesville, Virginia, United States of America.
Abstract
BACKGROUND: Alzheimer's disease (AD) is the most common form of dementia. AD is a degenerative brain disorder that causes problems with memory, thinking and behavior. It has been suggested that aggregation of amyloid-beta peptide (Aβ) is closely linked to the development of AD pathology. In the search for safe, effective modulators, we evaluated the modulating capabilities of erythrosine B (ER), a Food and Drug Administration (FDA)-approved red food dye, on Aβ aggregation and Aβ-associated impaired neuronal cell function.
METHODOLOGY/PRINCIPAL FINDINGS: In order to evaluate the modulating ability of ER on Aβ aggregation, we employed transmission electron microscopy (TEM), thioflavin T (ThT) fluorescence assay, and immunoassays using Aβ-specific antibodies. TEM images and ThT fluorescence of Aβ samples indicate that protofibrils are predominantly generated and persist for at least 3 days. The average length of the ER-induced protofibrils is inversely proportional to the concentration of ER above the stoichiometric concentration of Aβ monomers. Immunoassay results using Aβ-specific antibodies suggest that ER binds to the N-terminus of Aβ and inhibits amyloid fibril formation. In order to evaluate Aβ-associated toxicity we determined the reducing activity of SH-SY5Y neuroblastoma cells treated with Aβ aggregates formed in the absence or in the presence of ER. As the concentration of ER increased above the stoichiometric concentration of Aβ, cellular reducing activity increased and Aβ-associated reducing activity loss was negligible at 500 μM ER.
CONCLUSIONS/SIGNIFICANCE: Our findings show that ER is a novel modulator of Aβ aggregation and reduces Aβ-associated impaired cell function. Our findings also suggest that xanthene dye can be a new type of small molecule modulator of Aβ aggregation. With demonstrated safety profiles and blood-brain permeability, ER represents a particularly attractive aggregation modulator for amyloidogenic proteins associated with neurodegenerative diseases.
PMID: 21998691 [PubMed - in process] Free full text
Oncogene. 2011 Sep 26. doi: 10.1038/onc.2011.408. [Epub ahead of print]
PMID: 21996731 [PubMed - as supplied by publisher]
Related citations

# PubMed

NCBI Resources How To

Oncogene. 2011 Sep 26. doi: 10.1038/onc.2011.408. [Epub ahead of print]

**Breast cancer genome-wide association studies: there is strength in numbers.**

Fanale D, Amodeo V, Corsini LR, Rizzo S, Bazan V, Russo A.

Department of Surgical and Oncological Sciences, Section of Medical Oncology, University of Palermo, Palermo, Italy.

PLoS One. 2011;6(10):e25984. Epub 2011 Oct 6.

**Membrane-bound steel factor maintains a high local concentration for mouse primordial germ cell motility, and defines the region of their migration.**

Gu Y, Runyan C, Shoemaker A, Surani MA, Wylie C.

Division of Developmental Biology, Cincinnati Children's Hospital Medical Center, Cincinnati, Ohio, United States of America.


## Abstract

Steel factor, the protein product of the Steel locus in the mouse, is a multifunctional signal for the primordial germ cell population. We have shown previously that its expression accompanies the germ cells during migration to the gonads, forming a "travelling niche" that controls their survival, motility, and proliferation. Here we show that these functions are distributed between the alternatively spliced membrane-bound and soluble forms of Steel factor. The germ cells normally migrate as individuals from E7.5 to E11.5, when they aggregate together in the embryonic gonads. Movie analysis of Steel-dickie mutant embryos, which make only the soluble form, at E7.5, showed that the germ cells fail to migrate normally, and undergo "premature aggregation" in the base of the allantois. Survival and directionality of movement is not affected. Addition of excess soluble Steel factor to Steel-dickie embryos rescued germ cell motility, and addition of Steel factor to germ cells in vitro showed that a fourfold higher dose was required to increase motility, compared to survival. These data show that soluble Steel factor is sufficient for germ cell survival, and suggest that the membrane-bound form provides a higher local concentration of Steel factor that controls the balance between germ cell motility and aggregation. This hypothesis was tested by addition of excess soluble Steel factor to slice cultures of E11.5 embryos, when migration usually ceases, and the germ cells aggregate. This reversed the aggregation process, and caused increased motility of the germ cells. We conclude that the two forms of Steel factor control different aspects of germ cell behavior, and that membrane-bound Steel factor controls germ cell motility within a "motility niche" that moves through the embryo with the germ cells. Escape from this niche causes cessation of motility and death by apoptosis of the ectopic germ cells.



PMID: 21998739 [PubMed - in process] **Free full text**


4. Fanale D, Amodeo V, Corsini LR, Rizzo S, Bazan V, Russo A.
Oncogene. 2011 Sep 26. doi: 10.1038/onc.2011.408. [Epub ahead of print]
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Related citations

# PubMed

NCBI Resources How To

PubMed.gov
US National Library of Medicine
National Institutes of Health

PubMed "protein aggregation" Search

RSS Save search Limits Advanced

Display Settings: Summary, 20 per page, Sorted by Recently Added Send to:

Res

1.

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Neurochem Res. 2011 Oct 9. [Epub ahead of print]
PMID: 21984199 [PubMed - as supplied by publisher]
Related citations

PLoS One. 2011;6(10):e25752. Epub 2011 Oct 5.

## Xanthene Food Dye, as a Modulator of Alzheimer's Disease Amyloid-beta Peptide Aggregation and the Associated Impaired Neuronal Cell Function.

Wong HE, Kwon I.

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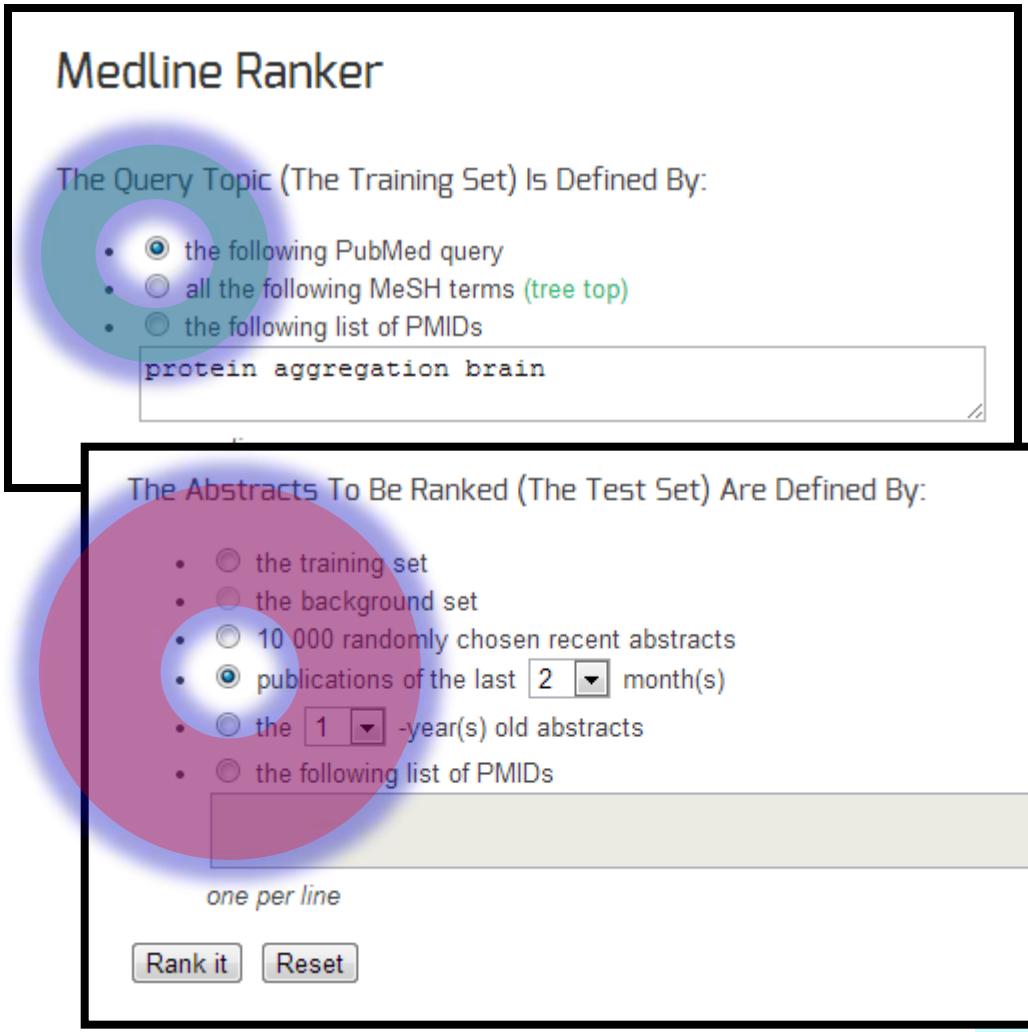
PMID: 21998691 [PubMed - in process] Free full text

# MedlineRanker

Jean-Fred Fontaine

**Discriminative words**

| rank | word | weight |
|---|---|---|
| 1 | aggregation | 7.13 |
| 2 | synuclein | 6.26 |
| 3 | misfolding | 5.59 |
| 4 | lewy | 5.33 |
| 5 | tangle | 5.07 |
| 6 | alzheimer | 4.98 |
| 7 | tau | 4.92 |
| 8 | neurodegeneration | 4.89 |
| 9 | amyloid | 4.79 |
| 10 | aggregate | 4.75 |
| 11 | app | 4.59 |
| 12 | huntington | 4.58 |
| 13 | fibril | 4.49 |
| 14 | prion | 4.47 |
| 15 | oligomer | 4.44 |
| 16 | ad | 4.26 |
| 17 | hallmark | 4.26 |




Rank MEDLINE according a topic

taine *et al.* 09) *Nucleic ls earch*

http://cbdm.mdc-berlin.de/tools/medlineranker/

# MedlineRanker

Jean-Fred Fontaine

**Discriminative words**

| rank | word | weight |
|---|---|---|
| 1 | | |
| 2 | | |
| 3 | | |
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| 5 | | |
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| Rank | PMID | Abstract Title | P-value |
|---|---|---|---|
| | | | 7.09e-05 |
| | | | 7.45e-05 |
| | | | 7.48e-05 |
| | | | 7.52e-05 |
| | | | 7.93e-05 |
| | | | 8.01e-05 |
| | | | 8.11e-05 |
| | | | 8.25e-05 |
| | | | 8.31e-05 |

**HIGHLIGHTER** CONTACT CBDM GROUP

**The Role of α-Synuclein in Neurodegenerative Diseases : From Molecular Pathways in Disease to Therapeutic Approaches.**

Parkinson disease (PD ) is the second most prevalent neurodegenerative disorder after Alzheimer 's disease (AD ). The formation of the cytoplasmic inclusions named "Lewy bodies" in the brain , considered to be a marker for neuronal degeneration in PD and dementia with Lewy bodies. However, Lewy bodies (LBs) are also observed in approximately 60 percent of both sporadic and familial cases with AD . LBs consist of fibrils mainly formed by post -translational modified α-synuclein (α-syn) protein . The modifications can be truncation, phosphorylation , nitration and mono-, di-, or tri-ubiquitination. Development of disease seems to be linked to events that increase the concentration of α-syn or cause its chemical modification, either of which can accelerate α-syn aggregation . Examples of such events include increased copy number of genes, decreased rate of degradation via the proteasome or other proteases, or modified forms of α-syn. As the aggregation of α-syn in the brain has been strongly implicated as a critical step in the development of several neurodegenerative diseases , the current search for disease -modifying drugs is focused on modification of the process of α-syn deposition in the brain . Recently researchers have screened and designed various molecules that are selectively focused on inhibiting or preventing α-syn aggregation and toxicity . Another strategy that has emerged is to target α-syn expression as a potential therapy for neurodegenerative diseases associated with LBs.

http://cbdm.mdc-berlin.de/tools/medlineranker/

# Génie

**Discriminative words**

| rank | word | weight |
|---|---|---|
| 1 | aggregation | 7.13 |
| 2 | synuclein | 6.26 |
| 3 | misfolding | 5.59 |
| 4 | lewy | 5.33 |
| 5 | tangle | 5.07 |
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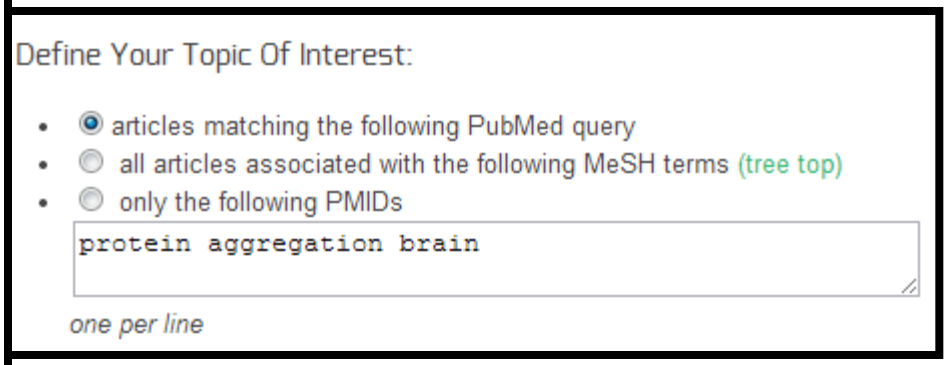
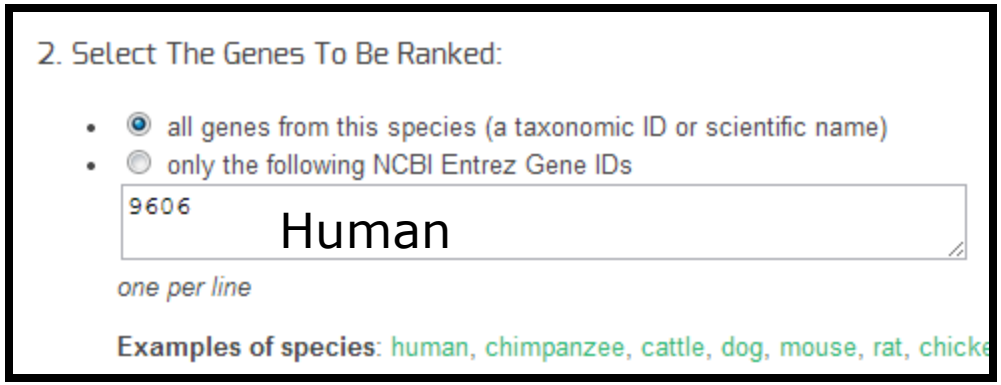
| 7 | tau | 4.92 |
| 8 | neurodegeneration | 4.89 |
| 9 | amyloid | 4.79 |
| 10 | aggregate | 4.75 |
| 11 | app | 4.59 |
| 12 | huntington | 4.58 |
| 13 | fibril | 4.49 |
| 14 | prion | 4.47 |
| 15 | oligomer | 4.44 |
| 16 | ad | 4.26 |
| 17 | hallmark | 4.26 |




Ranks a set of genes from a whole genome according to a topic




Fontaine *et al.* (2011) *Nucleic Acids Research*

http://cbdm.mdc-berlin.de/tools/genie/

# Génie

| Rank | GeneID | Symbol | Homologs | PMIDs | Hits | FDR | Top 10 abstracts |
|---|---|---|---|---|---|---|---|
| 1 | 6622 | SNCA | | | | | 21613474 (p=7.09e-05), 16651889 (p=7.17e-05), 15180968 (p=7.35e- |
| 2 | 351 | APP | | | | | 6e- |
| 3 | 4137 | MAPT | | | | | 0e- |
| 4 | 23621 | BACE1 | | | | | 5e- |
| 5 | 3064 | HTT | | | | | 3e- |
| 6 | 5663 | PSEN1 | | | | | 3e- |

HIGHLIGHTER CONTACT CBDM GROUP

**Induction of intracellular tau aggregation is promoted by α-synuclein seeds and provides novel insights into the hyperphosphorylation of tau .**

Intracytoplasmic proteinaceous inclusions , primarily composed of tau or α-synuclein (α-syn), are predominant pathological features of Alzheimer 's disease (AD ) and Parkinson 's disease (PD ), respectively. However, the coexistence of these pathological aggregates is identified in many neurodegenerative disorders , including spectrum disorders of AD and PD . Whereas α-syn can spontaneously polymerize into amyloidogenic fibrils , in vitro, tau polymerization requires an inducing agent. The current study presents a human-derived cellular model , in which recombinant, preformed α-syn fibrils cross-seed intracellular tau to promote the formation of neurofibrillary tangle -like aggregates . These aggregates were hyperphosphorylated, Triton insoluble, and thioflavin-S positive, either comingling with endogenously expressed α-syn aggregates or induced by only exogenously applied recombinant α-syn fibrils . Furthermore, filamentous, amyloidogenic tau took over the cellular soma, displacing the nucleus and isolating or displacing organelles, likely preventing cellular function . Although a significant proportion of wild-type tau formed these cellular inclusions , the P301L mutation in tau increased aggregation propensity resulting from α-syn seeds to over 50% of total tau protein . The role of phosphorylation on the development of these tau aggregates was investigated by coexpressing glycogen synthase kinase 3 β or microtubule-associated protein /microtubule affinity-regulating kinase 2. Expression of either kinase inhibited the formation of α-syn-induced tau aggregates . Analyses of phosphorylation sites suggest that multiple complex factors may be associated with this effect and that Triton-soluble versus Triton-insoluble tau may be independently targeted by kinases . The current work not only provides an exceptional cellular model of tau pathology , but also examines α-syn-induced tau inclusion formation and provides novel insights into hyperphosphorylation observed in disease .

http://cbdm.mdc-berlin.de/tools/genie/

# PESCADOR

Adriano Barbosa

**PESCADOR**

Platform for Exploration of Significant Concepts AssociateD to co-Occurrences Relationships.

Input | Concepts | Retrieval | Development | Help

MDC Berlin-Buch

INPUT **[Required]**

Paste below your list of PubMed IDs (one per line!):

17151287
18561034
17259179
12147333
15916898
18585350
15710903
19422822

Example: Alzheimer and Parkinson diseases i

Other examples of PMIDs lists related to:
Phosphorylation in Yeast
Host-Pathogen Interactions in *Arabidopsis t*
Cell Cycle in *Escherichia coli*

Clear

*Target Species*

Inform below the NCBI's Taxonomy ID for t

9606

Examples: 9606 (*H. sapiens*), 3702 (*A. thal*

**PLUS**

*Customized concepts*

Load below the biological concepts to be checked for (one per line!)

ALZHEIMER
AD
PARKINSON
PD
AGGREGATION
AMYLOID
CLEAVAGE

Example Clear

Start analysis

Extract interactions and filter by concepts

Barbosa-Silva et al. (2010) *BMC Bioinformatics*

Barbosa-Silva et al. (2011) *BMC Bioinformatics*

http://cbdm.mdc-berlin.de/tools/pescador/

# PESCADOR

**PESCADOR**

Platf

**Target Organism**: *Homo sapiens* (TAXID: 9606)

Click on a co-occurrence type to filter the type of co-occurrences displayed.

Selected PubMed ID: 10764738

**Sentence 1**: **Microtubule-associated protein 1B is a component of cortical Lewy bodies and binds alpha-synuclein filaments.**

**Sentence 2**: Lewy bodies, neuropathological hallmarks of Parkinson's disease and dementia with Lewy bodies, comprise alpha-synuclein filaments and other less defined proteins.

**Sentence 3**: Characterization of Lewy body proteins that interact with alpha-synuclein may provide insight into the mechanism of Lewy body formation.

**Sentence 4**: Double immunofluorescence labeling and confocal microscopy revealed approximately 80% of cortical Lewy bodies contained microtubule-associated protein 1B (MAP-1B) that overlapped with alpha-synuclein.

**Sentence 5**: Lewy bodies were isolated using an immunomagnetic technique from brain tissue of patients dying with dementia with Lewy bodies.

**Sentence 6**: Lewy body proteins were resolved by polyacrylamide gel electrophoresis.

**Sentence 7**: Immunoblotting confirmed the presence of MAP-1B and alpha-synuclein in purified Lewy bodies.

**Sentence 8**: Direct binding studies revealed a high affinity interaction (IC(50) approximately 20 nm) between MAP-1B and alpha-synuclein.

**Sentence 9**: The MAP-1B-binding sites were mapped to the last 45 amino acids of the alpha-synuclein C terminus.

**Sentence 10**: MAP-1B also bound in vitro assembled alpha-synuclein fibrils.

**Sentence 11**: Thus, MAP-1B may be involved in the pathogenesis of Lewy bodies via its interaction with monomeric and fibrillar alpha-synuclein.

# PESCADOR

**Type 1**

MAP-1B also bound in vitro assembled alpha-synuclein fibrils.

**Term + [Biointeraction] + Term**

**Type 2**

Direct binding studies revealed a high affinity interaction (IC(50) approximately 20 nm) between MAP-1B and alpha-synuclein.

**[Biointeraction] +Term + Term + [Biointeraction]**

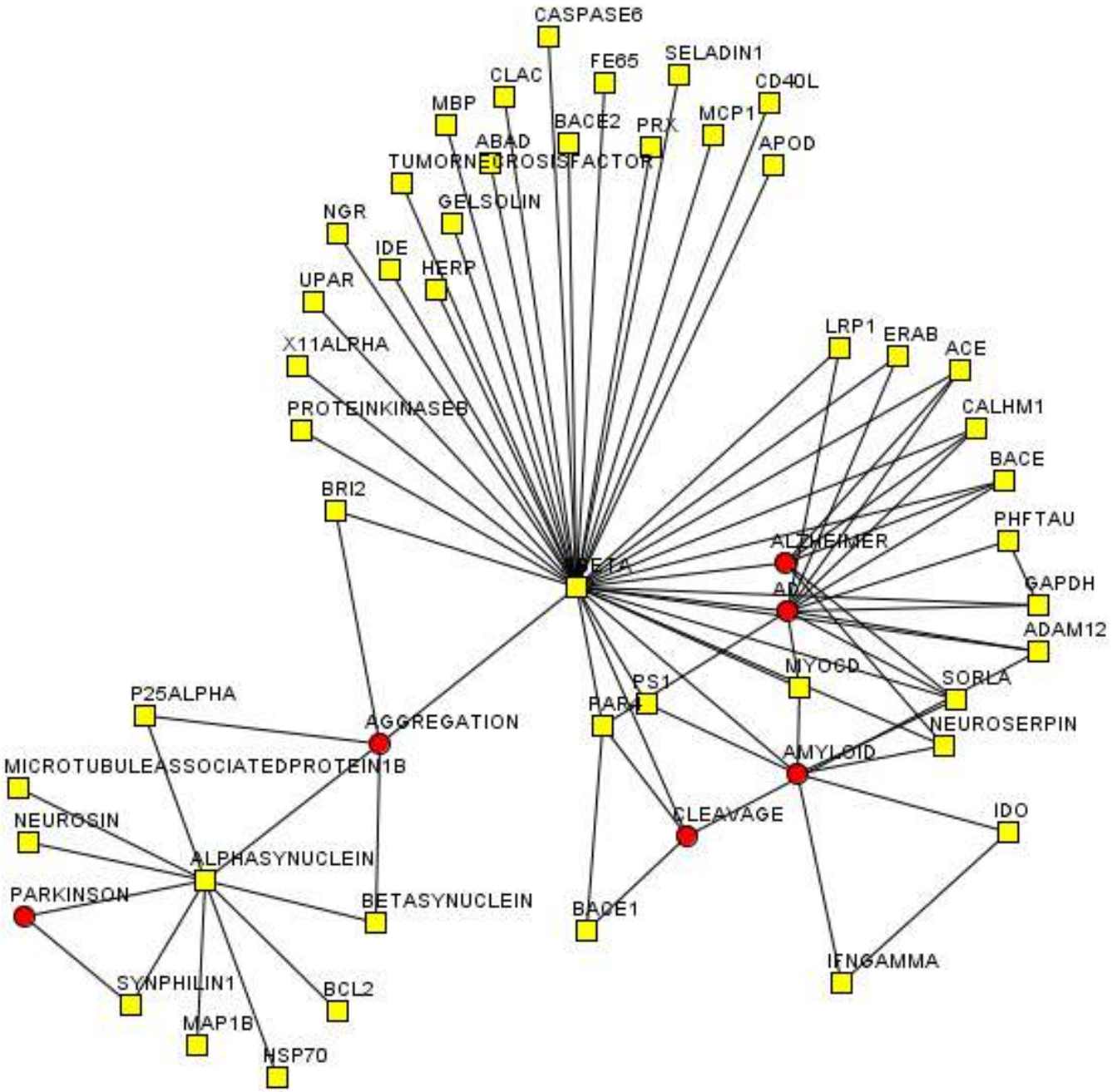
**Type 3**

Immunoblotting confirmed the presence of MAP-1B and alpha-synuclein in purified Lewy bodies.

**Term + Term**

Type 4

**co-occurrence in abstract**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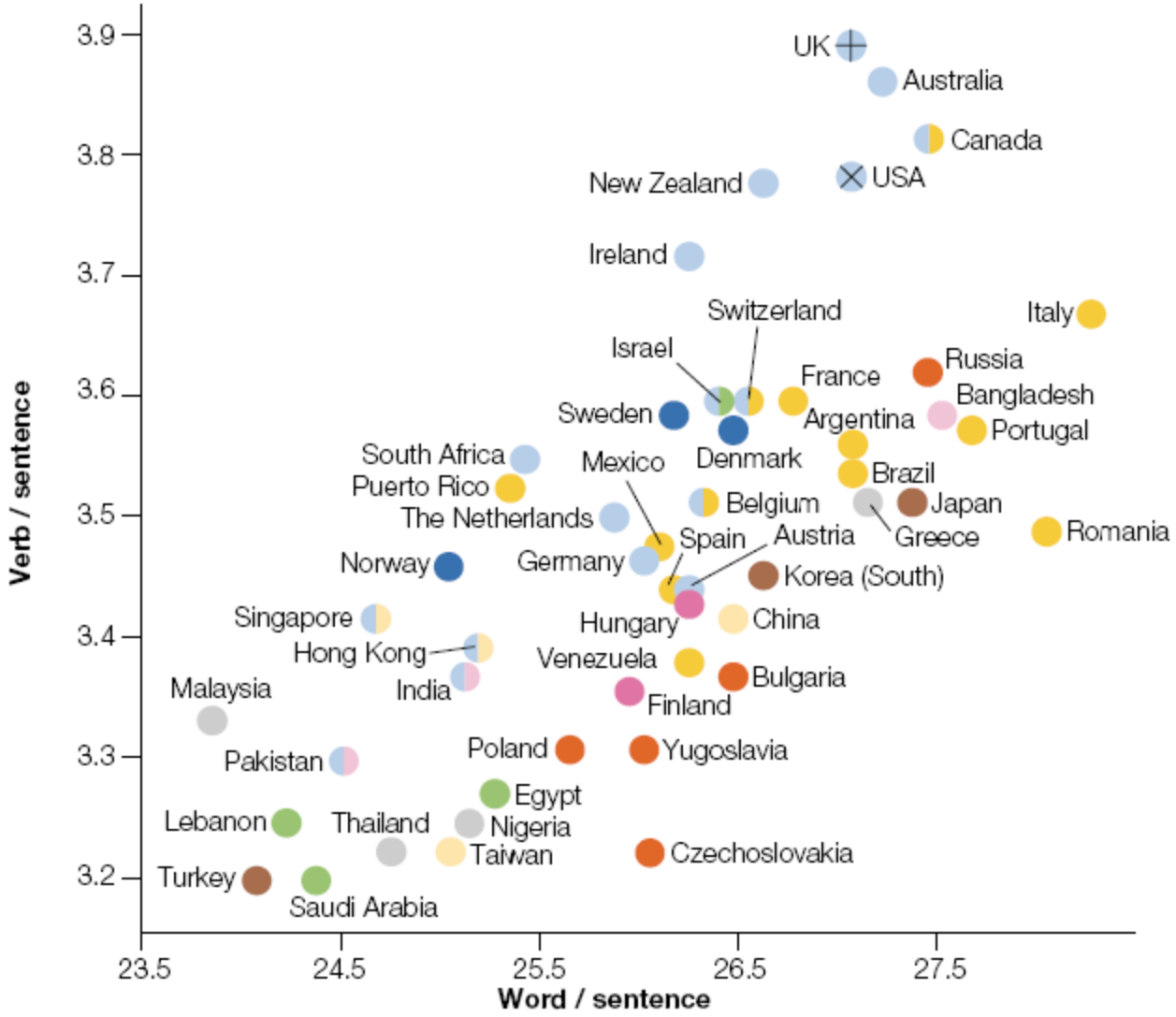
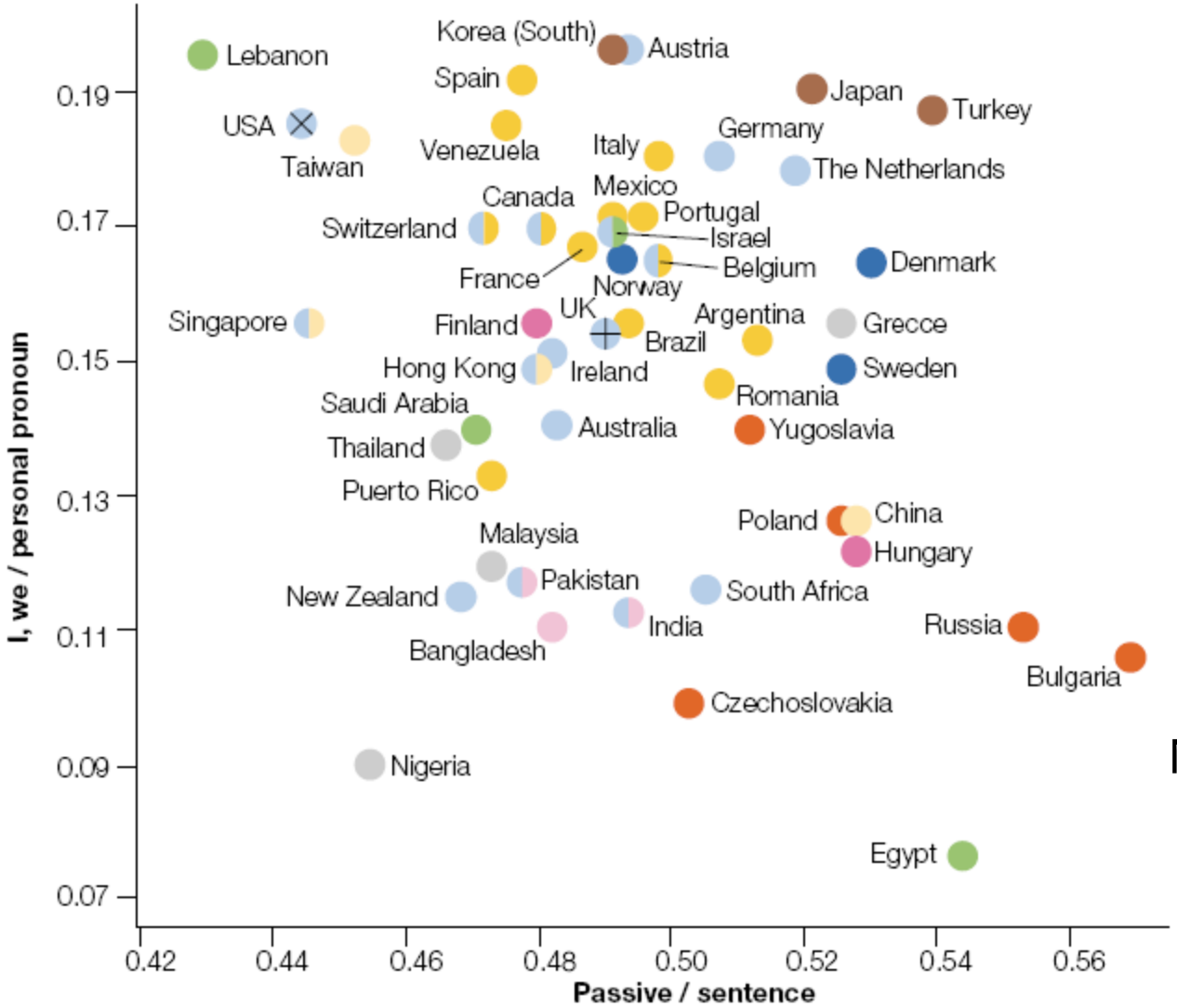
NGR
IDE
HERP
UPAR
X11ALRHA
LRP1
ERAB
ACE
PROTEINKINASEB
CALHM1
BACE
BRI2
PHFTAU
ALZHEIMER
GAPDH
AD
ADAM12
MYO6D
SORLA
P25ALPHA
PS1
PAR4
AGGREGATION
NEUROSERPIN
AMYLOID
MICROTUBULEASSOCIATEDPROTEIN1B
NEUROSIN
CLEAVAGE
IDO
ALPHASYNUCLEIN
PARKINSON
BETASYNUCLEIN
BACE1
IFNGAMMA
SYNPHILIN1
BCL2
MAP1B
HSP70

# Country-specific variations of English




Netzel et al. (2003)
*EMBO Reports*

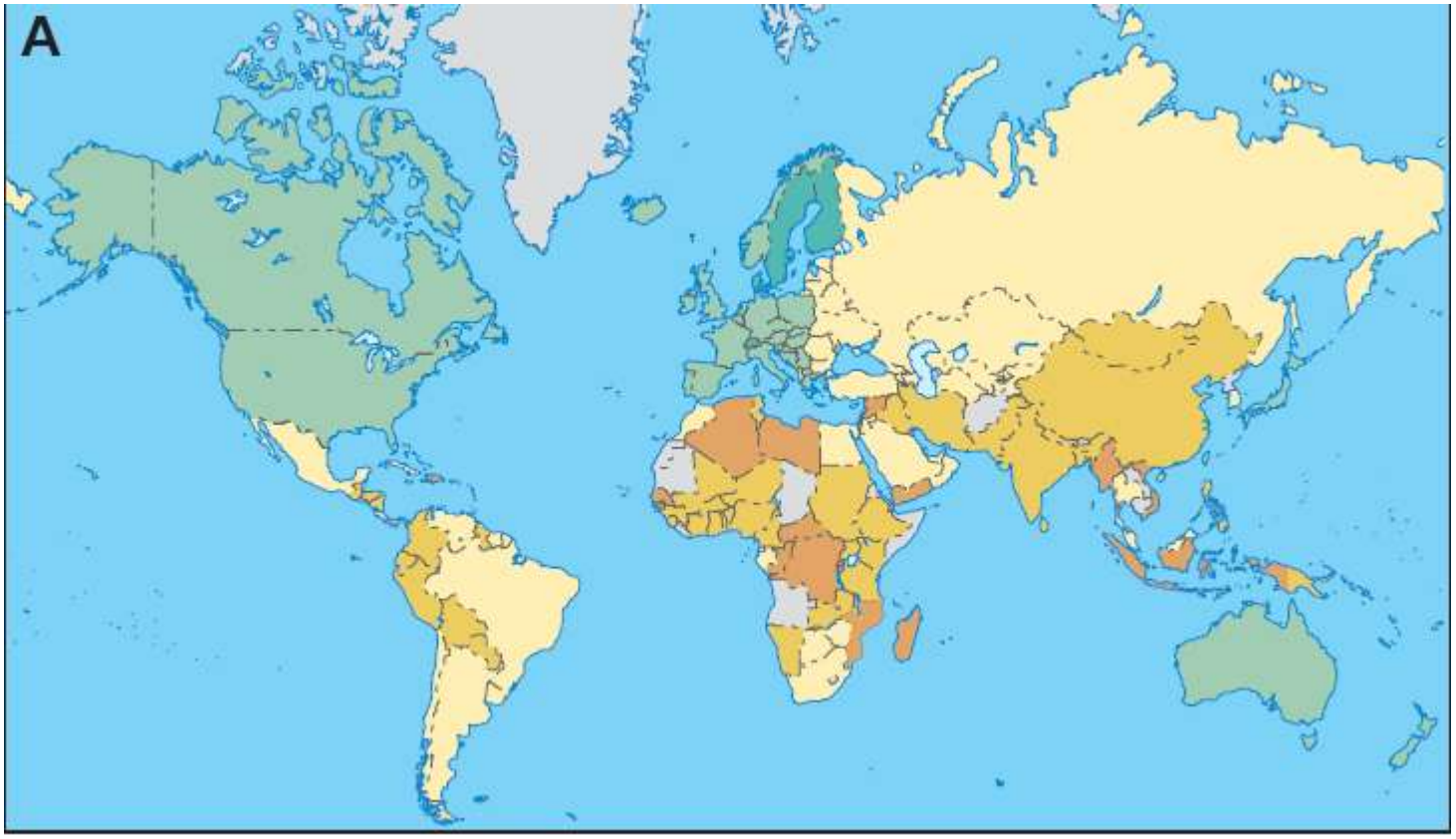
# Country-specific variations of English




Netzel et al. (2003)
*EMBO Reports*

# Worldwide scientific publishing activity

**Approximate amount of publications for the years 1996–2001 per million inhabitants by country:**

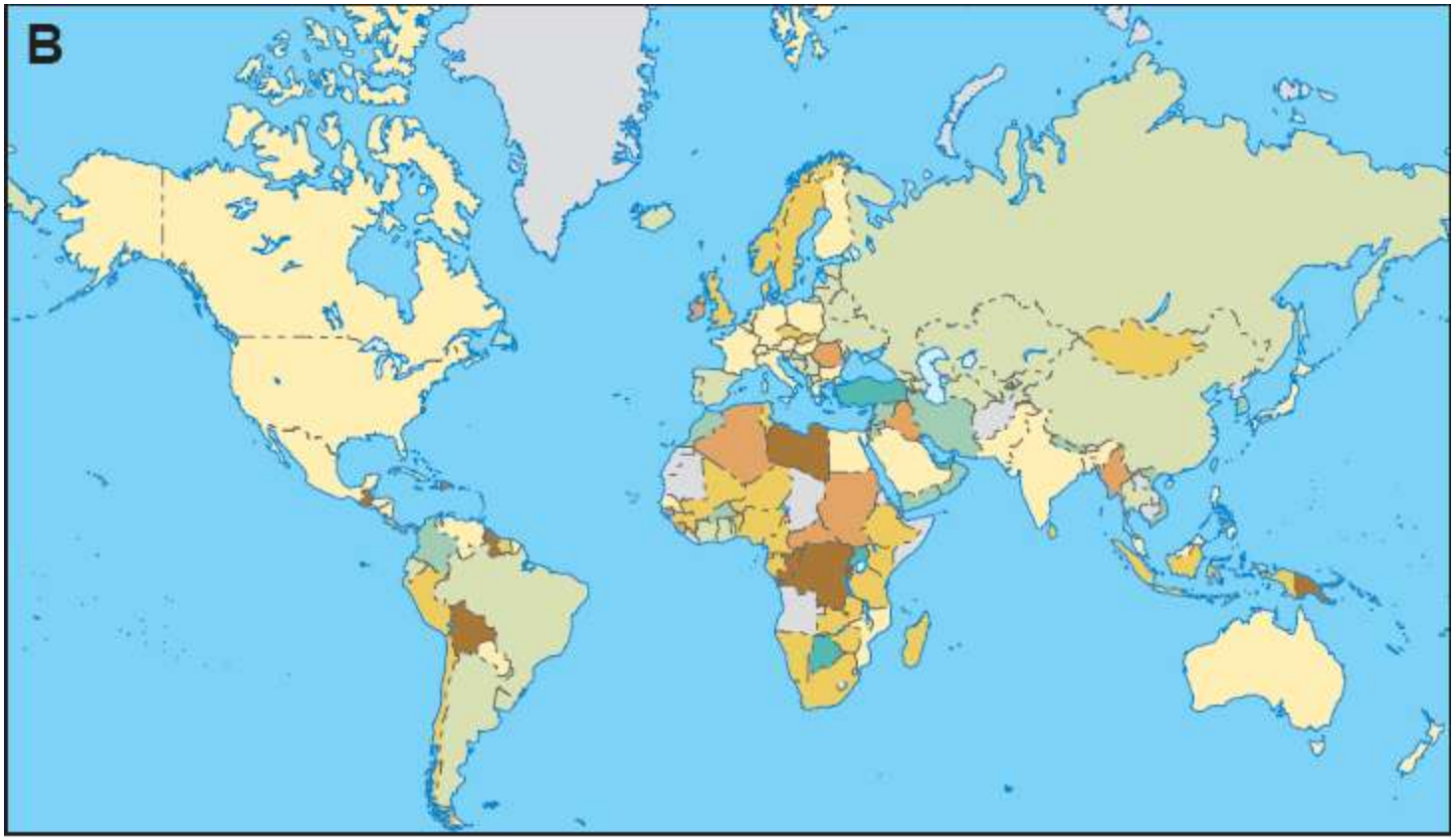



**10,000**
**1,000**
**100**
**10**
**1**

Perez-Iratxeta and Andrade (2002) *Science*

# Worldwide scientific publishing activity

**Ratio publications for 1996–2001 / 1989–95**

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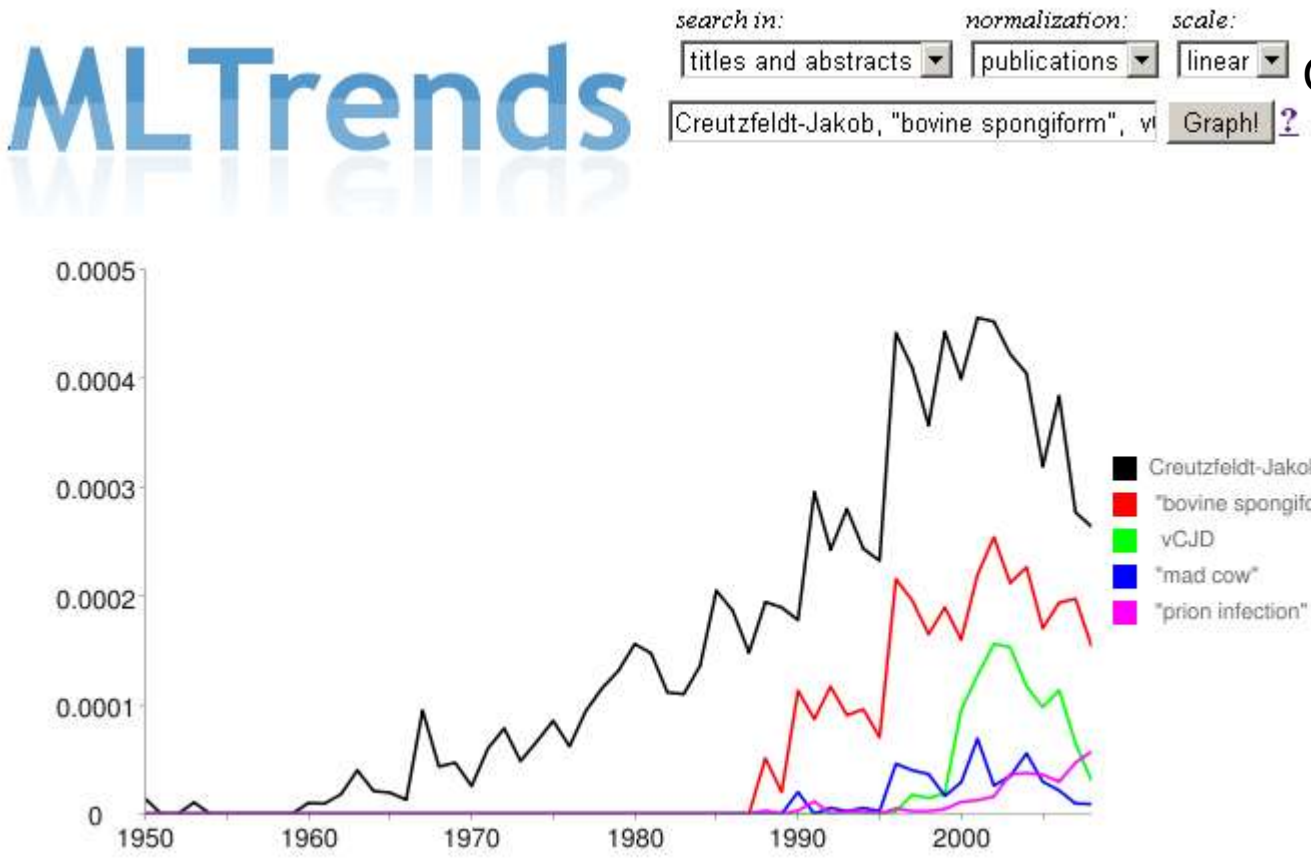


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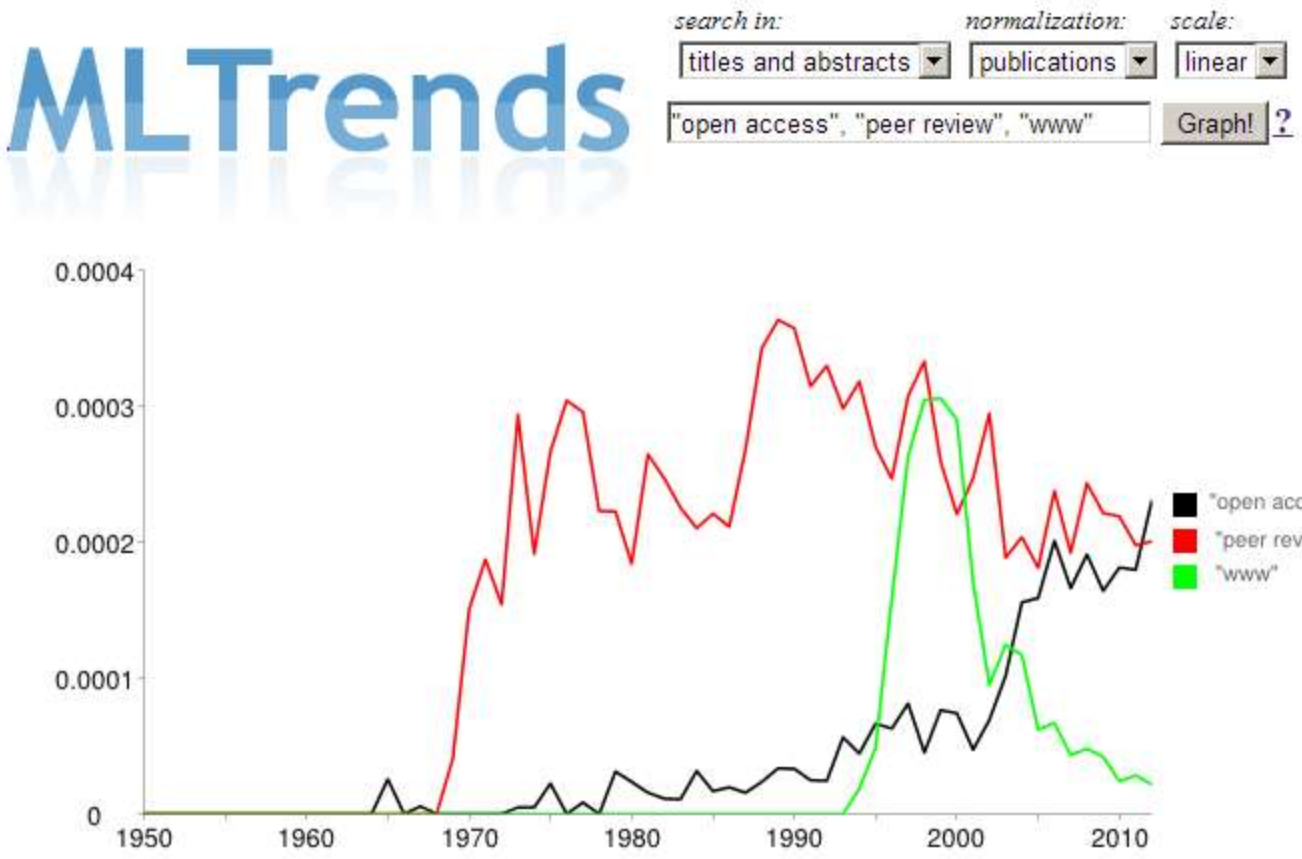


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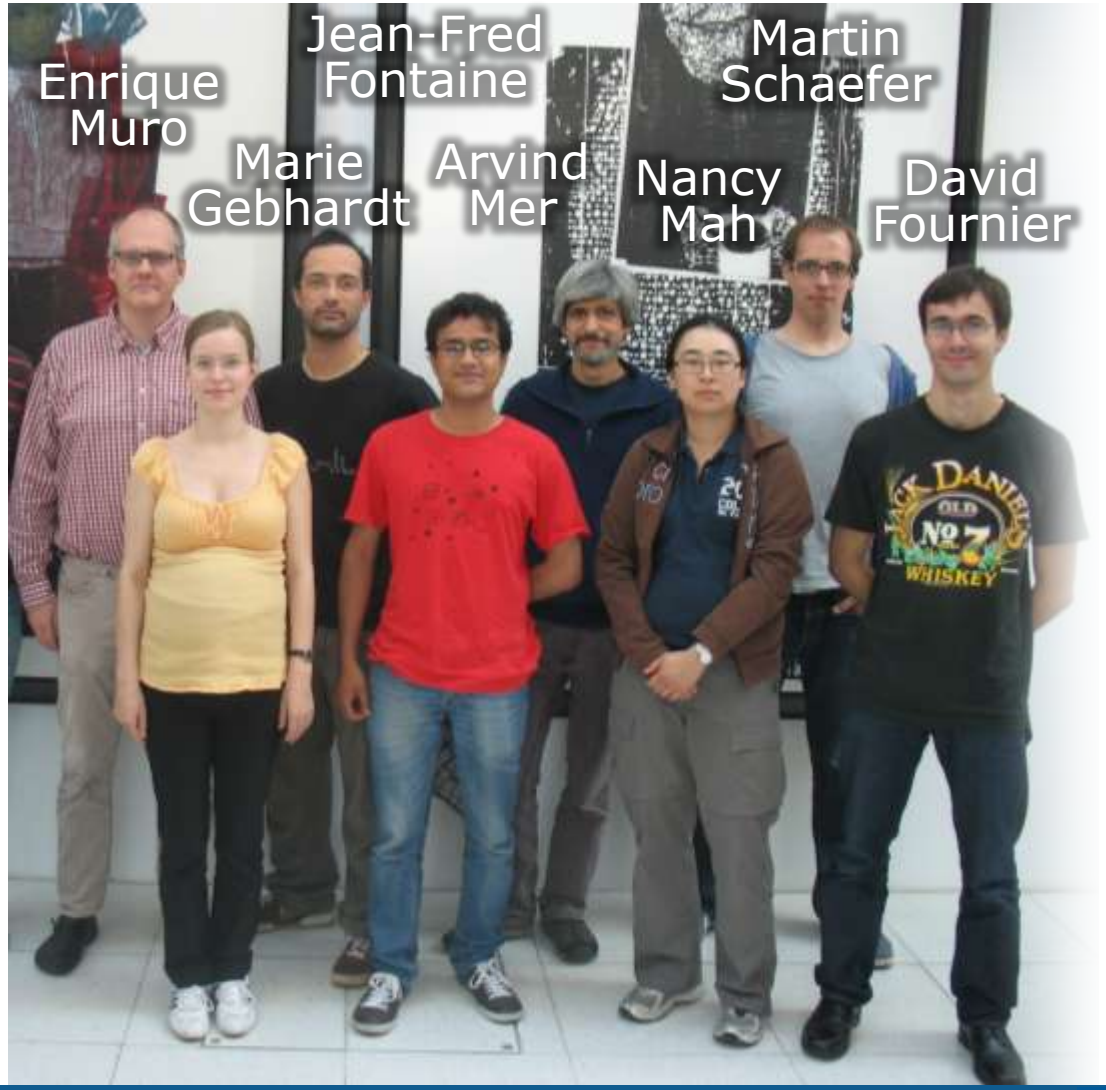


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