

BEL

Biological Expression Language

BEL Features



Share



Computable



Easy to learn

BEL makes it easier to re-use and share biological knowledge. Since it is an open standard supported by an active community, you can also aggregate any BEL content into your knowledgebase.

Makes biological knowledge computable. Drive prior-knowledge-based algorithms using a standard representation. Build your knowledgebase once.

BEL is easier than the Chemical Reaction Language which every high-school student learns.

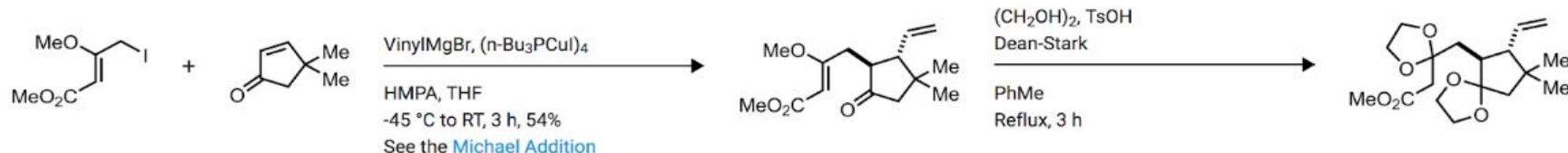
A photograph of a lightning strike over a field of tall grass. The sky is dark and filled with clouds, with a bright lightning bolt striking from the left side. The foreground is filled with the texture of tall grass blades.

Data Reproducibility

Photo by Eugene Triguba on Unsplash

BEL - Open Standard Language for Biology

Chemists have the Chemical Reaction Language



Biologists communicate inefficiently - journal papers, powerpoints and databases ...

pre-BEL: "... Northern blot analysis documented that two transcription factor genes chosen for further study, c-myc promoter-binding protein (MBP-1) [official symbol: ENO1] and X-box binding protein 1 (XBP-1), were up-regulated in U266 cells about 3-fold relative to the cell cycle-dependent beta-actin gene 12 h after IL-6 treatment ..."

BEL:

- $p(\text{HGNC:IL6}) \text{ increases } r(\text{HGNC:ENO1})$
- $p(\text{HGNC:IL6}) \text{ increases } r(\text{HGNC:XBP1})$

BEL, provides greatly enhanced readability, is computable and can be assembled into a visual network of knowledge (networks are the native language of biologists)



Assertions:

$p(\text{HGNC:MAPKAP1}) \text{ increases } p(\text{HGNC:AKT1}, \text{pmod(Ph, S, 473)})$

Context:

Citation: 16962653 [Pubmed]

Evidence: “*Genetic ablation of sin1 (MAPKAP1) abolished Akt-Ser473 phosphorylation and disrupted rictor-mTOR interaction but maintained Thr308 phosphorylation.*”

Annotations:

MESH:Atherosclerosis [Disease]

UBERON:"cardiovascular system endothelium" [Anatomy]

CL:"endothelial cell" [Cell]

MESH:"Muscle, Smooth, Vascular" [Anatomy]

EFO:U-266 [CellLine]

MESH:"Cell Membrane" [CellStructure]

TAX:9606 – human [Species]

Contradictory BEL Assertions

Nanopub 1

Using a modified form of the yeast two-hybrid system, we show in Cos cells that c-Jun can interact with the DNA binding domain/hinge region (CD regions) of the androgen receptor. Therefore, we propose that c-Jun functions as a mediator for androgen receptor-induced transactivation.

$\text{act}(\text{p(HGNC:JUN}), \text{ma(tscript)}) \text{ decreases } \text{act}(\text{p(HGNC:AR}), \text{ma(tscript)})$

Annotations: Species: human, Cell: endothelial cell, Anatomy: [lung, bronchoaveolar lavage fluid], CellStructure: cytoplasm, Disease: Leukemia, CellLine: EFO:U-266

Nanopub 2

proto-oncoprotein c-jun was increased by DHA treatment. A transient transfection found that forced expression of c-jun inhibited AR transactivation activity.

$\text{act}(\text{p(HGNC:JUN}), \text{ma(tscript)}) \text{ increases } \text{act}(\text{p(HGNC:AR}), \text{ma(tscript)})$

Annotations: Species: human, Cell: astrocyte, Anatomy: muscle tissue, CellStructure: cell nucleus, Disease: Head and Neck Neoplasms, CellLine: EFO:PC-10

Consistency Algorithm

- Develop algorithms to evaluate biological consistency using BEL
- Consistency types:
 - Logical inconsistencies
 - Data-driven consistency analysis
- Great Research Opportunity! Interested in collaborations

Powered by BEL



AIDAKA



Powering BEL



Fraunhofer
SCAI

BEL Content



Clarivate
Analytics

BEL Tooling (partial list)

- **BEL & BEL API** - python library/API for language services: parsing, completion, terminologies, etc (opensource/commercial support)
- **BioDati Studio** - Web application/API's for managing BEL-related content - curation, network building/storage/visualization (commercial support)
- **PyBEL/BELIEF** - BEL graph analytics and BEL text mining - Charlie Hoyt, et al, at Fraunhofer-SCAI
- **INDRA Project** - BEL Text mining/terminologies, knowledge engine (opensource) - John Bachman, et al, Harvard Medical School

Just launched - New BEL Language Website!

The screenshot shows the left sidebar of the website with navigation links like 'Language', 'Reference Section', 'Current (2.1.0)', 'Functions', 'Relations', 'Cheatsheet current', '2.0.0', 'Functions', 'Relations', 'Cheatsheet 2.0.0', 'Structure', 'Namespaces', 'Annotations', 'Tutorials', 'Contributing', 'Credits', and 'MORE'. Below 'MORE' are links to 'BEL Home', 'Discussions', 'Language Specifications', 'Enhancement Proposals', and 'Contact Us'. The main content area shows the URL 'BEL Language Website > Language > Reference Section > Current (2.1.0) > Functions > ...' and a purple button labeled 'abundance'. The title 'proteinAbundance (current)' is displayed, followed by 'Long form: proteinAbundance() Short form: p0'. A detailed description follows: 'proteinAbundance(ns:v) or p(ns:v) denotes the abundance of the protein designated by the value +v+ in the namespace +ns+, where +v+ references a gene or a named protein family.' Below this is a section titled 'Function Signatures' with two examples: 'proteinAbundance(NSArg, loc|frag()?, var|pmod()*)' and 'proteinAbundance(fus(), loc()?, var()*)'. Each example has a numbered list of parameters: 1. Namespace argument of following type(s): Protein, 2. Zero or one of each function(s): location, fragment, and 3. Zero or more of each function(s): variant, proteinModification. The right side of the content area features a large left arrow icon and a large right arrow icon. At the bottom, there's a dark bar with the text 'p(HGNC:AKT1)' and 'p(SFAM:"AKT Family")'.

• abundance

proteinAbundance (current)

Long form: proteinAbundance()
Short form: p0

proteinAbundance(ns:v) or **p(ns:v)** denotes the abundance of the protein designated by the value +v+ in the namespace +ns+, where +v+ references a gene or a named protein family.

Function Signatures

proteinAbundance(NSArg, loc|frag()?, var|pmod()*)

1. Namespace argument of following type(s): Protein
2. Zero or one of each function(s): location, fragment
3. Zero or more of each function(s): variant, proteinModification

proteinAbundance(fus(), loc()?, var()*)

1. One of following function(s): fusion
2. Zero or one of each function(s): location
3. Zero or more of each function(s): variant

Examples

p(HGNC:AKT1)

p(SFAM:"AKT Family")

BioDati Studio

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< Search Here

SEARCH

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Validation ▾

- Warning-Annotation (15)
- Error-Assertion (7)
- Warning-Assertion (6)

WORKSPACES

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KNOWLEDGE

Status ▾

- Finalized (32733)
- Fix (14)
- Approved (8)
- Draft (7)
- Review (3)
- Rejected (1)

Update Date ▾

- < 1 year (32766)
- < 3 month (27)
- < 1 month (27)

« » 1 - 50 of 32,766

Add To Workspace Edit Copy Delete Review Export Export All Draft Network | trash icon collapse icon

REFERENCES

- a(CHEBI:celecoxib) *decreases* r(RGD:Meox2)
- a(CHEBI:celecoxib) *increases* r(RGD:Ppp1r3b)
- a(CHEBI:lovastatin) *decreases* r(RGD:Cyp26b1)
- a(CHEBI:lovastatin) *increases* r(RGD:Ppp1r3b)
- p(HGNC:PTHLH) *increases* bp(GO:"cell proliferation")
- a(CHEBI:"reactive oxygen species") *increases* (act(p(RGD:F2), ma(cat)) increases p(RGD:Jak2, pmod(Ph, Y)))
- a(CHEBI:"reactive oxygen species") *increases* (act(p(RGD:F2), ma(cat)) increases p(RGD:Stat3, pmod(Ph)))
- a(SCHEM:Mevastatin) *decreases* (act(p(RGD:F2), ma(cat)) increases p(RGD:Stat3, pmod(Ph)))
- a(SCHEM:Mevastatin) *decreases* (act(p(RGD:F2), ma(cat)) increases act(p(RGD:Hras), ma(gtp)))
- act(p(RGD:F2), ma(cat)) *increases* bp(GO:"DNA replication")
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- act(p(RGD:F2), ma(cat)) *increases* p(RGD:Stat3, pmod(Ph))
- act(p(RGD:F2), ma(cat)) *increases* act(p(RGD:Hras), ma(gtp))
- p(EG:183) *increases* r(HGNC:CTGF)
- a(CHEBI:simvastatin) *decreases* (p(EG:183) increases r(HGNC:CTGF))
- p(EG:183) *increases* r(HGNC:CXCL8)
- a(SCHEM:"Geranylgeranyl pyrophosphate") *decreases* (a(CHEBI:simvastatin) decreases r(HGNC:PTGS2))
- a(CHEBI:simvastatin) *decreases* r(HGNC:PTGS2)
- a(SCHEM:Pravastatin) *decreases* r(MGI:Ccl2)

ASSERTIONS

Single Reference Knowledge Search

BIODATI

 10551823



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Validation

Error-Assertion (1)

Status

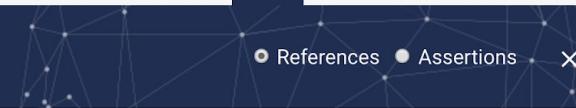
Finalized (8)
 Approved (1)

UpdateDate

< 1 year (9)
 < 3 month (1)
 < 1 month (1)
 < 1 week (0)
 < 1 day (0)

CreationDate

< 1 year (9)
 < 3 month (0)
 < 1 month (0)
 < 1 week (0)
 < 1 day (0)



References Assertions



« » 1 - 9 of 9

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REFERENCES	INFORMATION	ASSERTIONS
10551823 10551823 10551823 10551823 10551823 10551823 10551823 10551823 10551823	<h3>ABSTRACT</h3> <p>Recently, we identified the two myeloid related protein-8 (MRP8) (S100A8) and MRP14 (S100A9) as fatty acid-binding proteins (Klempt, M., Melkonyan, H., Nacken, W., Wiesmann, D., Holtkemper, U., and Sorg, C. (1997) FEBS Lett. 408, 81-84). Here we present data that the S100A8/A9 protein complex represents the exclusive arachidonic acid-binding proteins in human neutrophils. Binding and competition studies revealed evidence that (i) fatty acid binding was dependent on the calcium concentration; (ii) fatty acid binding was specific for the protein complex formed by S100A8 and S100A9, whereas the individual components were unable to bind fatty acids; (iii) exclusively polyunsaturated fatty acids were bound by S100A8/A9, whereas saturated (palmitic acid, stearic acid) and monounsaturated fatty acids (oleic acid) as well as arachidonic acid-derived eicosanoids (15-hydroxyeicosatetraenoic acid, prostaglandin E(2), thromboxane B(2), leukotriene B(4)) were poor competitors. Stimulation of neutrophil-like HL-60 cells with phorbol 12-myristate 13-acetate led to the secretion of S100A8/A9 protein complex, which carried the released arachidonic acid. When elevation of intracellular calcium level was induced by A23187, release of arachidonic acid occurred without secretion of S100A8/A9. In view of the unusual abundance in neutrophilic cytosol (approximately 40% of cytosolic protein) our findings assign an important role for S100A8/A9 as mediator between calcium signaling and arachidonic acid effects. Further investigations have to explore the exact function of the S100A8/A9-arachidonic acid complex both inside and outside of neutrophils.</p> <h3>EVIDENCES</h3>	a(SCHEM:Calcium) increases... a(SCHEM:Calcium) increases... p(HGNC:S100A8) decreases... p(HGNC:S100A9) increases... a(SCHEM:"Tetradecanoylph... a(SCHEM:Calcium) increases... a(SCHEM:Calcium) increases... act(p(SFAM:"PRKC Family")... act(p(HGNC:PRKCA), ma(ki... p(HGNC:S100A9) increases... p(HGNC:EGF increases p(H...

Add Knowledge



One assertion is required before a Nanopub can be saved.



HUMAN | BEL: 2.1.0 | ⓘ STATUS: Draft

SEARCH



KNOWLEDGE EDITOR

CITATION

CONTENT (01)

METADATA (00)

CITATION TYPE
Database

DATABASE NAME
PubMed

DATABASE ID
29658179

PUBMED INFO



TITLE
Vav1 downmodulates Akt in different breast cancer sub-

AUTHORS
Grassilli, Silvia;Brugnoli, Federica;Lattanzio, Rossano;Ma

SOURCE NAME
Molecular oncology

PUBLICATION DATE
06/01/2018

EVIDENCE
Only in ER-negative cell lines, the silencing of Vav1 induced the expression but not the activation of Akt2.

Comments

ABSTRACT

Targeting different members of the Akt pathways is a promising therapeutic chance in solid tumors including breast cancer. The variable expression levels of Akt isoforms with opposite effects on tumor growth and metastasis, however, make it difficult to select the inhibitors to be used for specific breast tumor subtypes. Using *in vitro* and *in vivo* models, we demonstrated here that Vav1, ectopically expressed in invasive breast tumors derived cells, downmodulates Akt acting at expression and/or activation levels depending on tumor subtype. The decreased p-Akt1 (Ser473) levels are a common effect of Vav1 upmodulation, suggesting that, in breast tumor-derived cells and independently of their phenotype, Vav1 interferes with signaling pathways ended to specifically recruit Akt1. Only in ER-negative cell lines, the silencing of Vav1 induced the

Internal Comments

Add BEL Assertion

BIODATI | One assertion is required before a Nanopub can be saved.

Only in ER-negative cell lines, the silencing of Vav1 induced the expression but not the activation of Akt2.

KNOWLEDGE EDITOR

CITATION

CONTENT (01)

METADATA (00)

Assertions (00)

SUBJECT: **act(p(vav))** 

Relation:  Object: 

Description
Denotes the abundance of a protein

Function Summary
`proteinAbundance(NSArg, loc|frag()?, var|pmod()*)`

Argument Help

1. Namespace argument of following type(s): Protein
2. Zero or one of each function(s): location, fragment
3. Zero or more of each function(s): variant, proteinModification

Assertion Details

Subject	Relation	Object
act(p(vav))	proteinAbundance	NSArg, loc frag()?, var pmod()*

Actions

Edit | Delete

Assertion Table

Index	Subject	Relation	Object
1	act(p(vav))	proteinAbundance	NSArg, loc frag()?, var pmod()*

Bad Assertion

 BIODATI | Owner: whayes | Created: | Modified: 07.24.19 |      

Vav1 downmodulates Akt in different breast cancer subtypes: a new promising chance to improve breast cancer out...

HUMAN | BEL: 2.1.0 | STATUS: Draft

EVIDENCE
Only in ER-negative cell lines, the silencing of Vav1 induced the expression but not the activation of Akt2.

KNOWLEDGE EDITOR

CITATION

CONTENT (02)

METADATA (01)

Assertions (01)

Subject	Relation	Object	CREATE
<input type="checkbox"/> act(p(HGNC:VAV1), ma(tscript))	decreases	r(HGNC:AKT2)	

Edit | Delete

Subject	Relation	Object
<input type="checkbox"/> act(p(HGNC:VAV1), ma(tscript))	decreases	r(HGNC:AKT2)

Assertions (01)

- Failed parse at position 52. Check that you have closed your parenthesis correctly before this point.
act(p(HGNC:VAV1), ma(tscript)) decreases r(HGNC:AKT2)
^
- Invalid BEL Statement – cannot parse

Search for Knowledge

BIODATI | Grid Search Add Export User Help

< Search Here References Assertions

FACETS

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Validation

- Warning-Annotation (15)
- Error-Assertion (7)
- Warning-Assertion (6)

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KNOWLEDGE

- Finalized (32733)
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- Draft (7)
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Update Date

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- < 1 month (27)

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REFERENCES

- a(CHEBI:celecoxib) *decreases* r(RGD:Meox2)
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- a(CHEBI:simvastatin) *decreases* r(HGNC:PTGS2)
- a(SCHEM:Pravastatin) *decreases* r(MGI:Ccl2)

REFERENCES Assertions

Creating a Network - searching for edges

BIODATI | 

Create Network

 CREATE NETWORK

FACETS i

[Clear All](#)

EdgeType ▾

- backbone (5848)
- primary (2569)
- orthologized (2047)
- computed (1583)
- original (1282)
- causal (1263)

Edge Species ▾

- mouse (3756)
- human (3198)
- None (1657)
- rat (1389)

Relations ▾

- transcribedTo (3795)
- translatedTo (2053)
- increases (1732)
- hasActivity (798)
- decreases (706)
- hasComponent (321)
- hasModification (253)

Search Query

Nearest Neighbors Shortest Path

STEPS: 1 | DIRECTION: ANY | Contains

Start Nodes

 Start Node

SUBMIT **RESET**

Create | Add (No active network)
1,000 edges returned (0 selected)

<input type="checkbox"/> Subject	Relation	Object	Project	Nanopub Url
<input type="checkbox"/> act(p(SFAM:"PRKC Famil...)	directlyIncreases	p(HGNC:CYTH2, pmod(Ph, S, ...)		a93b6def-13dd-446d-bd58-2cfe54
<input type="checkbox"/> g(EG:100533709)	transcribedTo	r(EG:100533709)		37a81a6e-d84b-4049-87c4-0a4f5e
<input type="checkbox"/> r(EG:571144)	translatedTo	p(EG:571144)		094a82f5-d22e-413c-ad33-941196
<input type="checkbox"/> g(MGI:4930456L15Rik)	transcribedTo	r(MGI:4930456L15Rik)		e002e6b5-623c-4bea-b428-23c75:
<input type="checkbox"/> r(MGI:Cbarp)	translatedTo	p(MGI:Cbarp)		dd48b8d4-2ccc-4394-bc18-c6a07:
<input type="checkbox"/> bp(GO:"cell differentiation")	increases	r(HGNC:ARL4D)		9b4b11a0-bddd-4b20-9182-e2585
<input type="checkbox"/> a(CHEBI:"cholic acid")	increases	r(MGI:Nab2)		64b5ff86-9c1b-4421-8aef-629fc81:
<input type="checkbox"/> g(EG:100417436)	transcribedTo	r(EG:100417436)		5b7771ed-a5e5-4a46-9562-fcb50c
<input type="checkbox"/> r(EG:571340)	translatedTo	p(EG:571340)		d69d80af-bc88-4cb8-9296-0817da
<input type="checkbox"/> r(MGI:100533709)	hasActivity	r(MGI:100533709)		90076-500-470-2000-2b-400

Provenance ALWAYS!

BIODATI

Create Network

FACETS ⓘ

CREATE NETWORK

EdgeType ▲

- backbone (5848)
- primary (2569)
- orthologized (2047)
- computed (1583)
- original (1282)
- causal (1263)

Edge Species ▲

- mouse (3756)
- human (3198)
- None (1657)
- rat (1389)

Relations ▲

- transcribedTo (3795)
- translatedTo (2053)
- increases (1732)
- hasActivity (798)
- decreases (706)
- hasComponent (321)

Search Query

Nearest Neighbors Shortest Path

STEPS: 1 DIRECTION: ANY Contiguous

SUBMIT **RESET**

Create | Add (No active network)
1,000 edges returned (0 selected)

<input type="checkbox"/> Subject	Relation
act(p(SFAM:"PRKC Family"))	directlyIncreases
g(EG:100533709)	transcribedTo
r(EG:571144)	translatedTo
g(MGI:4930456L15Rik)	transcribedTo
r(MGI:Cbarp)	translatedTo
bp(GO:"cell differentiation")	increases
a(ChEBI:"cholic acid")	increases
g(EG:100417436)	transcribedTo
r(EG:571340)	translatedTo

NANOPUB REVIEW

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Created:9/13/18 Modified:9/13/18, 12:07 AM Creator:Selventa

PhosphoElm data from PMID 15212693

CITATION INFORMATION

Source name: Current biology : CB
Database name: PubMed Database id: 10531036
Citation title: Regulation of ARNO nucleotide exchange by a PH domain electrostatic switch.
Authors: Santy, L C, Frank, S R, Hatfield, J C, Casanova, J E
Reference: Curr Biol 1999 Oct 21 9(20) 1173-6
Publication Date: 1999-10-21

INTERNAL COMMENTS

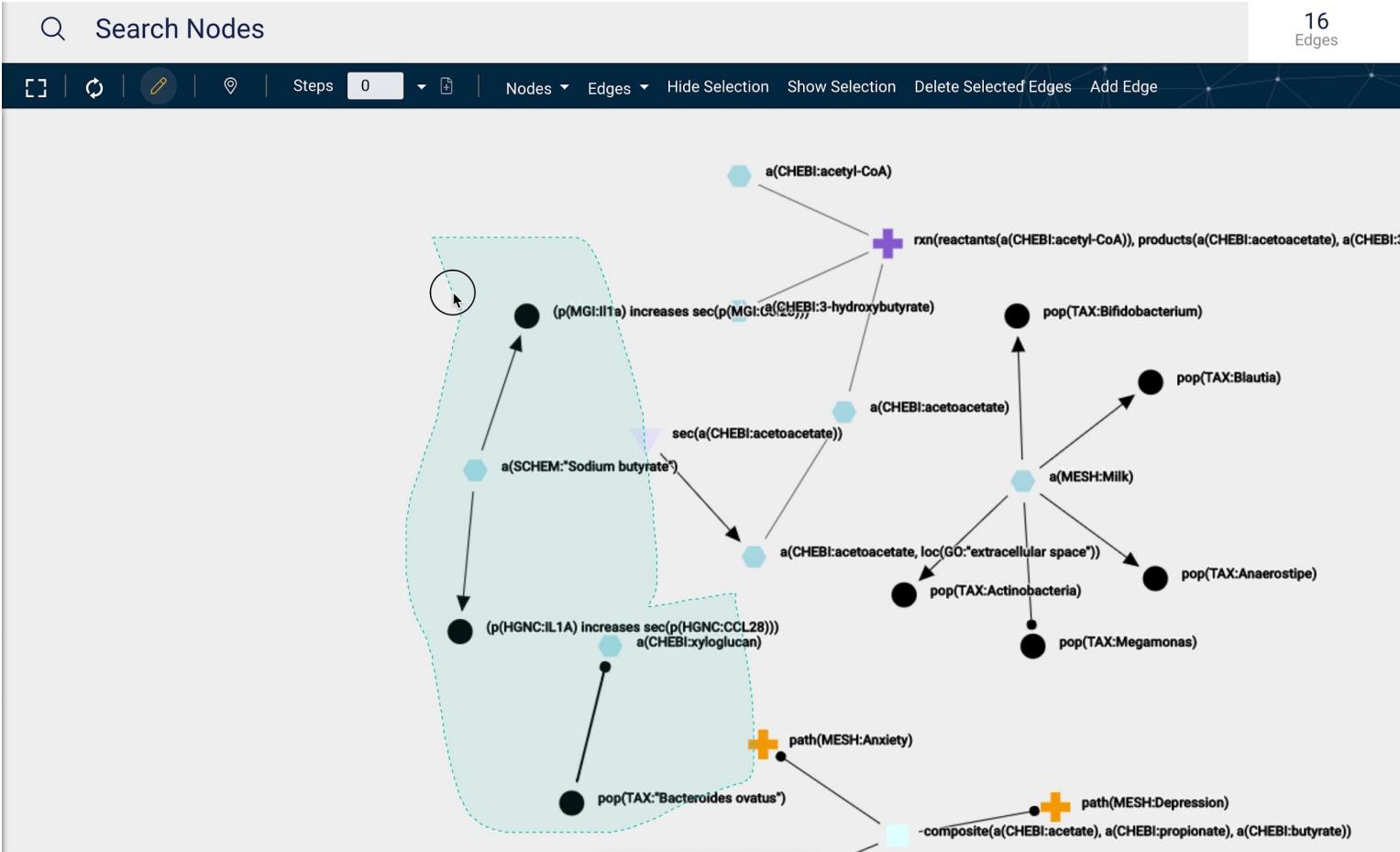
ASSERTIONS (1)

Subject	Relation	Object
act(p(SFAM:"PRKC Family"), ma(kin))	directlyIncreases	p(HGNC:CYTH2, pmod(Ph, S, 392))

ANNOTATIONS (7)

ID	Type	Label
TAX:9606	Species	human
CL:"endothelial cell"	Cell	endothelial cell
MESH:Myometrium	Anatomy	myometrium
MESH:Peroxisomes	CellStructure	peroxisome

Network Lasso





Thank you!

Acknowledgements

- PMI team
 - Julia Hoeng
 - Stephanie Boue
 - Marja Talikka
 - Justyna Szostak
- BEL Language Committee
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 - John Bachman, HMS
 - Natalie Catlett, PatientsLikeMe
 - William Hayes, BioDati
- BioDati
 - Anselmo di Fabio
 - Ally Rose
 - Grant Shih
- Dexter Pratt
- Natalie Catlett
- Renee Deehan-Kenney
- David de Graaf
- Many more!

BEL Resources

- BEL.bio -- <https://bel.bio>
- BEL Language -- <https://language.bel.bio>
- BioDati -- <https://biodati.com>
- PyBEL -- <https://pybel-tools.readthedocs.io>



Looking for BEL Language
Committee volunteers!
and
BEL Evangelists!



BEL – Biological Expression Language
(Open Standard)