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   </list0fCompartments>
   st0fSpecies>
     <species id="A" compartment="cell" initialConcentration="1"/>
     <species id="B" compartment="cell" initialConcentration="1"/>
   </listOfSpecies>
   <parameter id="k1" value="0.1"/>
   </listOfParameters>
   istOfReactions>
     <reaction id="r1" reversible="false">
     <speciesReference species="A"/>
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      <speciesReference species="B"/>
      <kineticLaw>
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            <times/>
```

Building the biological Babylon Tower with

</math>
</kineticLaw>

standard representations of pathways



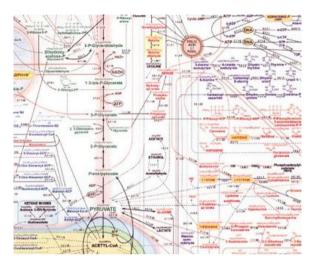


Biochemical pathways are old ...

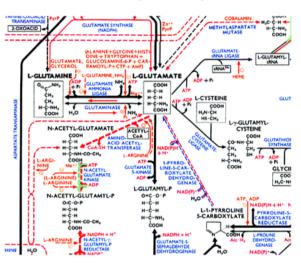
Gortner (1949)

Glycogen Phosphate Glucose Phosphate Glucose Phosphate Glucose Phosphate Glucose-6-phosphate Fructose-6-phosphate Phosphate ATP 2-Phosphopyruvic acid 2-Phosphopyruvic acid 3-Phosphoglyceric acid 3-Phosphoglyceric acid 1,3-Diphosphoglyceric acid 1,3-Diphosphoglyceric acid 1,3-Diphosphoglyceric acid 1,3-Diphosphoglyceric acid 1,3-Diphosphoglyceric acid

Nicholson (1970)



Michal (1984)



- "Hand drawing" on paper
- → no software-based browsing, processing and analysis

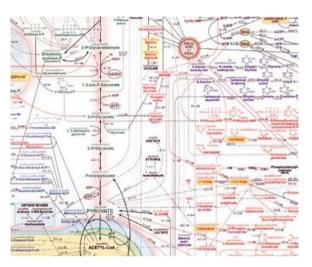


Biochemical pathways are old ... or not so much

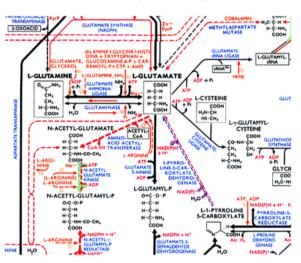
Gortner (1949)

Glycogen Phosphate Glucose-1-phosphate Glucose Phosphate Glucose Glucose Phosphate Glucose-6-phosphate Fructose-6-phosphate Phosphate ADP 2-Phosphopyruvic acid 2-Phosphopyruvic acid 3-Phosphopyruvic acid 1,3-Diphosphoglyceric acid 1,3-Diphosphoglyceric acid 1,3-Diphosphoglyceric acid 1,3-Diphosphoglyceric acid 1,3-Diphosphoglyceric acid 1,3-Diphosphoglyceric acid

Nicholson (1970)

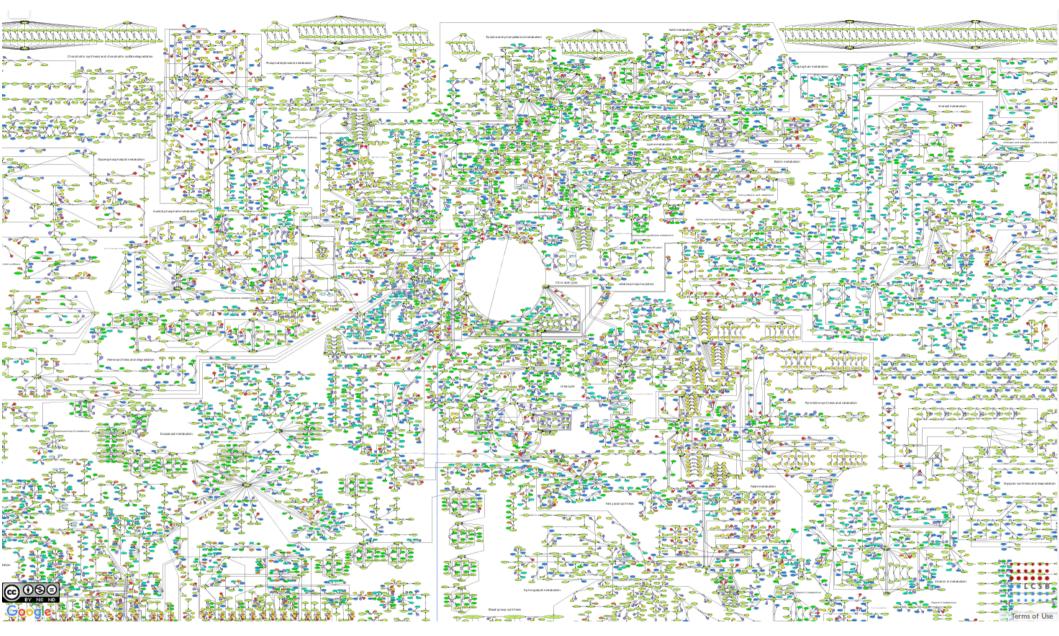


Michal (1984)



- 1990s: high-throughput data generation → Large amount of knowledge increase in computing power → automatic reconstruction, browsing and analysis
- Databases: EcoCyc (1994), KEGG (1995), Reactome (2000)
- Formats: BioPAX (2000), SBML (2000), PSI-MI (2002)



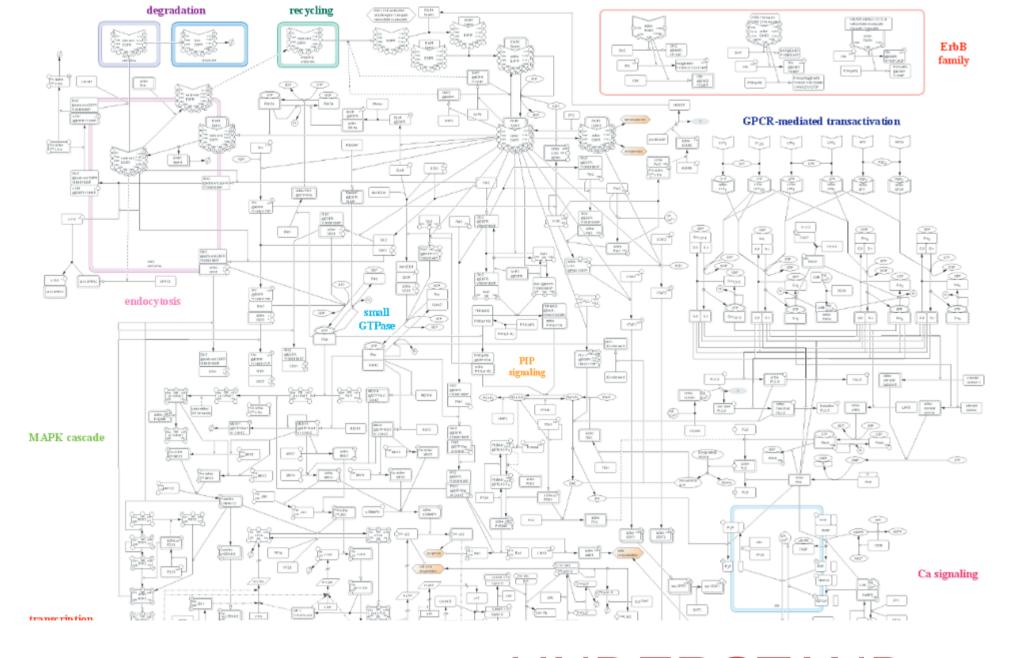


http://vmh.uni.lu

UNDERSTAND

Metabolic networks

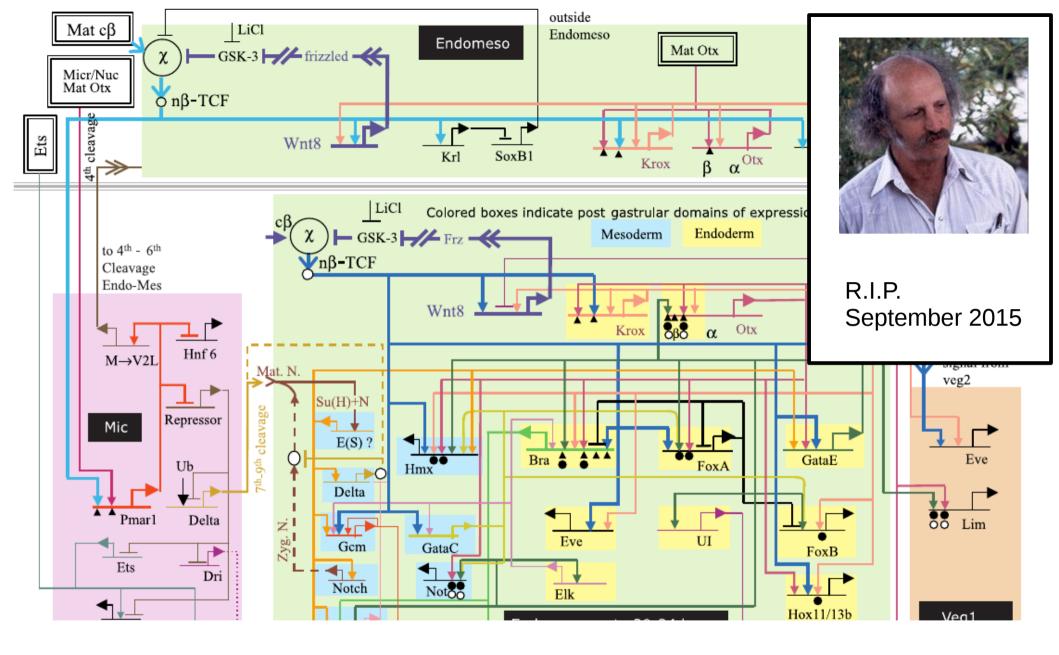




UNDERSTAND

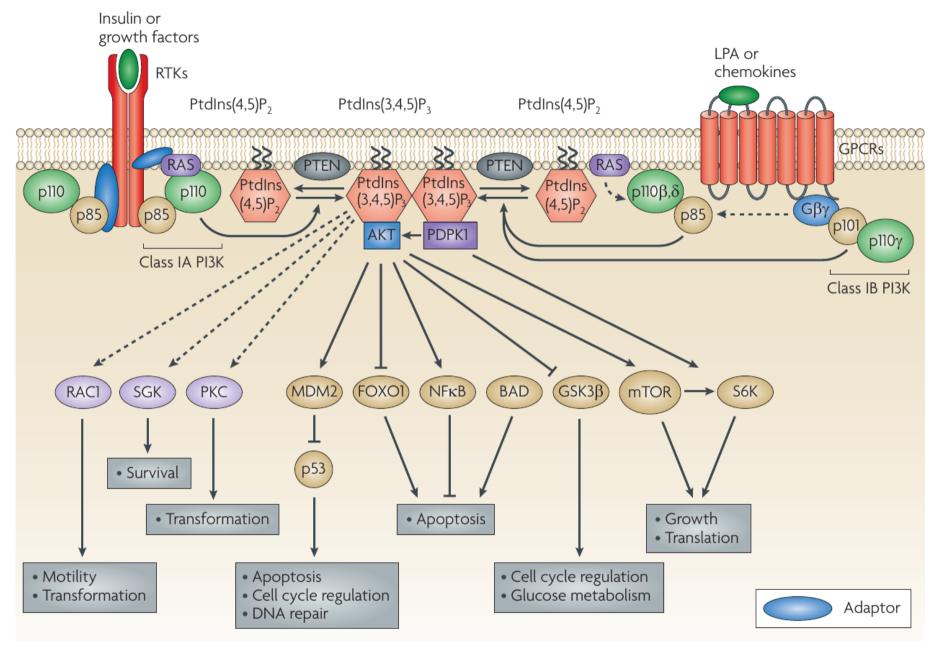
Signalling pathways





UNDERSTAND

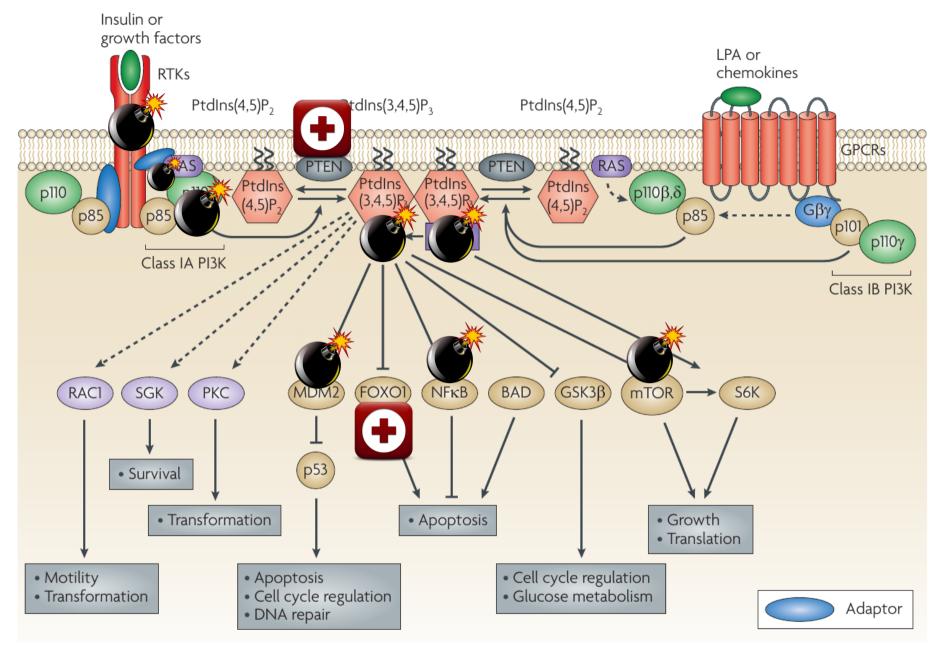
Gene regulatory networks





Liu et al (2009) Nat Rev Drug Discov

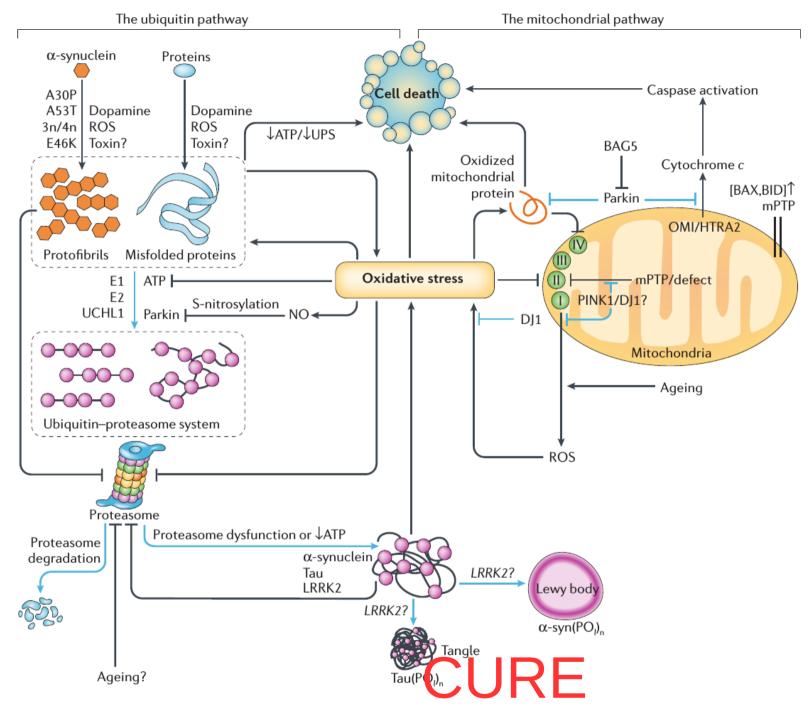




Liu et al (2009) Nat Rev Drug Discov

CURE
Breast cancer

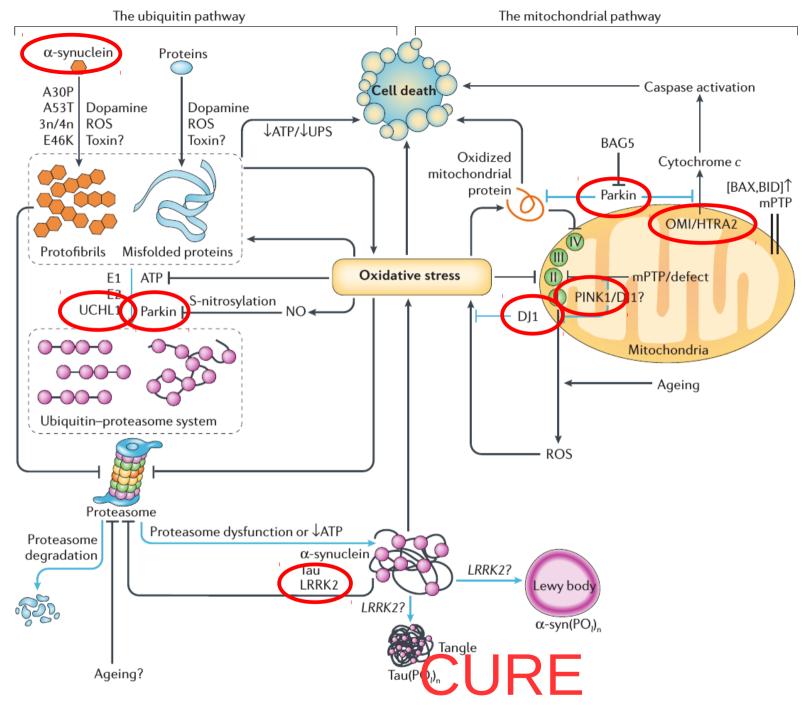




Abou-Sleiman et al (2006) Nat Rev Neurosci

Parkinson's disease





Abou-Sleiman et al (2006) Nat Rev Neurosci

Parkinson's disease



systems medicine disease maps

ABOUT
PROJECTS
GUIDELINES
PUBLISHED MAPS
TOOLS
EVENTS
GET INVOLVED
CONTACT

Welcome to the portal for communication, news, guidelines, and other information about the **Disease Maps Project**, an effort to comprehensively represent disease mechanisms for various diseases.

Upcoming events

The second Disease Maps Community Meeting is scheduled 2-3 October 2017 in Belval, Luxembourg, as a satellite event before the Impact of Big Data Analytics on Healthcare conference 4-5 October 2017.

What is a disease map?

A systems medicine disease map is a high-quality expert-approved representation of disease mechanisms, a conceptual model of a disease. The detailed layer consists of interconnected signalling, metabolic and gene regulatory pathways stored in standard formats such as SBGN, SBML and BioPAX. The involvement of domain experts (clinicians and biologists) ensures that all the disease hallmarks are covered and relevant knowledge is adequately represented.

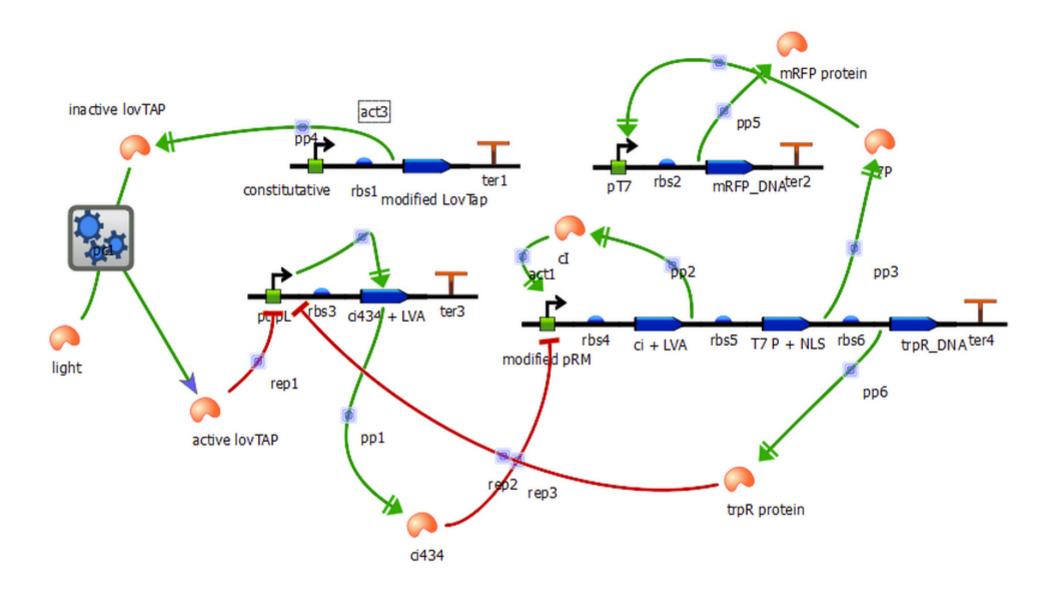
What are the disease maps for?

In translational medicine projects disease maps are used for advanced 'omics data interpretation and hypothesis generation, as the basis for mathematical models, for predictive analysis and for building clinical decision support systems (CDSS). The maps can be used for educational purposes as teaching aids.

The community

The Disease Maps Project is designed as a large-scale community effort. It is a network of groups that work together in order to better understand disease mechanisms. We exchange best practices, share information, develop tools to make it easier for all the involved groups to achieve their goals.





BUILD Synthetic Biology

Team Nanjing, iGEM (2013)



Ambiguity of usual representations





Ambiguity of usual representations



is transformed into

translocates (X "=" Y)

is degraded into

associates into

dissociates into

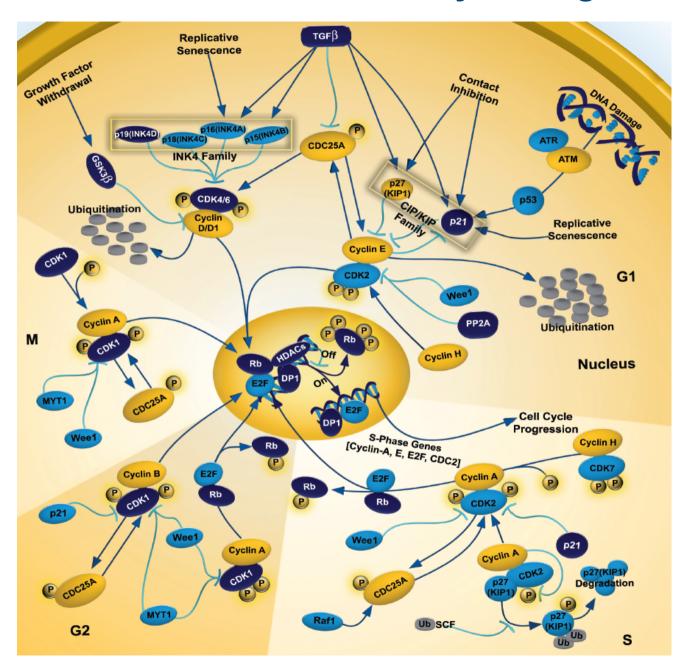
stimulates the activity of

stimulates the expression of

catalyses the formation of

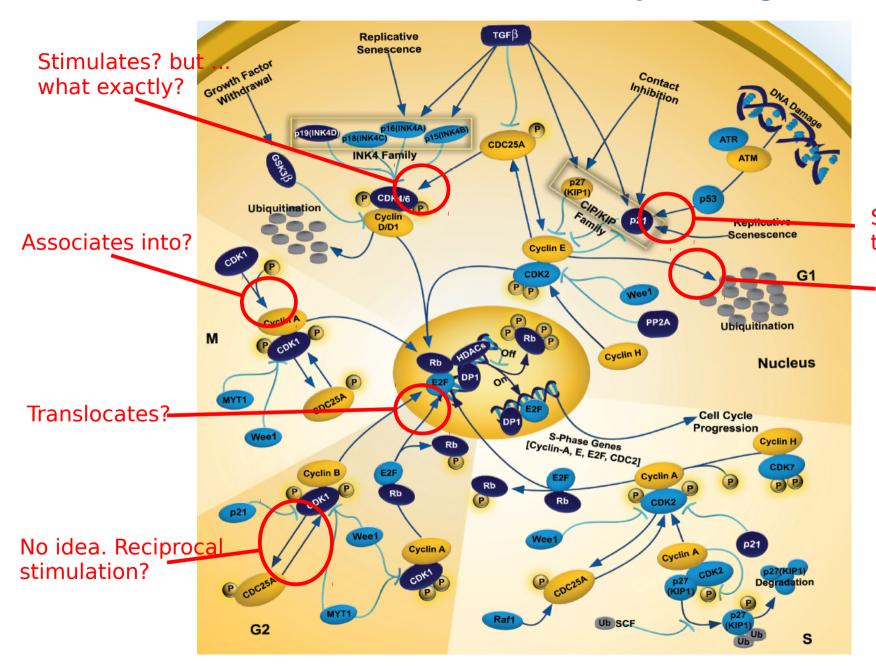


Can-this be understood by biologists?





Can-this be understood by biologists?



Stimulates gene transcription?

Is degraded?



Ambiguity of usual representations

X inhibits Y

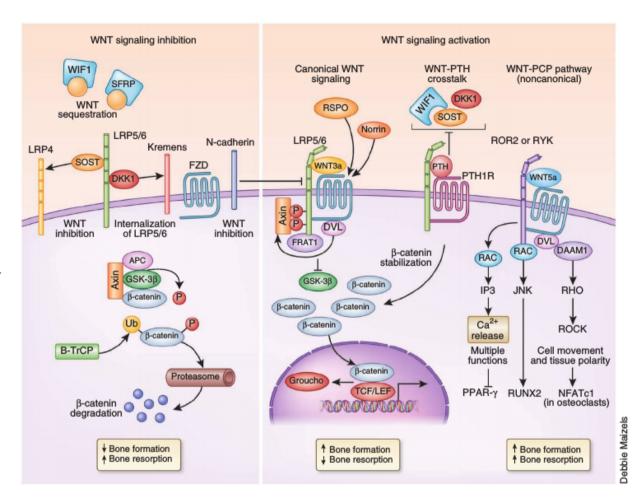


Ambiguity of usual representations

X inhibits Y inhibition



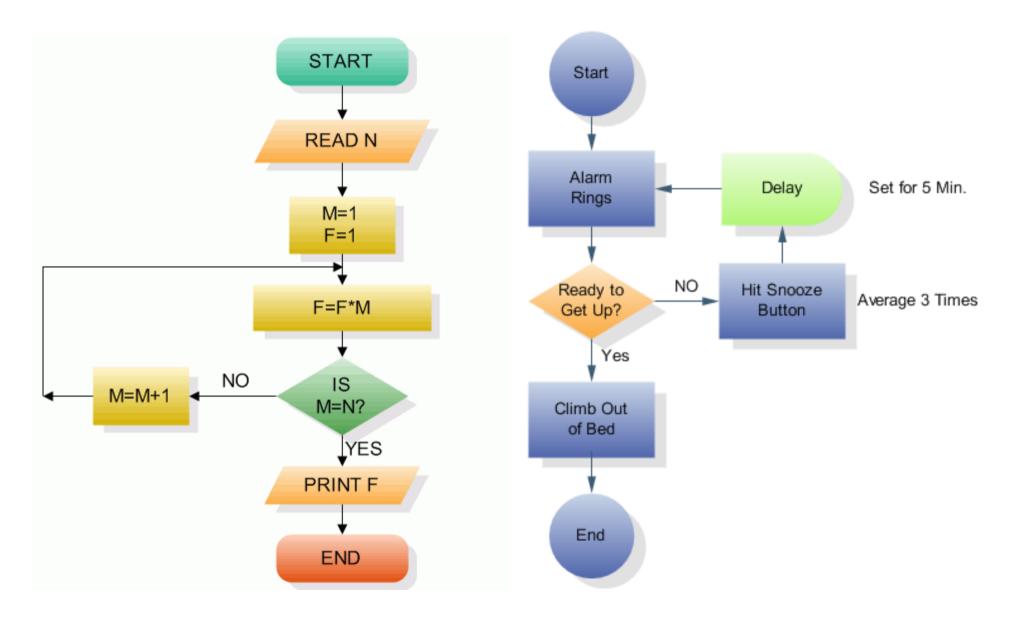
Figure 1 WNT signaling: a simplified view. In the absence of WNT, the amounts of B-catenin are low, except in adherens junctions, because of its constitutive targeting by a multiprotein destruction complex (left). The tumor suppressors axin and adenomatous polyposis (APC) bring B-catenin to GSK-3B and casein kinase 1 (CK1) (not shown), resulting in its phosphorylation (Ps in circles) at specific serine/threonine residues (left). Phosphorylated β-catenin is then targeted for polyubiquitination (Ub) (predominantly by the E3 ligase B-TrCP) and proteosomal destruction. T cell factor/lymphoid enhancer factor (TCF/LEF) transcription factors are repressed by Groucho in the nucleus. Binding of canonical WNT ligands to a dual receptor complex comprising the WNT co-receptors LRP5 or LRP6 (LRP5/6) and one of the seven transmembrane receptors of the FZD family (right) initiates WNT-β-catenin signaling. Axin moves to the LRP5/6 tail at the membrane through its interaction with dishevelled (DVL. also called DSH), which is recruited by FZD (right). This forms a complex that also includes FRAT1 and GSK-3B, which prevents phosphorylation of



β-catenin and its proteosomal degradation. β-catenin accumulates in the cytoplasm and translocates into the nucleus, where it associates with members of the TCF/LEF transcription factors while displacing Groucho to control target gene transcription. WNT signaling is modulated not only through fine tuning by a large number of WNT ligands and RSPO proteins and norrin (right) but also by extracellular antagonists such as DKK1, SOST and Wise, which bind LRP5/6 (left). Their antagonism is mediated or enhanced by receptors such as Kremen proteins and LRP4. In addition to sequestering β-catenin, N-cadherin also inhibits WNT–β-catenin signaling by interacting with LRP5 (left to right). In contrast, secreted frizzled-related proteins (SFRPs) and WNT inhibitory factor 1 (WIF1), which have ligand specificity, inhibit WNT signaling by directly sequestering WNT ligands and inhibiting both canonical and noncanonical WNT signaling (left). The PTH1 receptor can also activate the pathway in the absence of WNT ligands by forming a complex with LRP5/6 after PTH binding (right). In the WNT-PCP pathway (right), WNT binding to FZD also recruits DVL, which forms a complex with dishevelled associated activator of morphogenesis 1 (DAAM1) to trigger activation of the small G protein RHO, which in turn activates RHO-associated kinase (ROCK). Alternatively, DVL forms a complex with RAC, resulting in Jun kinase (JNK) activity. The WNT-Ca²⁺ pathway is activated by WNT5a binding to FZD and receptor-tyrosine-kinase-like orphan receptor (ROR). Intracellular calcium concentrations increase after WNT-induced coupled G protein activation of phospholipase C (PLC), resulting in dystroglycan 1 (DAG) and inositol 1,4,5-trisphosphate, type 3 (IP3) generation and cyclic GMP (cGMP)-specific phosphodiesterase (PDE) decreasing the amount of cGMP. NFATc1, nuclear factor of activated T cells, cytoplasmic, calcineurin dependent 1; PPAR-γ, peroxisome proliferator activated receptor-γ.

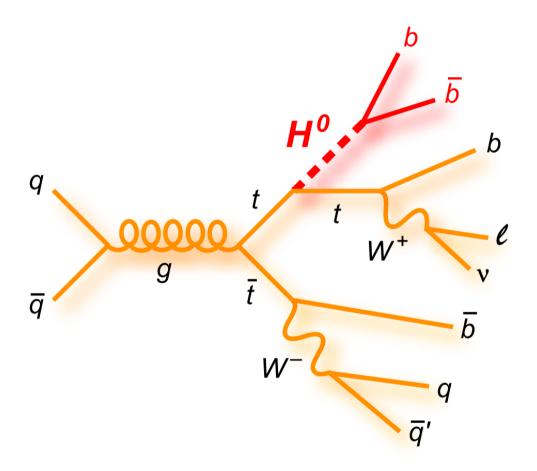


Every computer scientist understands those



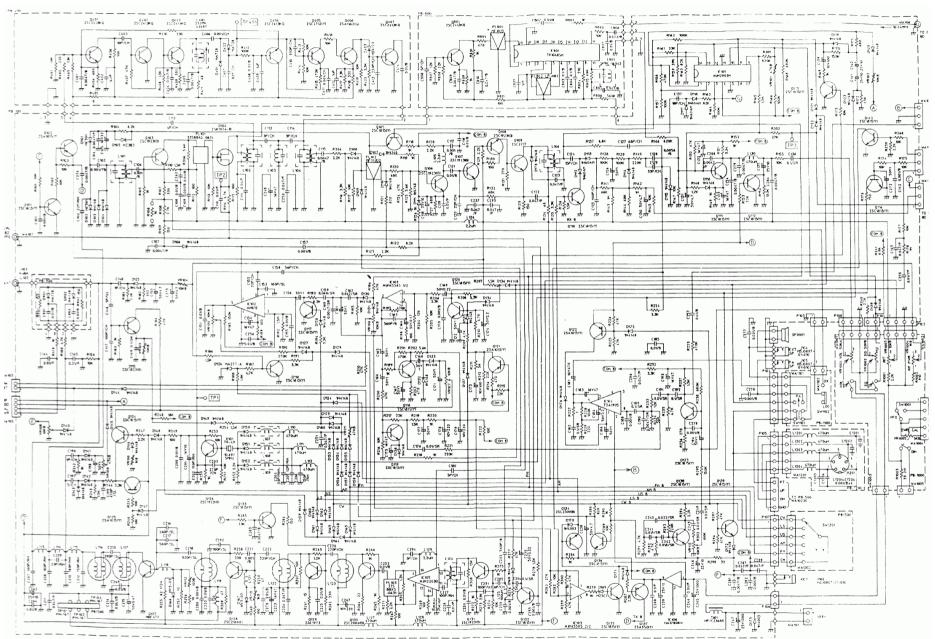


Every physicist understands this





Every engineer understands that





The Systems Biology Graphical Notation

- A tool to graphically describe and unambiguously interpret biochemical and cellular events
- Limited amount of symbols Smooth learning curve
- Can represent logical or mechanistic models, biochemical pathways, at different levels of granularity
- Detailed technical specification, precise data-models, standard API and growing software support
- Developed over ten years by a diverse community, including biologists, modellers, computer scientists etc.

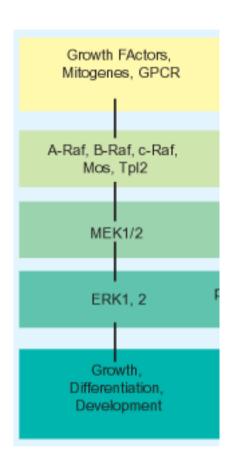






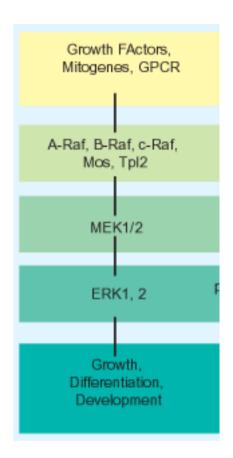


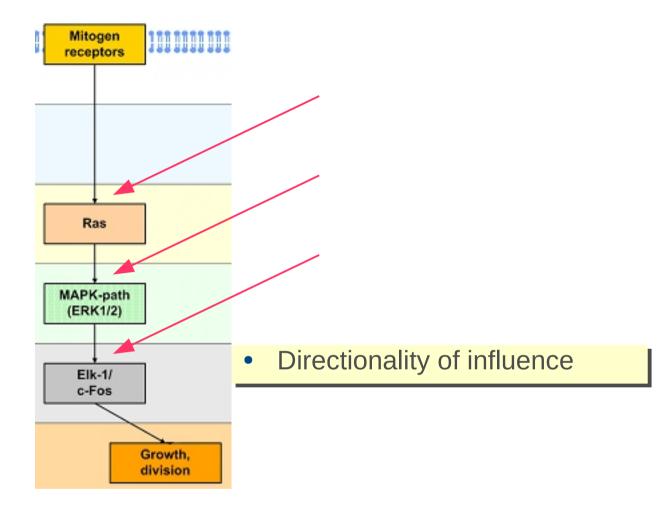
Different granularities of a pathway



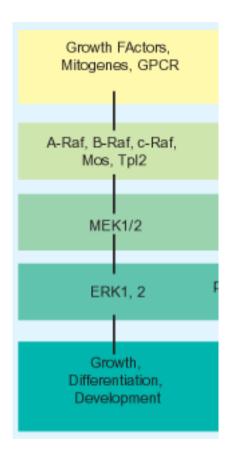
- No temporal sequence
- No directionality
- No biochemical effects
- No mechanistic descriptions

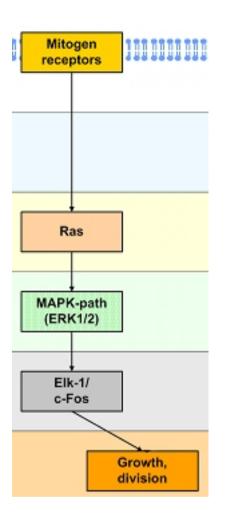




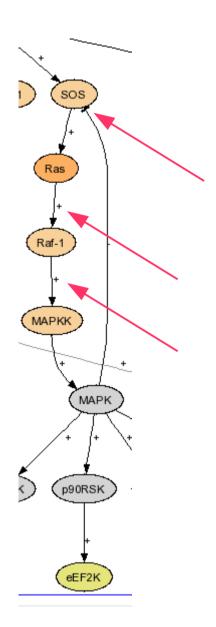




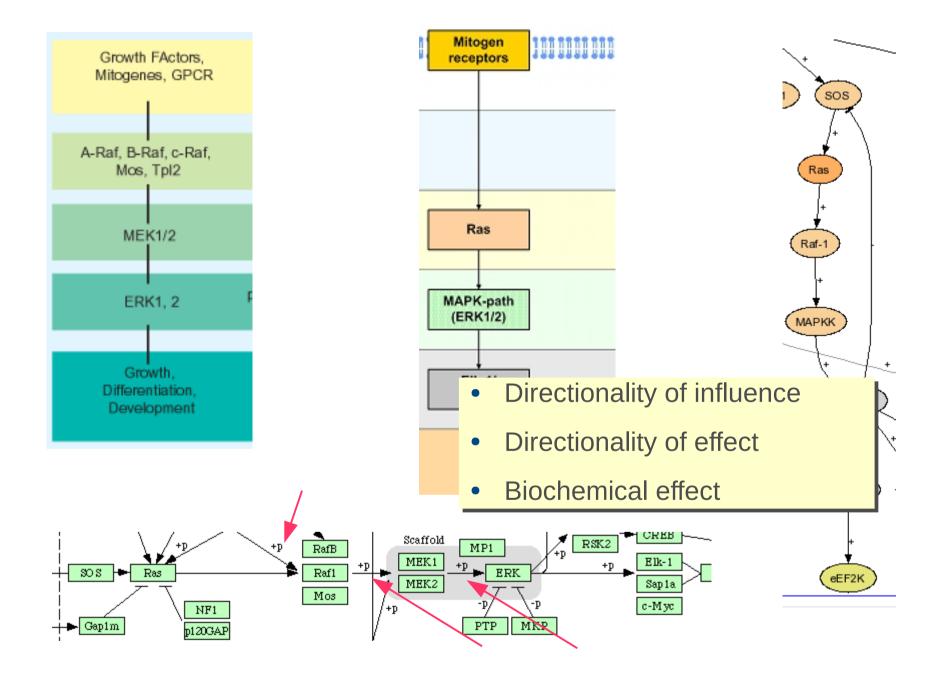




- Directionality of influence
- Directionality of effect



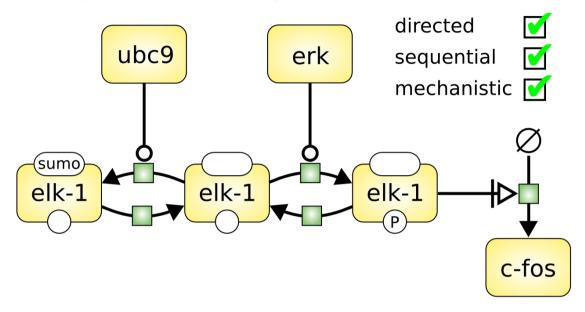




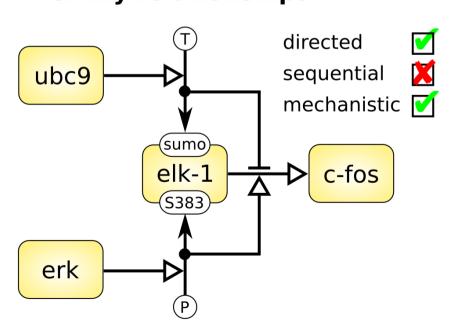


One notation – three languages

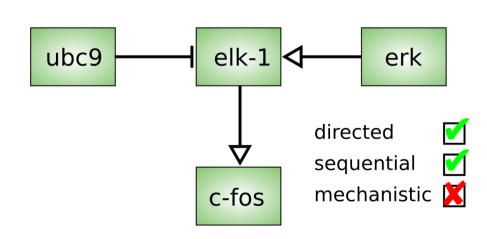
process descriptions



entity relationships

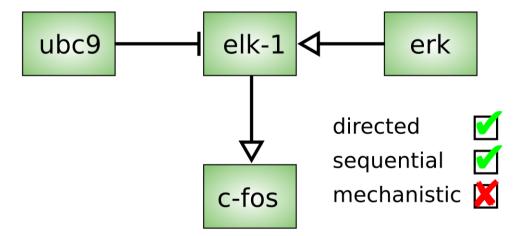


activity flows



Functional descriptions: the Activity Flows

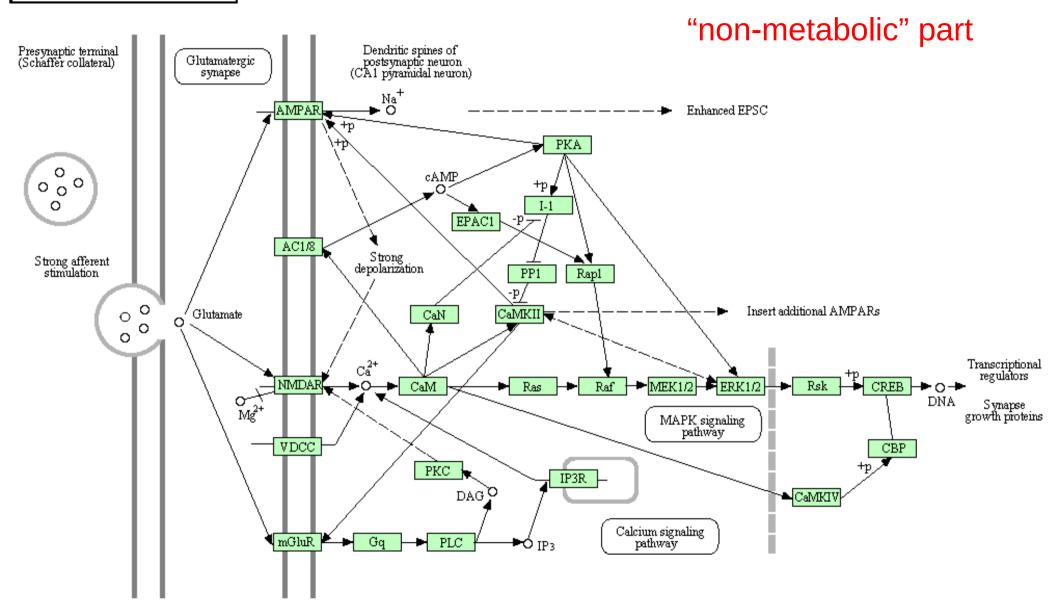
- Logical modelling
- Signalling pathways, gene regulatory networks





http://www.genome.jp/kegg/pathway.html

LONG-TERM POTENTIATION







> Print Version

Pathway Tools

> Glossary

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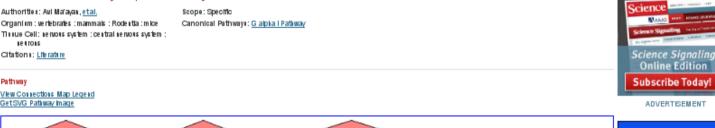
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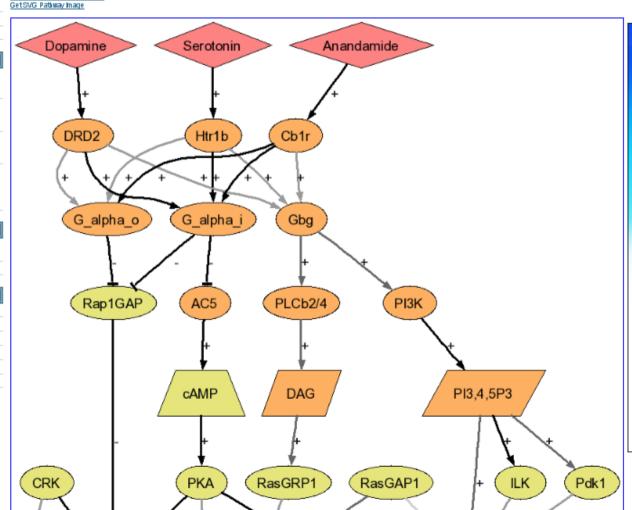
Feedback

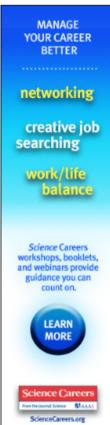
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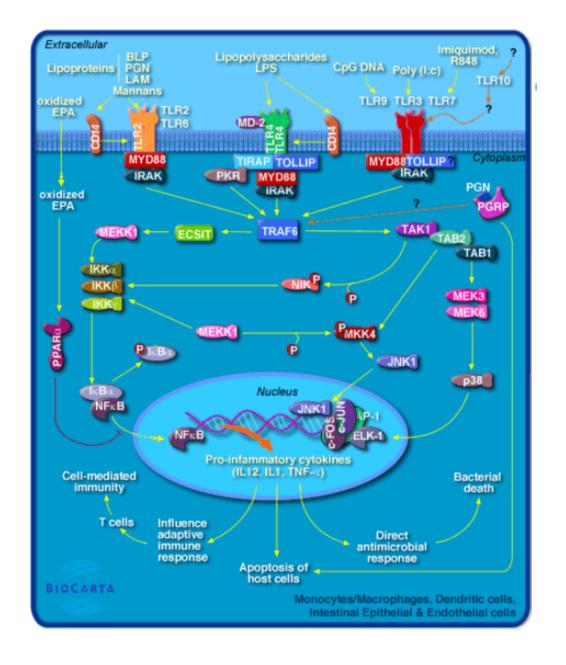




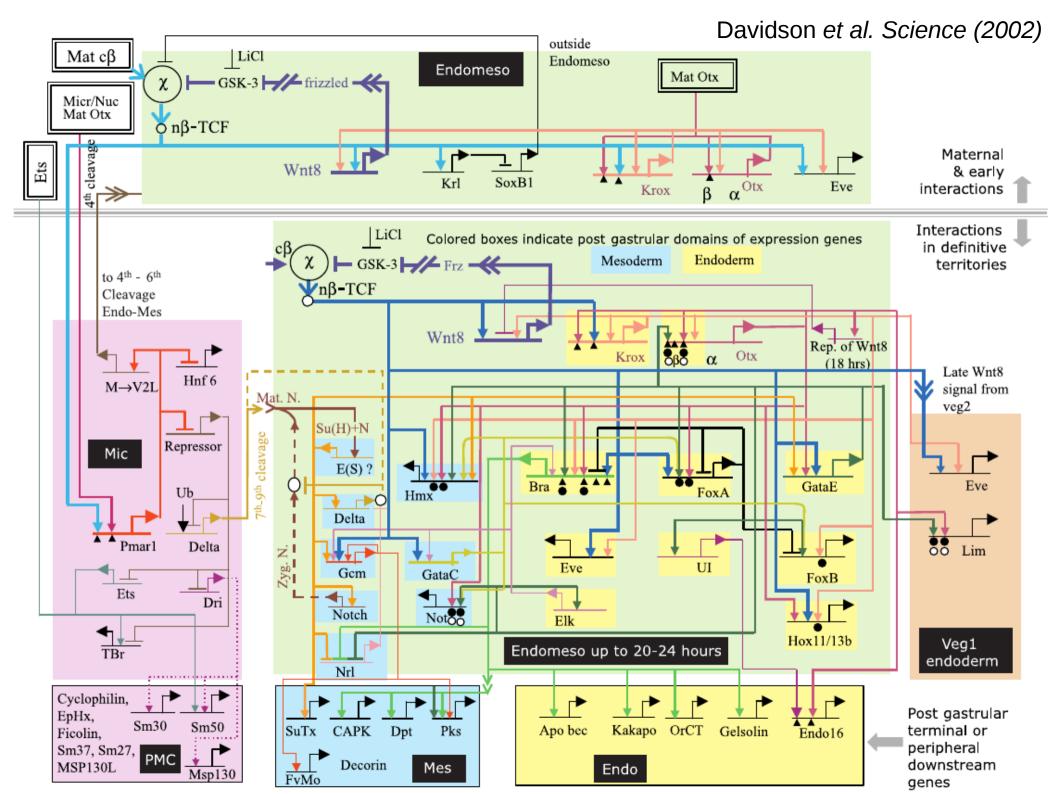
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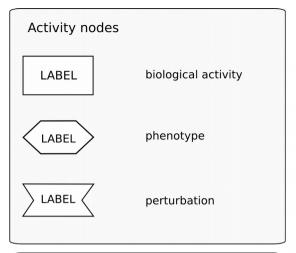
http://cgap.nci.nih.gov/Pathways/BioCarta.org

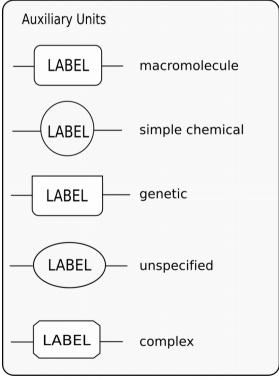


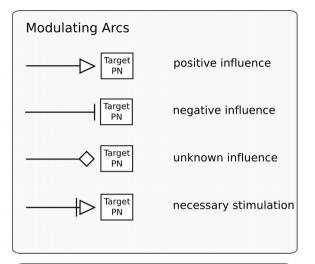


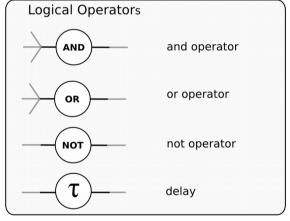


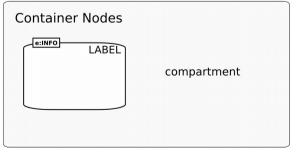
SBGN Activity Flows L1 reference card



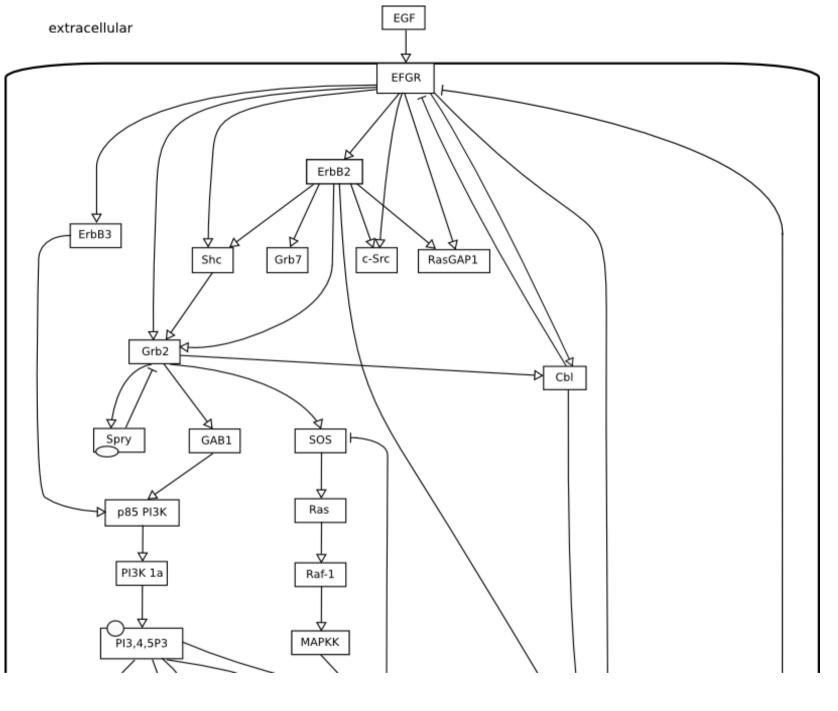








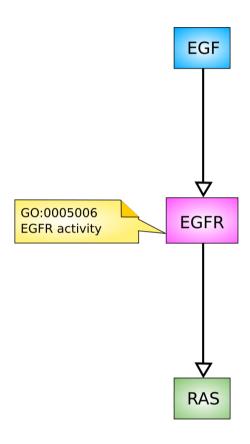




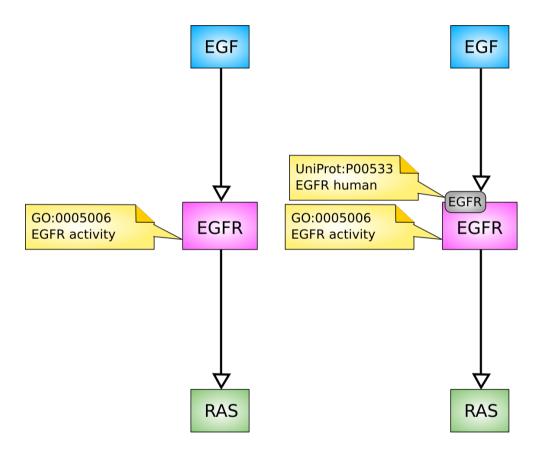
Example of Activity Flow map



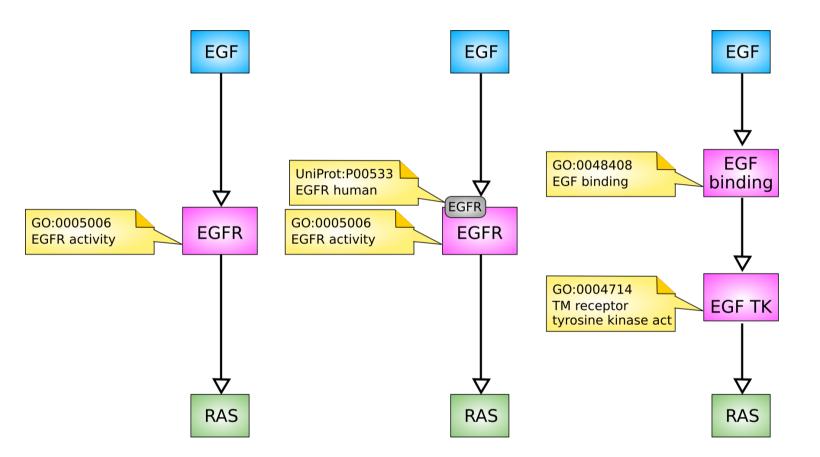
TIMTOWTDI (There Is More Than One Way To Do It)



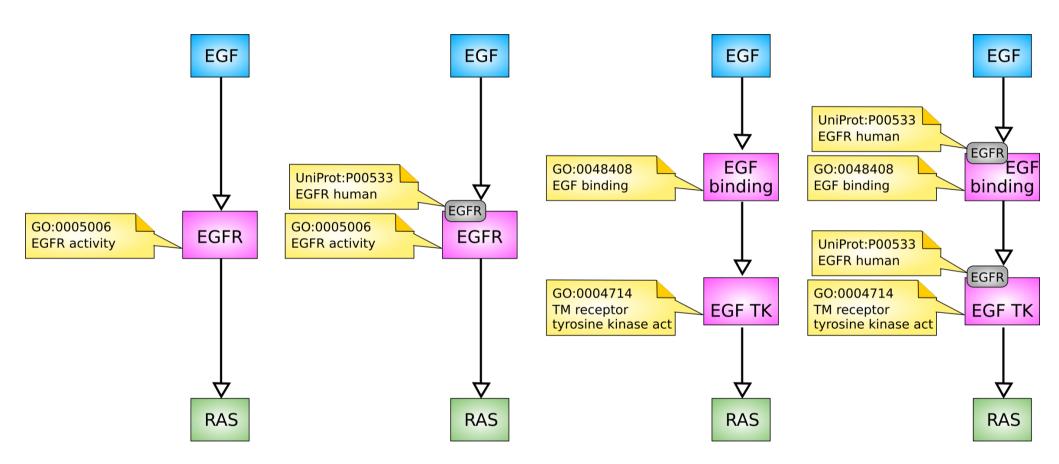




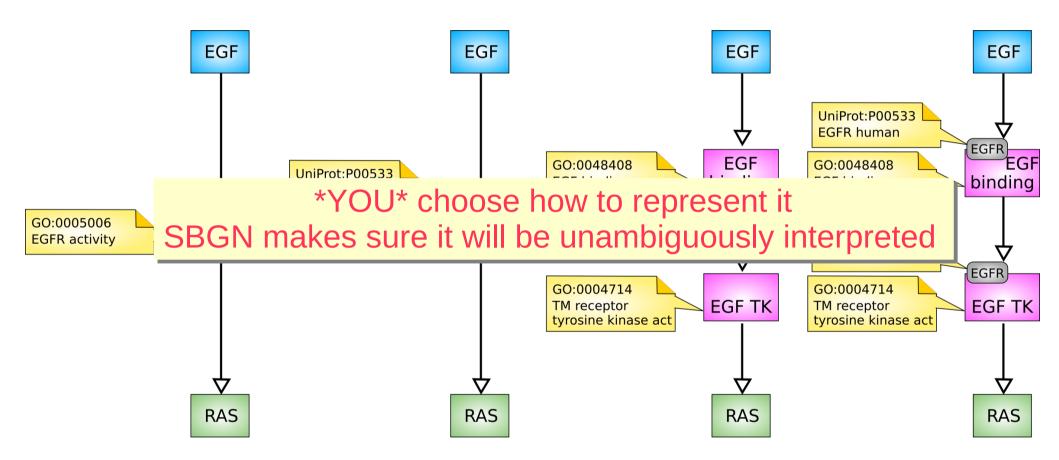






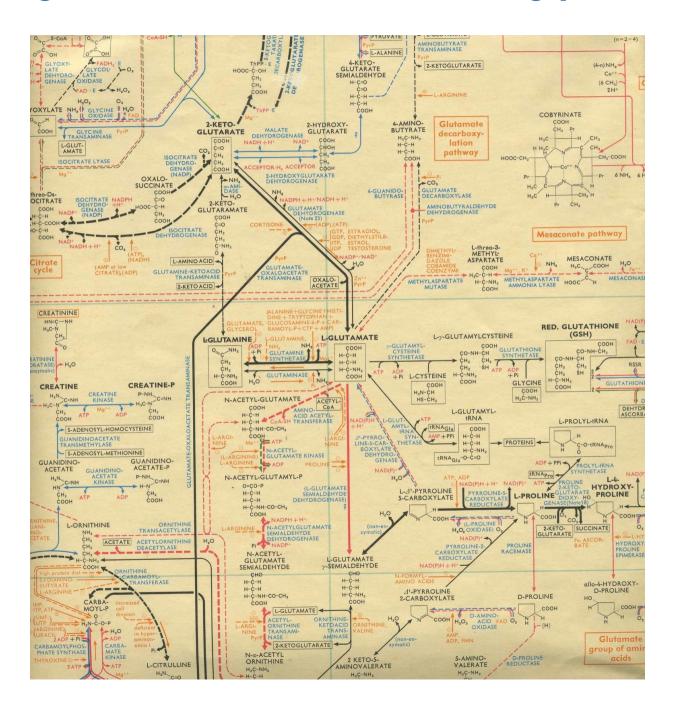






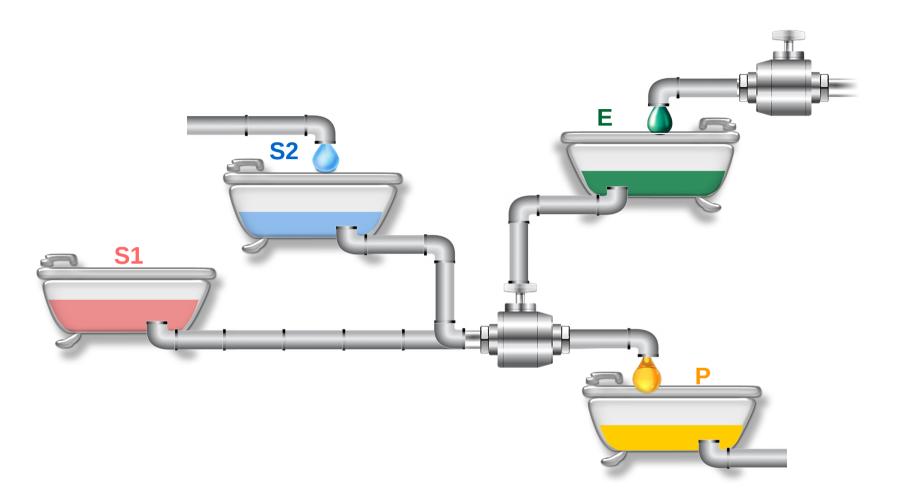


Puting in mechanisms: describing processes



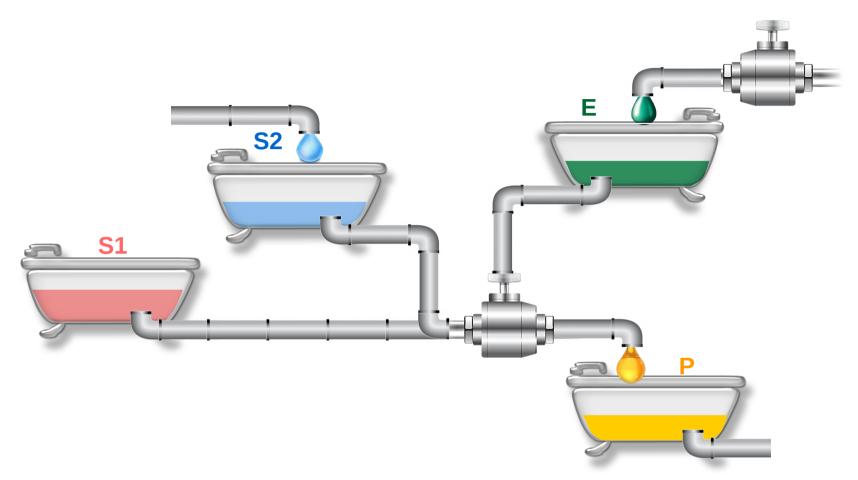


A biochemical reaction is a process





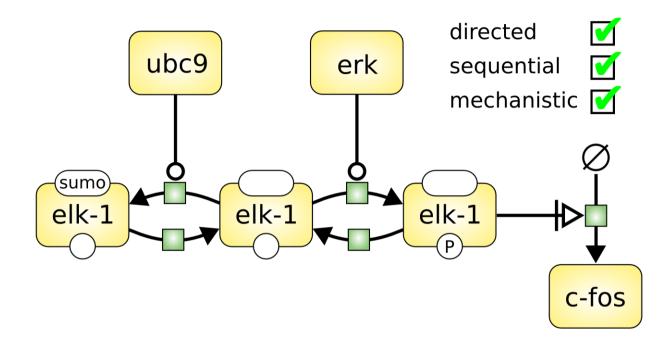
A biochemical reaction is a process



- → Reconstruction of state variable evolution from process descriptions:
- Processes can be combined in ODEs (for deterministic simulations);
 transformed in propensities (for stochastic simulations)
- Systems can be reconfigured quickly by adding or removing a process



SBGN Process Descriptions



- Process modelling
- Biochemistry, Metabolic networks
- Generally within "closed world"
- Subjected to combinatorial explosion



Open world

Anything not explicitly stated is unknown

Failure to observe does not imply non-existence

New pieces of knowledge do not affect prior pieces

Closed world

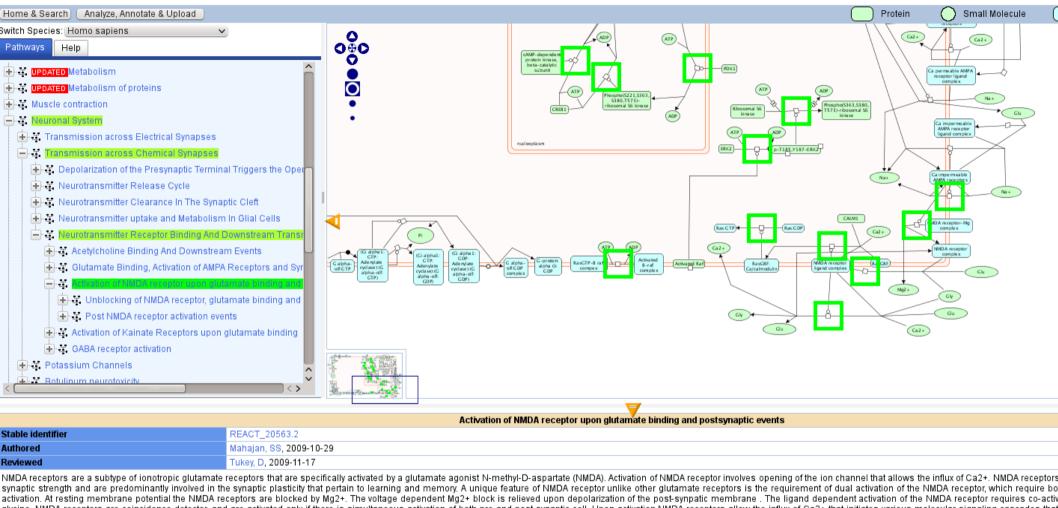
Anything not explicitly stated does not exist

Failure to observe implies non-existence

New pieces of knowledge might change the meaning of prior pieces



http://www.reactome.org



glycine. NMDA receptors are coincidence detector, and are activated only if there is simultaneous activation of both pre and post-synaptic cell. Upon activation NMDA receptors allow the influx of Ca2+ that initiates various molecular signaling cascades that memory.

cytoplasm GO nucleoplasm GO Cohen, S. Greenberg, ME Communication between the synapse and the nucleus in neuronal development, plasticity, and disease 2008 Annu Rev Cell Dev Biol PubMed Activation of NMDA receptor upon glutamate binding and postsynaptic events [Dictyostelium discoideum] Activation of NMDA receptor upon glutamate binding and postsynaptic events [Schizosaccharomyces pombe] Activation of NMDA receptor upon glutamate binding and postsynaptic events [Saccharomyces cerevisiae]

Activation of NMDA receptor upon glutamate binding and postsynaptic events [Caenorhabditis elegans]

Homo sapiens extracellular region GO plasma membrane GO

Organism

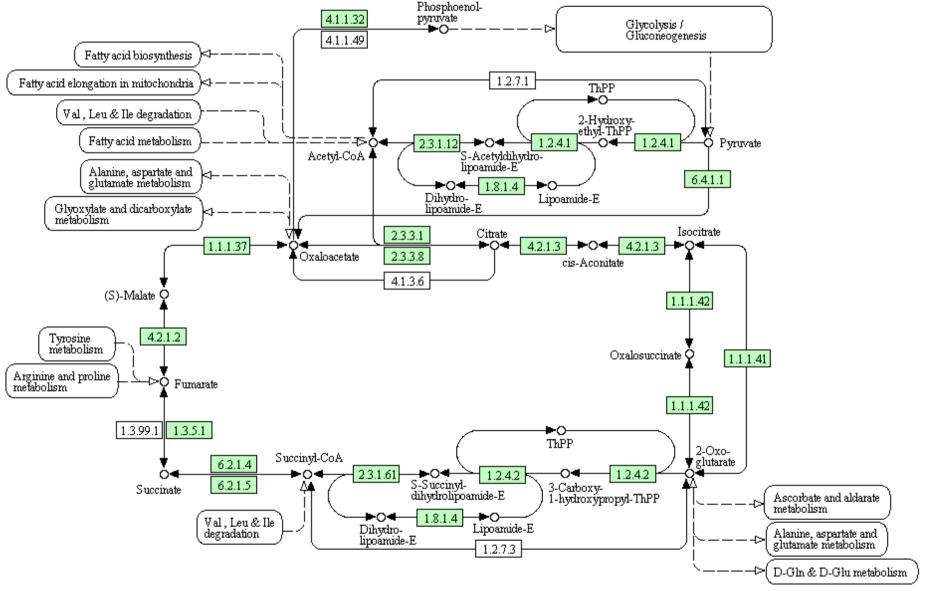
Cellular compartment

prins areases

http://www.genome.jp/kegg/pathway.html

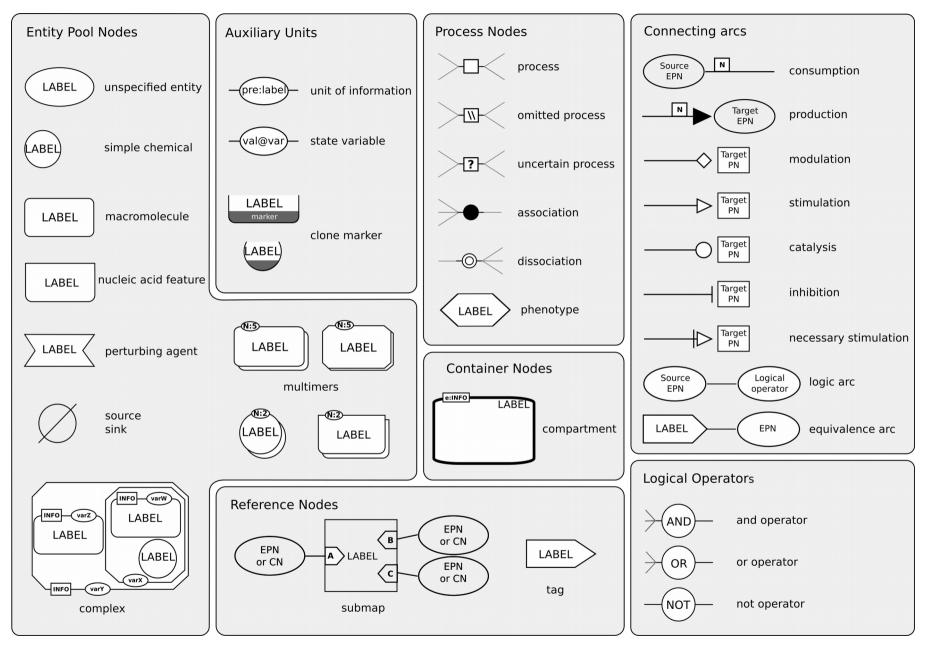
CITRATE CYCLE (TCA CYCLE)

"metabolic" part



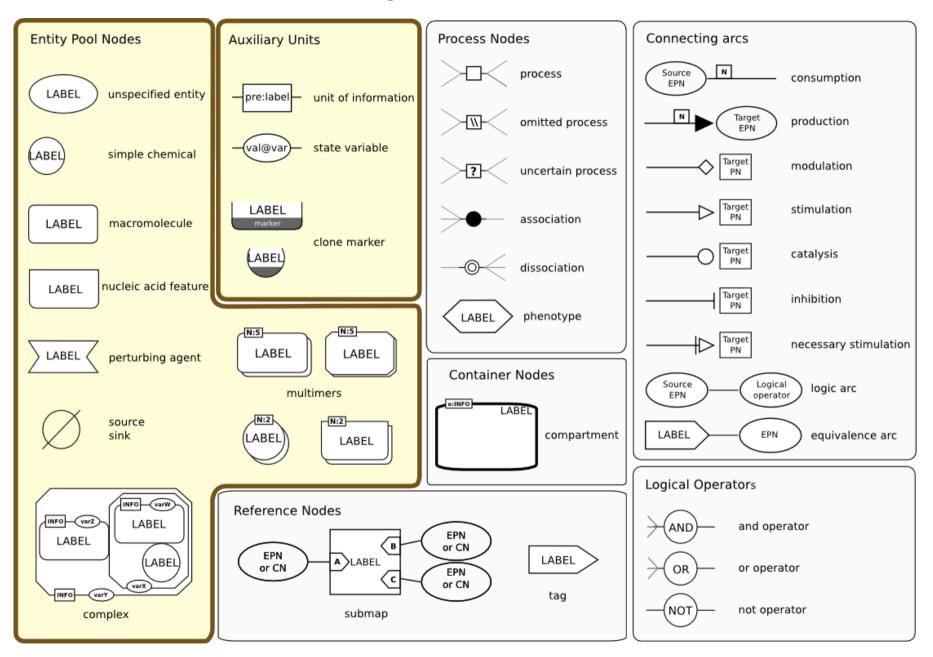


SBGN Process Diagram L1 reference card



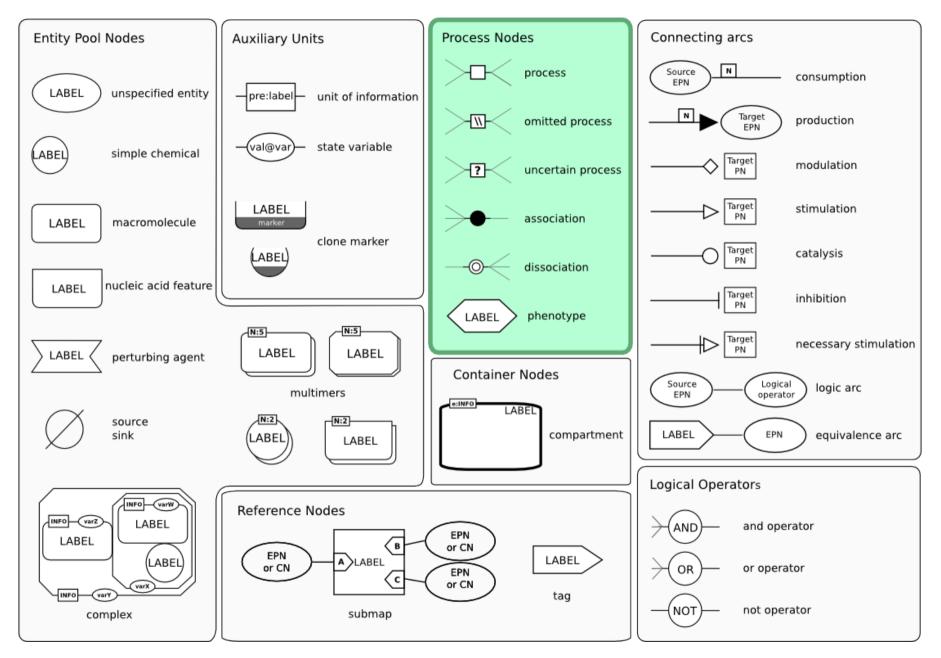


Entity Pool Nodes



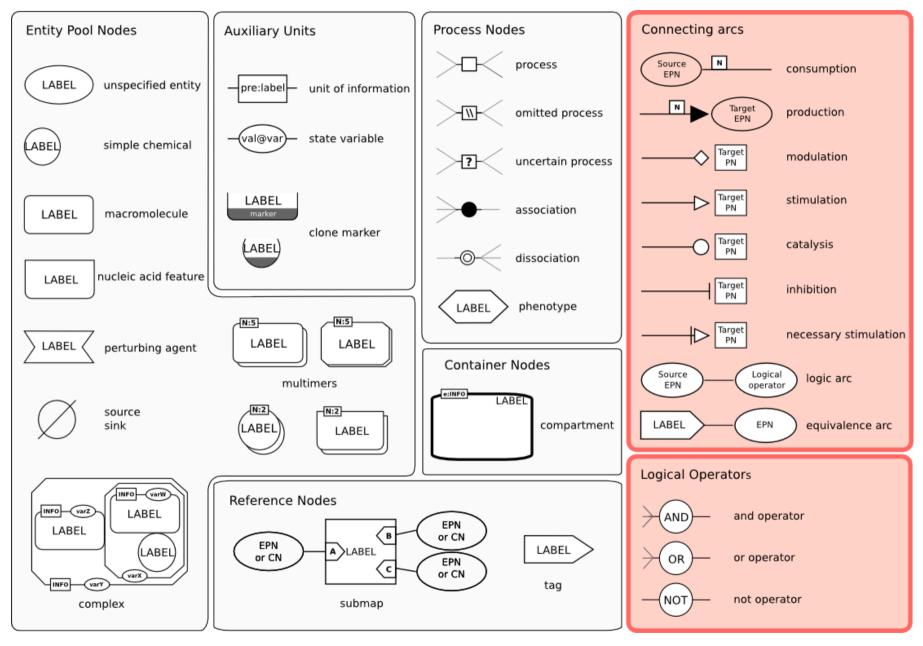


Process Nodes



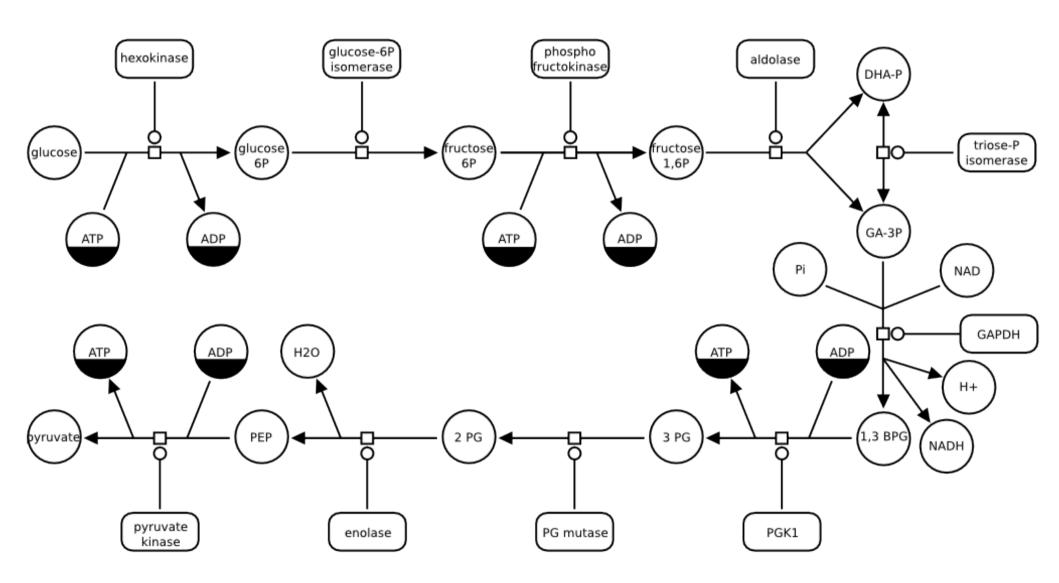


Connecting arcs



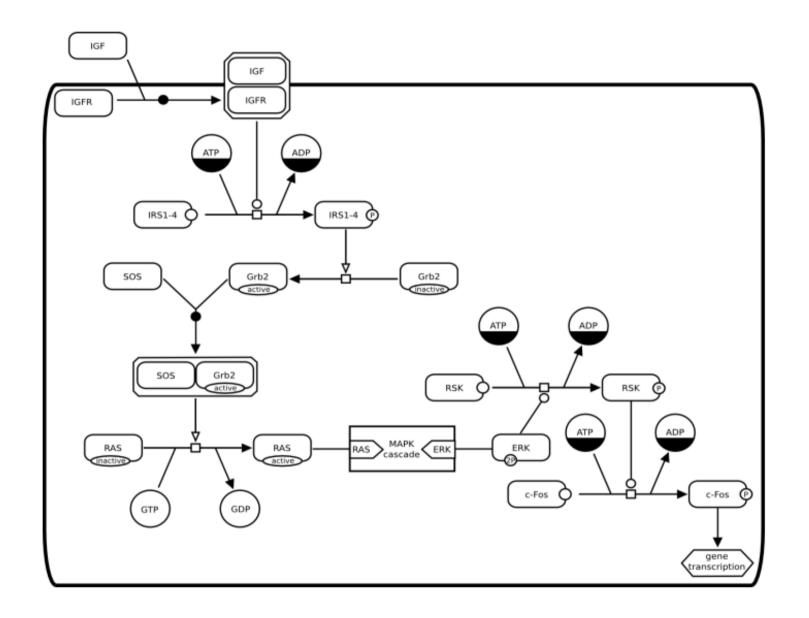


Metabolism in SBGN PD



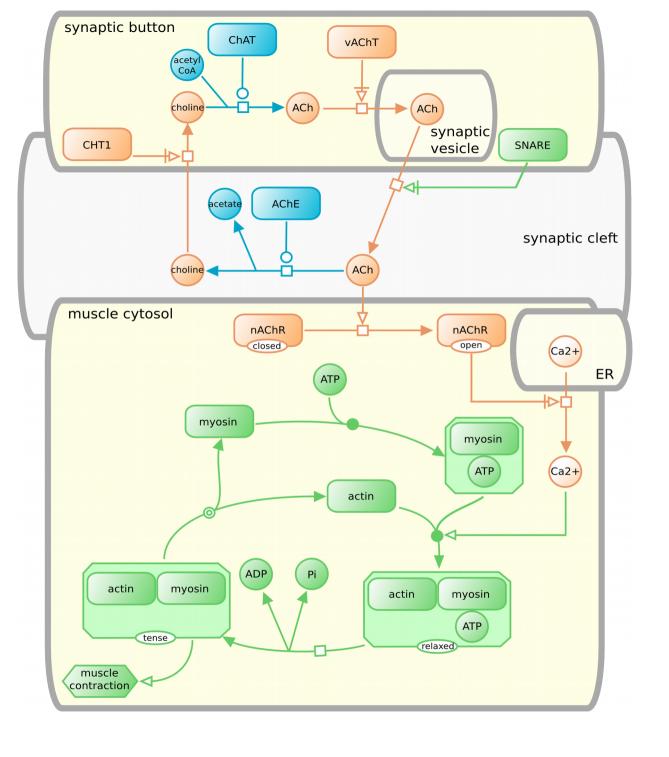


Signalling in SBDG PD



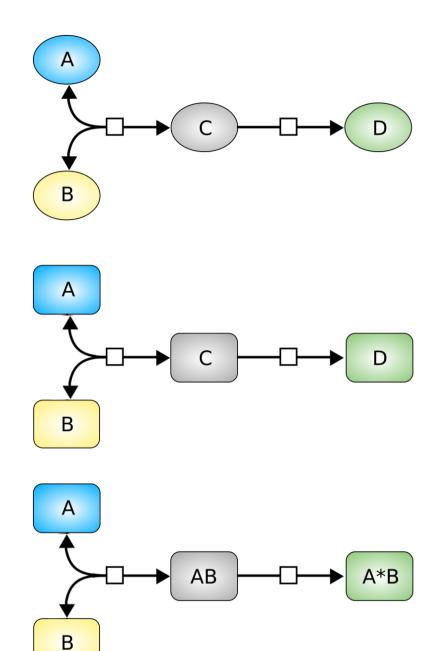


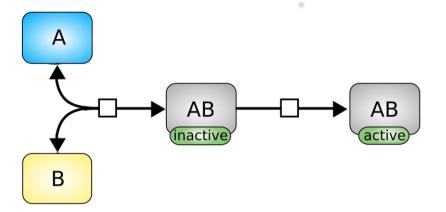
Multicellular processes

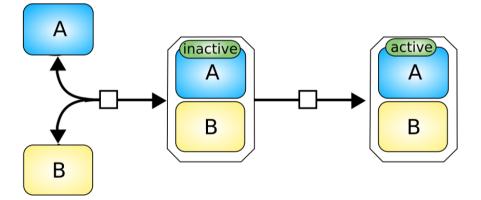




Variable granularity

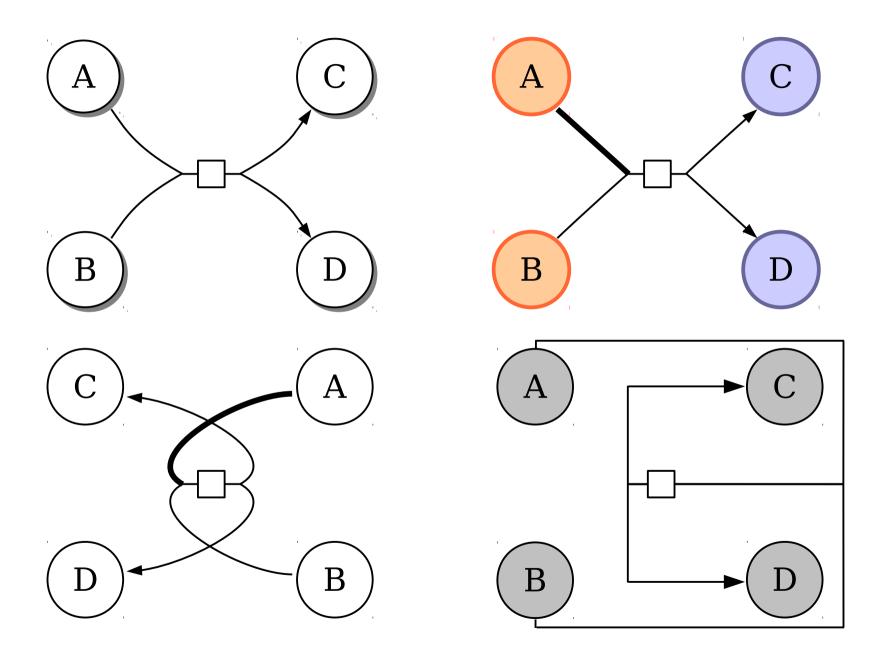




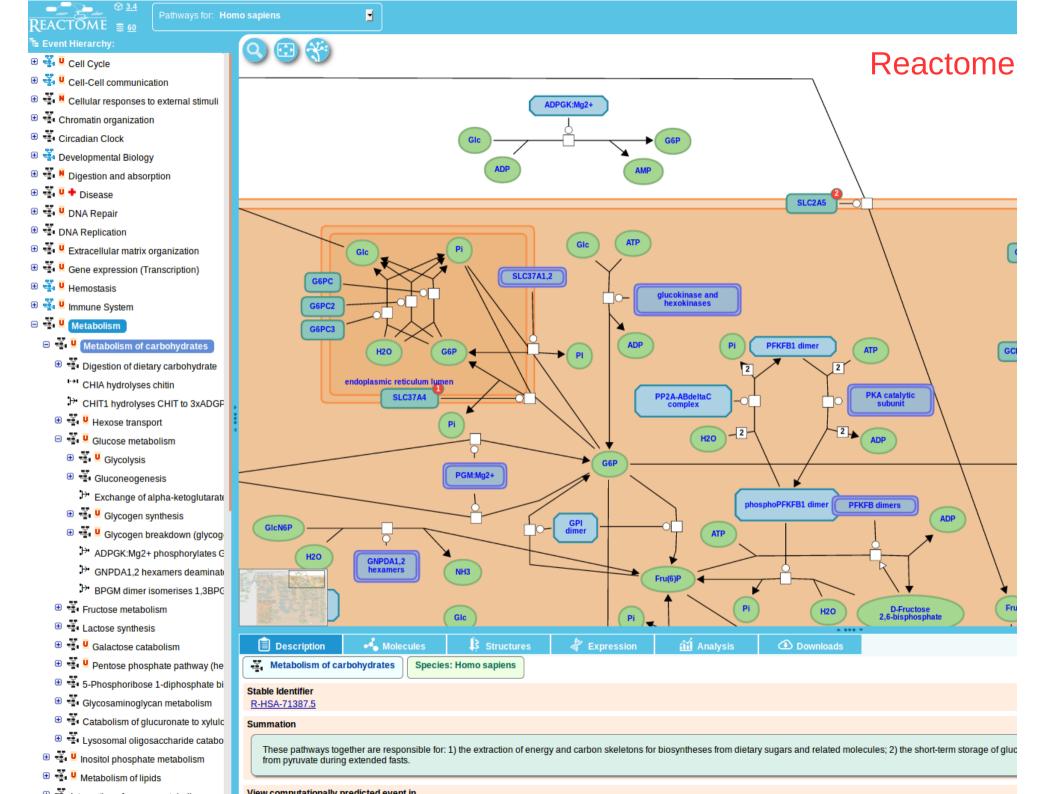


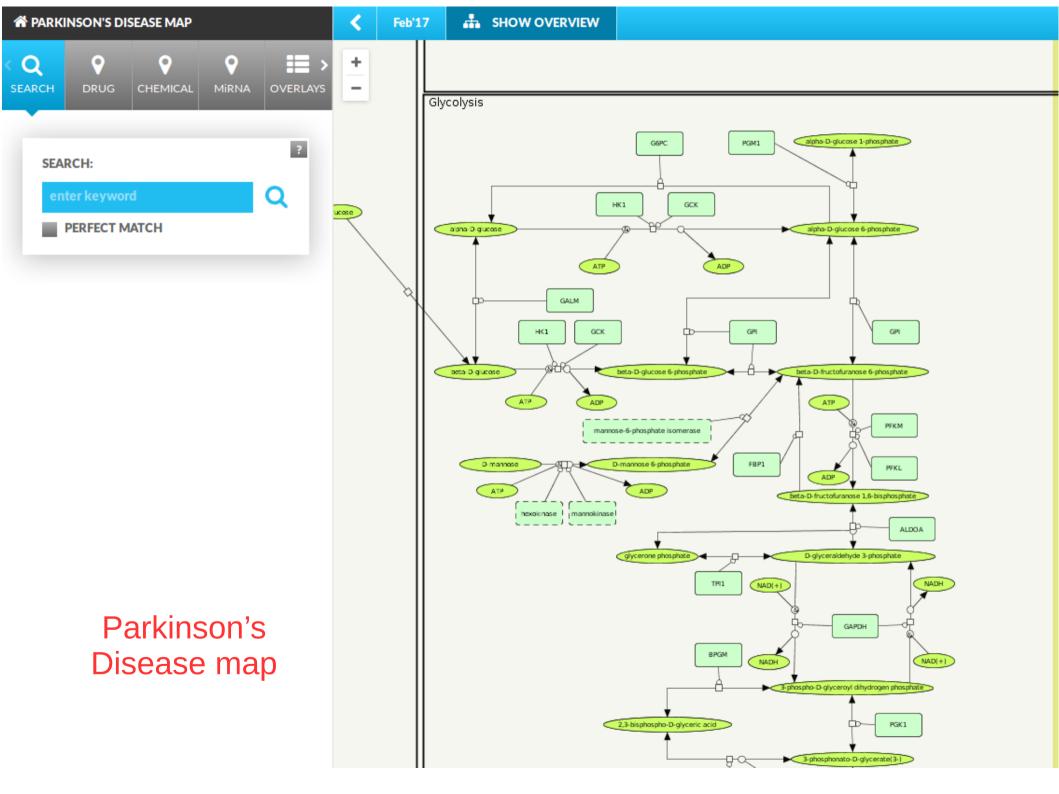


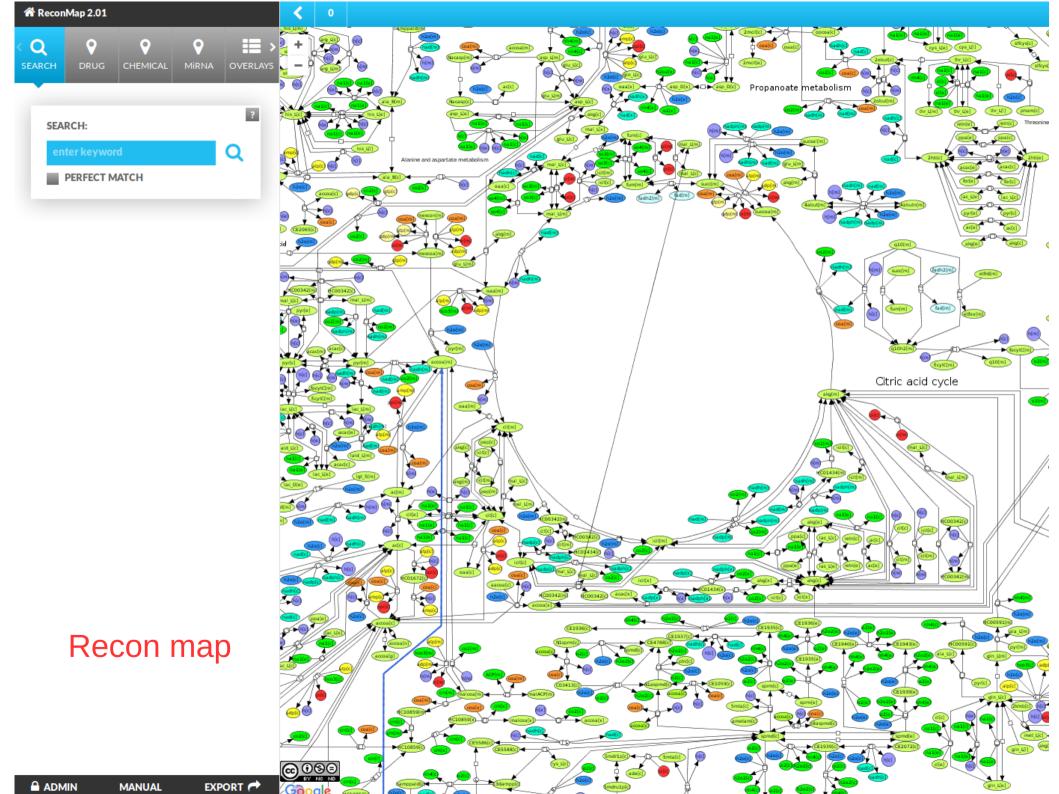
All those diagrams are identical

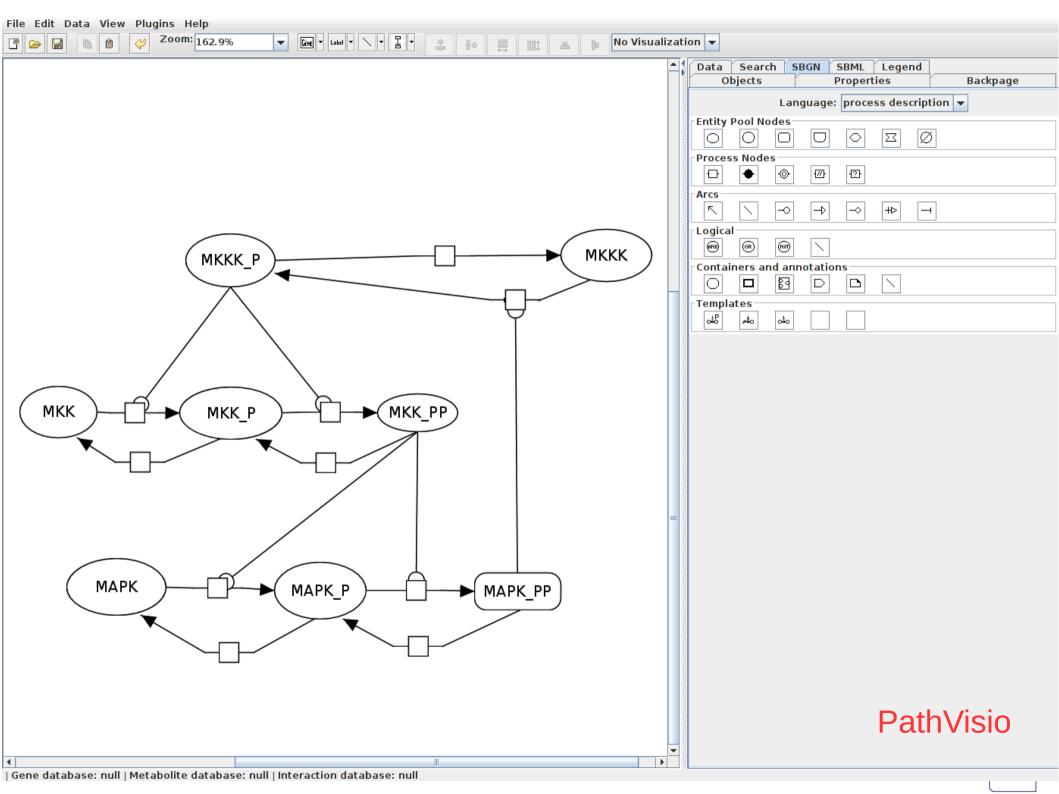


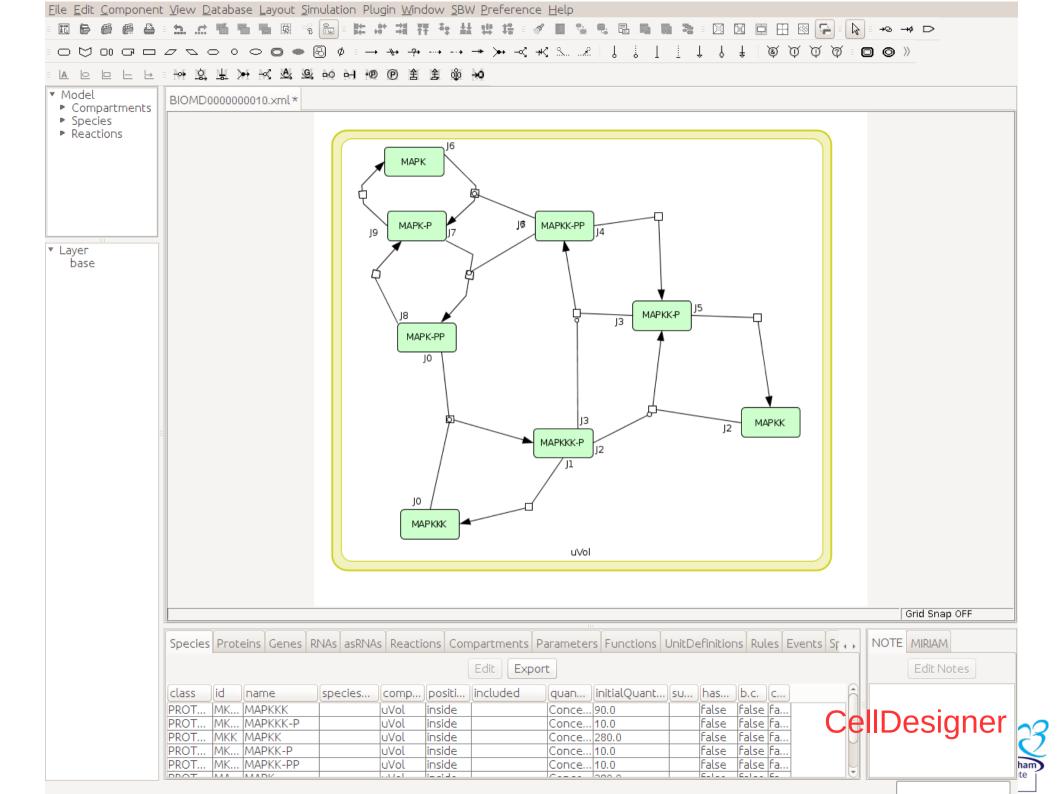


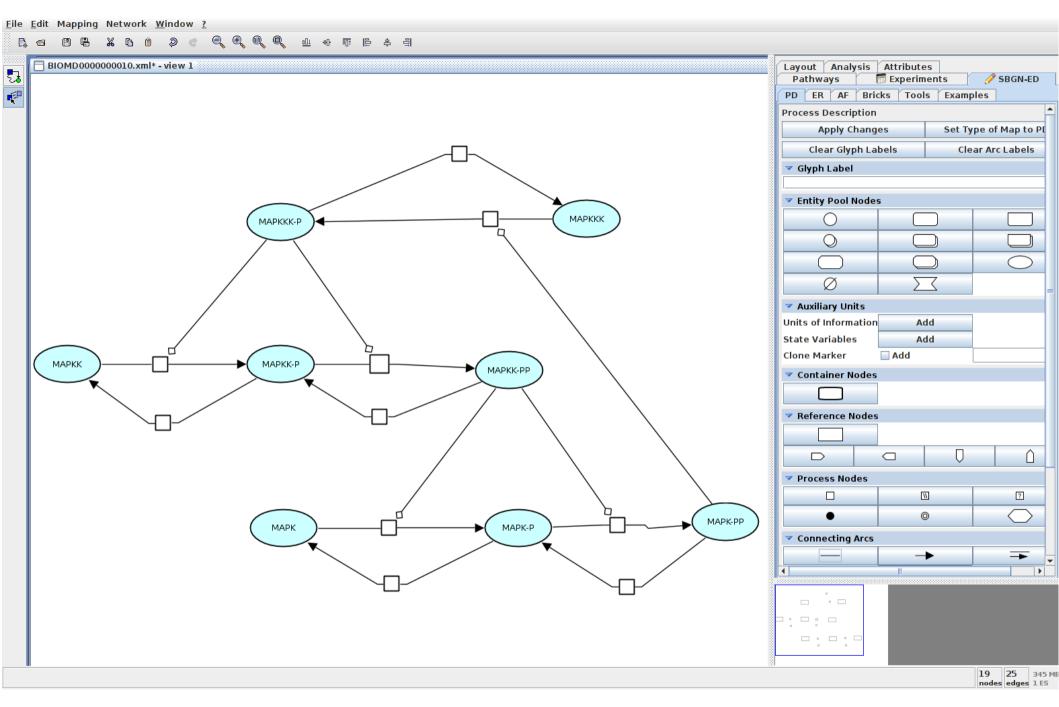










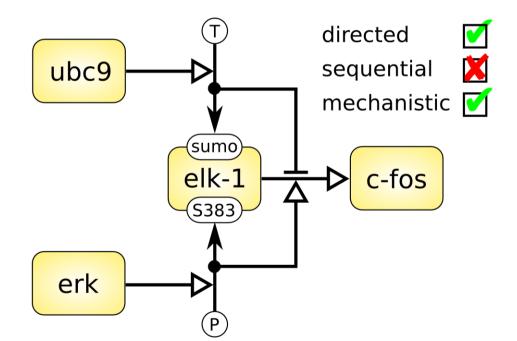


Vanted



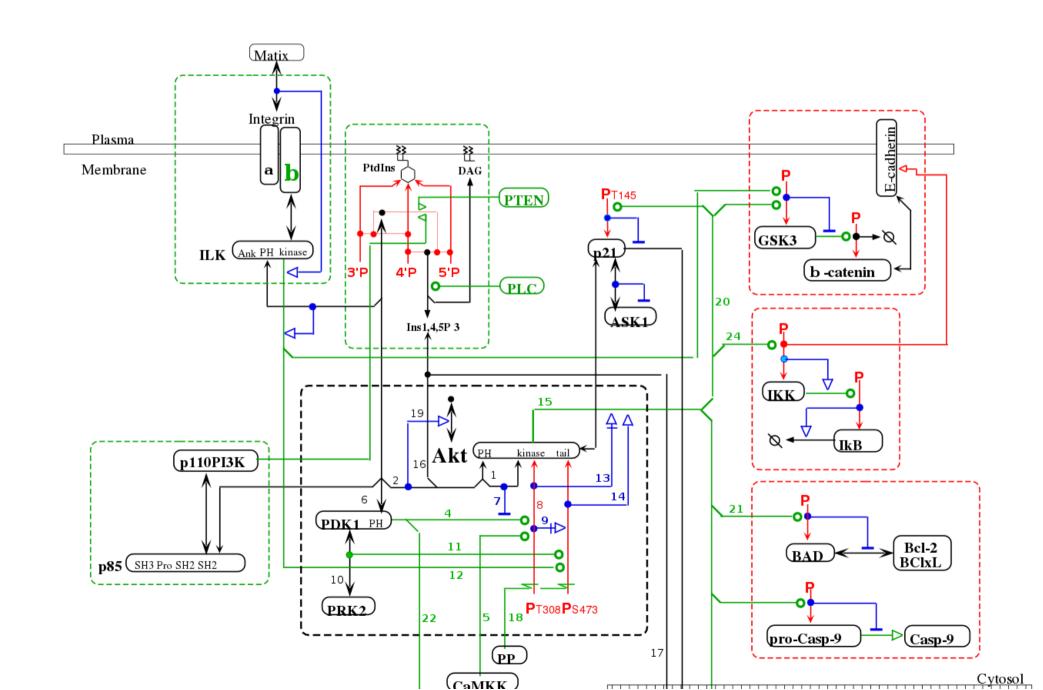
Entity Relationships

- Rule-based modelling
- Molecular Biology
- "Open world"
- Independent rules: no explosion

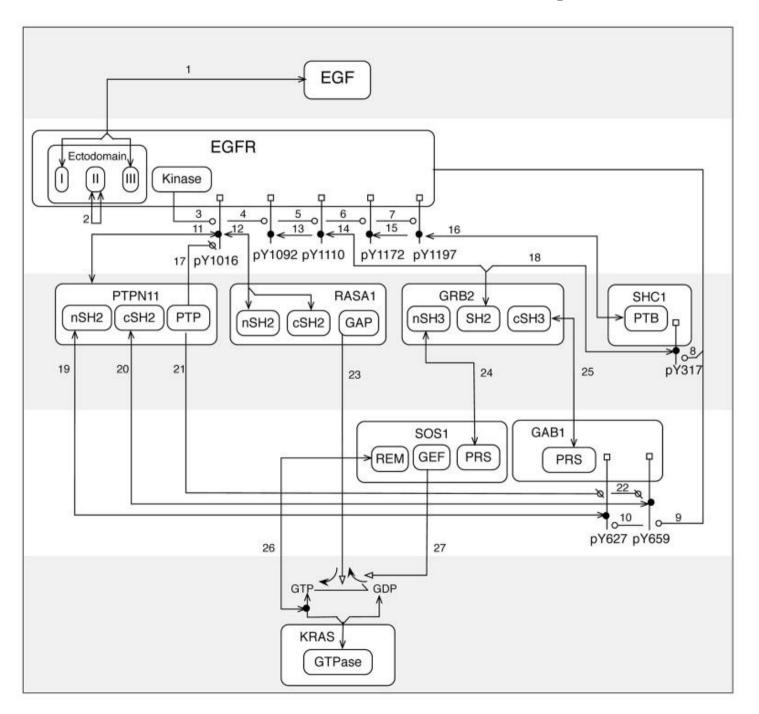




Molecular Interaction Maps



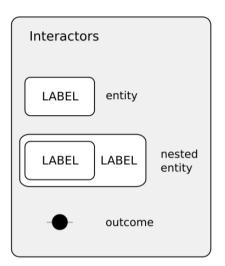
Extended Contact Maps

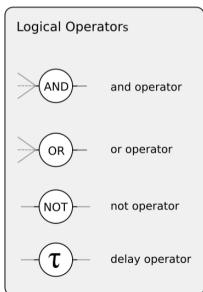


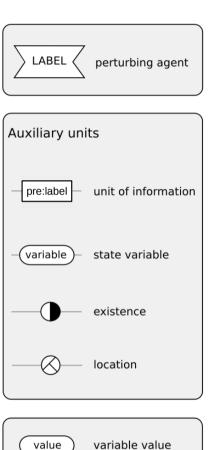


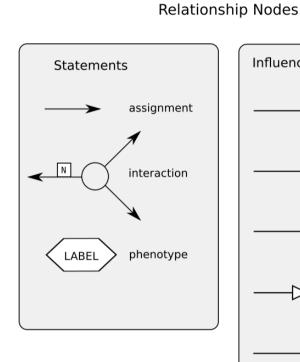
SBGN Entity Relationships L1 reference card

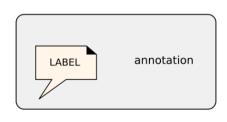
Entity Nodes



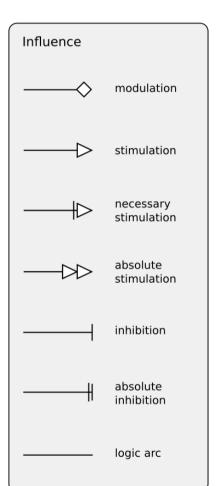








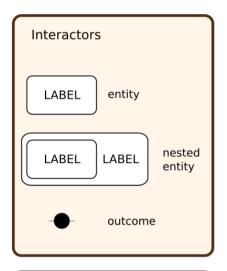
Reference Nodes

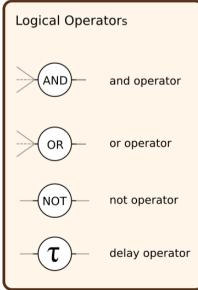


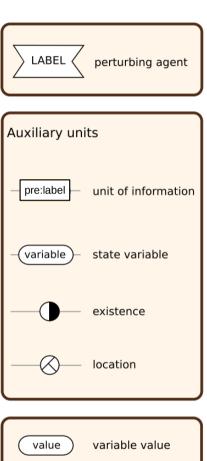


SBGN Entity Relationships L1 reference card

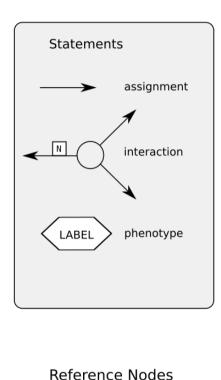
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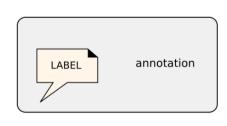


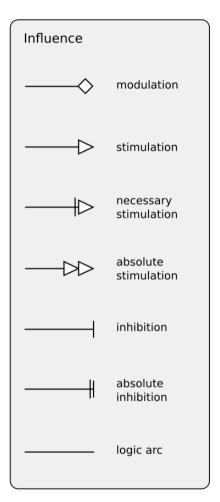




Relationship Nodes





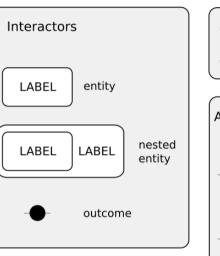


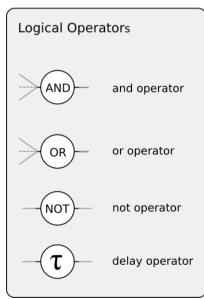
continuants, things that exists (or not)

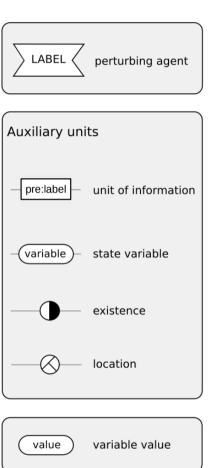


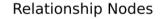
SBGN Entity Relationships L1 reference card

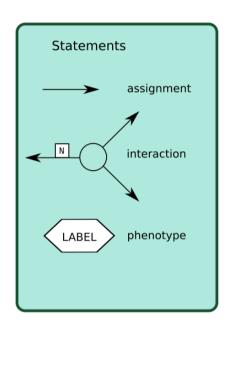
Entity Nodes

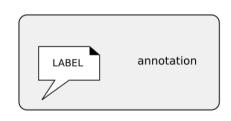




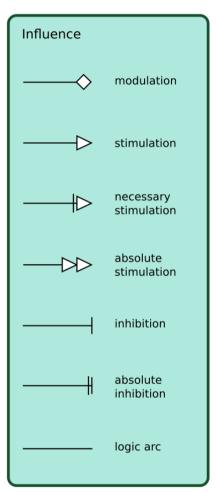








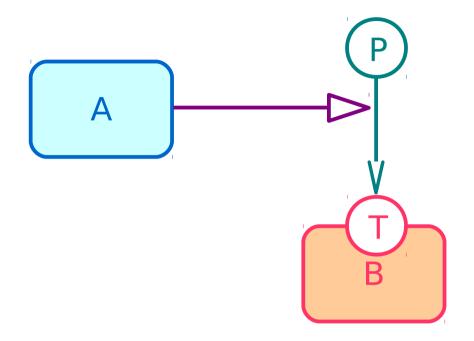
Reference Nodes



occurrents, events that may happen (or not)

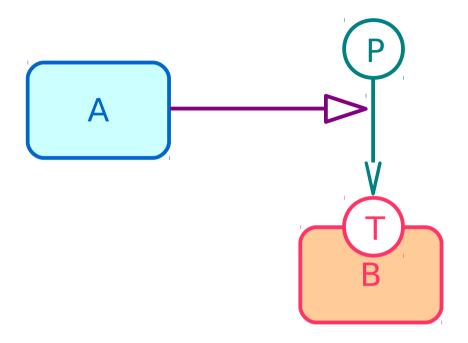


Entity Relationships can be viewed as rules





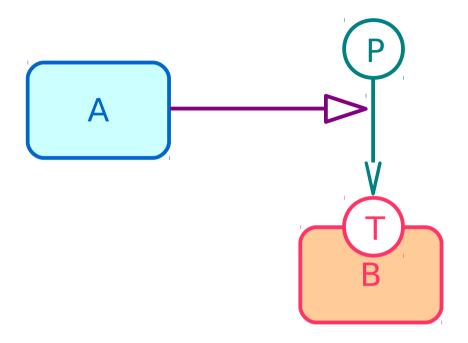
Entity Relationships can be viewed as rules



If A exists, the assignment of the value P to the state variable T of B is increased



Entity Relationships can be viewed as rules

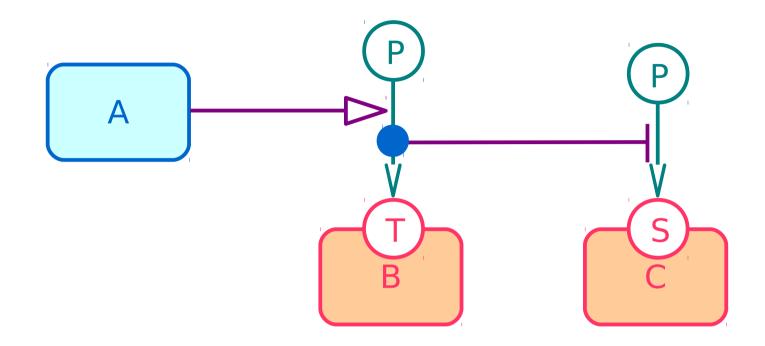


If A exists, the assignment of the value P to the state variable T of B is increased

(A stimulates the phosphorylation of B on the threonine)



Entity Relationships can be viewed as rules

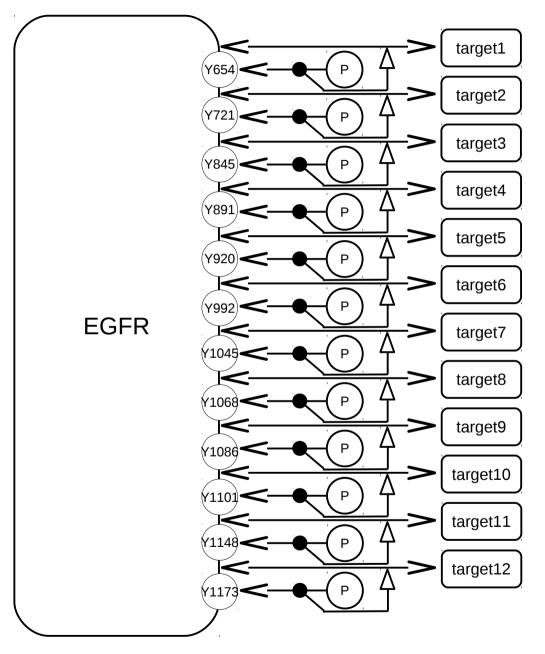


If A exists, the assignment of the value P to the state variable T of B is increased

If P is assigned to the state variable T of B, the assignment of the value P to the state variable S of B is decreased



Multistate and combinatorial explosion

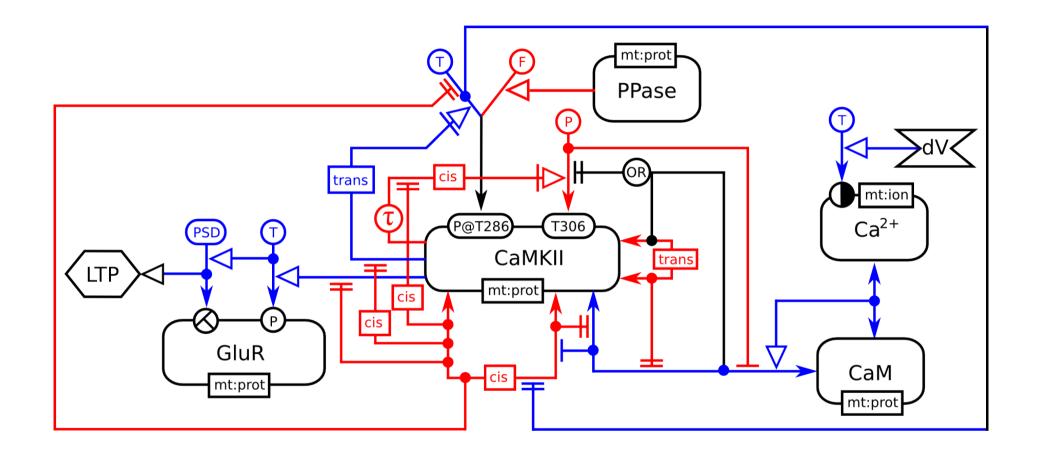


Process Descriptions: "once a state variable value, always a state variable value"

2¹² = 4096 states (i.e. EPN glyphs) for EGFR and 4096 complexes between EGFR and targets



Regulation of synaptic plasticity by calcium





Systems Biology Graphical Notation: Entity Relationship language Level 1

Version 2.0

Date: August 8, 2015

Anatoly Sorokin Nicolas Le Novère Augustin Luna Tobias Czauderna Emek Demir Robin Haw Huaiyu Mi Stuart Moodie Falk Schreiber Alice Villéger

Institute of Cell Biophysics RAS, RU Babraham Institute, UK Memorial Sloan-Kettering Institute, USA Monash University, Australia Memorial Sloan-Kettering Institute, USA Ontario Institute for Cancer Research, Canada University of Southern California, USA Eight Pillars Ltd, UK Monash University, Australia & MLU Halle, Germany



Editors

Freelance IT Consultant, UK

To discuss any aspect of SBGN, please send your messages to the mailing list sbgn-discuss@caltech.edu. To get subscribed to the mailing list or to contact us directly, please write to sbgn-editors@lists.sourceforge.net.

Bug reports and specific comments about the specification should be entered in the issue tracker http://sourceforge.net/p/sbgn/sbgn-er-11/.



Systems Biology Graphical Notation: Process Description language Level 1

Version 1.3 14 February, 2010

Stuart Moodie University of Edinburgh, UK EMBL European Bioinformatics Institute, UK Nicolas Le Novère Emek Demir Sloan-Kettering Institute, USA University of Southern California, USA University of Manchester, UK Huaiyu Mi Alice Villéger

To discuss any aspect of SBGN, please send your messages to the mailing list sbgn-discuss@sbgn.org. To get subscribed to the mailing list or to contact us directly, please write to sbgn-editors@lists.sourceforge.net.
Bug reports and specific comments about the specification should be entered in the issue tracker http://p.sf.net/sbgn/pd_tracker.



Bug reports and specific comments about the specification should be entered in the issue tracker http://sf.net/p/sbgn/sbgn-af-11/.



To discuss any aspect of SBGN, please send your messages to the mailing list sbgn-discuss@caltech.edu. To get subscribed to the mailing list or to contact us directly, please write to sbgn-editors@lists.sourceforge.net.

Systems Biology Graphical Notation:

Activity Flow language Level 1

Version 1.2

Date: July 27, 2015

Editors:

Huaiyu Mi Falk Schreiber

Stuart Moodie

Emek Demir Robin Haw Augustin Luna

Tobias Czauderna

Nicolas Le Novère Anatoly Sorokin Alice Villéger

 $\label{lem:university} \textit{University of Southern California, USA} \\ \textit{Monash University, Australia & MLU Halle, Germany}$

Memorial Sloan-Kettering Institute, USA
Ontario Institute for Cancer Research, Canada
Memorial Sloan-Kettering Institute, USA

Babraham Institute, UK Institute of Cell Biophysics RAS, RU Freelance IT Consultant, UK

Eight Pillars Ltd, UK

Monash University, Australia



SBGN AF L1V1.0 04-SEP-2009

L1V1.0

SBGN ER SBGN ER SBGN ER

03-SEP-2009 06-OCT-2010 14-APR-2011

L1V1.1

SBGN AF L1V1.2 27-JUL-2015

> **SBGN ER L1V2**

08-AUG-2015

SBGN PD L1V1.0

SBGN PD L1V1.1

SBGN PD L1V1.2

SBGN PD L1V1.3

L1V1.2

03-OCT-2010 14-FEB-2011 15-AUG-2008 02-SEP-2009



Decision to Create SBGN OCT-2005 Tokyo

SBGN 3 SEP-2007 Long Beach

SBGN 5 SEP-2009 Stanford

COMBINE SEP-2011 Heidelberg

HARMONY MAY-2013 Farmington

HARMONY APR-2015 Wittenberg

SBGN 1 FEB-2006

Tokyo

JAN-2008 Okinawa

SBGN 3.5

SBGN 5.5 APR-2010 Wittenberg

MAY-2012 Maastricht

HARMONY

SEP-2013 Paris

COMBINE

COMBINE OCT-2015 Salt Lake City

SBGN 2 OCT-2006 SBGN 4

COMBINE COMBINE

AUG-2012

HARMONY APR-2014

HARMONY

Yokohama

OCT-2008 Rostock

OCT-2010 Edinburg

Toronto

Manchester

JUN-2016 Auckland

SBGN 2.5 SBGN 4.5

MAR-2007 APR-2009 Heidelberg Waiheke

HARMONY SBGN 9 APR-2011 MAY-2013 New-York Edinburgh

COMBINE AUG-2014 Los Angeles COMBINE SEP-20162

Newcastles

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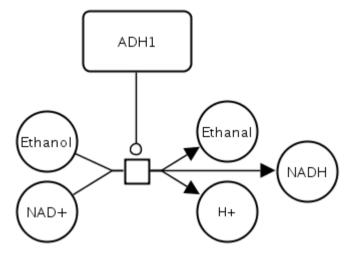
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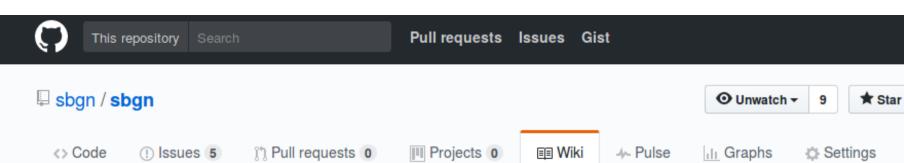
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```

SBGN-ML







LibSBGN

Matthias König edited this page on 30 Sep 2016 · 13 revisions

LibSBGN library

LibSBGN is the library for writing and reading SBGN-ML, a XML-based file format dedicated to the description of SBGN maps.

Source code: https://github.com/sbgn/libsbgn

Latest release: https://github.com/sbgn/libsbgn/releases

Features

LibSBGN is a library that deals with SBGN maps. It currently supports:

- Reading / writing the SBGN-ML file format (XML-based format for description of SBGN maps)
- Validation of semantical and syntactical correctness
- Conversion to other formats such as SBML and BioPAX
- Support for Java and C++

Documentation



Ÿ Fork

New Page

Edit

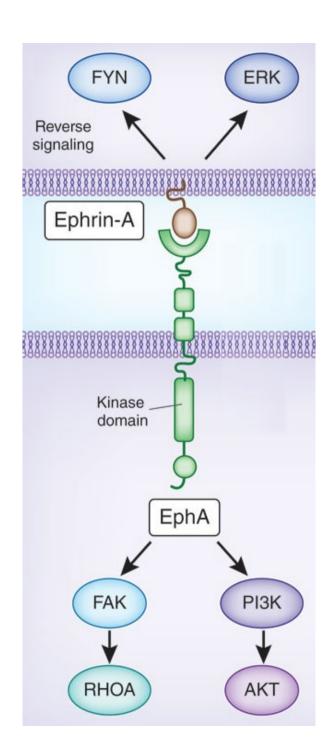
Are we done? Everything is solved, right?

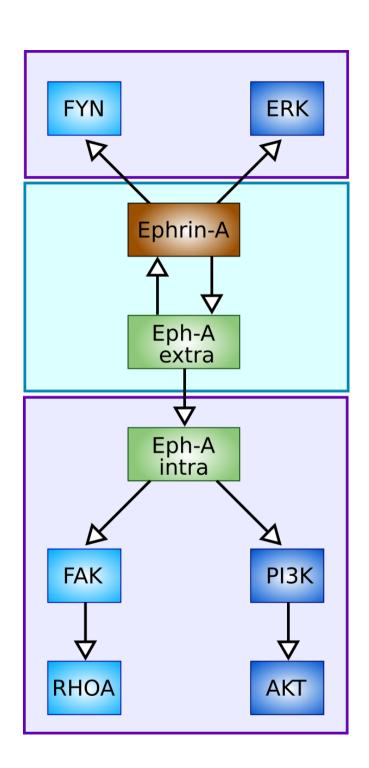


FYN ERK Reverse signaling Ephrin-A Kinase domain EphA FAK PI3K RHOA **AKT**

Multi-compartment entities

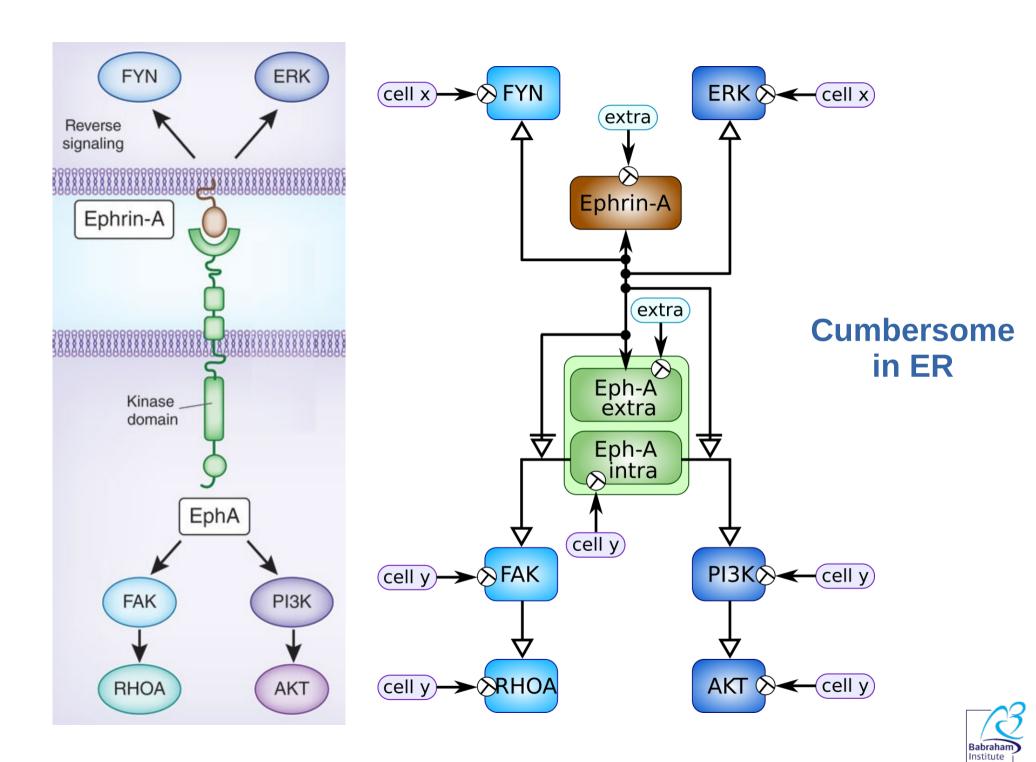


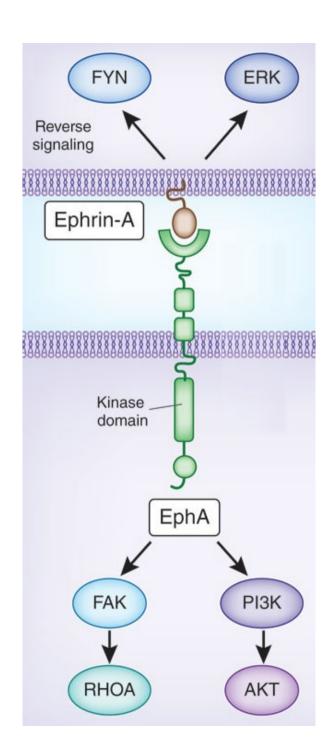


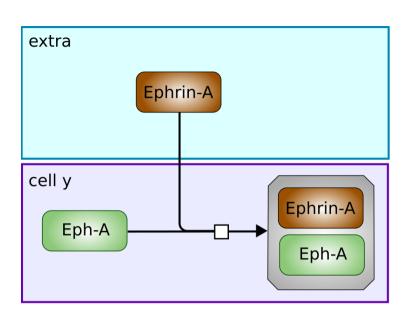


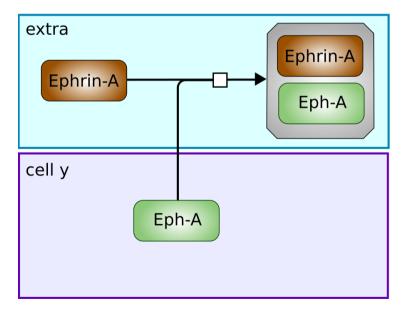
Easy in AF







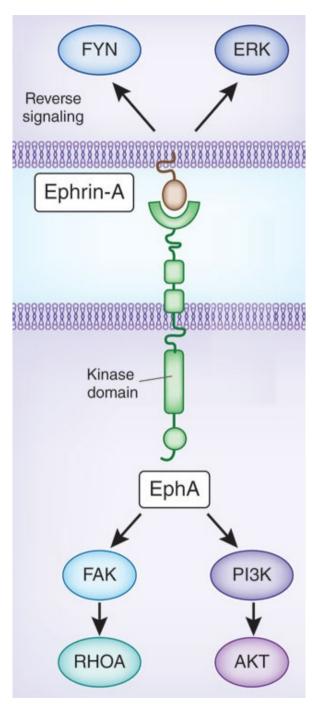


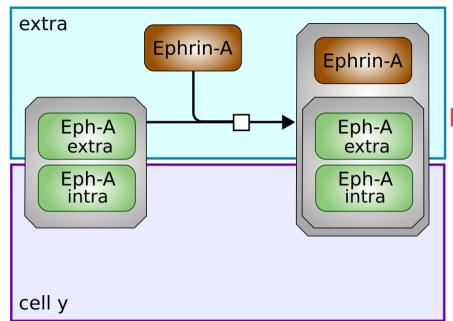


impossible In PD

molecular species (entity pool nodes) belong to a single compartment

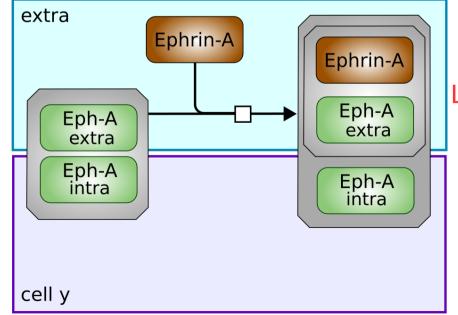






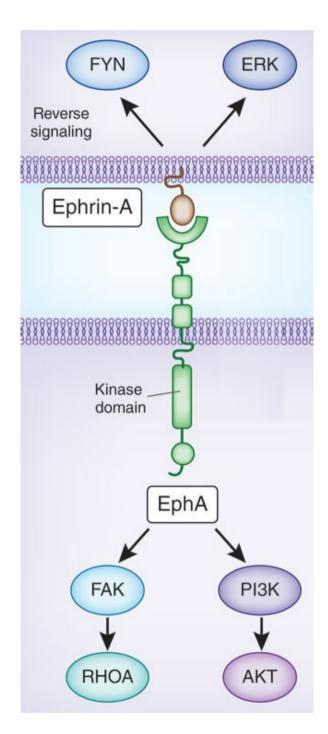
Loose the fact that Ephrin-A binds to Extracell part

Multi-compartment complexes do not solve the problem, since they represent Non-covalent assemblies

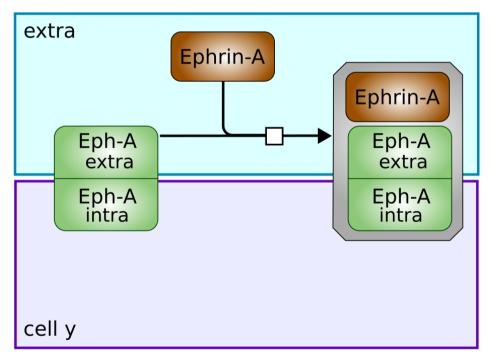


Loose the fact that Eph-A is a Multi-domain molecule



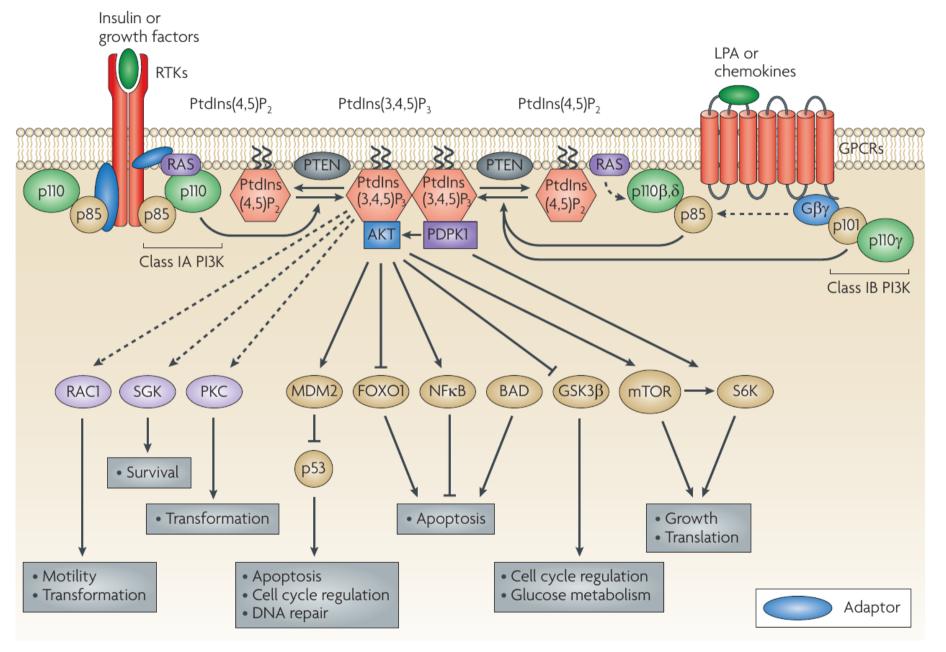


- New glyph: "Domain"; generalisation of the current "Nucleic acid feature".
- Domains can be associated in multi-domain entities pool nodes, the location being carried by the domains
- Allow complexes to be compartment-less
- Move content of complex from "decorations" to bona fide entity pool nodes



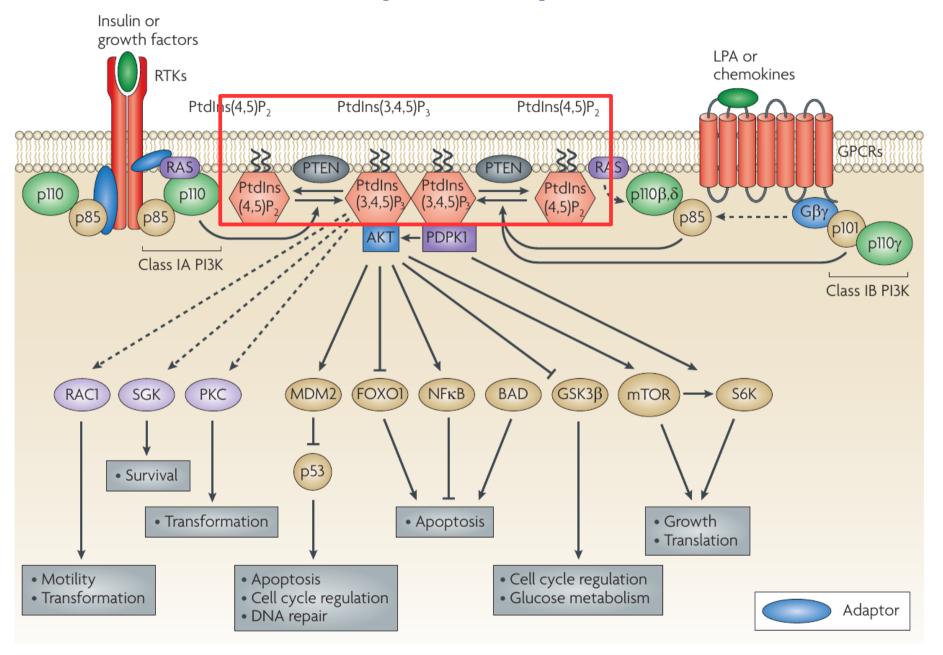


Hybrid maps

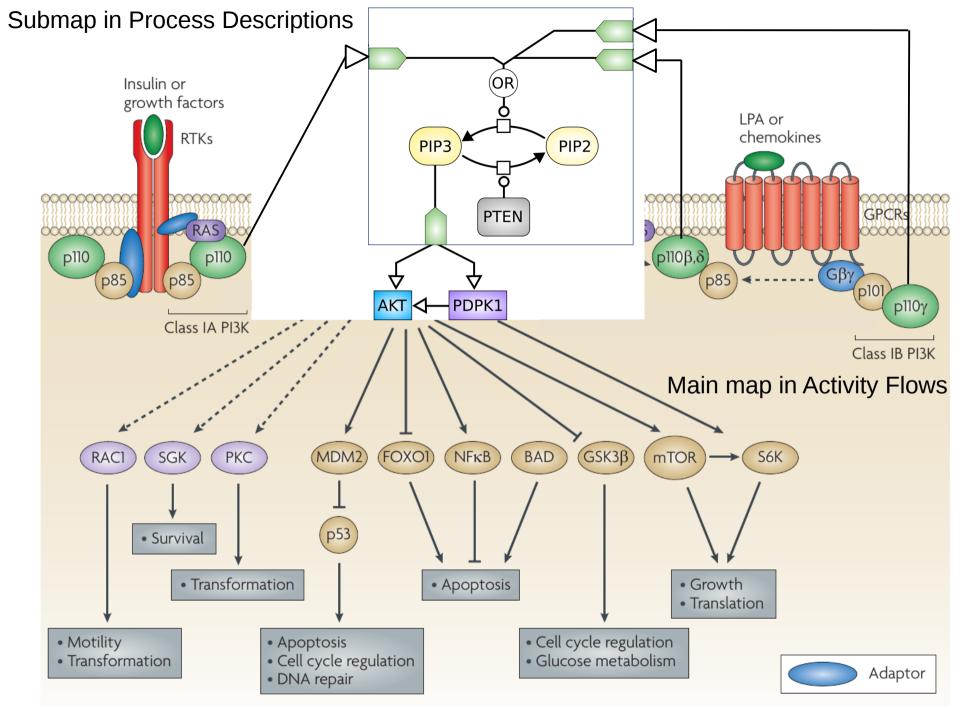




Hybrid maps



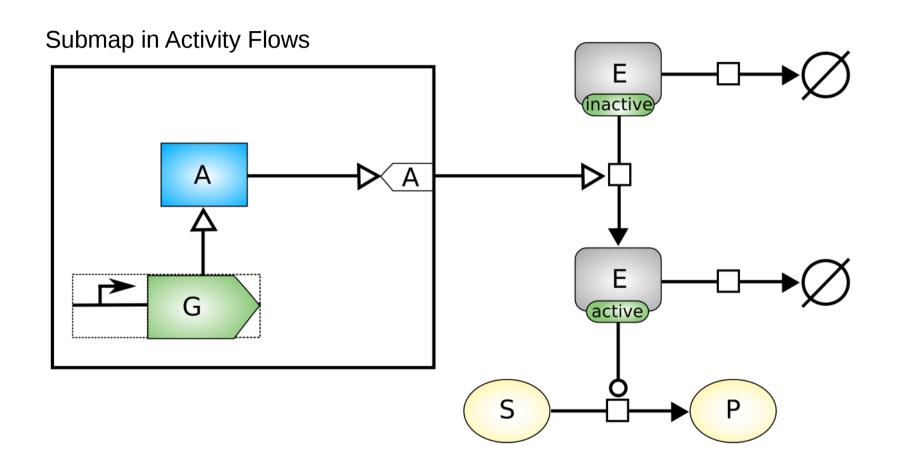




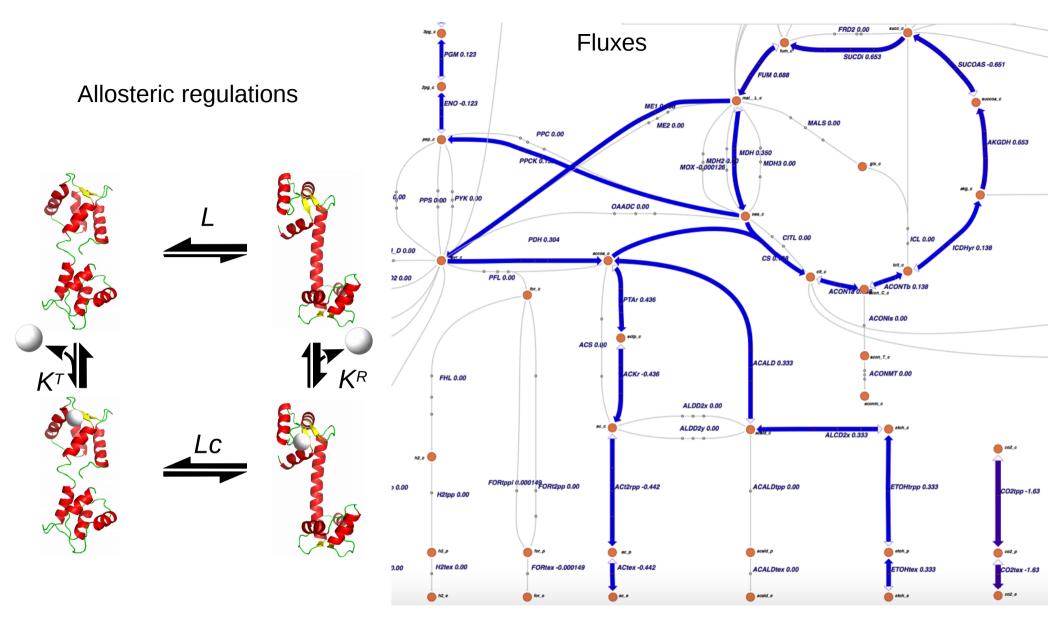




Main map in Process Descriptions

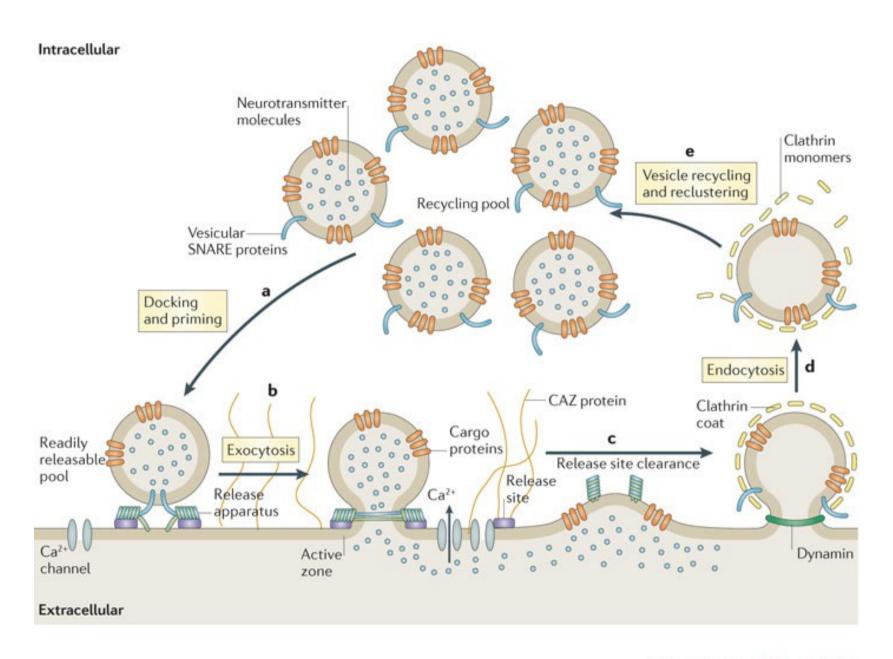




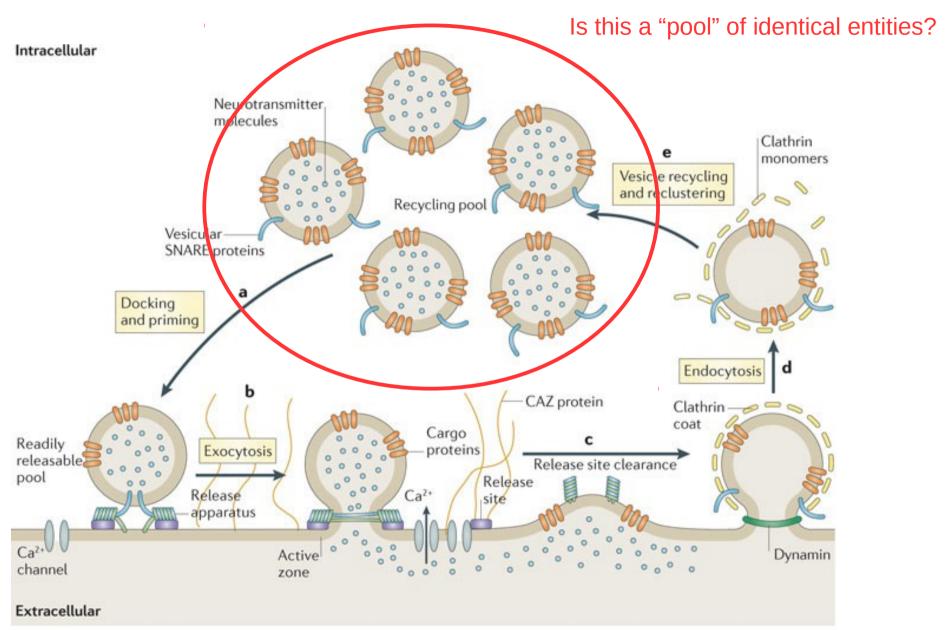


Intensities and amounts

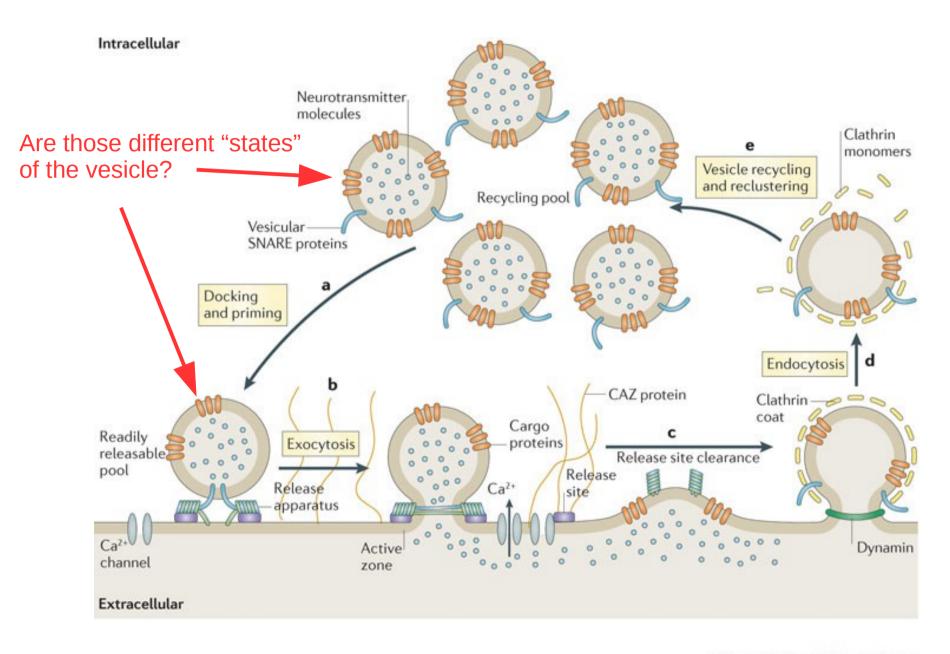




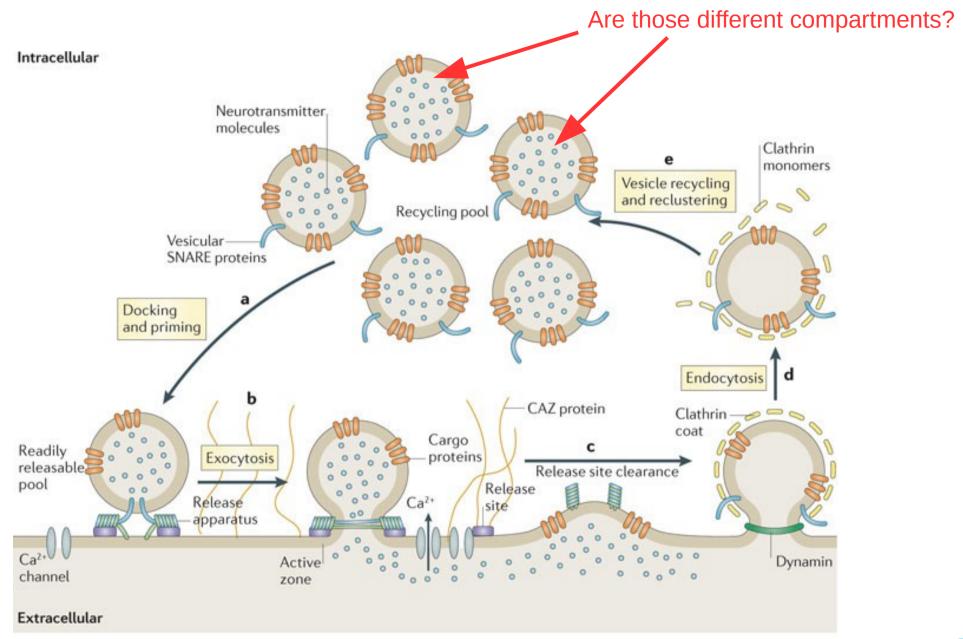




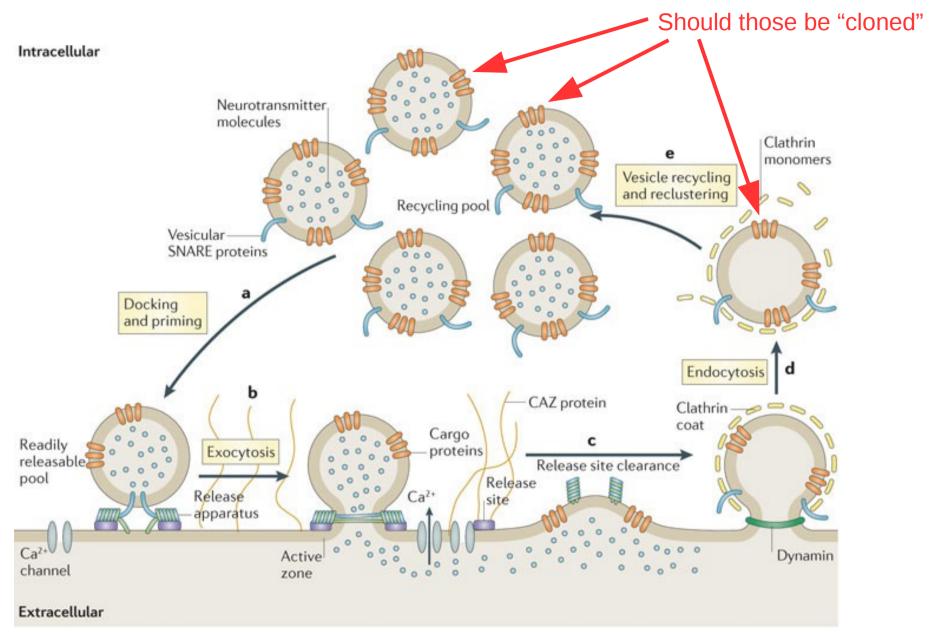














Temporal order and Dynamics?

Multi-scale?

Topologies?

Data integration?





Systems Biology Graphical Notation

Learn To Use SBGN

Symbols

Example Diagrams

Publications

Software Support

Specifications

Events

FAQ

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Contact

Competition

Contribute

SBGN Development

Join the conversation

http://sbgn.org

Welcome to the global portal for documentation, news, and other information about the **Systems Biology Graphical Notation** (SBGN) project, an effort to standardize the graphical notation used in maps of biological processes.

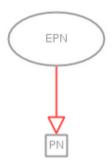
Quick start

Learn how to use SBGN

Get involved

Symbol Highlight

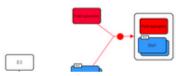
Stimulation



Pathway Highlight

This pathway is an SBGN diagram from the following paper published in the July, 2013 issue of the Cell magazine (PubMed ID: 23791384).

Park S., Kukushkin Y., Gupta R., Chen T., Konagai A., Hipp M., Hayer-Hartl M., and Hartl F. (2013) PolyQ Proteins Interfere with Nuclear Degradation of Cytosolic Proteins by Sequestering the Sis1p Chaperone. Cell 154, 134-145.



sbgn-discuss@googlegroups.com

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