BioPAX Pathway Services

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POJO beans – bound to OWL using reflection.
- Once in the Java model very time and memory efficient.
- A lot more friendly for other OO tools.
- Fail-fast validation.
- Implements equality methods for identity semantics.
- JPA and Lucene provide state-of-the-art persistence and querying.

- Hand-coded, an OO interpretation of the OWL description.
- Current I/O layer uses Jena thus heavyweight.
Paxtools based
Each rule as separate classes
Interweaved into the model, using AOP
Customizable Error/Reporting
Easy to define new validation rules
Easy to externally change validation policy
Policy files can be shared.
Extensible to manipulation rules
Pathway Commons is a convenient point of access to biological pathway information collected from public pathway databases, which you can browse or search. Pathways include biochemical reactions, complex assembly, transport and catalysis events, and physical interactions involving proteins, DNA, RNA, small molecules and complexes. more...

Pathway Commons currently contains the following data sources:

- **Cancer Cell Map, Release: 1.0**
  [19-May-06]
  [Browse]

- **HumanCyc, Release: 10.5**
  [18-Sep-06]
  [Browse]

- **NCI / Nature Pathway Interaction Database**
  [01-Jan-07]
  [Browse]

- **Reactome, Release: 19**
  [16-Nov-06]
  [Browse]

### Pathway Commons Quick Stats:

- Number of Pathways: 921
- Number of Interactions: 9,924
- Number of Physical Entities: 15,515
- Number of Organisms: 10

Integration of additional data sources is planned in the near future. For a comprehensive directory of interaction and pathway databases, please refer to Pathguide.

**Biologists**: Browse and search pathways across multiple valuable public pathway databases.

**Computational biologists**: Download an integrated set of pathways in BioPAX format for global analysis.

**Software developers**: Build software on top of Pathway Commons using our soon-to-be released web service API. Download and install the cPath software to create a local mirror.

Search Pathway Commons:

Enter a gene name, gene identifier or pathway name in the text box above. For example: p53, P38398 or mTOR.

To restrict your search to specific data sources or specific organisms, update your global filter settings.
**Pathway Commons**

Pathway Commons is a work in progress. We welcome your feedback. Email us at: pc-info@pathwaycommons.org.

**Searched for: p53**

Pathway Commons completed your search for "p53" and found 22 relevant records:

**Narrow Results by Type:**
- All Types (45)
- Pathway (22)
- Protein (23)

**Narrow Results by Data Source:**
- All Data Sources (22)
- Cancer Cell Map (2)
- NCI / Nature Pathway Interaction Database (3)
- Reactome (17)

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Showing Results 1 - 10 of 22 | Next 10
```

**Pathway: Transcriptional activation of p53 responsive genes**

**Summary:**

p53 causes G1 arrest by inducing the expression of a cell cycle inhibitor, p21 (El-Deiry et al, 1993; Harper et al, 1993; Xiong et al, 1993). P21 binds and inactivates Cyclin-Cdk complexes that mediate G1/S progression, resulting in lack of phosphorylation of Rb, E2F sequestration and cell cycle arrest at the G1/S transition. Mice with a homozygous deletion of p21 gene are deficient in their ability to undergo a G1/S arrest in response to DNA damage (Deng et al, 1995).

**Data Sources:**
- Reactome

- **... p53 causes G1 arrest by inducing the expression of a cell cycle inhibitor, p21 (El-Deiry et al, 1993; Harper et al, 1993; Xiong et al, 1993).**

**Pathway: Stabilization of p53**

... ATM also regulates the phosphorylation of p53 at other sites, especially Ser-20, by activating other serine/threonine kinases in response to IR (Chehab et al, 2000).

**Pathway: p53-Dependent G1 DNA Damage Response**

- Most of the damage-induced modifications of p53 are dependent on the ATM kinase. ... The first link between ATM and p53 was predicted based on the earlier studies that showed that AT cells exhibit a reduced and delayed induction of p53 following exposure to IR (Kastan et al, 1992 and Khanna and Lavin, 1993). ... Under normal conditions, p53 is a short-lived protein.

**Pathway: p53-Dependent G1/S DNA damage checkpoint**

- The arrest at G1/S checkpoint is mediated by the action of a widely known tumor suppressor protein, p53. ... Loss of p53 functions, as a result of mutations in cancer prevent the G1/S checkpoint (Kurowski et al, 1992). ... p53 is rapidly induced in response to damaged DNA.

**Pathway: p53-Independent G1/S DNA damage checkpoint**

- The G1 arrest induced by DNA damage has been ascribed to the transcription factor and tumor suppressor protein p53.

**Pathway: G1/S DNA Damage Checkpoints**

- In the G1 phase there are two types of DNA damage responses, the p53-dependent and the p53-independent pathways. ... The p53-dependent responses inhibit CDKs through the up-regulation of genes encoding CKIs mediated by the p53 protein, whereas the p53-independent mechanisms inhibit CDKs through the inhibitory T14Y15 phosphorylation of Cdk2.

**Pathway: Cell Cycle Checkpoints**

http://pathwaycommons.org
Integration is difficult
- Different notions and representations.
- Need for concurrency.
- Different levels of detail.
- Incomplete/Ambiguous knowledge
Horizontal Integration
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BioPAX community!
Editors to the last level: Gary Bader, Ken Fukuda
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