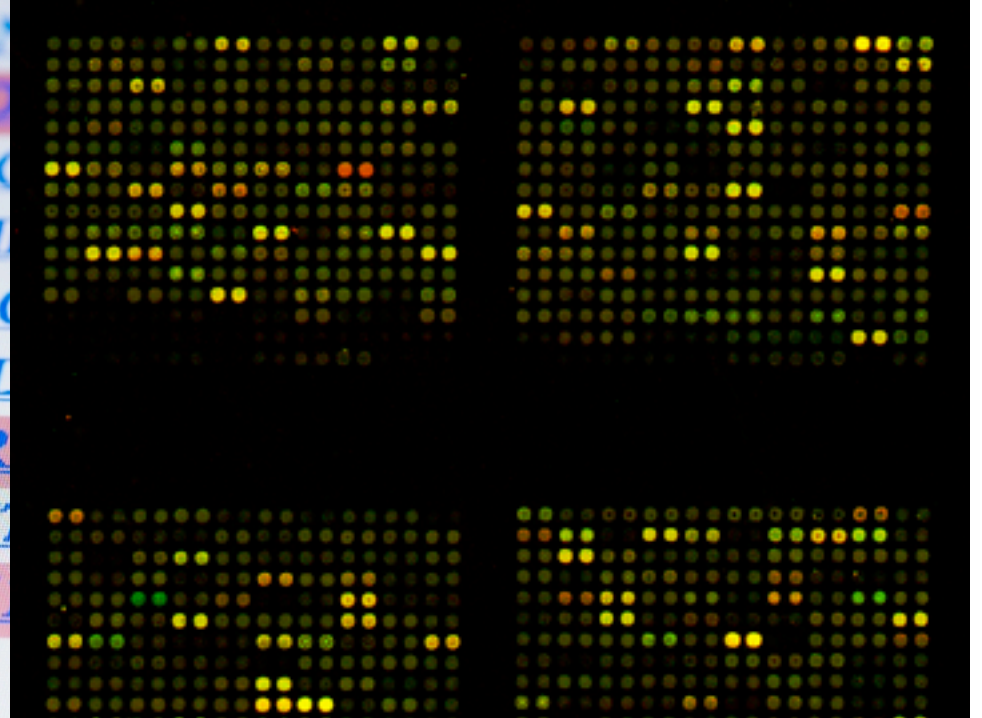
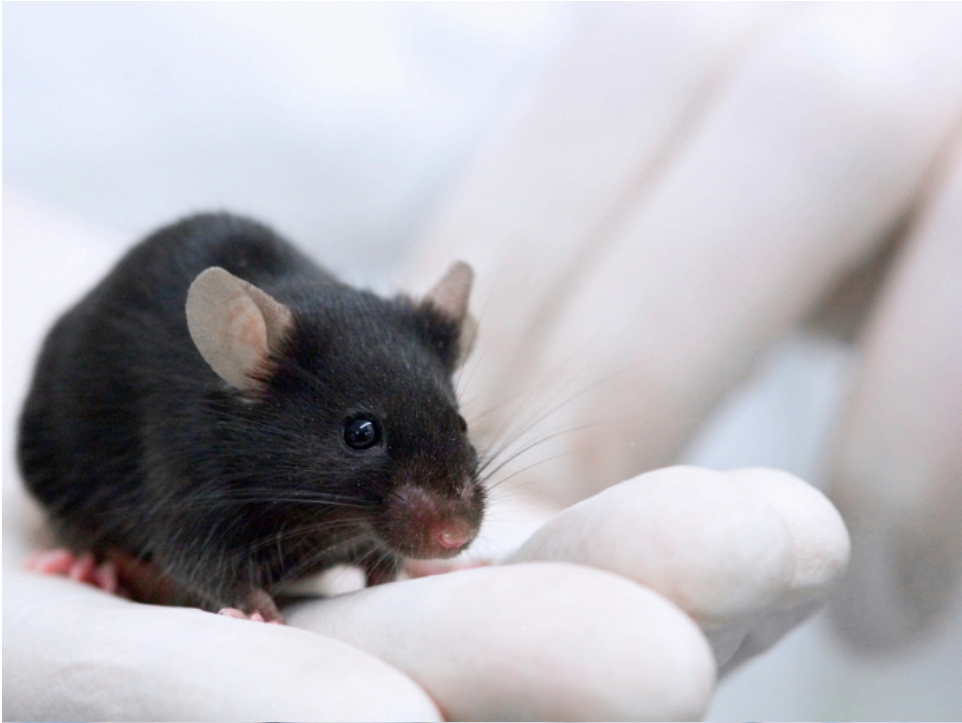


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oai5  
20 april 2007

john wilbanks  
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# 1. copyrights and open archives



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*describing copyright in RDF*

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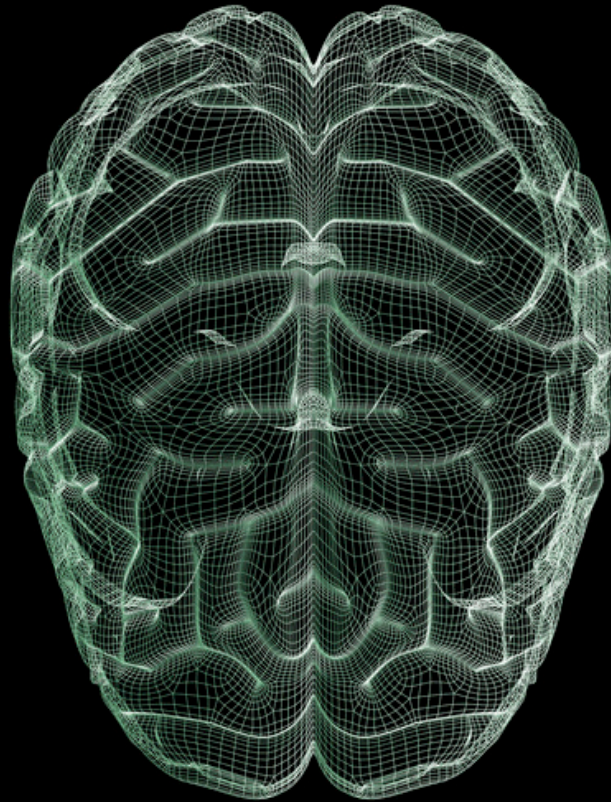
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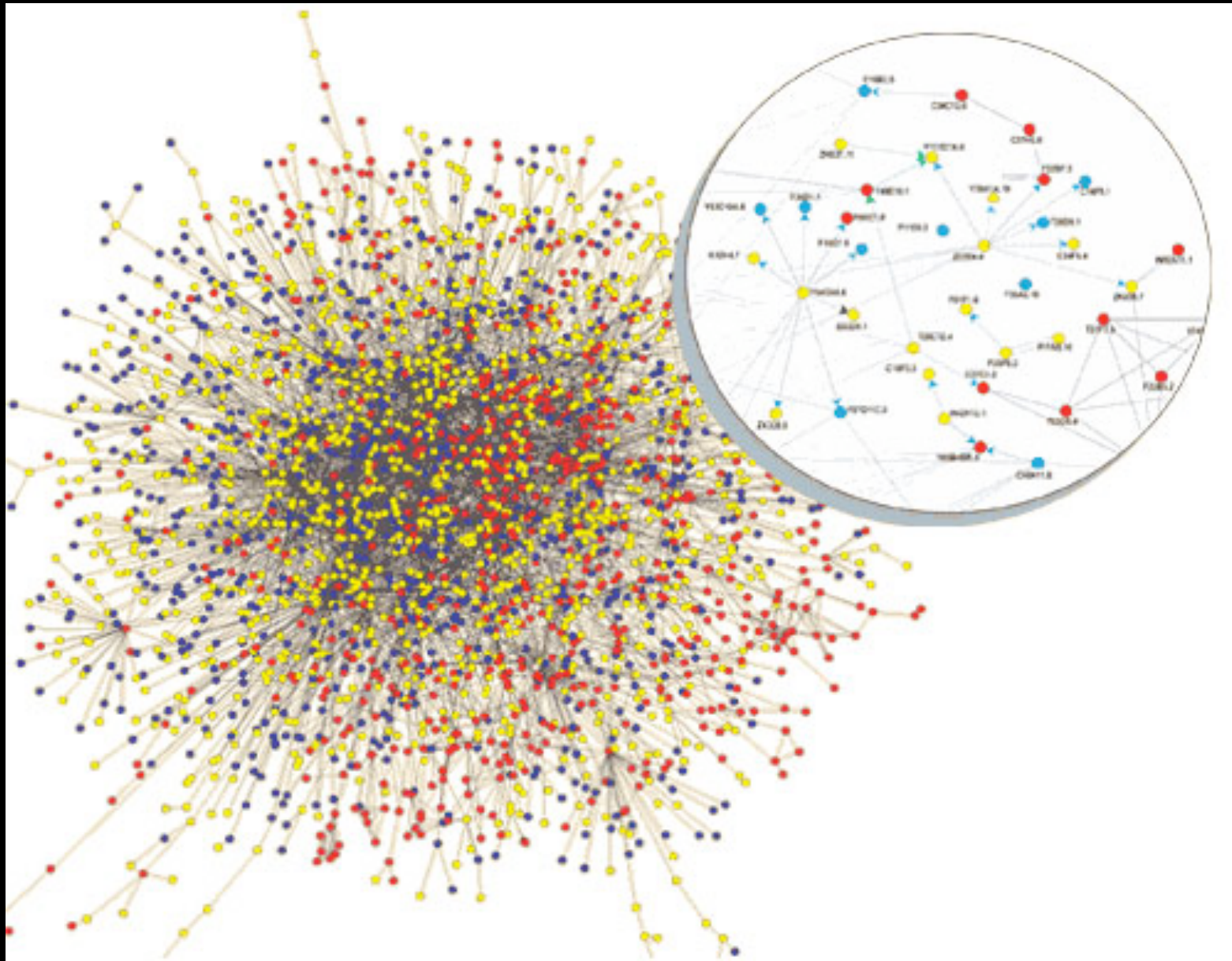
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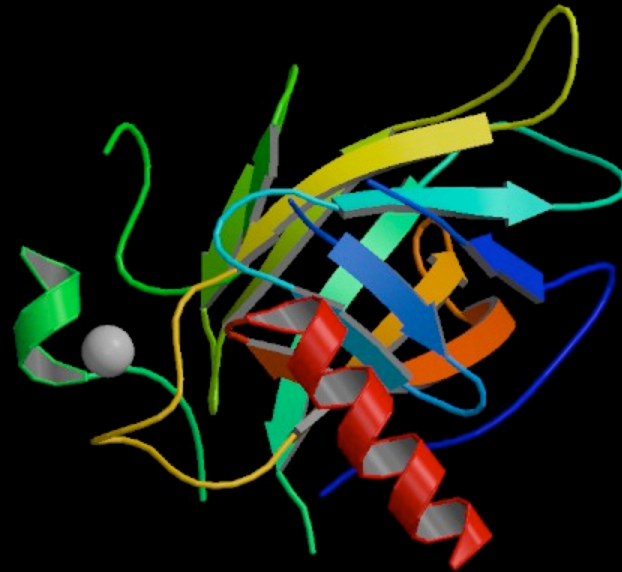






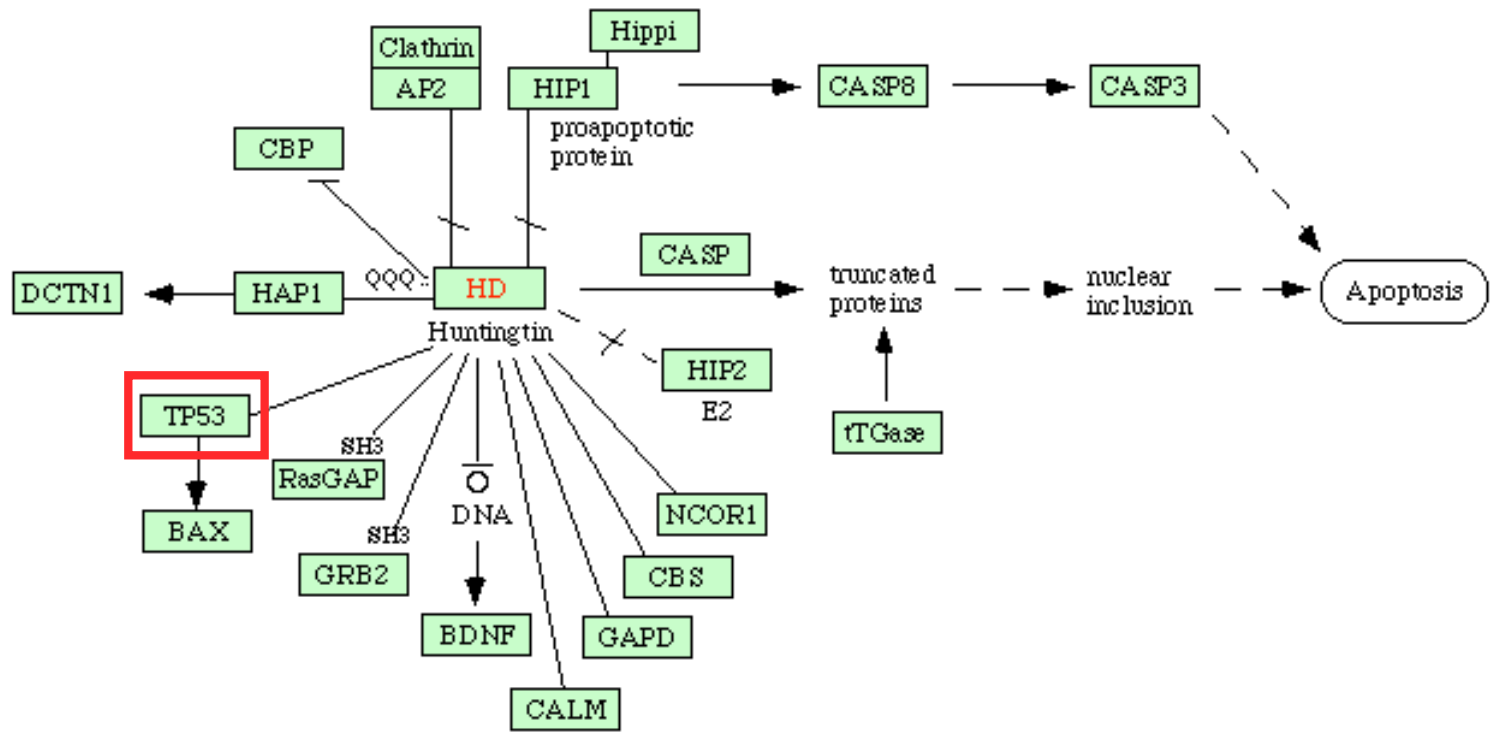
“protein-protein interaction networks”

“keystone species” of the genome  
knowledge ecology



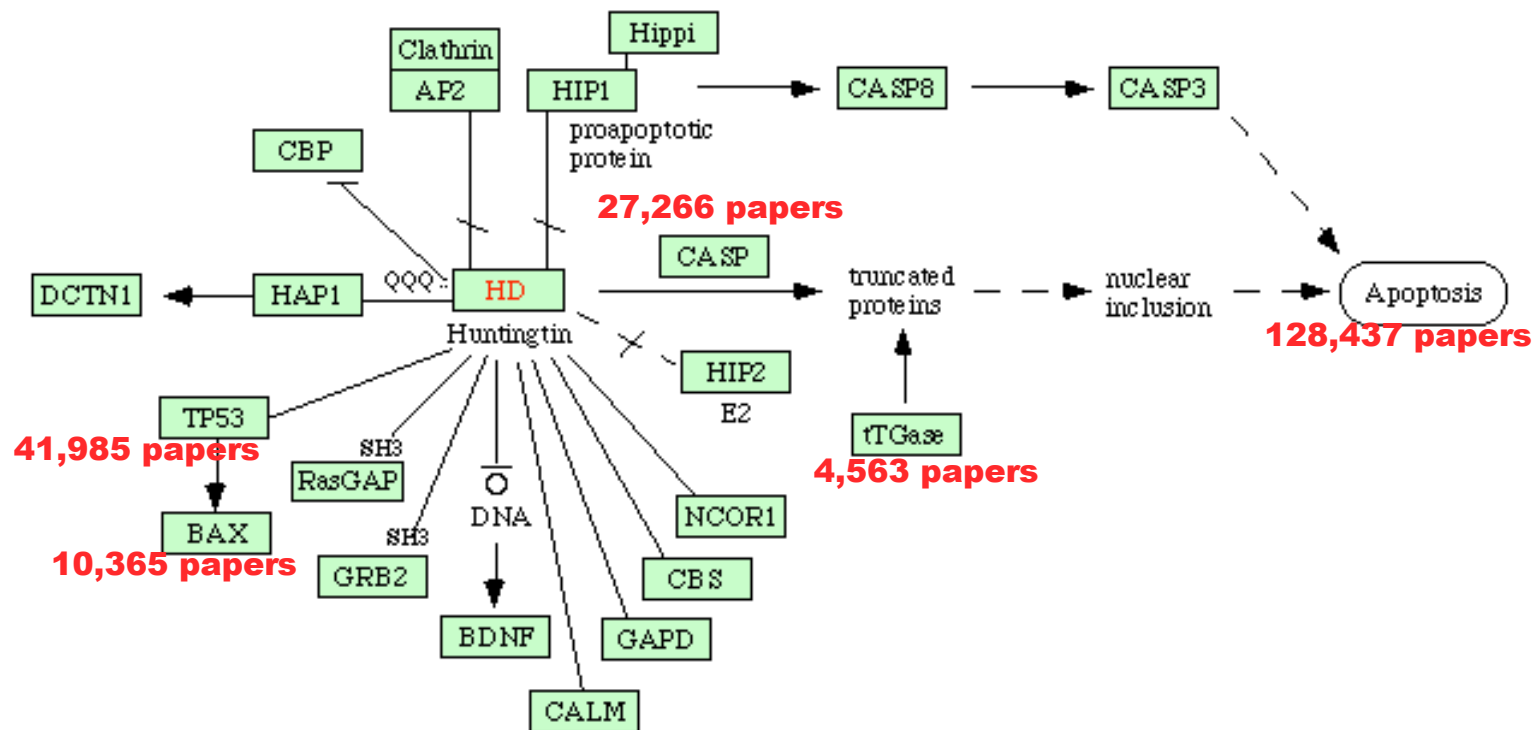
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# Huntington's Disease



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Huntington's Disease



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Department of Pharmacology, Loyola University Medical Center, Maywood, Illinois 60153, USA.

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we need to treat the literature itself as  
data, because we need computers to  
process it for us



**Identification and characterization of regulator of G protein signaling 4 (RGS4) as a novel inhibitor of tubulogenesis: RGS4 inhibits mitogen-activated protein kinases and vascular endothelial growth factor signaling.**

**Albig AR, Schiemann WP.**

Program in Cell Biology, Department of Pediatrics, National Jewish Medical and Research Center, Denver, CO 80206, USA.

Tubulogenesis by epithelial cells regulates kidney, lung, and mammary development, whereas that by endothelial cells regulates vascular development. Although functionally dissimilar, the processes necessary for tubulation by epithelial and endothelial cells are very similar. We performed microarray analysis to further our understanding of tubulogenesis and observed a robust induction of regulator of G protein signaling 4 (RGS4) mRNA expression solely in tubulating cells, thereby implicating RGS4 as a potential regulator of tubulogenesis. Accordingly, RGS4 overexpression delayed and altered lung epithelial cell tubulation by selectively inhibiting G protein-mediated p38 MAPK activation, and, consequently, by reducing epithelial cell proliferation, migration, and expression of vascular endothelial growth factor (VEGF). The tubulogenic defects imparted by RGS4 in epithelial cells, including its reduction in VEGF expression, were rescued by overexpression of constitutively active MKK6, an activator of p38 MAPK. Similarly, RGS4 overexpression abrogated endothelial cell angiogenic sprouting by inhibiting their synthesis of DNA and invasion through synthetic basement membranes. We further show that RGS4 expression antagonized VEGF stimulation of DNA synthesis and extracellular signal-regulated kinase (ERK)1/ERK2 and p38 MAPK activation as well as ERK1/ERK2 activation stimulated by endothelin-1 and angiotensin II. RGS4 had no effect on the phosphorylation of Smad1 and Smad2 by bone morphogenic protein-7 and transforming growth factor-beta, respectively, indicating that RGS4 selectively inhibits G protein and VEGF signaling in endothelial cells. Finally, we found that RGS4 reduced endothelial cell response to VEGF by decreasing VEGF receptor-2 (KDR) expression. We therefore propose RGS4 as a novel antagonist of epithelial and endothelial cell tubulogenesis that selectively antagonizes intracellular signaling by G proteins and VEGF, thereby inhibiting cell proliferation, migration, and invasion, and VEGF and KDR expression.

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neurocommons text mining pilot:

PubMed abstracts @ 16,000,000 >  
CNS classified abstracts @ 874,727 >  
text mining recognized @ 368,688 >  
text mining processed @ 94,381

extracted graph of 30,000+ relationships  
and 5,500 genes and proteins

no ir's harvested

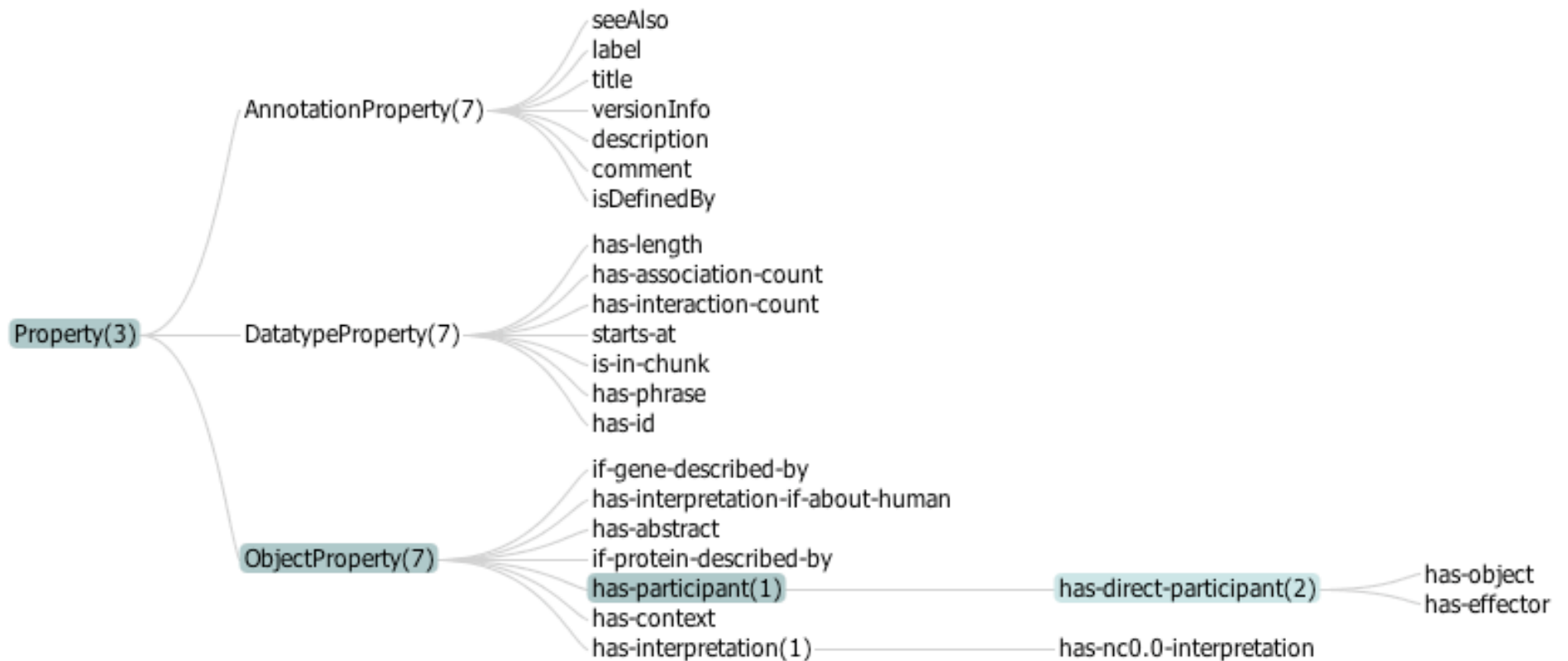
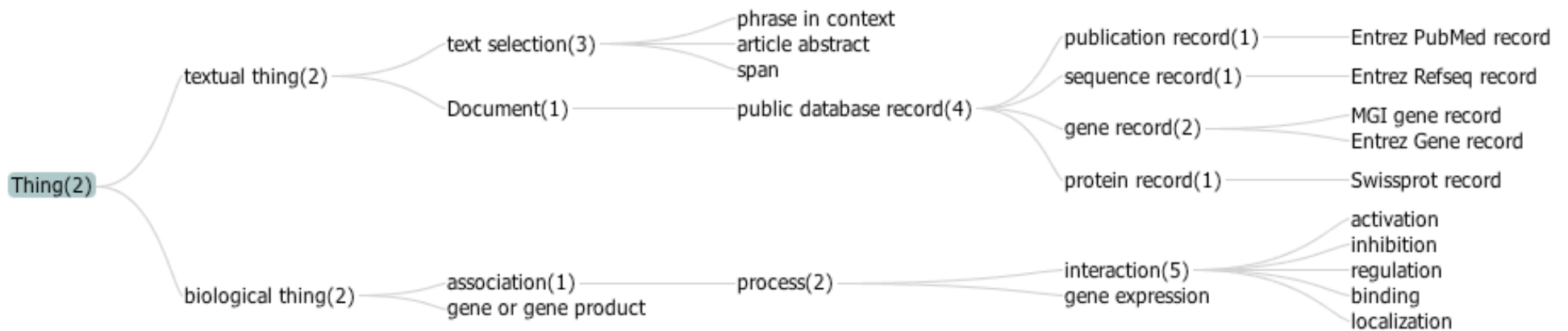
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looking forward to OAI-ORE







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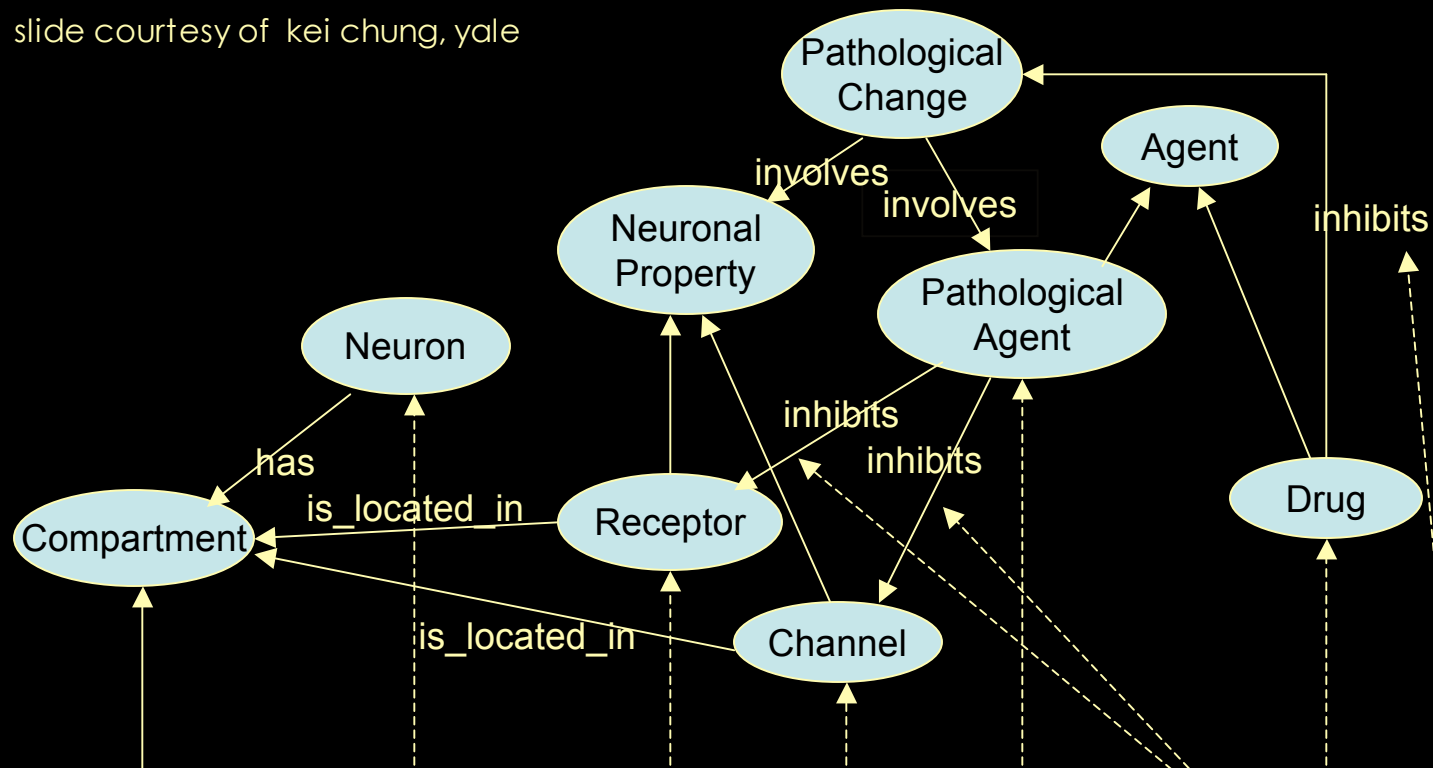
“wire” primary public databases to the  
public graph

open source analytics: a “concordance  
engine” for genes



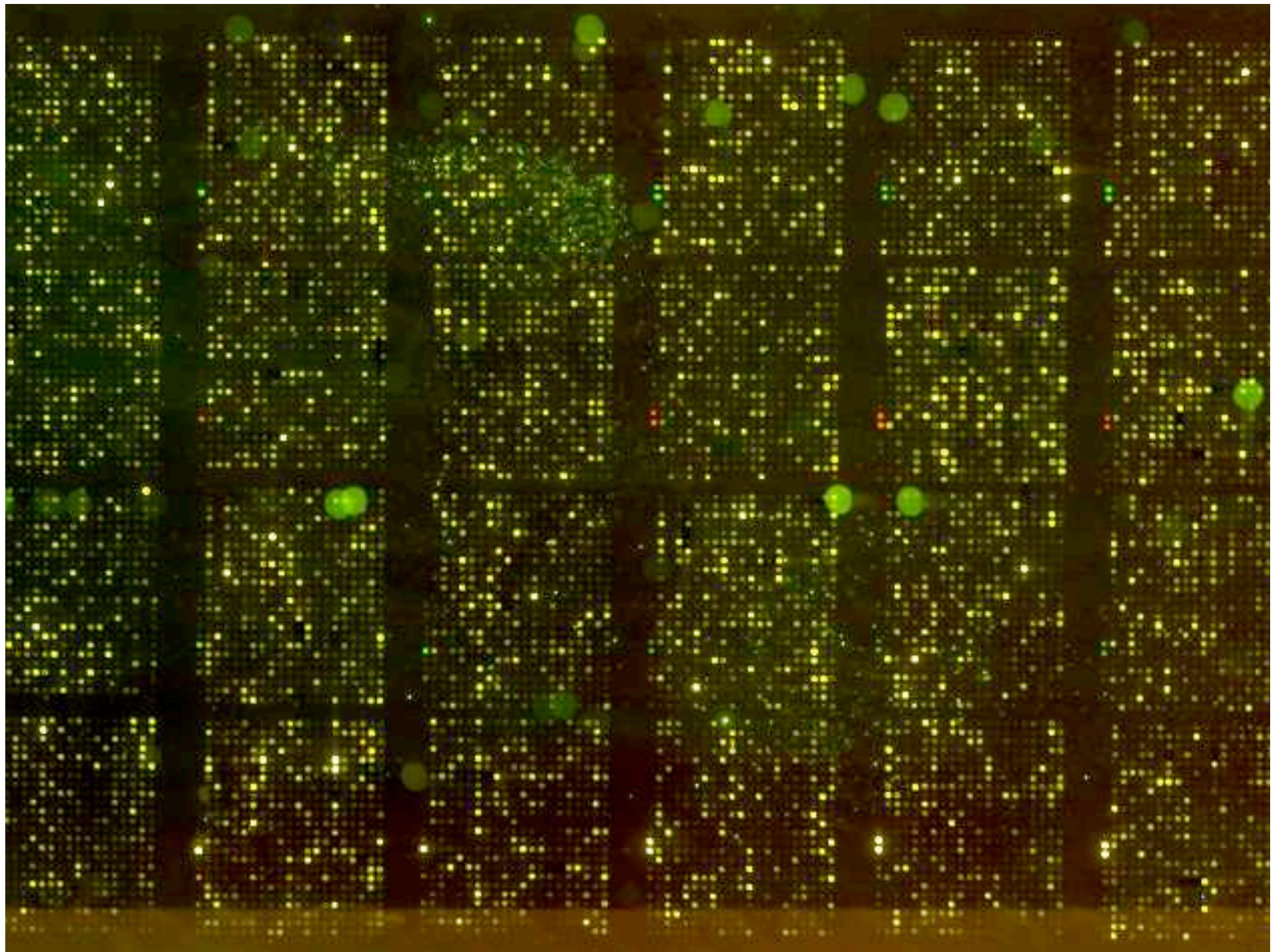
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Compartment	Cell: NeuronDB	Receptor	Channel	Pathological Agent (PA)	PA Action	Drug	Drug Action	Stage	Note	Detail
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	<a href="#">Olfactory bulb mitral cell</a>	<a href="#">GabaA</a>						<a href="#">Early</a>	<a href="#">View</a>	<a href="#">66750</a>
Dendrite	<a href="#">CA1 pyramidal neuron</a>		<a href="#">I A</a>	<a href="#">beta Amyloid</a>	<a href="#">Inhibits</a>			<a href="#">Early</a>	<a href="#">View</a>	<a href="#">66240</a>
	<a href="#">Olfactory bulb mitral cell</a>	<a href="#">GabaA</a>						<a href="#">Early</a>	<a href="#">View</a>	<a href="#">66750</a>
Unspecified	<a href="#">Oocyte</a>		<a href="#">I L high threshold</a>	<a href="#">beta Amyloid</a>	<a href="#">Inhibits</a>			<a href="#">Early</a>	<a href="#">View</a>	<a href="#">66252</a>
								<a href="#">Early</a>	<a href="#">View</a>	<a href="#">66753</a>
	<a href="#">CA1 pyramidal neuron</a>			<a href="#">beta Amyloid</a>	<a href="#">Inhibits</a>			<a href="#">Early</a>	<a href="#">View</a>	<a href="#">66758</a>
	<a href="#">CA1 pyramidal neuron</a>	<a href="#">NMDA</a>	<a href="#">I Calcium</a>	<a href="#">beta Amyloid</a>	<a href="#">Inhibits</a>		<a href="#">Inhibits</a>		<a href="#">View</a>	<a href="#">66250</a>





Category Name	size	log p
structural constituent of ribosome	140	-30.4
cytosolic large ribosomal subunit	36	-17.7
cytosolic small ribosomal subunit	27	-17.4
translation initiation factor activity	46	-9.0
heterogeneous nuclear ribonucleoprotein	13	-8.3
regulation of translational initiation	23	-6.5
pre-mRNA splicing factor activity	59	-6.2
mRNA splicing	45	-5.9
Arp2/3 protein complex	7	-4.5
spliceosome complex	34	-3.9
mRNA splice site selection	6	-3.8
thyroid hormone receptor binding	14	-3.8
eukaryotic translation initiation factor 3 complex	10	-3.6
nuclear pore	38	-3.6
androgen receptor signaling pathway	11	-3.2
vitamin D receptor binding	9	-3.1

hypothesis direction from the literature  
("pay attention to the ribosome")

moreso, legally allowed to use the next  
killer technologies at will...





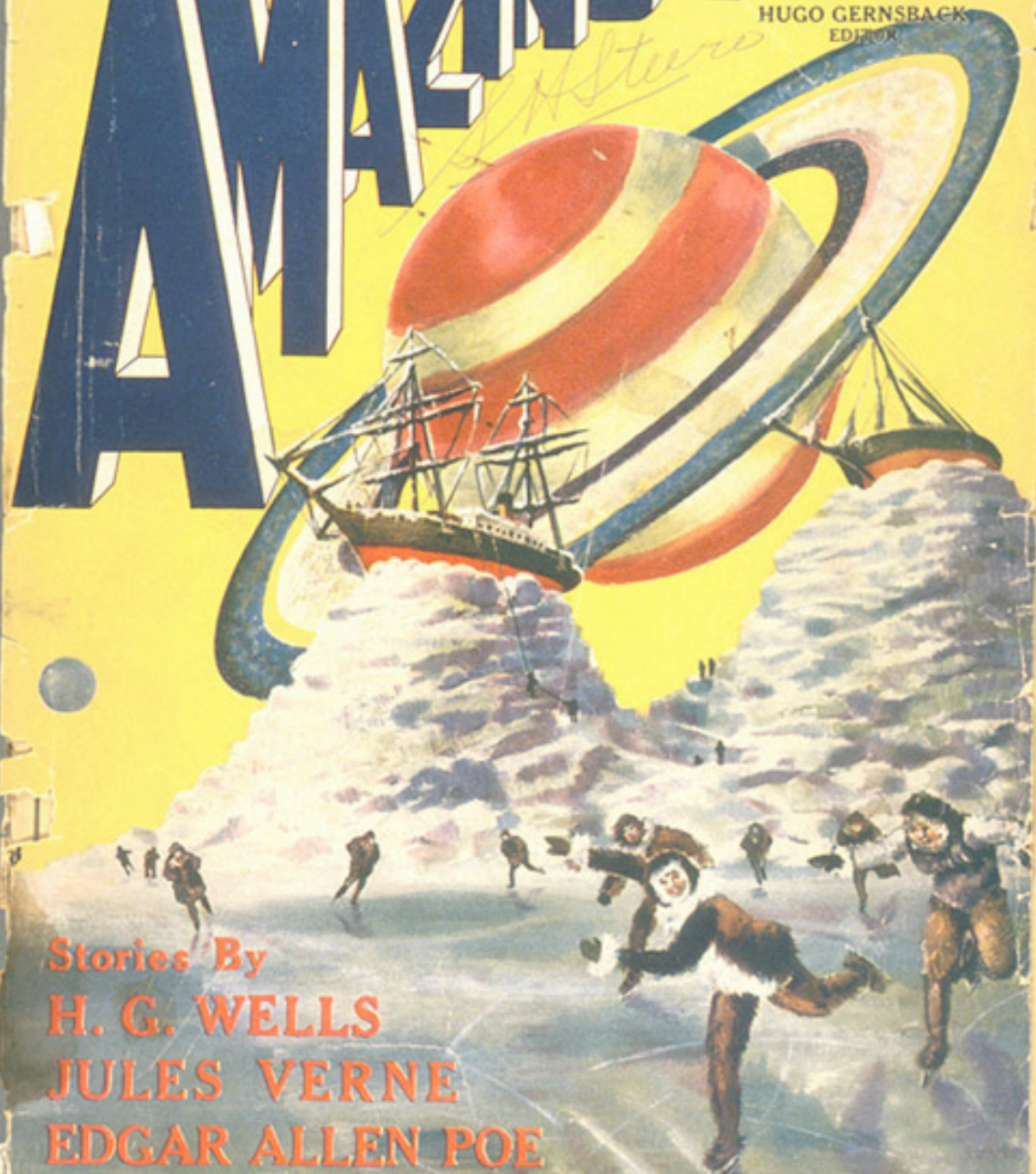
April, 1926

25 Cents

26

# AMAZING STORIES

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