Don’t’ Talk to Laypeople

(they may adopt your pet technology)

‘Knowledge is like love: it multiplies when shared’

Barend Mons

Biosemantics Group LUMC and EMC
LS integrator Nehterlands eScience Center
Chair of DTL-data
Head of ELIXIR node NL
EC member of Open PHACTS

1. DON’T MIX UP EVERYTHING……
2. Don’t try to make helicopters that dig…..
3. Bigger is not better !
4. If you get everything almost right…….
   you have everything wrong (so much for ontologies)

elixir Data for Life

Search

about | organisation | pilot projects | events | news & media | members area

Welcome to ELIXIR

Building a sustainable European infrastructure for biological information, supporting life science research and its translation to medicine, agriculture, bioindustries and society.

"ELIXIR unites Europe's leading life science organisations in managing and safeguarding the massive amounts of data being generated every day by publicly funded research. It is a pan-European research infrastructure for biological information.

"ELIXIR will provide the facilities necessary for life science researchers - from bench biologists to cheminformaticians - to make the most of our rapidly growing store of information about living systems, which is the foundation on which our understanding of life is built."

- Dr Niklas Blomberg, ELIXIR Director

Group shot

Data interoperability and exchange

Compute and storage infrastructure services

Training & Education

ELIXIR's NL node is hosted by the Dutch Techcenter for Life sciences (DTL), a public private partnership that aims to jointly establish a world-class Next Generation Life Sciences cross technology & cross sector capability including a federated data infrastructure.

The ELIXIR NL node acts as the gateway of ELIXIR capabilities and expertise to all the associated partners in DTL. The NL node focuses its contribution to ELIXIR in three core areas: data interoperability, compute & storage infrastructure services and training.

Collaborating organisations

University Medical Centers
- Academic Medical Centre (AMC)
- Erasmus Medical Centre Rotterdam (EMC)
- Leiden University Medical Centre (LUMC)
- Radboud University Nijmegen Medical Centre (UMCN)
- University of Groningen Medical Centre (UMCG)
- Utrecht University Medical Centre (UMCU)
- VU University Medical Centre (VUMC)
- Maastricht UMC+

Institutes
- Centrum voor Wiskunde en Informatica (CWI)
- CBS-KNAW
- Hubrecht Institute
- Netherlands Cancer Institute (NKI)
- Netherlands eScience Centre
- Plant Research International (PRI)
- RIKILT – Institute of Food Safety
- Royal Tropical Institute (KIT)
- SURFnet & SURFsara

Universities
- Delft University of Technology (TU-Delft)
- Eindhoven University of Technology (TUe)
- Leiden University (UL)
- Maastricht University (UM)
- Radboud University Nijmegen (RU)
- University of Amsterdam (UvA)
- University of Groningen (RUG)
- Utrecht University (UU)
- VU University of Amsterdam (VU)
- Wageningen University (WU)

Private sector partners
- DSM
- Philips
- TNO
- Unilever
- SME's

Data interoperability and exchange

Several Dutch groups have specialized in data capture standards, software, semantic web standards and formats to enable meaningful exchange and integration of biological information. ELIXIR NL will focus on implementing and developing professional capturing, publishing and hosting of data in standard (semantically interoperable) format that will be offered in a public-private partnership in close collaboration with other ELIXIR nodes and the Hub

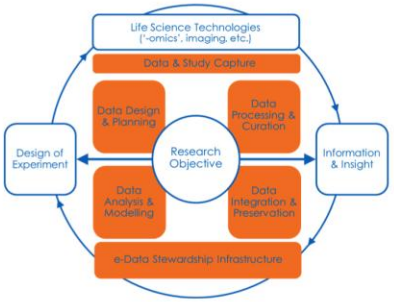
Compute and storage infrastructure services

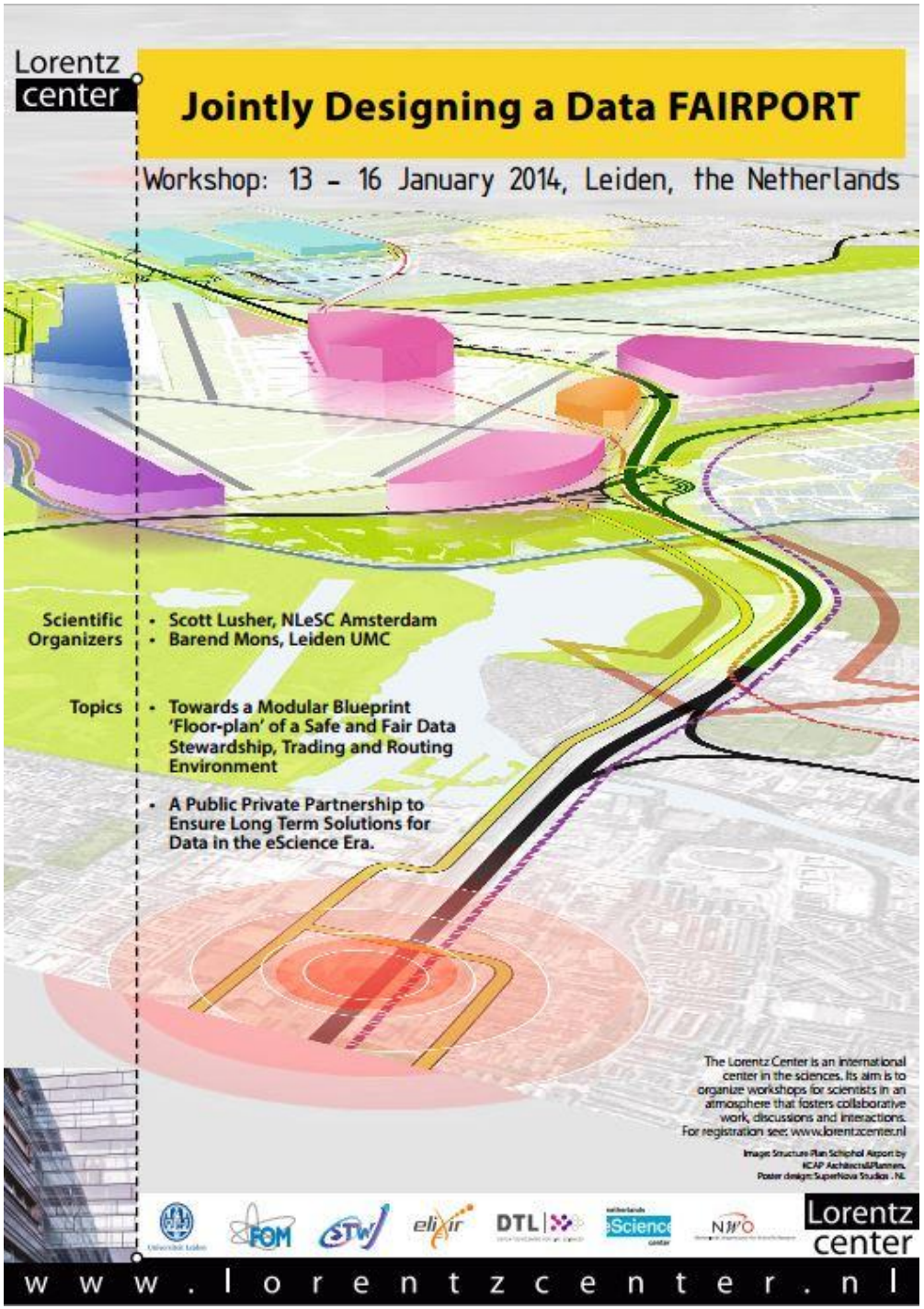
The e-infrastructure capabilities of the Dutch national compute, data and ultra high speed network infrastructure are a clear strength of the ELIXIR NL Node, with extensive experience in running a shared compute and storage environment for collaborative life science projects. The ELIXIR NL node will focus on supporting complex data/compute-intensive life science projects, in collaboration with, and complementary to the offerings of other ELIXIR nodes.

Training

ELIXIR-NL will contribute extensive experience and capacity in bioinformatics training built up within NBIC, and will leverage broad education & training capabilities of the broader DTL partnership in a comprehensive portfolio in the broader scope of the ELIXIR train programme.

ELIXIR NL: focus within the Data Cycle




Lorentz center
Jointly Designing a Data FAIRPORT
Workshop: 13 - 16 January 2014, Leiden, the Netherlands
Scientific Organizers
• Scott Lusher, NLeSC Amsterdam
• Barend Mons, Leiden UMC
Topics
• Towards a Modular Blueprint 'Floor-plan' of a Safe and Fair Data Stewardship, Trading and Routing Environment
• A Public Private Partnership to Ensure Long Term Solutions for Data in the eScience Era.
The Lorentz Center is an international center in the sciences. Its aim is to organize workshops for scientists in an atmosphere that fosters collaborative work, discussions and interactions.
For registration see: www.lorentzcenter.nl
Image: Structure Plan Schiphol Airport by KCAP Architects&Planners.
Poster design: SuperNova Studios . NL
FOM
STW
elixir
DTL
eScience
NWO
Lorentz center
www.lorentzcenter.nl

Why a FAIRPORT ?

WORLD ECONOMIC FORUM : Data, the new currency >>>>>

COMMITTED TO IMPROVING THE STATE OF THE WORLD

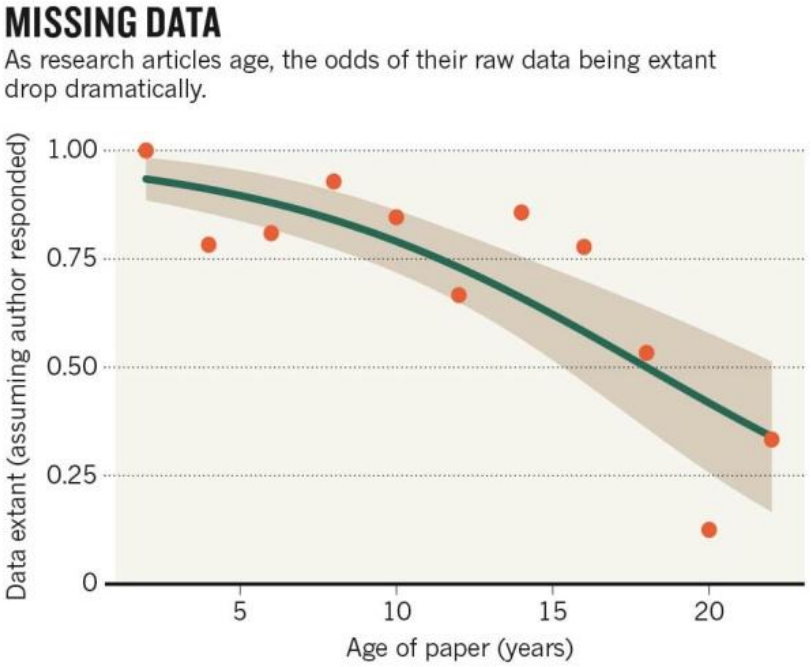
Neelie Kroes (@NeelieKroesEU)
16-03-12 14:25
'Data is the new oil': I urge @ePSIplatform conference to go out & make case for #opendata youtu.be/9Jq4Qy1UeAE

Neelie Kroes is Vice-President of the European Commission, responsible for the “Digital Agenda” for the European union. When she announced the EU’s Open Data Strategy she opened with “Data is the New Gold”. We wish it were that simple.
http://blog.bigml.com

The value of data

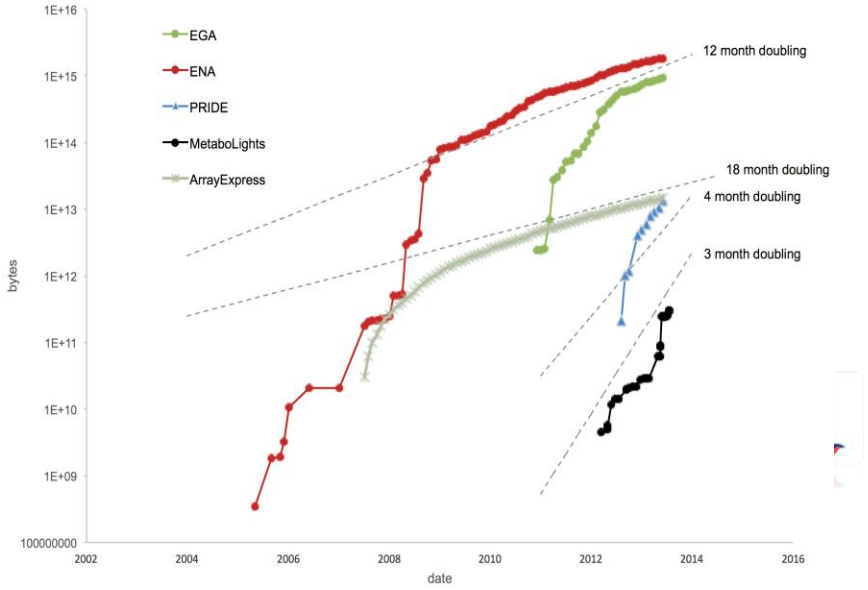
Barend Mons[1–4], Herman van Haagen[1], Christine Chichester[2,4], Peter-Bram ‘t Hoen[1,4], Johan T den Dunnen[1], Gertjan van Ommen[1,4], Erik van Mulligen[3,4], Bharat Singh[2,3], Rob Hooft[2,4], Marco Roos[1,2,4], Joel Hammond[5], Bruce Kiesel[5], Belinda Giardine[6], Jan Velterop[4,7], Paul Groth[4,8] & Erik Schultes[1,4]

Data loss is real and significant…



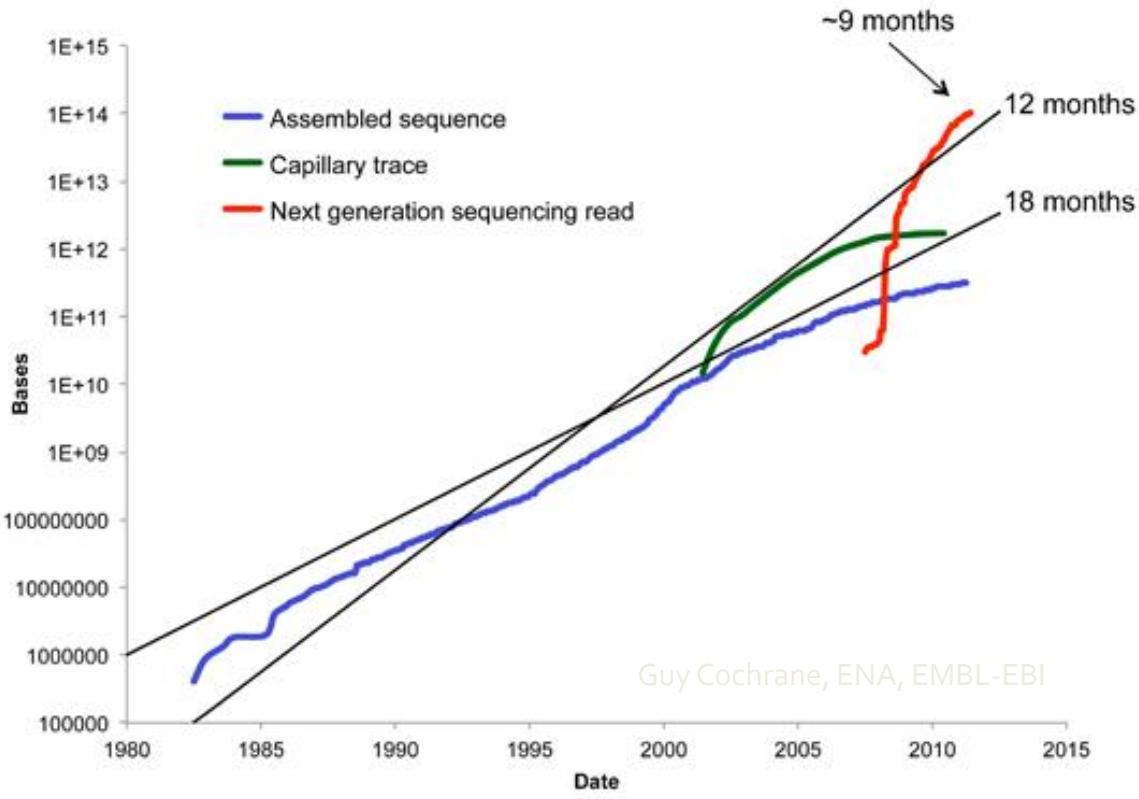
Nature news, 19 December 2013

…and so is Data growth

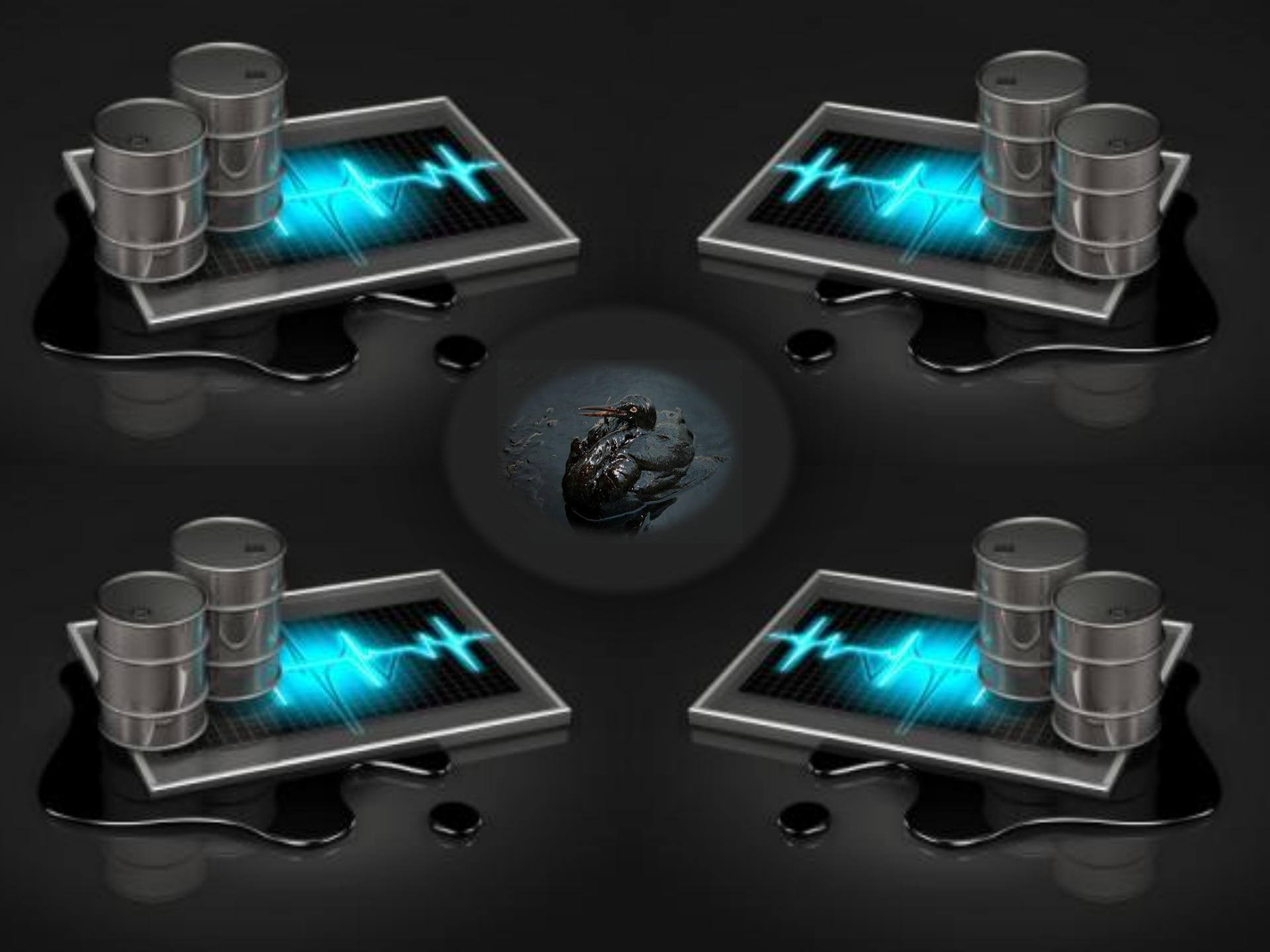


The Data Challenge

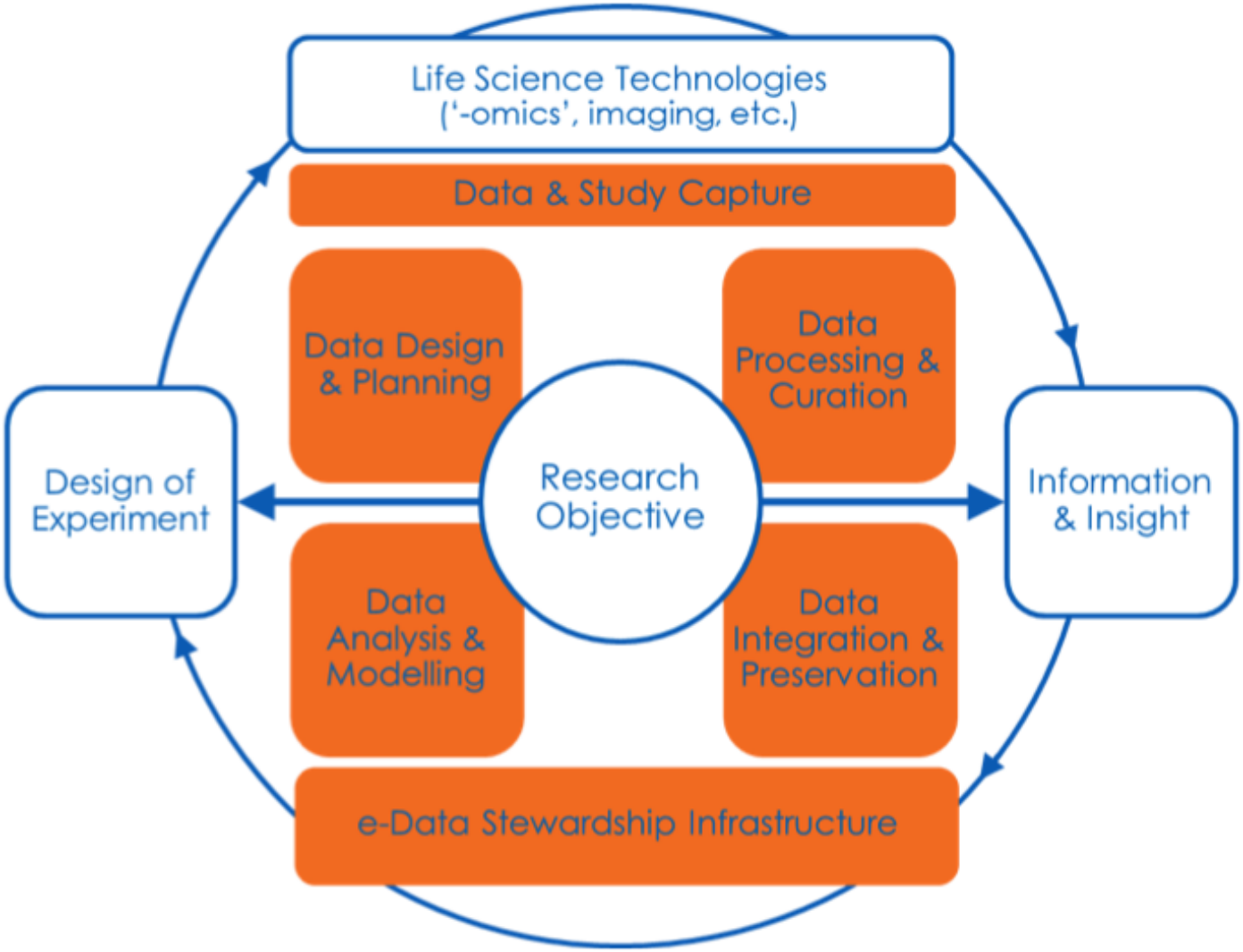
- Computer speed and storage capacity is **doubling every 18 months** and this rate is steady
- DNA sequence data is **doubling every 6-8 months** over the last 3 years and looks to continue for this decade

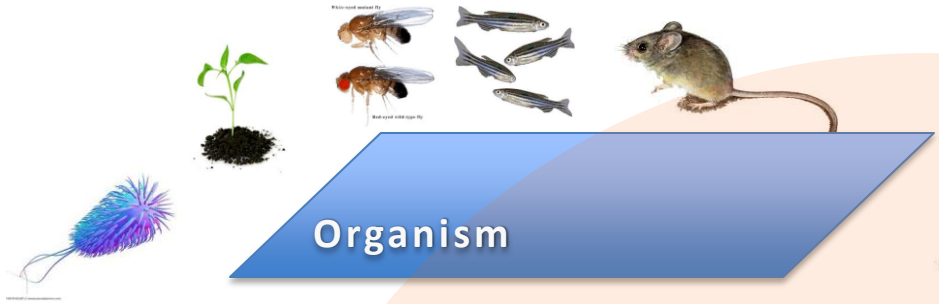


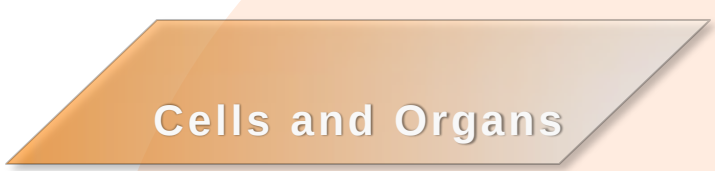

DATA
is the new oil

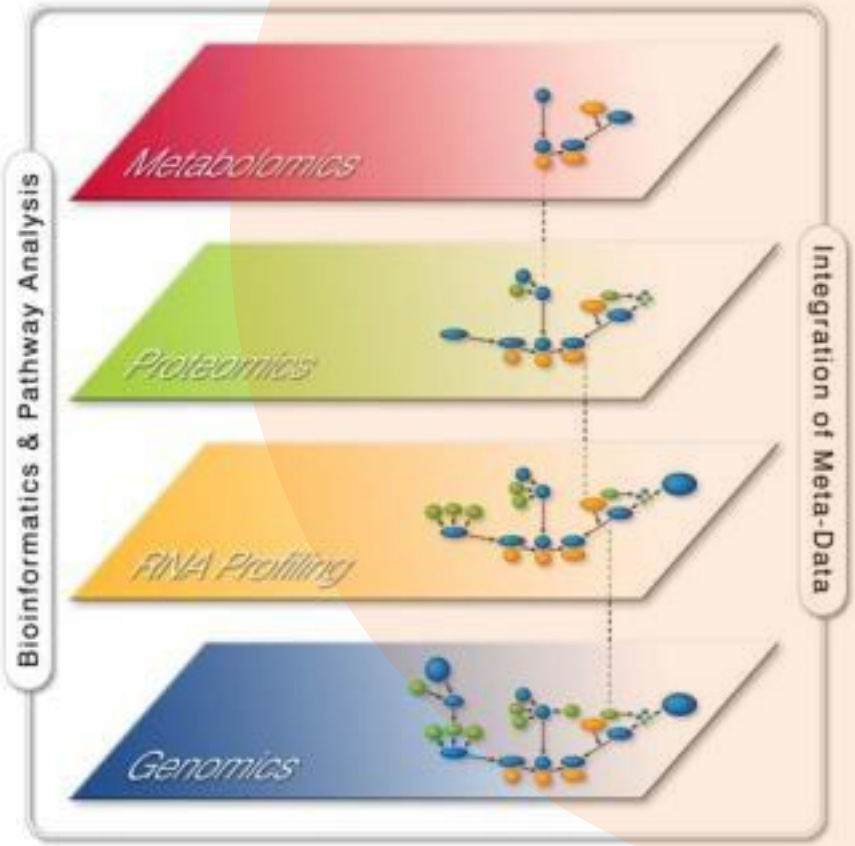


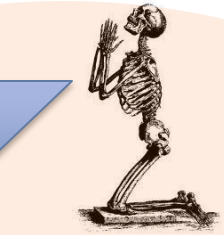
The Data cycle in eScience

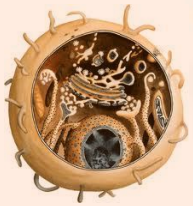


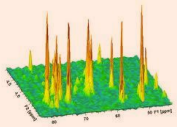

Organism

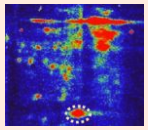

Cells and Organs

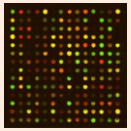

Bioinformatics & Pathway Analysis
Metabolomics
Proteomics
RNA Profiling
Genomics
Integration of Meta-Data

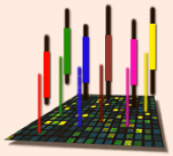










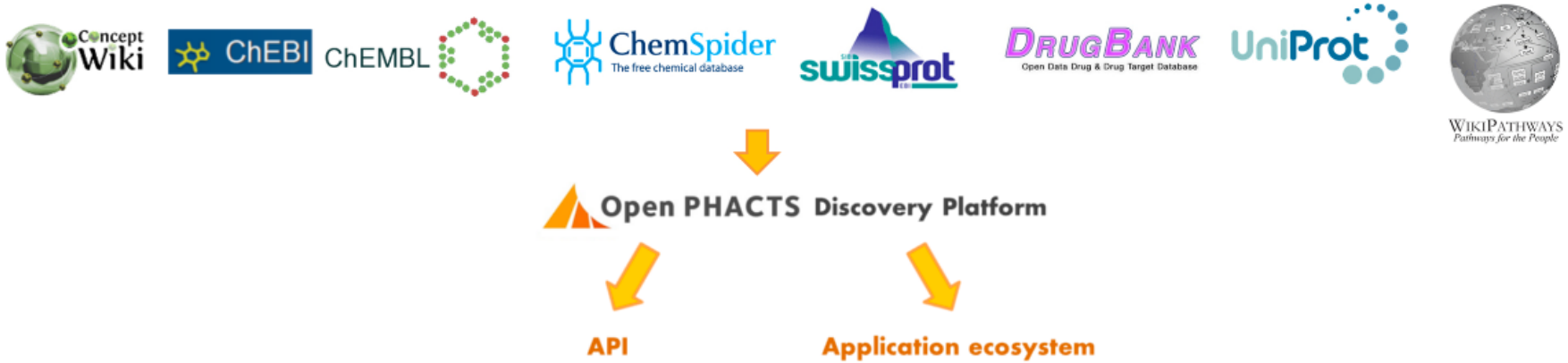


The Open PHACTS Foundation

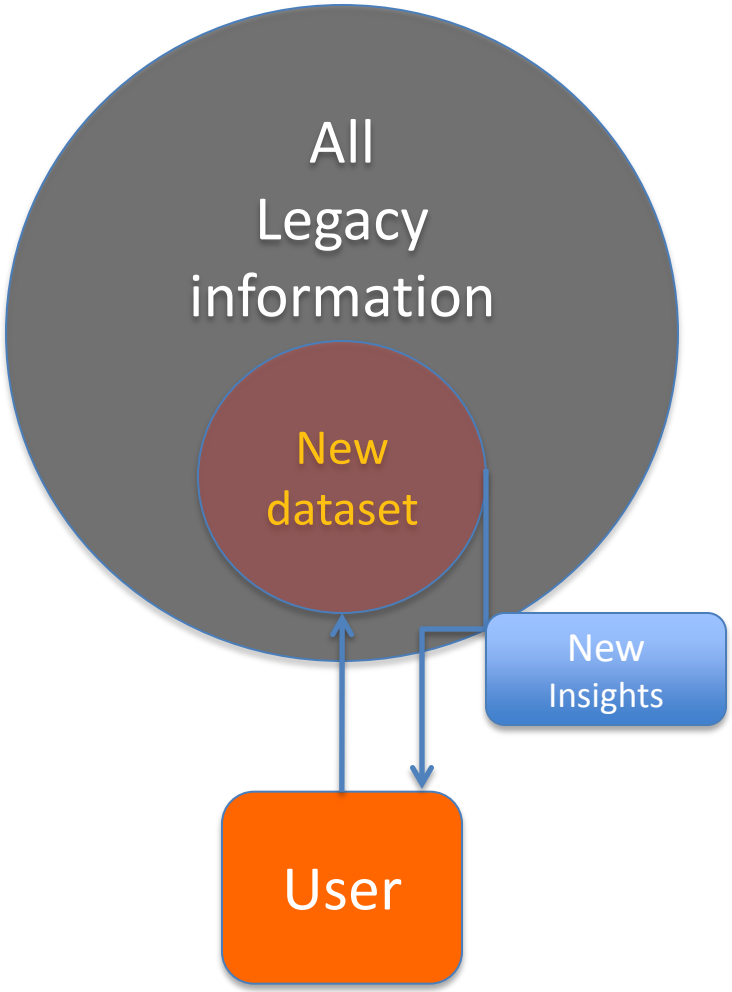
OPF is a not-for-profit membership organisation, supporting the Open PHACTS Discovery Platform:
A sustainable, open, vibrant and interoperable information infrastructure for applied life science research and development.

To reduce the barriers to drug discovery in industry, academia and for small businesses, the Open PHACTS Discovery Platform provides tools and services to interact with multiple integrated and publicly available data sources. To integrate this data, extensive cross-referencing of scientific concepts is needed across all databases.

The Open PHACTS Foundation ensures the sustainability of the Open PHACTS Discovery Platform infrastructure and acts as a hub for relevant scientific research and development.

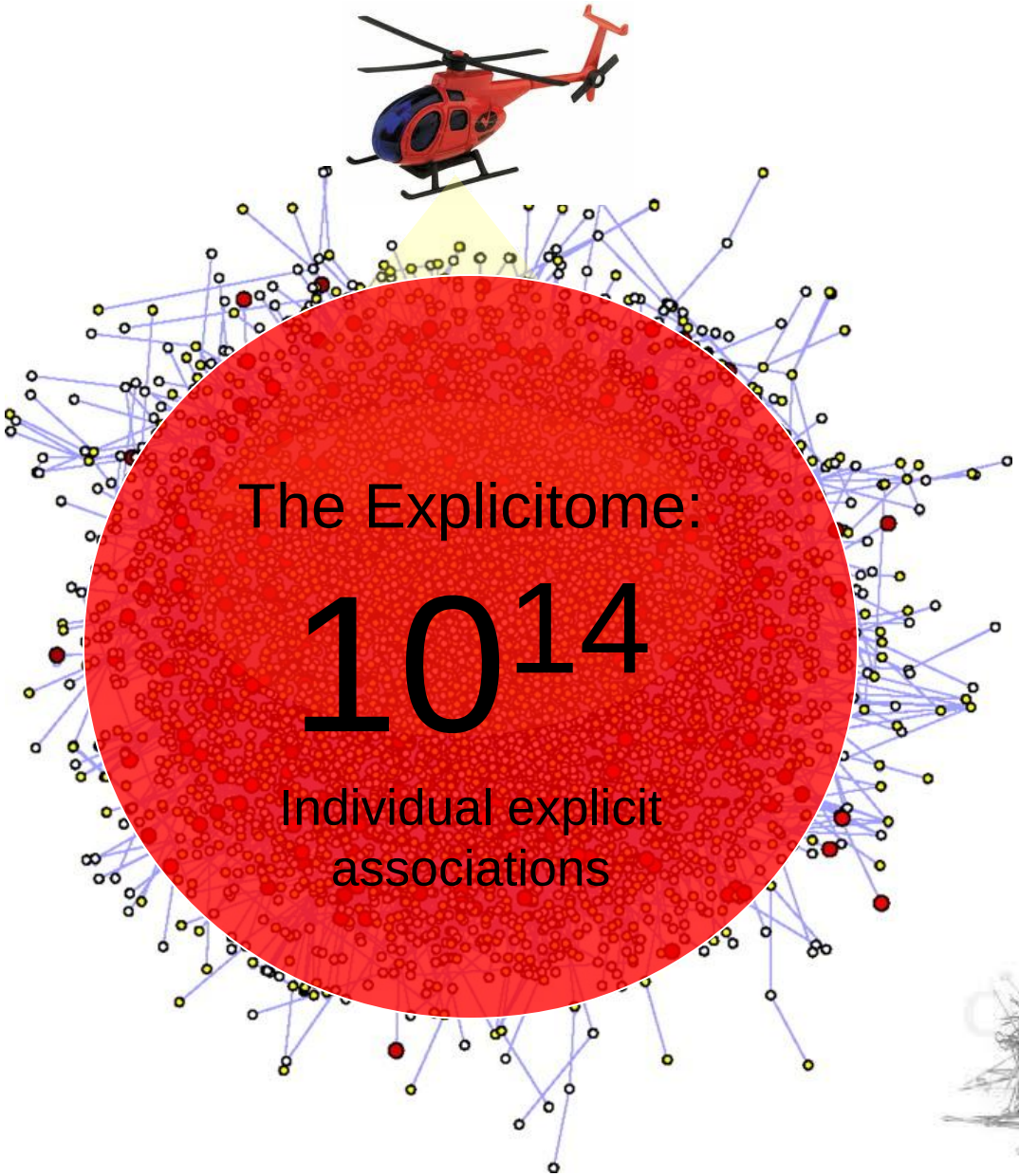


Simplified eScience

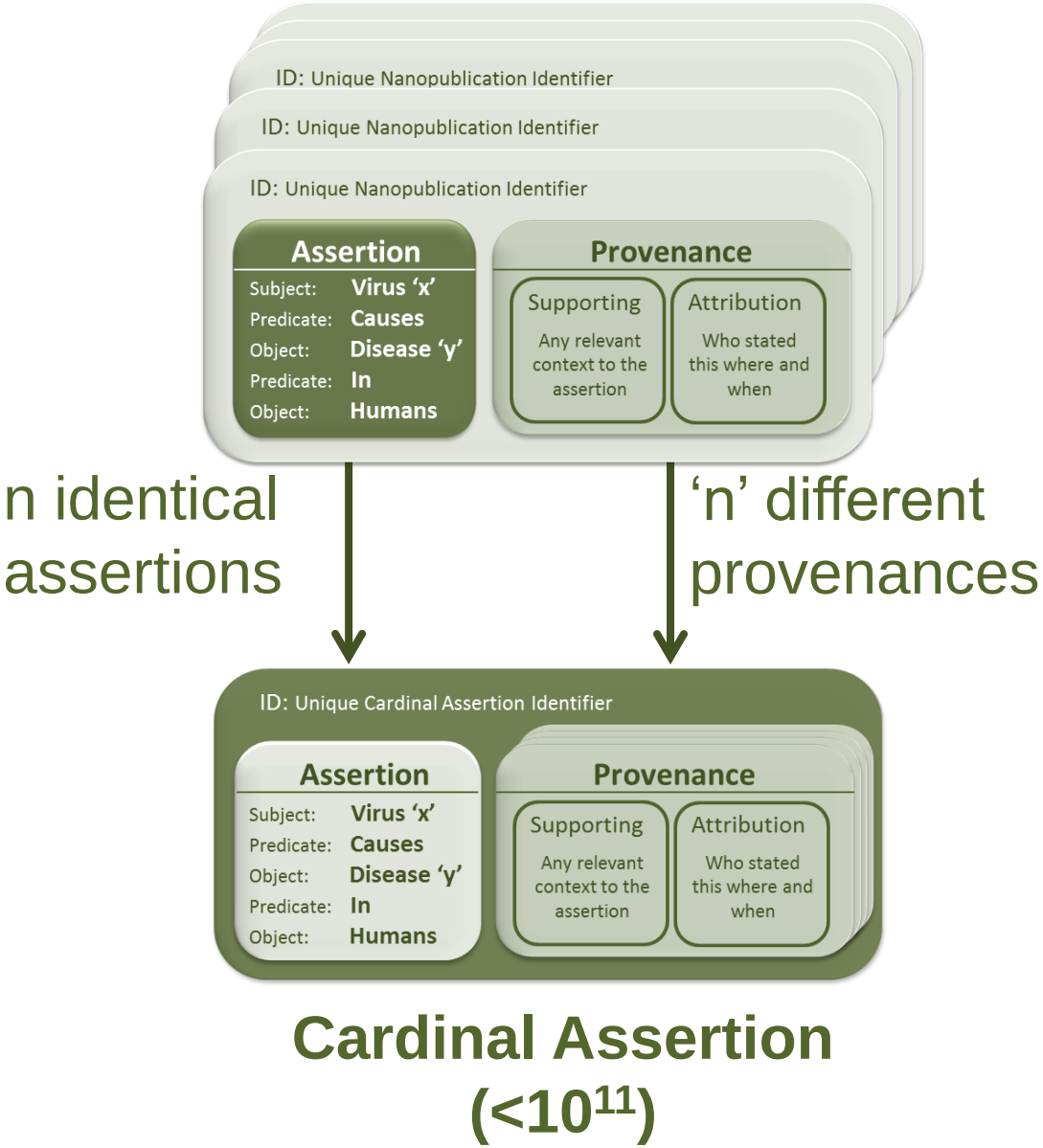


AREAL SURVEY

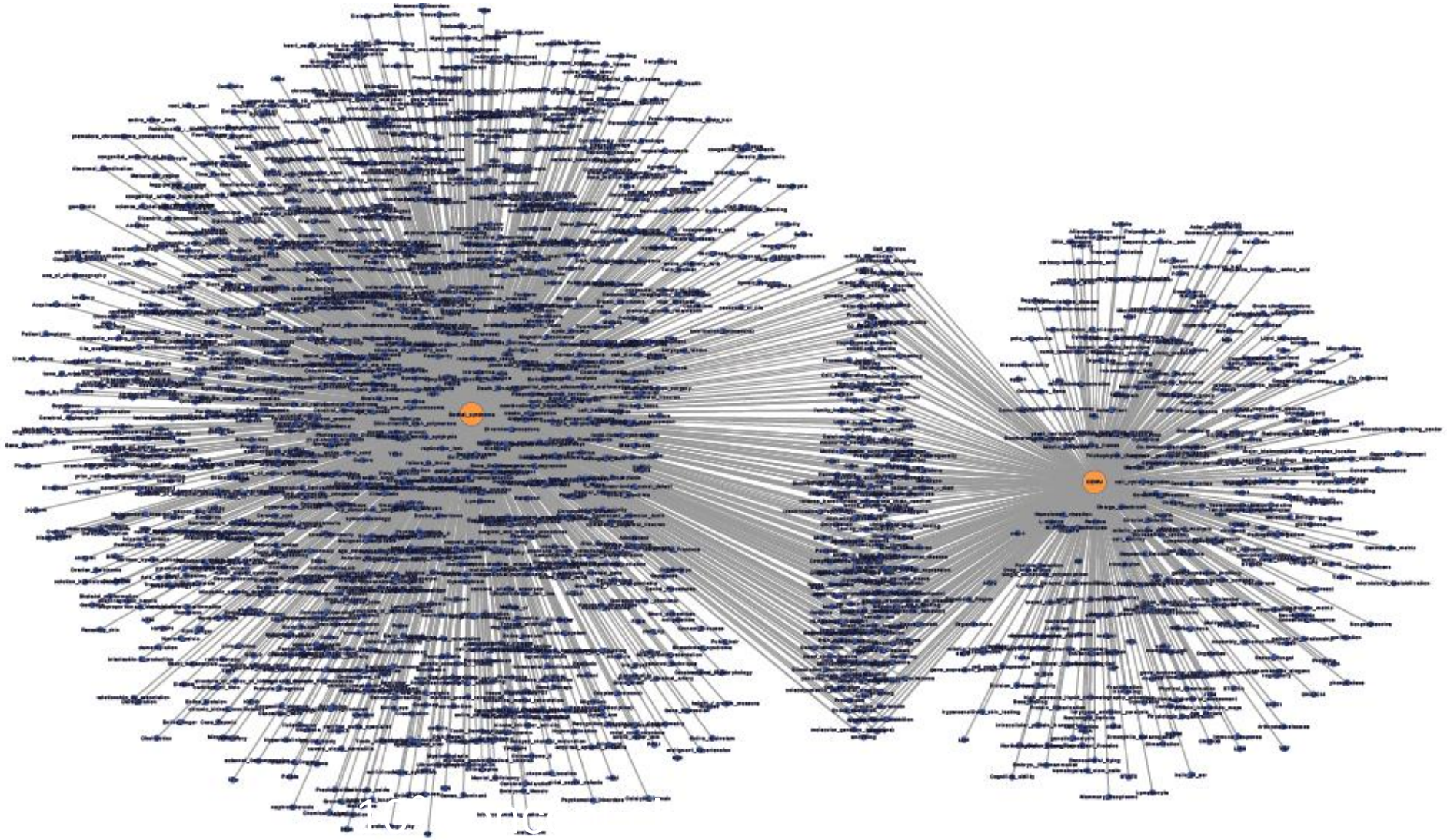
DEEP EXCAVATION


The Explicitome:
10^{14}
Individual explicit associations

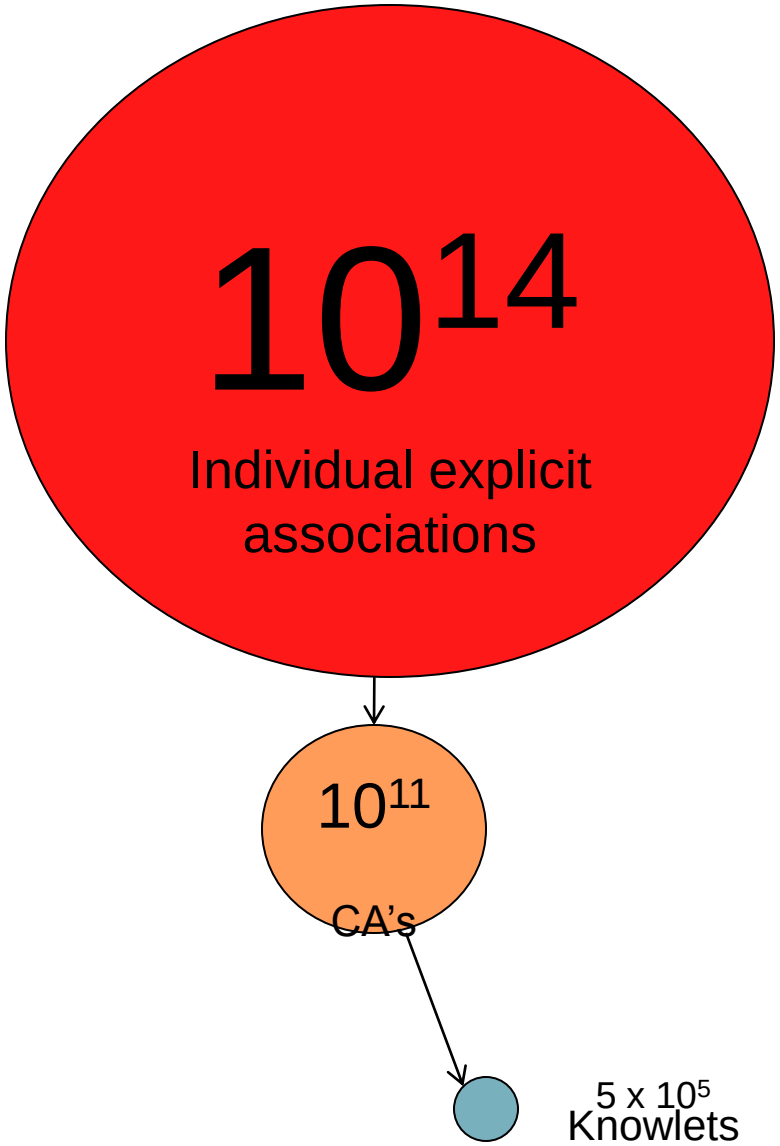
A Semantic Web approach to interoperability

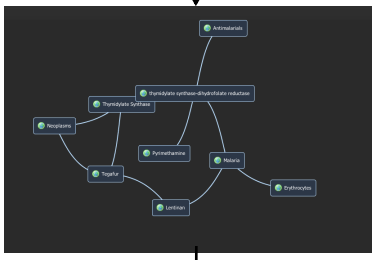


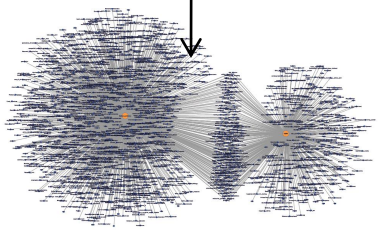
We publish about less than a million LS Concepts !



Zipping the tome



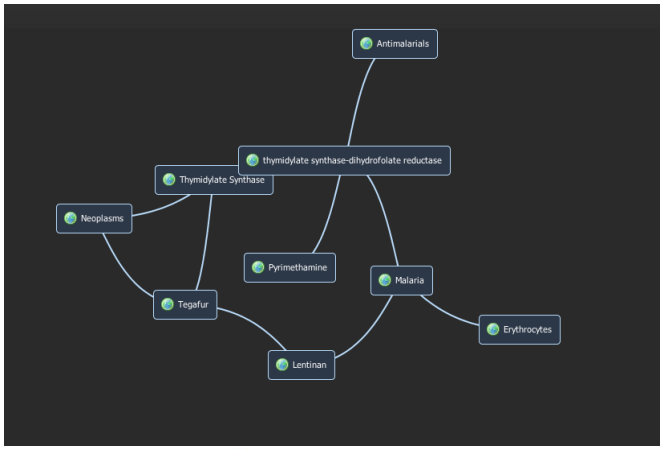


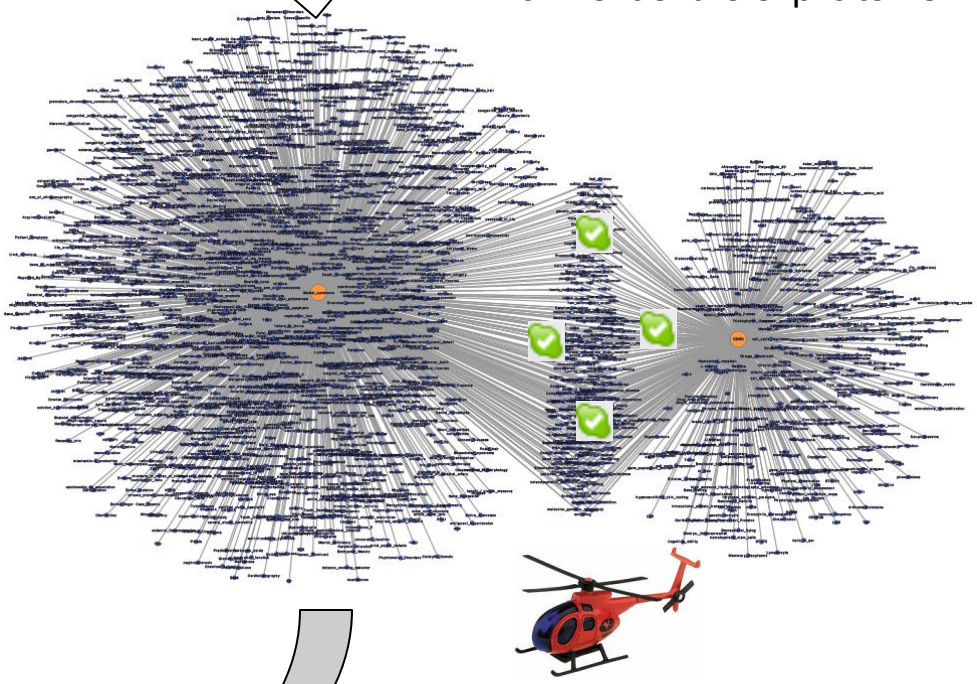


In silico knowledge discovery for the millions..

experimentation

Enrichment of the explicitome



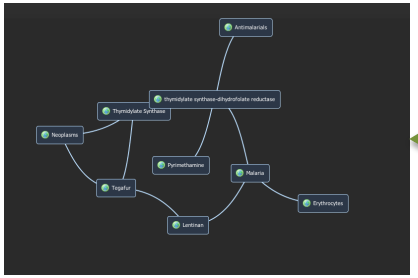


In cerebro rationalisation
And confirmational reading

In silico hypothesis generation

BRAIN[Ξ]

Biological pathways	Phenotypes	Genetics	Proteins	Phenotypes	Pharmacology	Other
WikiPathways	DisGenet	EntrezGene	UniProt	DisGenet	ChEMBL	Pubmed
Reactome	OMIM	SNP db	Human Protein Atlas	OMIM	Chemspider	Clinicaltrial
HMDB	Genes/Diseases	CTD	Enzyme	Genes/Diseases	Drugbank	USPTO
KEGG	DBGaP	LOVD	Antibodypedia	DBGaP	PubChem	
BioGRID		GWAS Central	Proteine Data Bank		SuperLigands	
		Jaspar DB	Secreted Proteine db			
		Tiger Epigenomics	Binding db			



ID: Unique Nanopublication Identifier

Assertion

Subject:	Virus ‘x’
Predicate:	Causes
Object:	Disease ‘y’
Predicate:	In
Object:	Humans

Provenance

Supporting	Attribution
Any relevant context to the assertion	Who stated this where and when

ing latency. Their immunophenotypical characterization as well as their role in HCC development warrant further studies [19].

Activation of Ras/MAPK signalling represents a common hallmark in cancer [20]. It is a dominant network, promoting cell proliferation and survival. Binding of multiple growth factors (e.g. EGF and IGF) to their receptors (e.g. EGFR, IGF1R) induces activation of Ras and downstream effectors (B-Raf, MAPK). Phosphorylation of ERK induces activation of transcription factors (e.g. c-Jun) which, in turn promote transcription of genes involved in cell growth and proliferation.

Sorafenib is a multikinase inhibitor targeting B-Raf activity, amongst others. The presence of pERK in tumor cells, and its role as predictive marker of response to sorafenib, has recently been reported [21]. We tested for ERK/pERK, by means of immunohistochemistry. This revealed that it was weakly expressed and non-phosphorylated in tumoral cells, while pERK was positively expressed in peritumoral endothelial cells; a condition that has not been elucidated yet.

frontiers in IMMUNOLOGY

REVIEW ARTICLE
published: 26 September 2013
doi: 10.3389/fimmu.2013.00298

Immune activation and collateral damage in AIDS pathogenesis

Frank Miedema[1]*, Mette D. Hazenberg[2], Kiki Tesselaar[1], Debbie van Baarle[1], Rob J. de Boer[3] and José A. M. Borghans[1]

[1] *Department of Immunology, University Medical Center Utrecht, Utrecht, Netherlands*
[2] *Department of Internal Medicine and Hematology, Academic Medical Center, Amsterdam, Netherlands*
[3] *Theoretical Biology and Bioinformatics, Utrecht University, Utrecht, Netherlands*

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In the past decade, evidence has accumulated that human immunodeficiency virus (HIV)-induced chronic immune activation drives progression to AIDS. Studies among different monkey species have shown that the difference between pathological and non-pathological infection is determined by the response of the immune system to the virus, rather than its cytopathicity. Here we review the current understanding of the various mechanisms driving chronic immune activation in HIV infection, the cell types involved, its effects on HIV-specific immunity, and how persistent inflammation may cause AIDS and the wide spectrum of non-AIDS related pathology. We argue that therapeutic relief of inflammation may be beneficial to delay HIV-disease progression and to reduce non-AIDS related pathological side effects of HIV-induced chronic immune stimulation.

Keywords: AIDS, pathogenesis, immune activation, TLR, Immunity, therapy

CHRONIC IMMUNE ACTIVATION IS THE PRIMARY DRIVER IN HIV PATHOGENESIS

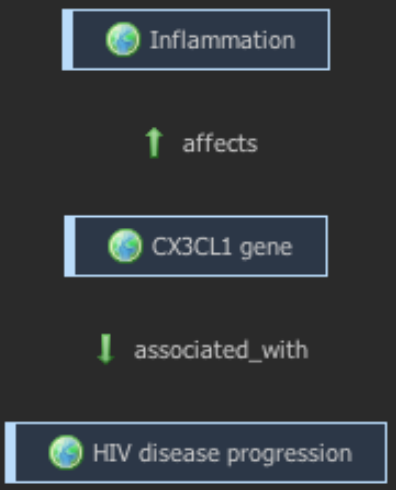
out, however, that T-cell proliferation rates drop concomitant with the loss of virus, even when $CD4^+$ T-cell numbers are still far below

Based on meticulous reading of 221 articles

Brain Home | Frank | **Path predictor** | Shortest route | CX3CL1 gene -> associated_wi

Statement prediction: Inflammation - HIV disease progression (Value: 2.959106910331007 - Prediction: 0.79)

Path visualisation



Path overview (30)

# of statements	Path value ▲
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2	27.36383500660193
2	27.648922177276003
2	27.837785067527275
2	27.837785067527275
2	27.837785067527275
2	27.850928597853745
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2	28.157981907545764
2	28.295468212384968
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2	28.614728503568738
2	28.614728503568738
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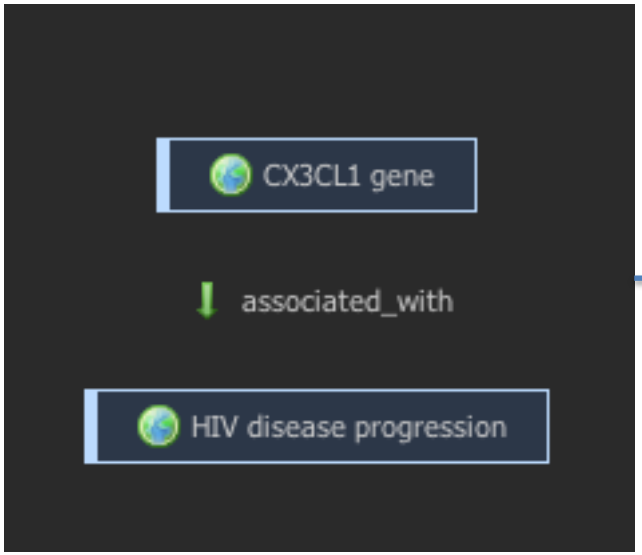
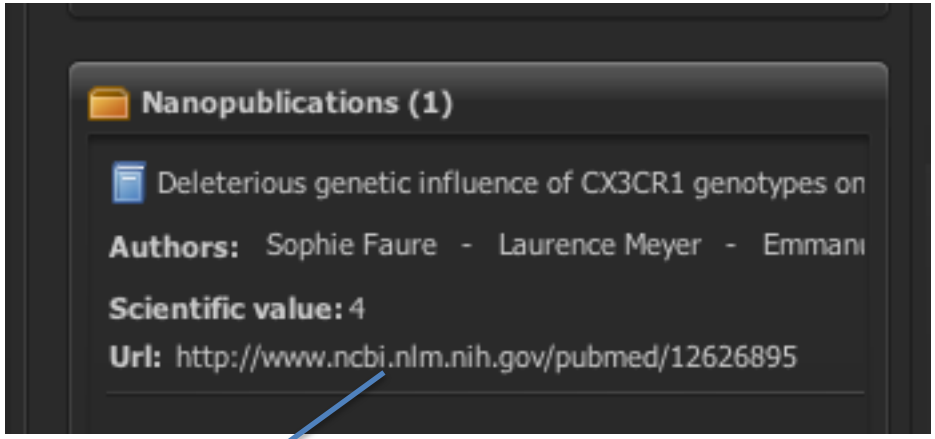




Not mentioned in the Miedema paper.

Display Settings: Abstract

Send to:

J Acquir Immune Defic Syndr. 2003 Mar 1;32(3):335-7.

Deleterious genetic influence of CX3CR1 genotypes on HIV-1 disease progression.

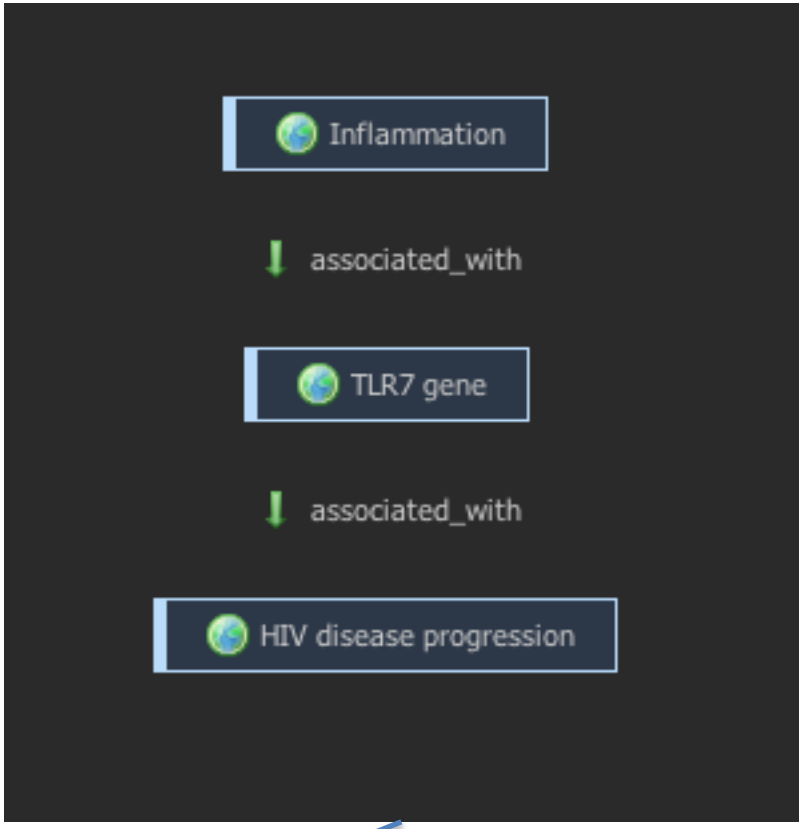
Faure S, Meyer L, Genin E, Pellet P, Debré P, Théodorou I, Combadière C; SEROCO Study Group.

INSERM U543, Hôpital Salpêtrière, Paris, France.

Abstract

We previously reported that patients homozygous for a specific mutation (M280) in the chemokine receptor CX3CR1 progressed to AIDS more rapidly than those with other genotypes. This deleterious effect would predict that a cohort of prevalent patients would be depleted in M280 carriers, because these patients would have disappeared before recruitment. This hypothesis is confirmed in this new study based on the French SEROCO cohort showing that patients homozygous for the M280 allele were rare among the seroprevalent group. These results may explain the conflicting results published on the impact of CX3CR1 polymorphism in seroconverters.

PMID: 12626895 [PubMed - indexed for MEDLINE]



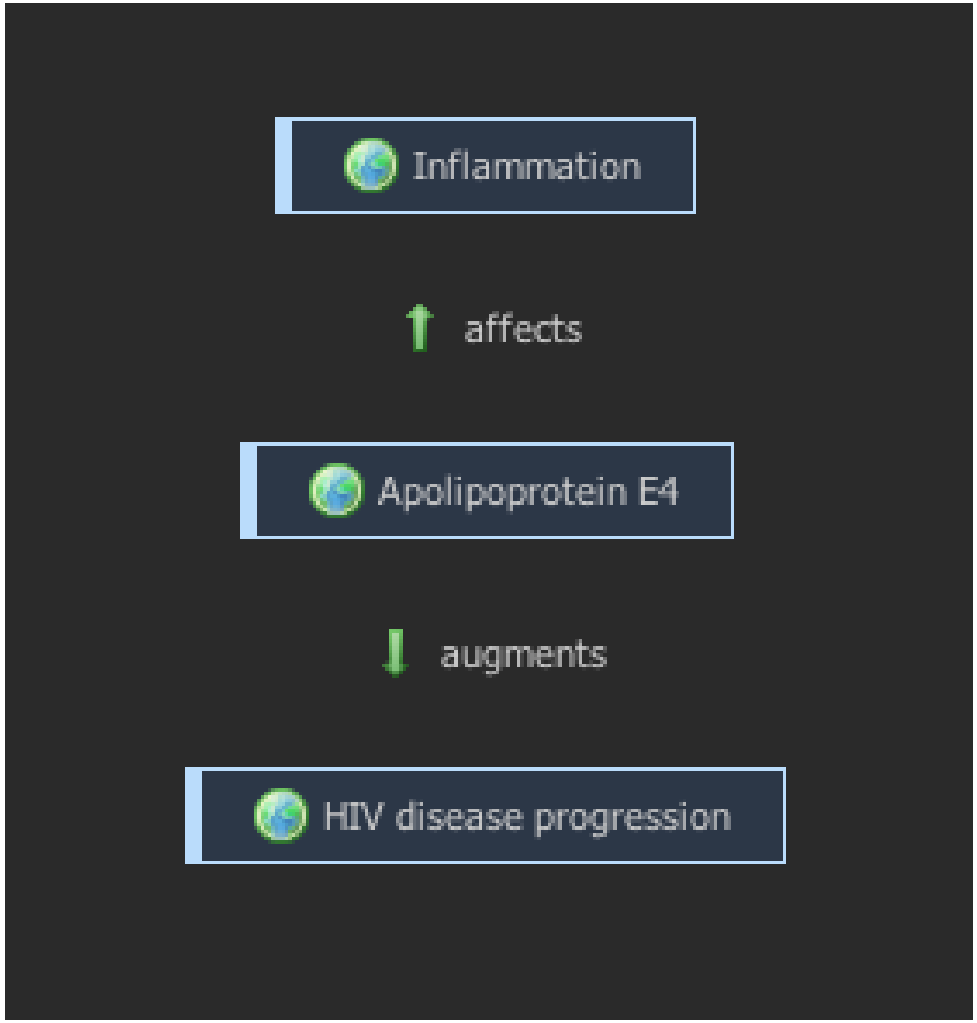
Mentioned in the Miedema paper.

AIDS. 2009 Jan 28;23(3):297-307. doi: 10.1097/QAD.0b013e32831fb540.

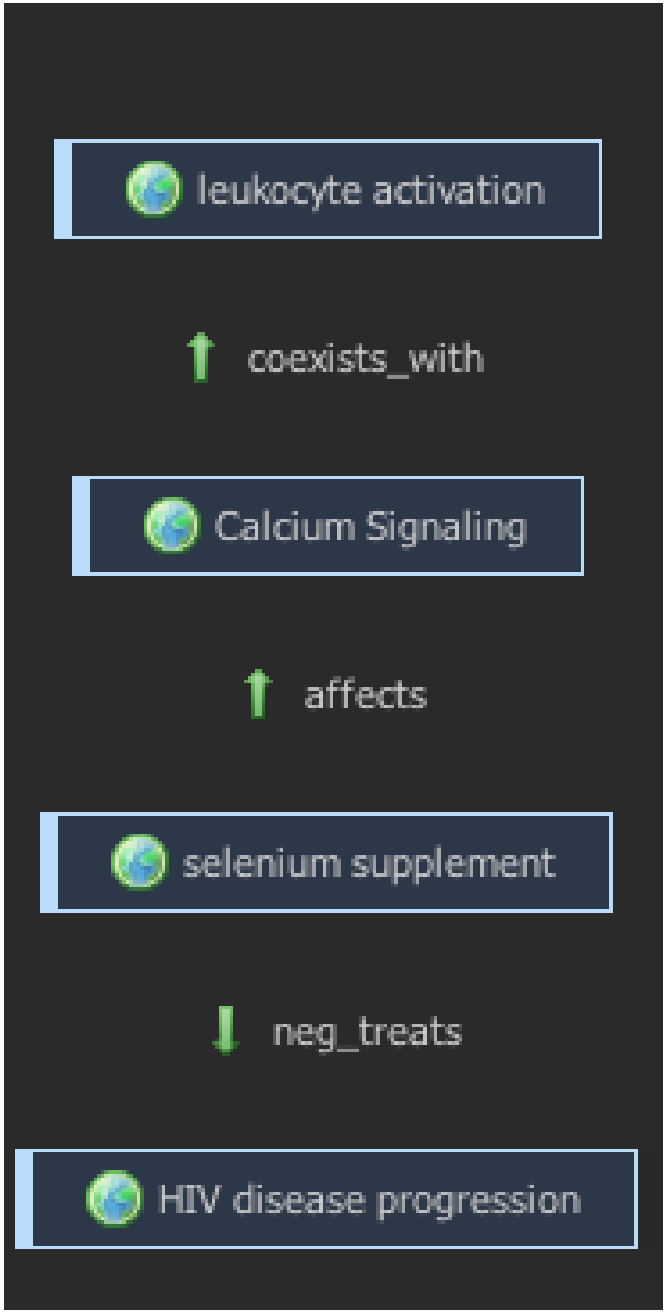
A frequent functional toll-like receptor 7 polymorphism is associated with accelerated HIV-1 disease progression.

Oh DY, Baumann K, Hamouda O, Eckert JK, Neumann K, Kücherer C, Bartmeyer B, Poggensee G, Oh N, Pruss A, Jessen H, Schumann RR.

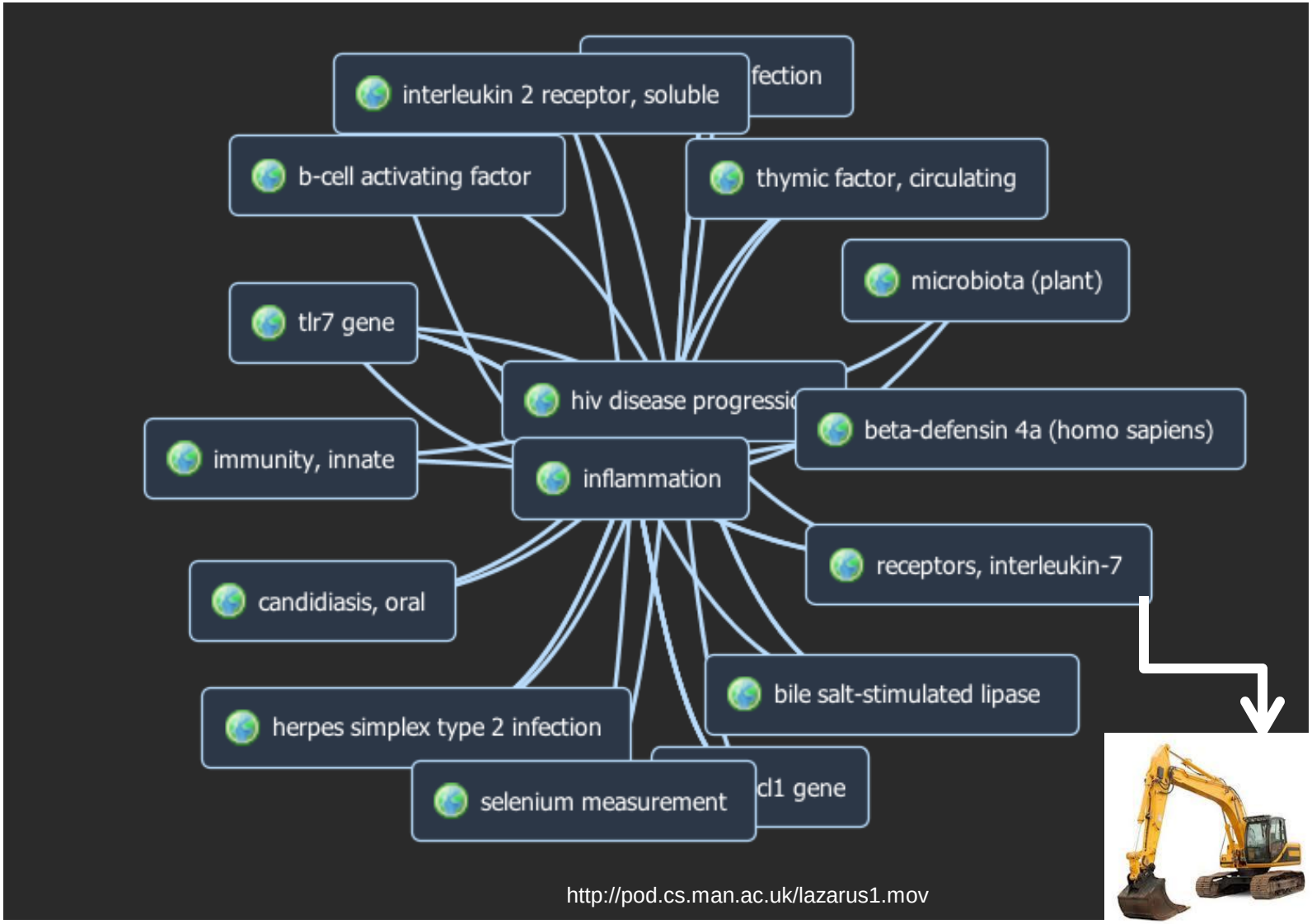
Institute for Microbiology and Hygiene, Charité University Medical Center, Berlin, Germany.



Not mentioned in the Miedema paper.



Not mentioned in the Miedema paper.


interleukin 2 receptor, soluble
fection
b-cell activating factor
thymic factor, circulating
microbiota (plant)
tlr7 gene
hiv disease progressi
beta-defensin 4a (homo sapiens)
immunity, innate
inflammation
receptors, interleukin-7
candidiasis, oral
bile salt-stimulated lipase
herpes simplex type 2 infection
cl1 gene
selenium measurement
http://pod.cs.man.ac.uk/lazarus1.mov

Brain v1.0 (current workspace: sorafenib.)

Exit Brain | Add Workspace

Current workspace

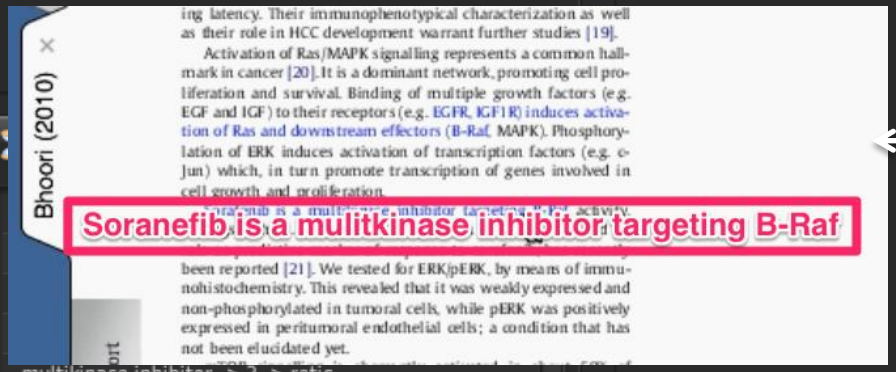
sorafenib.

Search terms and relations

Term: b-raf

Relation: ex. transmitted by

Term: retic



multikinase inhibitor -> ? -> retic

renal neoplasms -> ? -> retic

sorafenib -> ? -> retic

Workspaces (8)

Testlab

tegafur

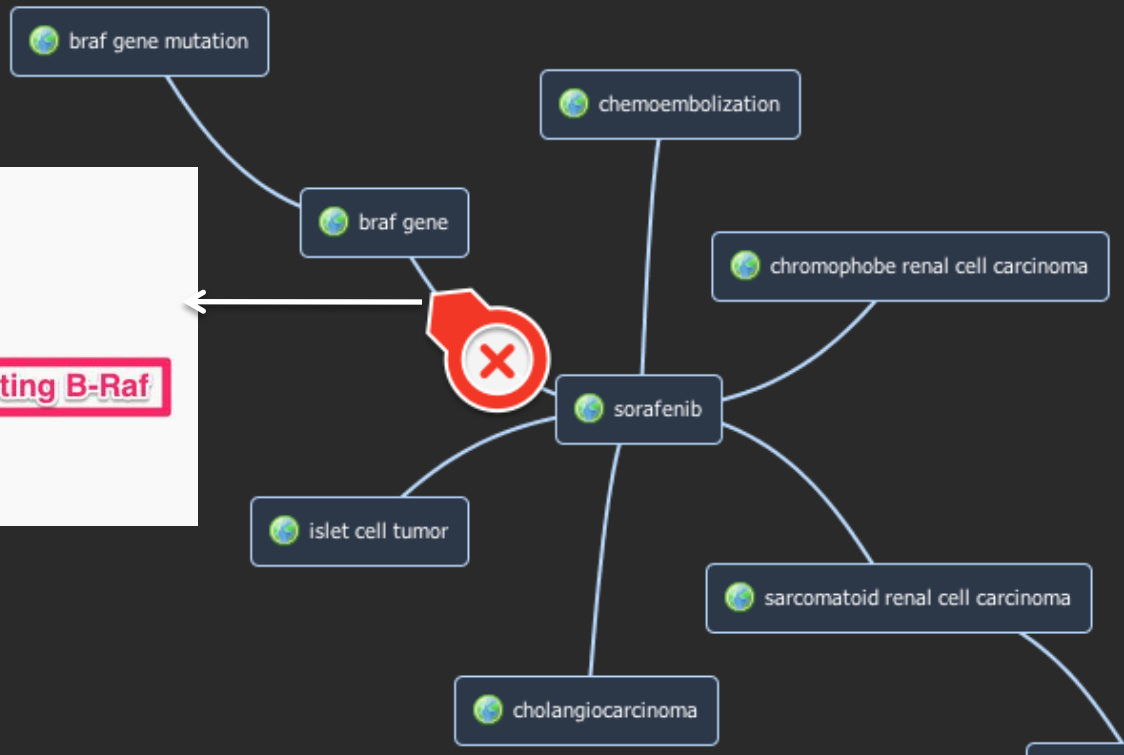
Frank

Marc

Fractalkine

sorafenib.

Brain Home | sorafenib. | Statements visualisation | sorafenib -> inhibits -> braf gene | braf



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Arch Dermatol. 2012 May;148(5):628-33. doi: 10.1001/archdermatol.2012.125.

Cutaneous toxic effects associated with vemurafenib and inhibition of the BRAF pathway.

Huang V[1], Hepper D, Anadkat M, Cornelius L.

Author information

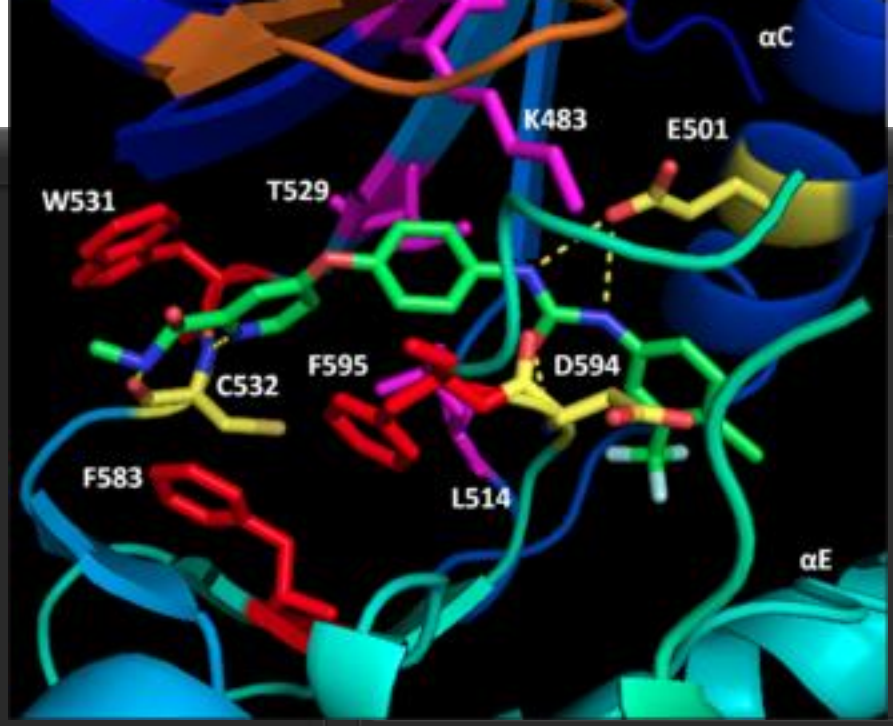
Abstract

BACKGROUND: The development of a novel BRAF inhibitor, vemurafenib, has been associated with impressive tumor r
BRAF-positive stage IV melanoma. In the phase 3 clinical trials, dermatologic toxic effects associated with vemurafenib w
development of eruptive squamous cell carcinomas. Herein, 3 cases are presented that highlight the development of squ
other cutaneous sequelae that have not been previously reported and are reminiscent of those observed with administrat
inhibitor sorafenib tosylate. In addition, the current understanding of the molecular mechanisms underlying these toxic eff

OBSERVATIONS: The development of keratosis pilaris-like eruptions; seborrheic dermatitis-like rashes; and hyperkerato
reminiscent of those seen in sorafenib-associated hand-foot skin reaction, as well as squamous cell carcinomas, is prese
vemurafenib-based treatment of metastatic melanoma.

CONCLUSIONS: The development of sorafenib-like cutaneous sequelae (squamous cell carcinomas, keratosis pilaris-lik
dermatitis-like rashes, and hand-foot skin reaction) associated with vemurafenib administration suggests that BRAF inhib
to induce these changes.

PMID: 22431713 [PubMed - indexed for MEDLINE]



sorafenib

inhibits

braf gene

Not True !
But who cares ?

Nanopublications (6)

Cutaneous toxic effects associated with vemurafeni

Authors: Huang, Victor (V) - Hepper, Donna (D) -

Scientific value: 4

Url: http://www.ncbi.nlm.nih.gov/pubmed/22431713

Development of a novel chemical class of BRAF inhi

Authors: Smalley, Keiran S M (KS) - Flaherty, Keith

Scientific value: 4

Url: http://www.ncbi.nlm.nih.gov/pubmed/19663727

Identification and characterization of novel oncoge

Authors: Cools, J (J)

Embargoed till March 2014

EDITORIAL

nature genetics

Call for data analysis papers

Community standards for data access, interoperability and metadata only make sense if data are creatively reused to further research. We are therefore inviting the submission of Analysis papers that reformat and integrate existing data sets to generate substantial novel insights into gene expression in cell differentiation transitions and different cell fates.

in the year. We will also apply advice on data interoperability from a range of experts, including but not limited to BioSharing (http://www.biosharing.org/), the Global Alliance for sharing data (http://www.ebi.ac.uk/about/news/press-releases/Global-Alliance), ELIXIR (http://www.elixir-europe.org/) and the US National Institutes of Health (NIH) Big Data to Knowledge initiative (http://www.bd2k.nih.gov/), and offer guidance to authors on adding value to proposed analyses.

Demonstruction time

- BRAIN
- Utopia
- Time tracking of concept in the Concept Web