



Collaborative Publishing with Authorship Tracking and Reputation System

Robert Hoffmann
NETTAB, Biological Wikis, Naples, Italy, 2010

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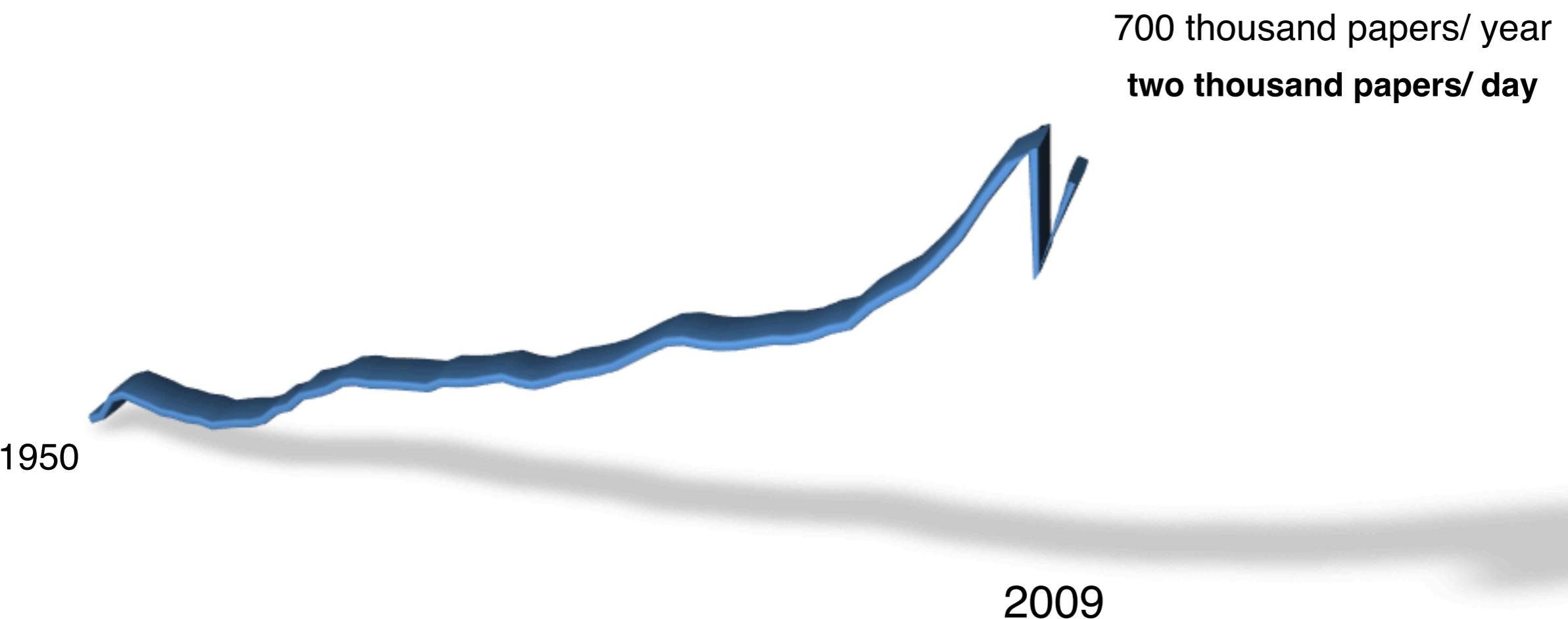
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Robert Hoffmann, hoffmann@cbio.mskcc.org



- Text-mining
- Semantic Web
- Collaborative publishing (~Wiki)

Collaborative publishing

- Community driven
- Integration of facts and views from different sources
- Self-organizing



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Nature Genetics **40**, 1047 - 1051 (2008)
Published online: 27 August 2008 | doi:10.1038/ng.f.217

A wiki for the life sciences where authorship matters

Robert Hoffmann¹

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wikigenes

evolutionary knowledge

Hoffmann, R. A wiki for the life sciences where authorship matters. *Nature Genetics*.

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Gene Review

PTEN - phosphatase and tensin homolog

Synonyms: 10q23del, BZS, DEC, MGC11227, MHAM, ...

[Maehama, T.](#), et al., [Gu, J.](#), et al., [Waite, K.A.](#), et al., [Radu, A.](#), et al., [Howe, J.R.](#), et al., et al.

Homo sapiens

[edit this page]

Disease relevance of PTEN

- Mutations that impair PTEN function result in a marked increase in cellular levels of PIP3 and constitutive activation of Akt survival signaling pathways, leading to inhibition of apoptosis, hyperplasia, and tumor formation [1].
- Frequent somatic mutations in PTEN and TP53 are mutually exclusive in the stroma of breast carcinomas [2].
- Hereditary mutation of PTEN causes tumor-susceptibility diseases such as Cowden disease [3].
- The PTEN and TSC2 tumor suppressors inhibit mammalian target of rapamycin (mTOR) signaling and are defective in distinct hamartoma syndromes [4].
- In colon cancer cells, PTEN stimulates Cdx-2 protein expression and the transcriptional activity of the Cdx-2 promoter [5].

Psychiatry related information on PTEN

- Interestingly, germline mutations in PTEN have also been found in about 50% of a related but distinct disorder, Bannayan-Ruvalcaba-Riley syndrome (BRR), which is characterised by neonatal-onset macrocephaly, mental retardation, Hashimoto's thyroiditis, lipomatosis, haemangiomas, hamartomatous polyps, and pigmented macules of the glans penis [6].
- Nothing is known regarding the pattern of PTEN expression during human development [7].
- Activation of Akt/PKB, increased phosphorylation of Akt substrates and loss and altered distribution of Akt and PTEN are features of Alzheimer's disease pathology [8].

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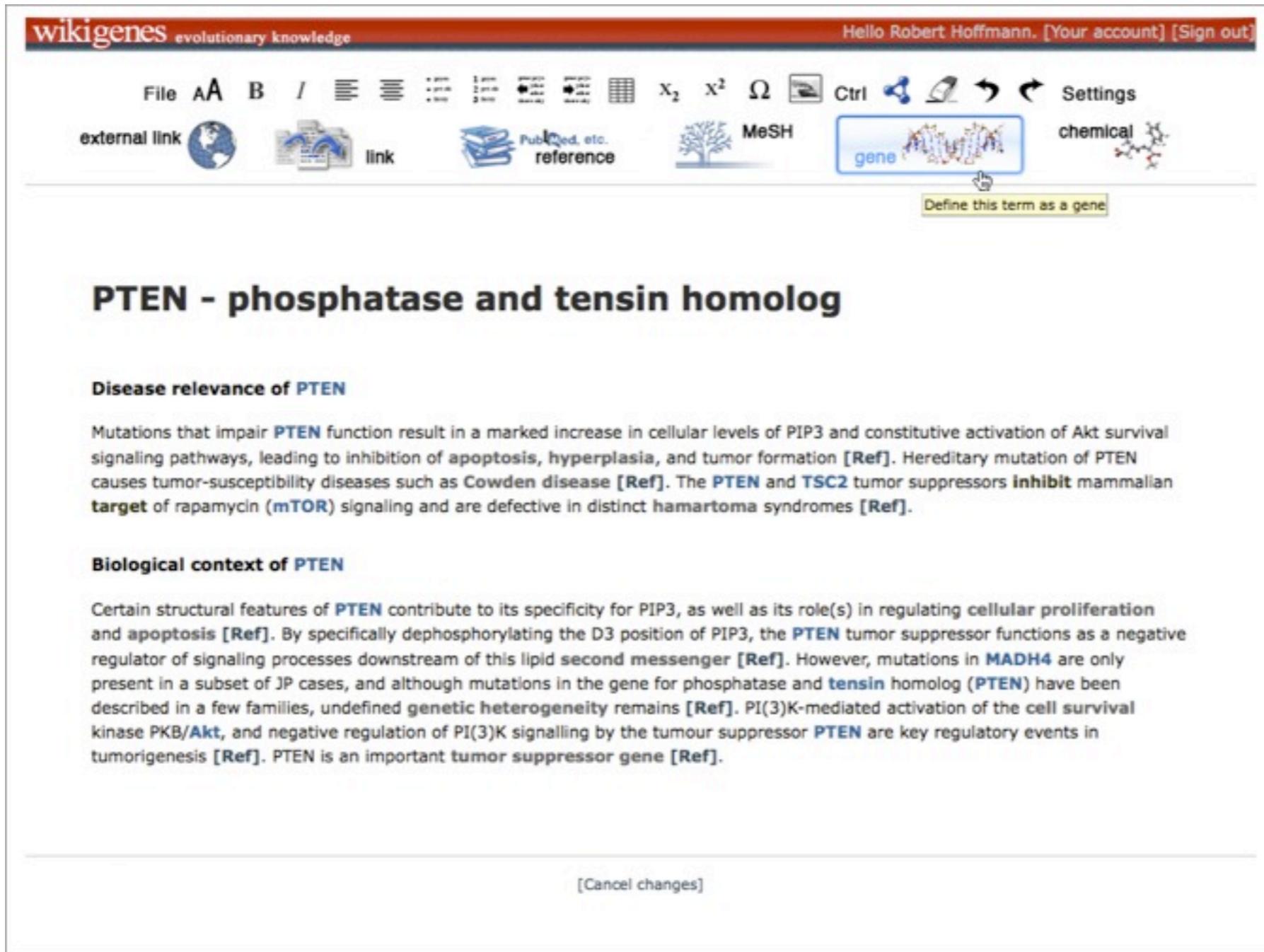
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Yale

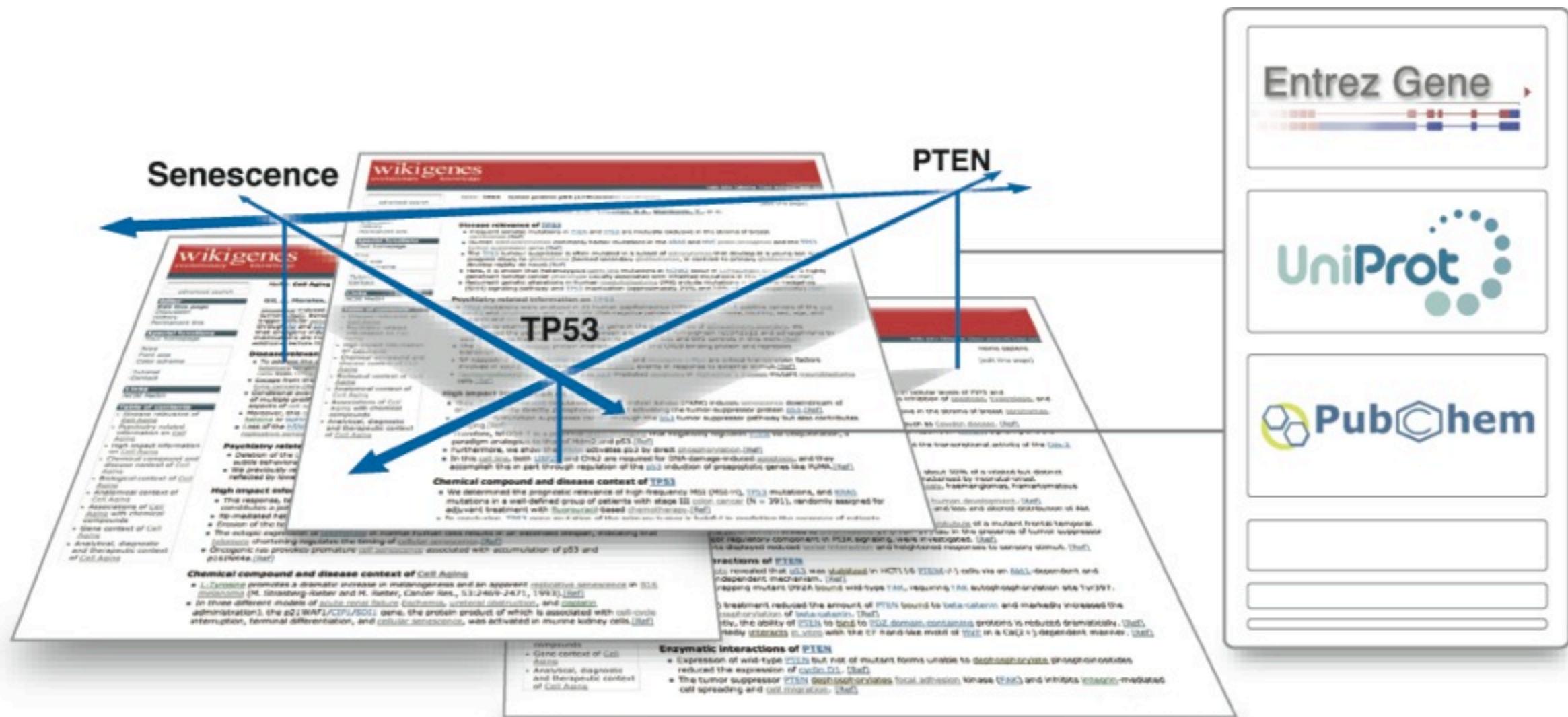
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Open access articles
80.000 genes and proteins
32.000 chemical compounds
12.000 diseases, biomedical concepts



The screenshot shows the WikiGenes interface for the gene **PTEN**. The top navigation bar includes links for File, AA, B, I, various document icons, Ctrl, Settings, and a user account dropdown. Below the navigation are links for external link, link, PubMed etc. reference, MeSH, and gene (which is highlighted with a blue border). A tooltip says "Define this term as a gene". The main content area features the title **PTEN - phosphatase and tensin homolog**. Under the heading **Disease relevance of PTEN**, it states: "Mutations that impair **PTEN** function result in a marked increase in cellular levels of PIP3 and constitutive activation of Akt survival signaling pathways, leading to inhibition of apoptosis, hyperplasia, and tumor formation [Ref]. Hereditary mutation of **PTEN** causes tumor-susceptibility diseases such as Cowden disease [Ref]. The **PTEN** and **TSC2** tumor suppressors inhibit mammalian target of rapamycin (**mTOR**) signaling and are defective in distinct hamartoma syndromes [Ref].". Under the heading **Biological context of PTEN**, it describes the gene's role in regulating cellular proliferation and apoptosis [Ref], its function as a negative regulator of PI(3)K-mediated activation of the cell survival kinase PKB/Akt, and its key regulatory events in tumorigenesis [Ref]. At the bottom of the page is a "[Cancel changes]" button.





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Provenance / Authorship

Charles Darwin – Wikipedia, the free encyclopedia

Charles Darwin – Wikipedia, the free encyclopedia

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Charles Darwin

From Wikipedia, the free encyclopedia

Charles Robert Darwin FRS (12 February 1809 – 19 April 1882) was an English naturalist^[1] who established that all species of life have descended over time from common ancestry, and proposed the scientific theory that this branching pattern of evolution resulted from a process that he called natural selection. He published his theory with compelling evidence for evolution in his 1859 book *On the Origin of Species*.^{[1][2]} The scientific community and much of the general public came to accept evolution as a fact in his lifetime,^[3] but it was not until the emergence of the modern evolutionary synthesis from the 1930s to the 1950s that a broad consensus developed that natural selection was the basic mechanism of evolution.^[4] In modified form, Darwin's scientific discovery is the unifying theory of the life sciences, explaining the diversity of life.^{[5][6]}

Darwin's early interest in nature led him to neglect his medical education at the University of Edinburgh; instead, he helped to investigate marine invertebrates. Studies at the University of Cambridge encouraged his passion for natural science.^[7] His five-year voyage on HMS *Beagle* established him as an eminent geologist whose observations and theories supported Charles Lyell's uniformitarian ideas, and publication of his journal of the voyage made him famous as a popular author.^[8]

Puzzled by the geographical distribution of wildlife and fossils he collected on the voyage, Darwin investigated the transmutation of species and conceived his theory of natural selection in 1838.^[9] Although he discussed his ideas with several naturalists, he needed time for extensive research and his geological work had priority.^[10] He was writing up his theory in 1858 when Alfred Russel Wallace sent him an essay which described the same idea, prompting immediate joint publication of both of their theories.^[11] Darwin's work established evolutionary descent with modification as the dominant scientific explanation of diversification in nature.^[3] In 1871, he examined human evolution and sexual selection in *The Descent of Man, and Selection in Relation to Sex*, followed by *The Expression of the Emotions in Man and Animals*. His research on plants was published in a series of books, and in his final book, he examined earthworms and their effect on soil.^[12]

In recognition of Darwin's pre-eminence as a scientist, he was one of only five nineteenth-century non-royal personages from the United Kingdom to be honoured by a state funeral,^[13] and was buried in Westminster Abbey, close to John Herschel and Isaac Newton.^[14]

Responses to the publication

The book aroused international interest, with less controversy than had greeted the popular *Vestiges of Creation*.^[15] Though Darwin's illness kept him away from the public debates, he eagerly scrutinised the scientific response, commenting on press cuttings, reviews, articles, satires and caricatures, and corresponded on it with colleagues worldwide.^[16] Darwin had only said "Light will be thrown on the origin of man",^[17] but the first review claimed it made a creed of the "men from monkeys" idea from *Vestiges*.^[18] Amongst early favourable responses, Huxley's reviews swiped at Richard Owen, leader of the scientific establishment Huxley was trying to overthrow.^[19] In April, Owen's review attacked Darwin's friends and confidently dismissed his ideas, angering Darwin.^[20] In May, Owen and others began to

Charles Darwin – Wikipedia, the free encyclopedia

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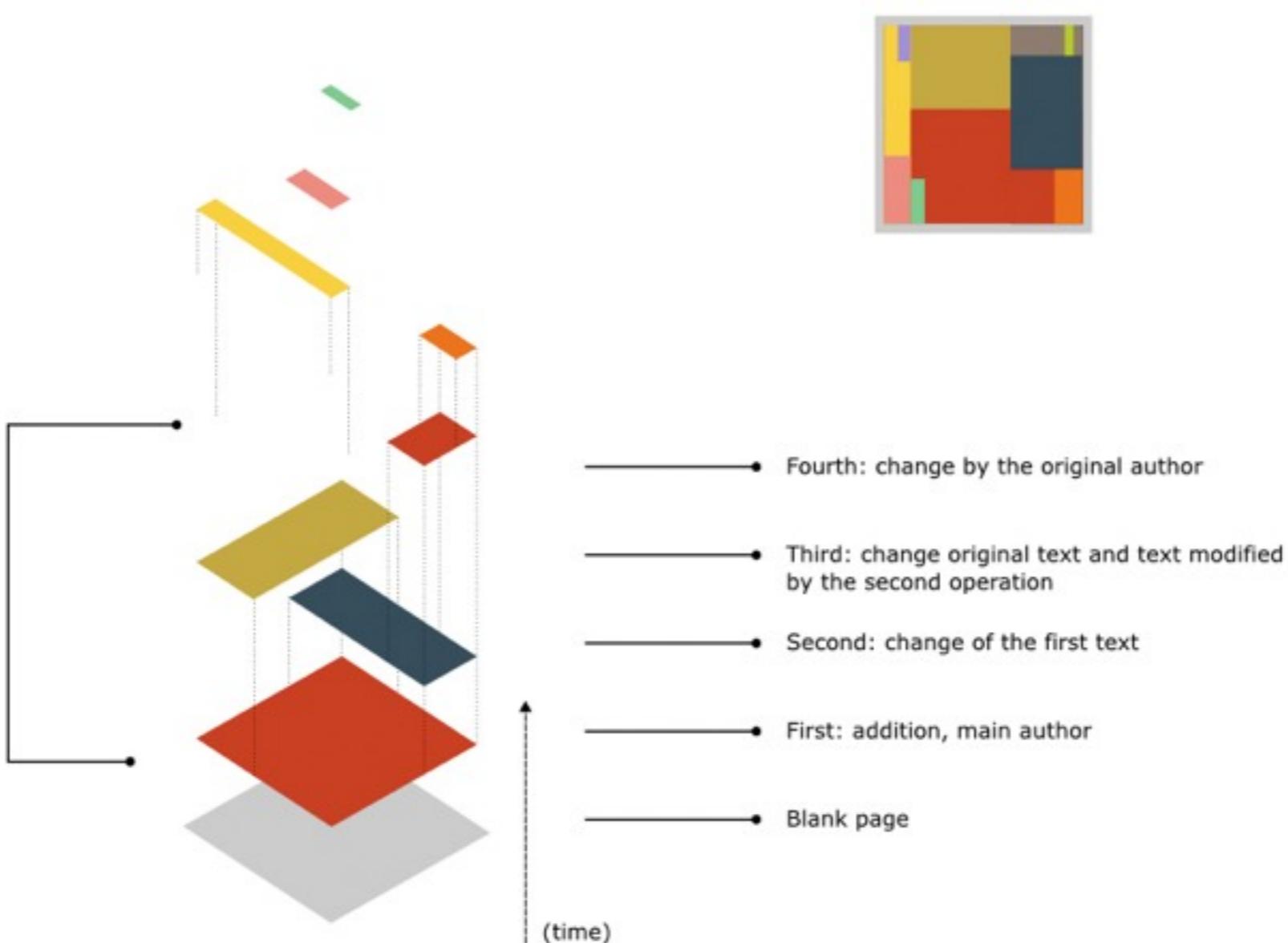
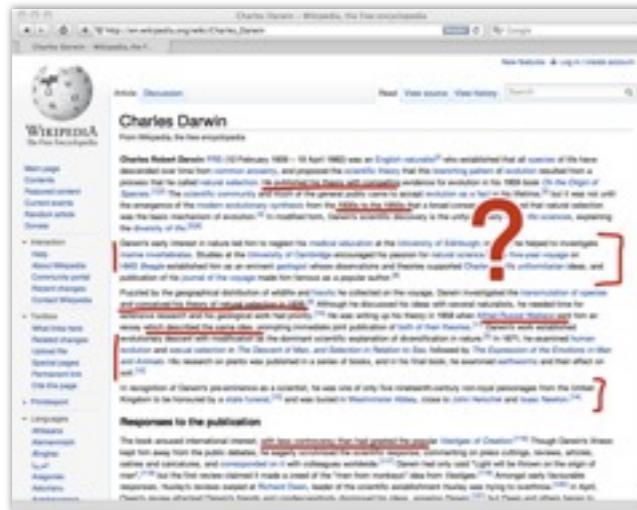
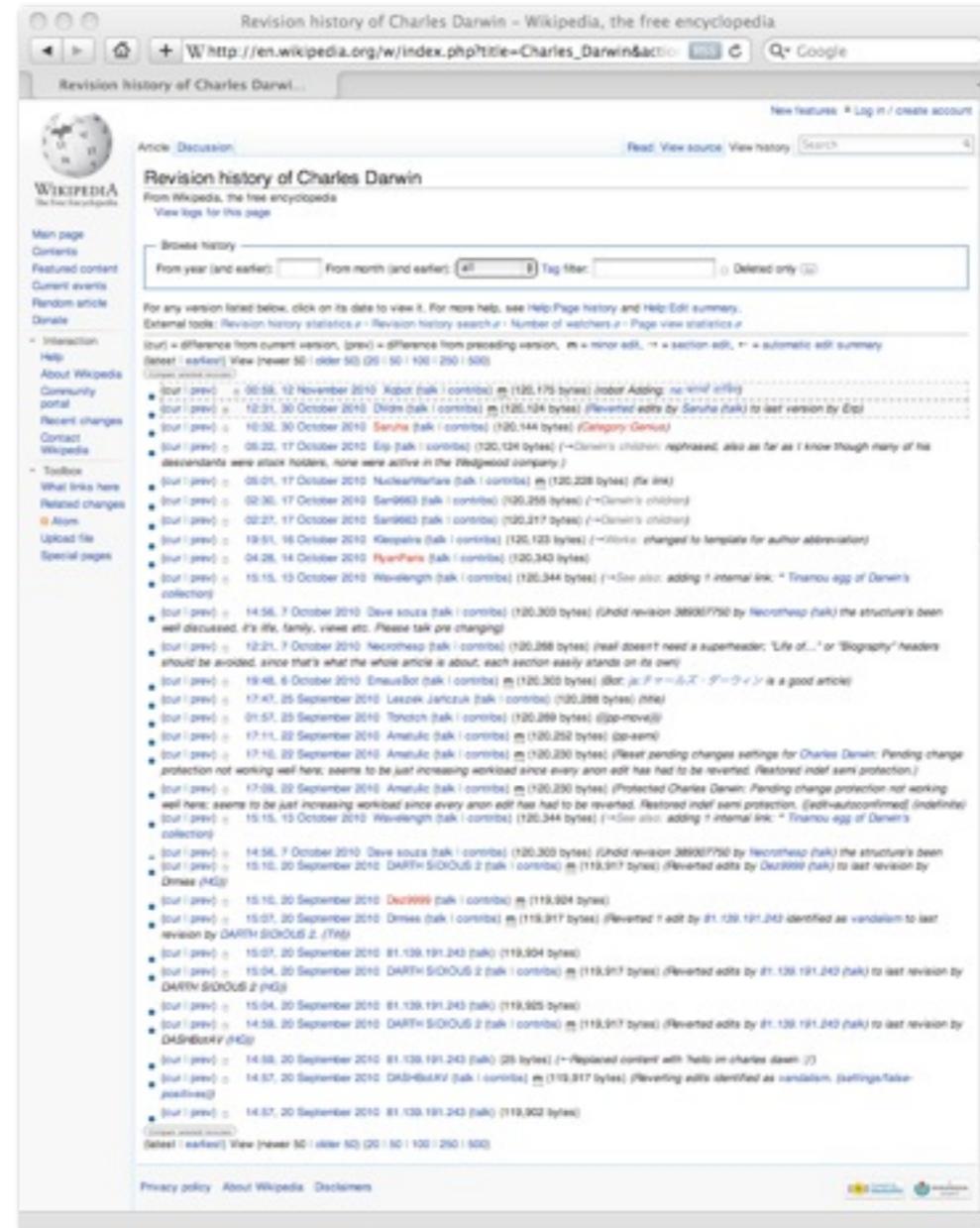


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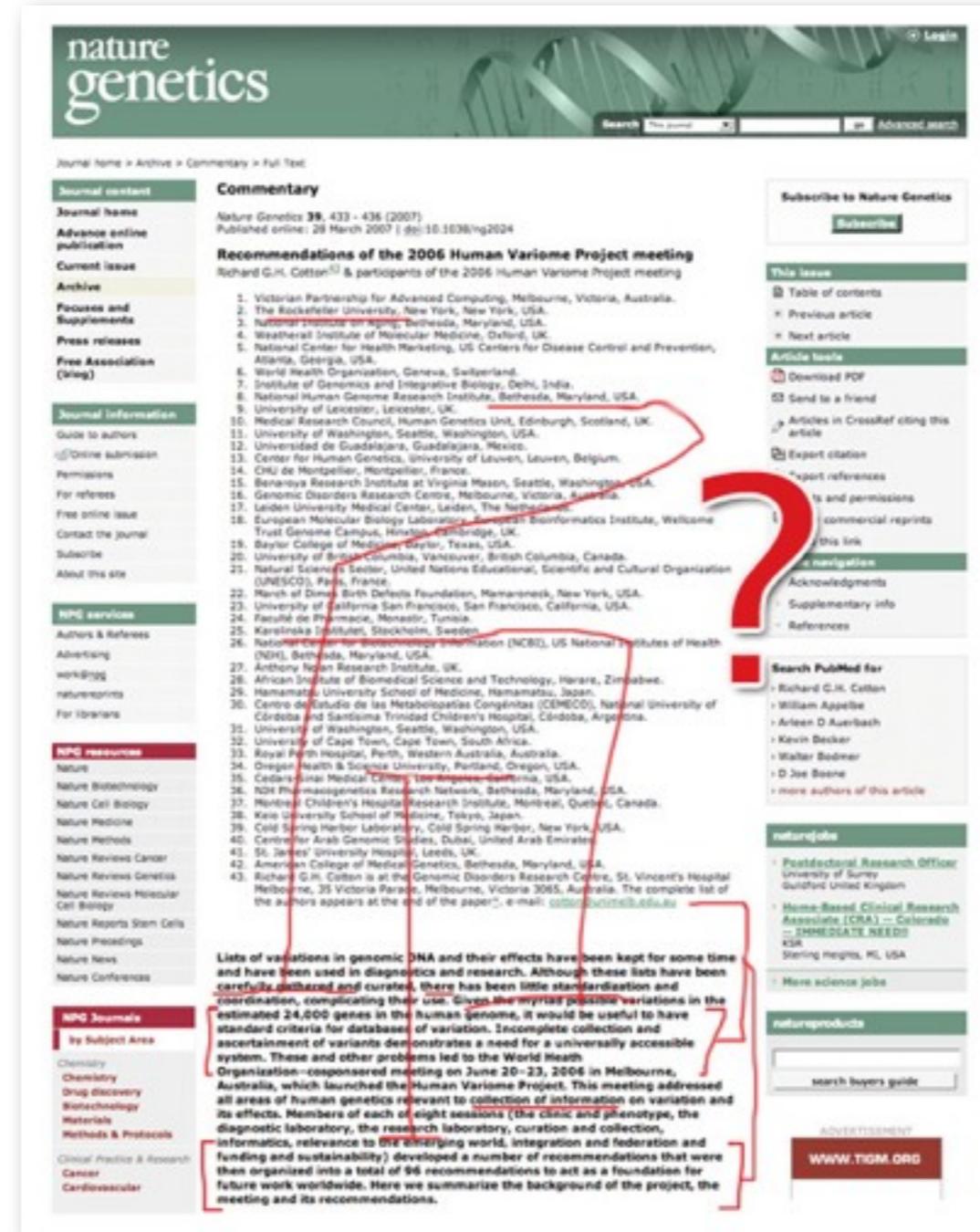
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The screenshot shows the Wikipedia article for Charles Darwin. A red box highlights a section of text where the word "Darwin" is underlined, indicating it has been edited by a user named "Darwin". Another red box highlights a note at the bottom of the page: "Proposed by the geographical distribution of wildlife and fossils he collected on the voyage, Darwin investigated the interconnectedness of species and formulated his theory of natural selection.¹⁰² Although he discussed his ideas with several naturalists, he needed time for extensive research and to geological work first手稿. He was writing up his theory in 1858 when Alfred Russel Wallace sent him an essay, which described the same idea, to the Linnean Society. Darwin and Wallace published their joint paper at the same time. Darwin's work was eventually published in 1859 in his book On the Origin of Species, which presented the evidence for the common ancestry of all life and the process of evolution by natural selection. In 1871, he published a follow-up book, The Descent of Man, and Selection in Relation to Sex, followed by The Expression of the Emotions in Man and Animals. His research on plants was published in a series of books, and in the final issue, he examined earthworms and their effect on soil.¹⁰³"



The screenshot shows the revision history for the Charles Darwin Wikipedia page. A red box highlights the first few revisions, showing edits made by users like "Kobet" and "Darwin". The interface includes a sidebar with navigation links and a footer with standard Wikipedia links.



The screenshot shows the *nature genetics* journal website. The main content is a **Commentary** titled "Recommendations of the 2006 Human Variome Project meeting" by Richard G.H. Cotton and participants. The commentary discusses the need for a standardized database of human variants. A large red question mark is overlaid on the page, pointing to the list of recommendations at the bottom of the article.

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Clinical Practice & Research

- Cancer
- Cardiovascular

Commentary

Recommendations of the 2006 Human Variome Project meeting (Richard G.H. Cotton) & participants of the 2006 Human Variome Project meeting

1. Victorian Partnership for Advanced Computing, Melbourne, Victoria, Australia.
 2. The Rockefeller University, New York, New York, USA.
 3. National Institutes of Aging, Bethesda, Maryland, USA.
 4. Weatherhead Institute of Molecular Medicine, Oxford, UK.
 5. National Committee for Health Marketing, US Centers for Disease Control and Prevention, Atlanta, Georgia, USA.
 6. World Health Organization, Geneva, Switzerland.
 7. Institute of Genomics and Integrative Biology, Delhi, India.
 8. National Human Genome Research Institute, Bethesda, Maryland, USA.
 9. University of Leicester, Leicester, UK.
 10. Medical Research Council, Human Genetics Unit, Edinburgh, Scotland, UK.
 11. University of Washington, Seattle, Washington, USA.
 12. Universidad de Guadalajara, Guadalajara, Mexico.
 13. Center for Human Genetics, University of Leuven, Leuven, Belgium.
 14. CHU de Montpellier, Montpellier, France.
 15. Benaroya Research Institute at Virginia Mason, Seattle, Washington, USA.
 16. Genomic Disorders Research Centre, Heidelberg, Victoria, Australia.
 17. Leiden University Medical Center, Leiden, The Netherlands.
 18. European Molecular Biology Laboratory, European Bioinformatics Institute, Wellcome Trust Genome Campus, Hinxton, Cambridge, UK.
 19. Baylor College of Medicine, Houston, Texas, USA.
 20. University of British Columbia, Vancouver, British Columbia, Canada.
 21. Natural Sciences Sector, United Nations Educational, Scientific and Cultural Organization (UNESCO), Paris, France.
 22. March of Dimes Birth Defects Foundation, White Plains, New York, USA.
 23. University of California San Francisco, San Francisco, California, USA.
 24. Faculté de Pharmacie, Monastir, Tunisia.
 25. Karolinska Institutet, Stockholm, Sweden.
 26. National Center for Biotechnology Information (NCBI), US National Institutes of Health (NIH), Bethesda, Maryland, USA.
 27. Anthony Nolan Research Institute, UK.
 28. African Institute of Biomedical Sciences and Technology, Harare, Zimbabwe.
 29. Hamamatsu University School of Medicine, Hamamatsu, Japan.
 30. Centro de Estudios de las Metabolicas Congénitas (CEMECO), National University of Córdoba and Santiago Trinidad Children's Hospital, Córdoba, Argentina.
 31. University of Washington, Seattle, Washington, USA.
 32. University of Cape Town, Cape Town, South Africa.
 33. Royal Perth Hospital, Perth, Western Australia, Australia.
 34. Oregon Health & Science University, Portland, Oregon, USA.
 35. Cedars-Sinai Medical Center, Los Angeles, California, USA.
 36. NIH Pharmacogenetics Research Network, Bethesda, Maryland, USA.
 37. Montreal Children's Hospital Research Institute, Montreal, Quebec, Canada.
 38. Keio University School of Medicine, Tokyo, Japan.
 39. Cold Spring Harbor Laboratory, Cold Spring Harbor, New York, USA.
 40. Centre for Arab Genetic Studies, Dubai, United Arab Emirates.
 41. St. James' University Hospital, Leeds, UK.
 42. American College of Medical Genetics, Bethesda, Maryland, USA.
 43. Richard G.H. Cotton is at the Genetic Disorders Research Centre, St. Vincent's Hospital Melbourne, 35 Victoria Parade, Melbourne, Victoria 3065, Australia. The complete list of the authors appears at the end of the paper*. e-mail: cotton@unimelb.edu.au

Lists of variations in genomic DNA and their effects have been kept for some time and have been used in diagnostics and research. Although these lists have been carefully gathered and curated, there has been little standardization and coordination, complicating their use. Given the myriad possible variations in the estimated 24,000 genes in the human genome, it would be useful to have standard criteria for databases of variation. Incomplete collection and ascertainment of variants demonstrates a need for a universally accessible system. These and other problems led to the World Health Organization-cosponsored meeting on June 20–23, 2006 in Melbourne, Australia, which launched the Human Variome Project. This meeting addressed all areas of human genetics relevant to collection of information on variation and its effects. Members of each of eight sessions (the clinic and phenotype, the diagnostic laboratory, the research laboratory, curation and collection, informatics, relevance to the emerging world, integration and federation and funding and sustainability) developed a number of recommendations that were then organized into a total of 98 recommendations to act as a foundation for future work worldwide. Here we summarize the background of the project, the meeting and its recommendations.

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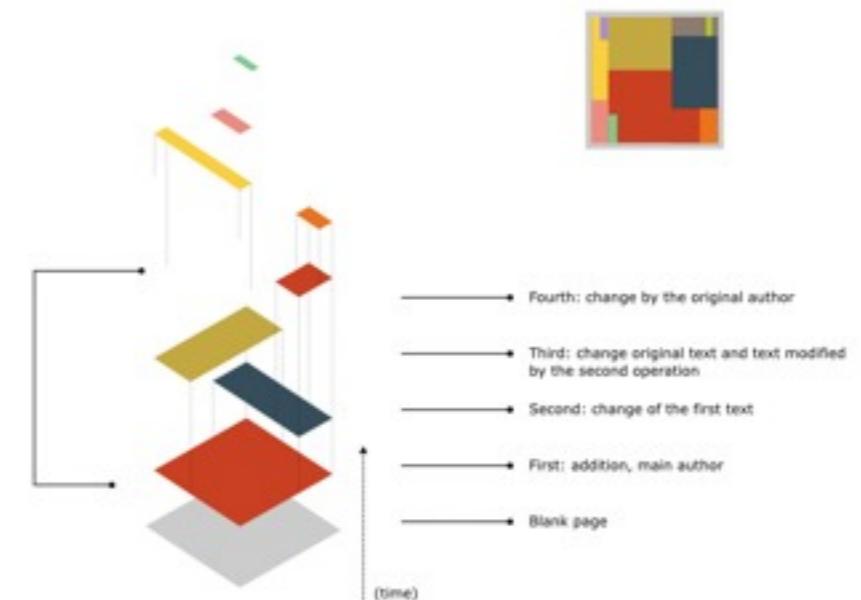
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PTEN - phosphatase and tensin homolog

Disease relevance of PTEN

Mutations that impair **PTEN** function result in a marked increase in cellular levels of PIP3 and constitutive activation of Akt survival signaling pathways, leading to inhibition of apoptosis, hyperplasia, and tumor formation [Ref]. Hereditary mutation of PTEN causes tumor-susceptibility diseases such as Cowden disease [Ref]. The **PTEN** and **TSC2** tumor suppressors inhibit mammalian target of rapamycin (**mTOR**) signaling and are defective in distinct hamartoma syndromes [Ref].

Biological context of PTEN

Certain structural features of **PTEN** contribute to its specificity for PIP3, as well as its role(s) in regulating cellular proliferation and apoptosis [Ref]. Specifically dephosphorylating the D3 position of PIP3, the **PTEN** tumor suppressor functions as a negative regulator of signaling processes downstream of this lipid second messenger [Ref]. However, mutations in **MADH4** are only present in a subset of JP cases, and although mutations in the gene for phosphatase and **tensin** homolog (**PTEN**) have been described in a few families, undefined genetic heterogeneity remains [Ref]. PI(3)K-mediated activation of the cell survival kinase **PKB/Akt**, and negative regulation of PI(3)K signalling by the tumour suppressor **PTEN** are key regulatory events in tumorigenesis [Ref]. **PTEN** is an important tumor suppressor gene [Ref].

In colon cancer cells, **PTEN** stimulates **Cdx-2** protein expression and the transcriptional activity of the **Cdx-2** promoter [Ref].

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Hoffmann, R. A wiki for the life sciences where authorship matters. *Nature Genetics*.

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PTEN - phosphatase and tensin homolog

Kim, S.
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contribute to its specificity for PIP3, as well as its role(s) in apoptosis [1]. In colon cancer cells, PTEN stimulates Cdx-2 protein expression and the transcriptional activity of the Cdx-2 promoter [2]. By specifically dephosphorylating the D3 position of PIP3, the PTEN tumor suppressor functions as a negative regulator of signaling processes downstream of this lipid second messenger [1]. PI(3)K-mediated activation of the cell survival kinase PKB/Akt, and negative regulation of PI(3)K signalling by the tumour suppressor PTEN are key regulatory events in tumorigenesis [3]. PTEN is an important tumor suppressor gene [4].

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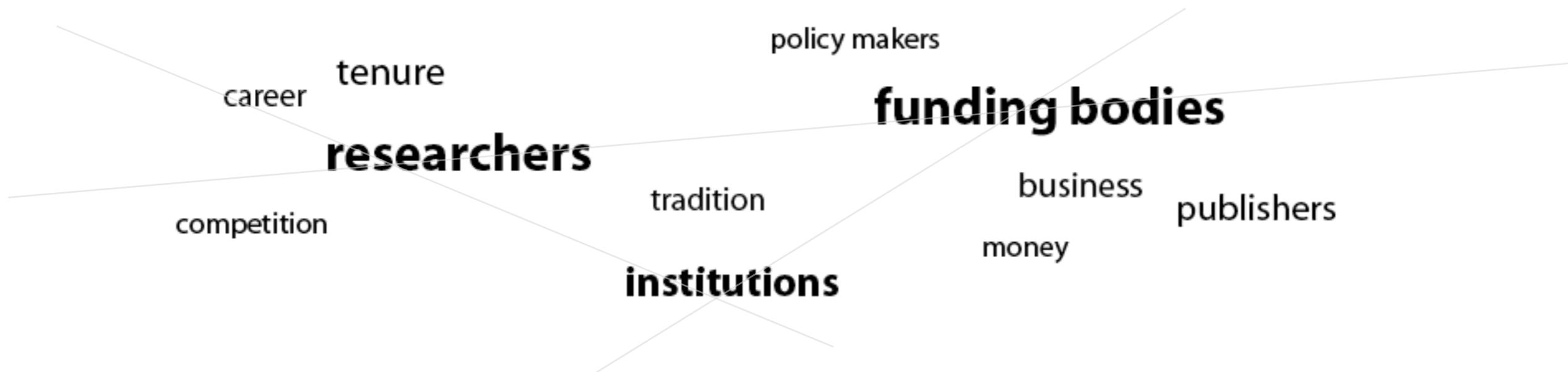
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- Democratic decision making based on reputation system
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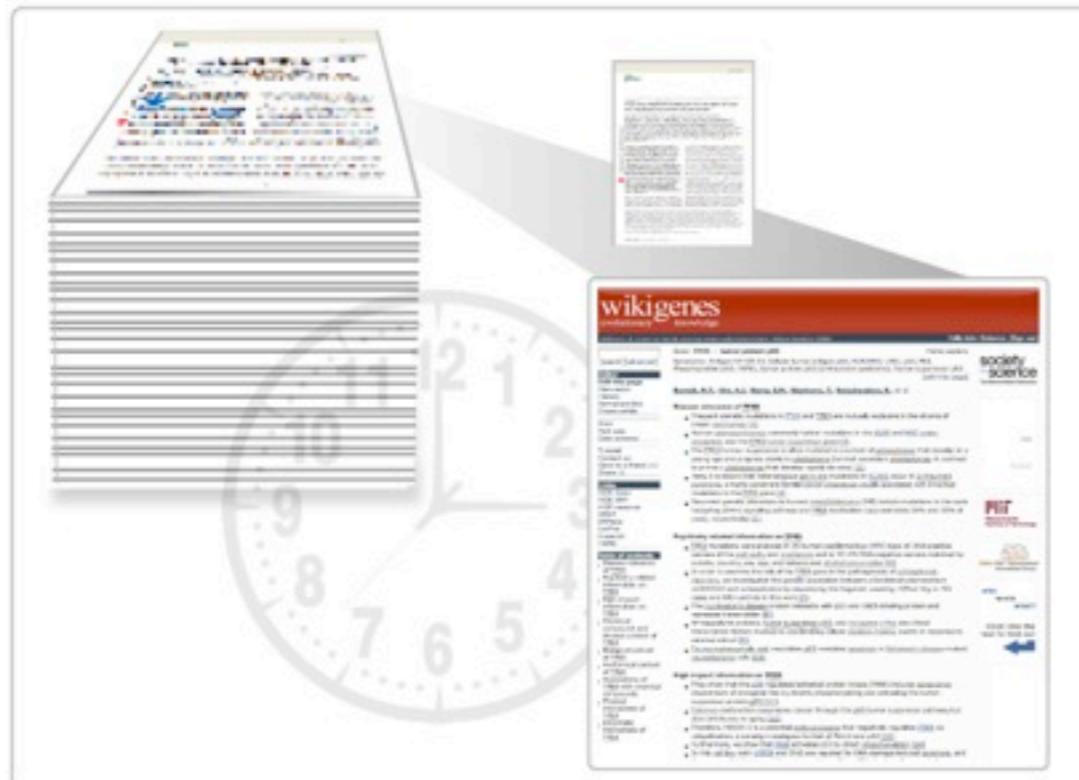
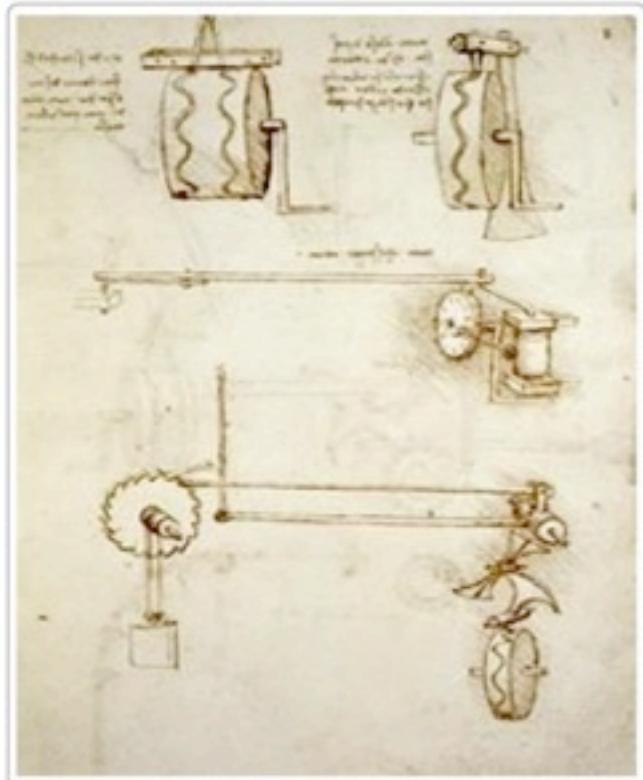


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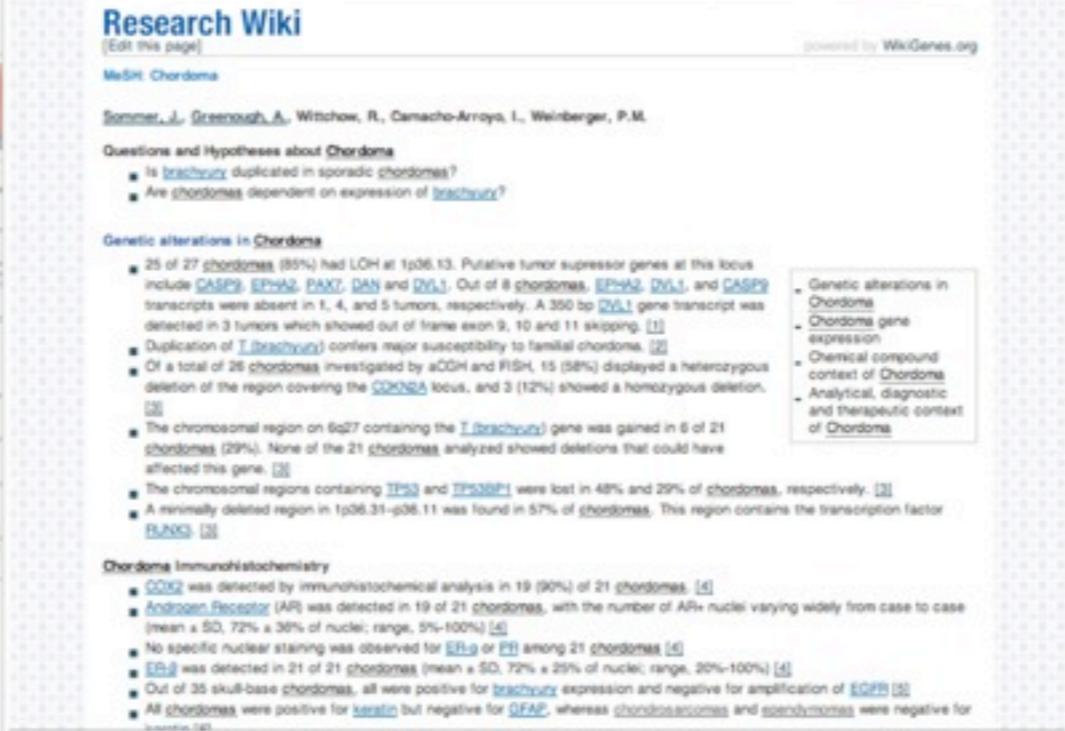
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2. Communities as drivers



The Chordoma Foundation homepage features a large photo of a young boy with a chordoma. Navigation links include About Us, About Chordoma, Research, Treatment, News, Get Involved, and Resources. A "Donate Now" button is prominently displayed.



The Research Wiki page for Chordoma is powered by WikiGenes.org. It includes a sidebar with links to Contact us, Search, and various research resources. The main content discusses Questions and Hypotheses about Chordoma, Genetic alterations in Chordoma, and Chordoma Immunohistochemistry. A sidebar on the right provides information on Genetic alterations in Chordoma, Chordoma gene expression, Chemical compound context of Chordoma, Analytical, diagnostic and therapeutic context of Chordoma, and Psychiatry related information on T.



The Wikigenes page for Chordoma includes sections on Chordoma, Chordoma gene expression, Chemical compound context of Chordoma, and Analytical, diagnostic and therapeutic context of Chordoma. It also features a sidebar with links to Contact us, Search, and various research resources.

3. Collaborations with publishers

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Wikigenes joins our experiment in collaborative authoring of standards

Now there is another way to make your contribution to the standards document "Principles for the post-GWAS functional characterisation of risk loci" described in the [previous post](#), automatically becoming a co-author in the process. Wikigenes creator Robert Hoffmann has set up a splash page, introducing the paper and a link to the [editable version](#) at Wikigenes.

We do not anticipate problems in assigning authorship and author roles since it is obvious whether you have contributed conceptual input or corrected punctuation. Corresponding author Ian Mills and editor Myles Axton will check and if necessary edit author names and roles for the final version to be submitted for publication after December 20th 2010.

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Myles Axton, Editor, *Nature Genetics*

A dense word cloud composed of numerous names in different colors, including purple, yellow, orange, and grey. The names are arranged in a non-linear, overlapping pattern across the page. Some names are accompanied by small, semi-transparent text labels below them, which appear to be additional names or titles. The overall effect is a visual representation of a large, diverse group of individuals.

Names visible in the word cloud include:

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- Michael Kleen
- Juan C. Cigudosa
- Dietrich Rebholz
- Peter Schuster
- Ernst Hafen
- Olaf Kubler
- Marta Ramirez
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- Nikolaus Wick
- Robert Badgett
- Timo Hannay
- Hanah Margalit
- Jose M. Fernandez
- Michael Tress
- Rolf Apweiler
- Florian Leitner
- Gianni Cesareni
- Norbert Gretz
- Marlin Nowak
- Kathy Kwon
- Les Grivell
- Urban Liebel
- Arabella Meixner
- Danny Weitzner
- Elspeth Bradford
- Branco Weiss
- Ben Gross
- Luis Rico
- Anton Beyer
- Gary Bader
- Chris Sander
- Alfonso Valencia
- Thomas Pfeiffer
- Claire Bird
- Helga Nowotny
- Dick Cotton
- Josh Sommer
- David Shotton
- Christian Forst
- Gerd Folkers
- Clem Starnon
- Florence Serraz
- Maria Perisco
- Rohan D. Teasdale
- Luciano Milanesi
- Morten Lindow
- Hiroaki Kitano
- Lukas Huber