Towards Collaborative Publishing in Science

Decentralized Information Group - Cambridge, September 2008.

Robert Hoffmann

roberth@MIT.edu

‘Conventional’ publications

- Stone, paper, silicon, …
- Closed number of authors
- Date of publication
- Final version
- ‘static’

Scientific discourse

- Over a series of static publications

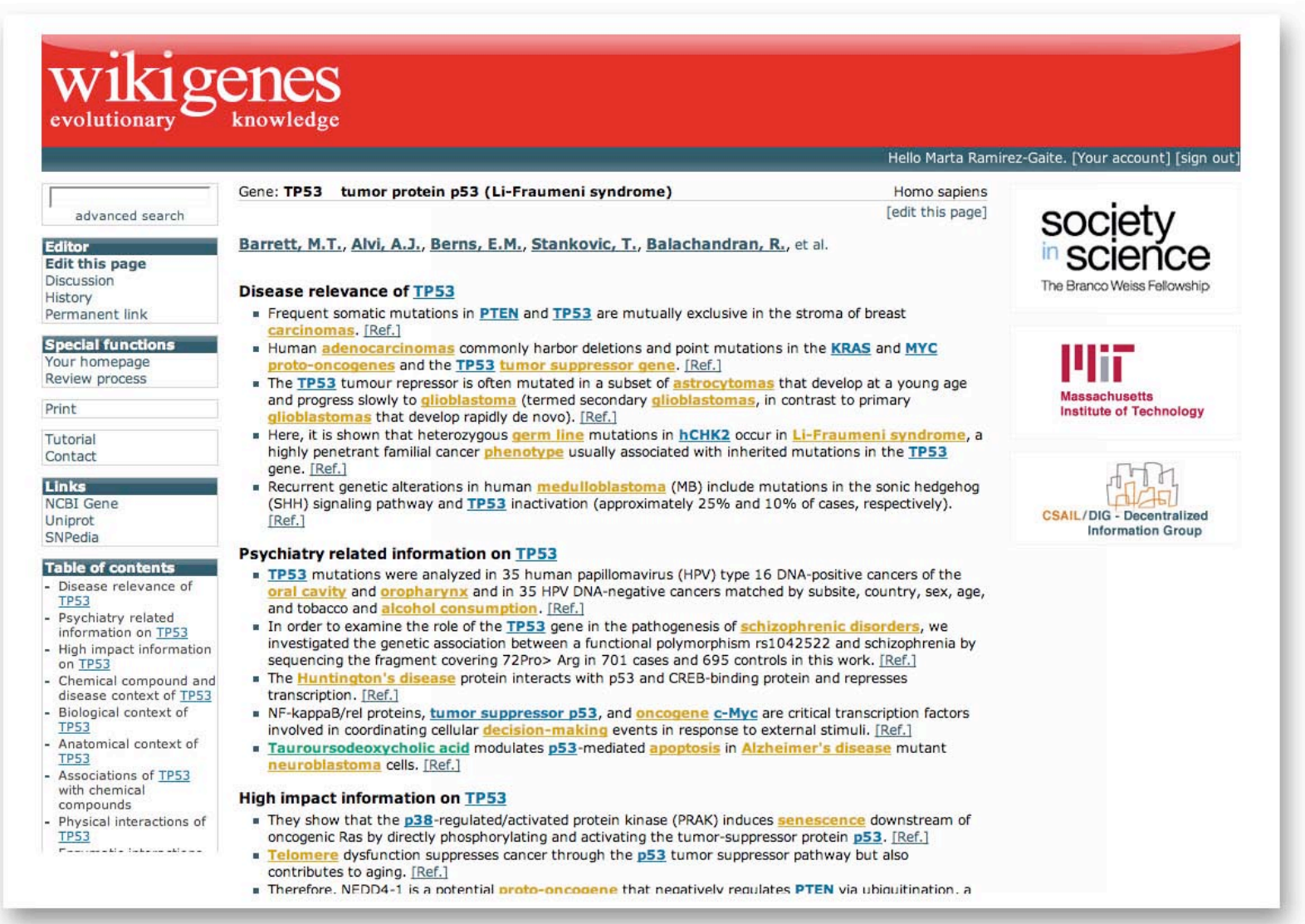
roberth@MIT.edu

‘Dynamic’ publications

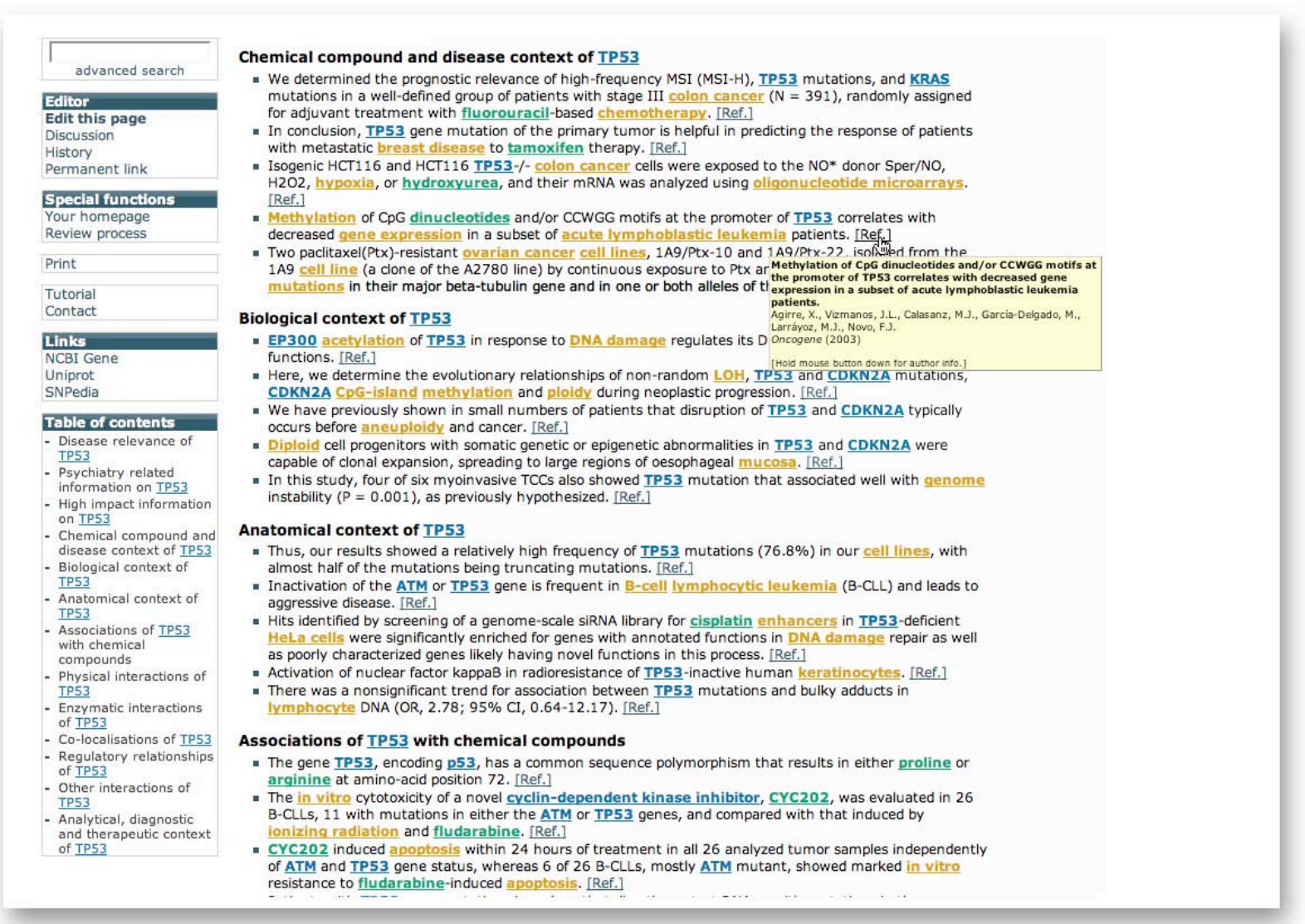
- [Open number of authors]
- Versions but no final date of publication
- Emphasis may shift over time
- Evolve with the subject
- Potentially always up-to-date
- Incorporate scientific discourse

roberth@MIT.edu

Towards Collaborative Publishing in Science



Towards Collaborative Publishing in Science



advanced search

Editor
Edit this page
Discussion
History
Permanent link

Special functions
Your homepage
Review process

Print

Tutorial
Contact

Links
NCBI Gene
Uniprot
SNPedia

Table of contents
- Disease relevance of TP53
- Psychiatry related information on TP53
- High impact information on TP53
- Chemical compound and disease context of TP53
- Biological context of TP53
- Anatomical context of TP53
- Associations of TP53 with chemical compounds
- Physical interactions of TP53
- Enzymatic interactions of TP53
- Co-localisations of TP53
- Regulatory relationships of TP53
- Other interactions of TP53
- Analytical, diagnostic and therapeutic context of TP53

Chemical compound and disease context of TP53

- We determined the prognostic relevance of high-frequency MSI (MSI-H), TP53 mutations, and KRAS mutations in a well-defined group of patients with stage III colon cancer (N = 391), randomly assigned for adjuvant treatment with fluorouracil-based chemotherapy. [Ref.]
- In conclusion, TP53 gene mutation of the primary tumor is helpful in predicting the response of patients with metastatic breast disease to tamoxifen therapy. [Ref.]
- Isogenic HCT116 and HCT116 TP53-/- colon cancer cells were exposed to the NO* donor Sper/NO, H2O2, hypoxia, or hydroxyurea, and their mRNA was analyzed using oligonucleotide microarrays. [Ref.]
- Methylation of CpG dinucleotides and/or CCWGG motifs at the promoter of TP53 correlates with decreased gene expression in a subset of acute lymphoblastic leukemia patients. [Ref.]
- Two paclitaxel(Ptx)-resistant ovarian cancer cell lines, 1A9/Ptx-10 and 1A9/Ptx-22, isolated from the 1A9 cell line (a clone of the A2780 line) by continuous exposure to Ptx ar… mutations in their major beta-tubulin gene and in one or both alleles of t…

Methylation of CpG dinucleotides and/or CCWGG motifs at the promoter of TP53 correlates with decreased gene expression in a subset of acute lymphoblastic leukemia patients.
Agirre, X., Vizmanos, J.L., Calasanz, M.J., García-Delgado, M., Larráyoz, M.J., Novo, F.J.
Oncogene (2003)
[Hold mouse button down for author info.]

Biological context of TP53

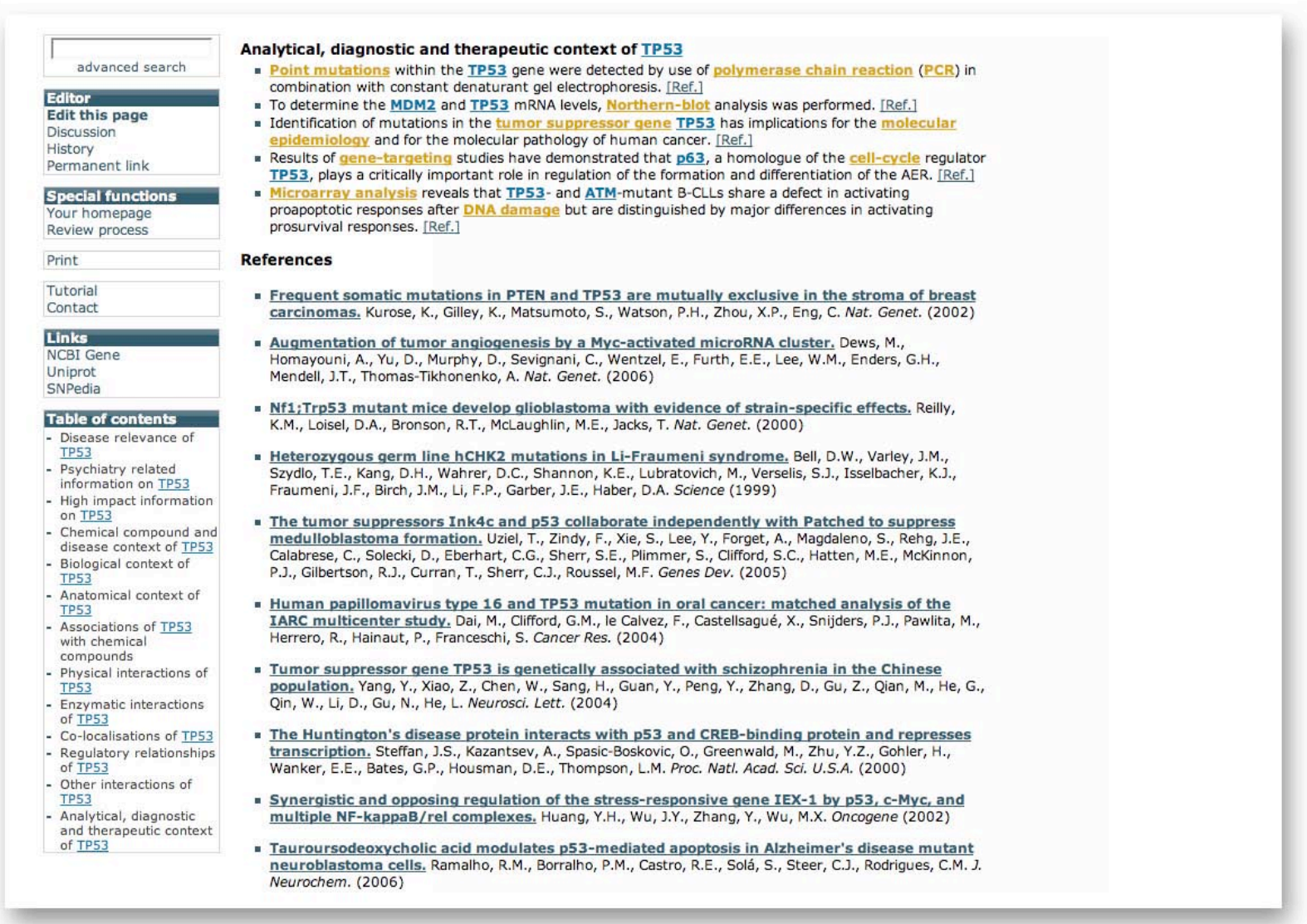
- EP300 acetylation of TP53 in response to DNA damage regulates its D… functions. [Ref.]
- Here, we determine the evolutionary relationships of non-random LOH, TP53 and CDKN2A mutations, CDKN2A CpG-island methylation and ploidy during neoplastic progression. [Ref.]
- We have previously shown in small numbers of patients that disruption of TP53 and CDKN2A typically occurs before aneuploidy and cancer. [Ref.]
- Diploid cell progenitors with somatic genetic or epigenetic abnormalities in TP53 and CDKN2A were capable of clonal expansion, spreading to large regions of oesophageal mucosa. [Ref.]
- In this study, four of six myoinvasive TCCs also showed TP53 mutation that associated well with genome instability (P = 0.001), as previously hypothesized. [Ref.]

Anatomical context of TP53

- Thus, our results showed a relatively high frequency of TP53 mutations (76.8%) in our cell lines, with almost half of the mutations being truncating mutations. [Ref.]
- Inactivation of the ATM or TP53 gene is frequent in B-cell lymphocytic leukemia (B-CLL) and leads to aggressive disease. [Ref.]
- Hits identified by screening of a genome-scale siRNA library for cisplatin enhancers in TP53-deficient HeLa cells were significantly enriched for genes with annotated functions in DNA damage repair as well as poorly characterized genes likely having novel functions in this process. [Ref.]
- Activation of nuclear factor kappaB in radioresistance of TP53-inactive human keratinocytes. [Ref.]
- There was a nonsignificant trend for association between TP53 mutations and bulky adducts in lymphocyte DNA (OR, 2.78; 95% CI, 0.64-12.17). [Ref.]

Associations of TP53 with chemical compounds

- The gene TP53, encoding p53, has a common sequence polymorphism that results in either proline or arginine at amino-acid position 72. [Ref.]
- The in vitro cytotoxicity of a novel cyclin-dependent kinase inhibitor, CYC202, was evaluated in 26 B-CLLs, 11 with mutations in either the ATM or TP53 genes, and compared with that induced by ionizing radiation and fludarabine. [Ref.]
- CYC202 induced apoptosis within 24 hours of treatment in all 26 analyzed tumor samples independently of ATM and TP53 gene status, whereas 6 of 26 B-CLLs, mostly ATM mutant, showed marked in vitro resistance to fludarabine-induced apoptosis. [Ref.]



advanced search

Editor
Edit this page
Discussion
History
Permanent link

Special functions
Your homepage
Review process

Print

Tutorial
Contact

Links
NCBI Gene
Uniprot
SNPedia

Table of contents
- Disease relevance of TP53
- Psychiatry related information on TP53
- High impact information on TP53
- Chemical compound and disease context of TP53
- Biological context of TP53
- Anatomical context of TP53
- Associations of TP53 with chemical compounds
- Physical interactions of TP53
- Enzymatic interactions of TP53
- Co-localisations of TP53
- Regulatory relationships of TP53
- Other interactions of TP53
- Analytical, diagnostic and therapeutic context of TP53

Analytical, diagnostic and therapeutic context of TP53

- **Point mutations** within the **TP53** gene were detected by use of **polymerase chain reaction** (**PCR**) in combination with constant denaturant gel electrophoresis. [Ref.]
- To determine the **MDM2** and **TP53** mRNA levels, **Northern-blot** analysis was performed. [Ref.]
- Identification of mutations in the **tumor suppressor gene** **TP53** has implications for the **molecular epidemiology** and for the molecular pathology of human cancer. [Ref.]
- Results of **gene-targeting** studies have demonstrated that **p63**, a homologue of the **cell-cycle** regulator **TP53**, plays a critically important role in regulation of the formation and differentiation of the AER. [Ref.]
- **Microarray analysis** reveals that **TP53**- and **ATM**-mutant B-CLLs share a defect in activating proapoptotic responses after **DNA damage** but are distinguished by major differences in activating prosurvival responses. [Ref.]

References

- **Frequent somatic mutations in PTEN and TP53 are mutually exclusive in the stroma of breast carcinomas.** Kurose, K., Gilley, K., Matsumoto, S., Watson, P.H., Zhou, X.P., Eng, C. *Nat. Genet.* (2002)
- **Augmentation of tumor angiogenesis by a Myc-activated microRNA cluster.** Dews, M., Homayouni, A., Yu, D., Murphy, D., Sevignani, C., Wentzel, E., Furth, E.E., Lee, W.M., Enders, G.H., Mendell, J.T., Thomas-Tikhonenko, A. *Nat. Genet.* (2006)
- **Nf1;Trp53 mutant mice develop glioblastoma with evidence of strain-specific effects.** Reilly, K.M., Loisel, D.A., Bronson, R.T., McLaughlin, M.E., Jacks, T. *Nat. Genet.* (2000)
- **Heterozygous germ line hCHK2 mutations in Li-Fraumeni syndrome.** Bell, D.W., Varley, J.M., Szydlo, T.E., Kang, D.H., Wahrer, D.C., Shannon, K.E., Lubratovich, M., Verselis, S.J., Isselbacher, K.J., Fraumeni, J.F., Birch, J.M., Li, F.P., Garber, J.E., Haber, D.A. *Science* (1999)
- **The tumor suppressors Ink4c and p53 collaborate independently with Patched to suppress medulloblastoma formation.** Uziel, T., Zindy, F., Xie, S., Lee, Y., Forget, A., Magdaleno, S., Rehg, J.E., Calabrese, C., Solecki, D., Eberhart, C.G., Sherr, S.E., Plimmer, S., Clifford, S.C., Hatten, M.E., McKinnon, P.J., Gilbertson, R.J., Curran, T., Sherr, C.J., Roussel, M.F. *Genes Dev.* (2005)
- **Human papillomavirus type 16 and TP53 mutation in oral cancer: matched analysis of the IARC multicenter study.** Dai, M., Clifford, G.M., le Calvez, F., Castellsagué, X., Snijders, P.J., Pawlita, M., Herrero, R., Hainaut, P., Franceschi, S. *Cancer Res.* (2004)
- **Tumor suppressor gene TP53 is genetically associated with schizophrenia in the Chinese population.** Yang, Y., Xiao, Z., Chen, W., Sang, H., Guan, Y., Peng, Y., Zhang, D., Gu, Z., Qian, M., He, G., Qin, W., Li, D., Gu, N., He, L. *Neurosci. Lett.* (2004)
- **The Huntington's disease protein interacts with p53 and CREB-binding protein and represses transcription.** Steffan, J.S., Kazantsev, A., Spasic-Boskovic, O., Greenwald, M., Zhu, Y.Z., Gohler, H., Wanker, E.E., Bates, G.P., Housman, D.E., Thompson, L.M. *Proc. Natl. Acad. Sci. U.S.A.* (2000)
- **Synergistic and opposing regulation of the stress-responsive gene IEX-1 by p53, c-Myc, and multiple NF-kappaB/rel complexes.** Huang, Y.H., Wu, J.Y., Zhang, Y., Wu, M.X. *Oncogene* (2002)
- **Tauroursodeoxycholic acid modulates p53-mediated apoptosis in Alzheimer's disease mutant neuroblastoma cells.** Ramalho, R.M., Borralho, P.M., Castro, R.E., Solá, S., Steer, C.J., Rodrigues, C.M. *J. Neurochem.* (2006)

roberth@MIT.edu

Towards Collaborative Publishing in Science

nature genetics

Commentary

Nature Genetics **39**, 433 - 436 (2007)
Published online: 28 March 2007 | doi:10.1038/ng2024

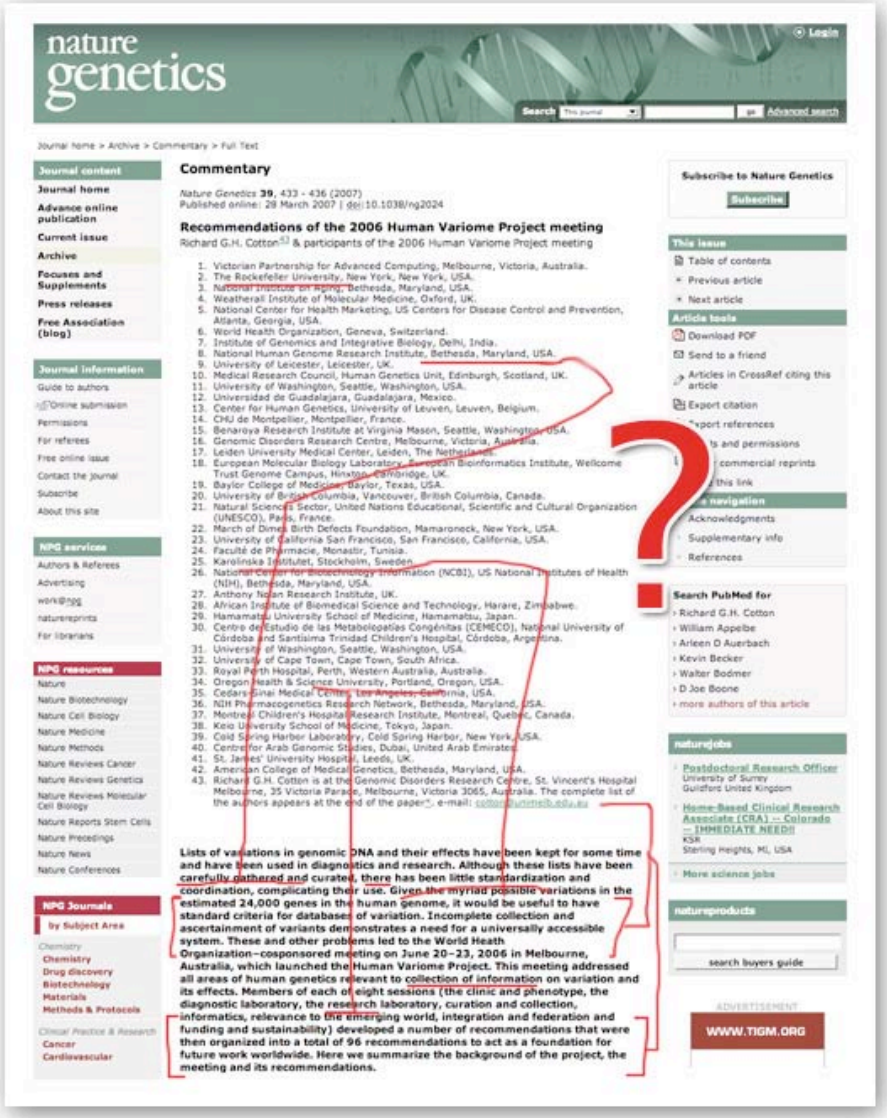
Recommendations of the 2006 Human Variome Project meeting

Richard G.H. Cotton[43] & 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 Institute on Aging, Bethesda, Maryland, USA.
4. Weatherall Institute of Molecular Medicine, Oxford, UK.
5. National Center 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, Melbourne, 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, Baylor, 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, Mamaroneck, 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 Science and Technology, Harare, Zimbabwe.
29. Hamamatsu University School of Medicine, Hamamatsu, Japan.
30. Centro de Estudio de las Metabolopatías Congénitas (CEMECO), National University of Córdoba and Santísima 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 Genomic 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 Genomic 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@utmelb.edu.au

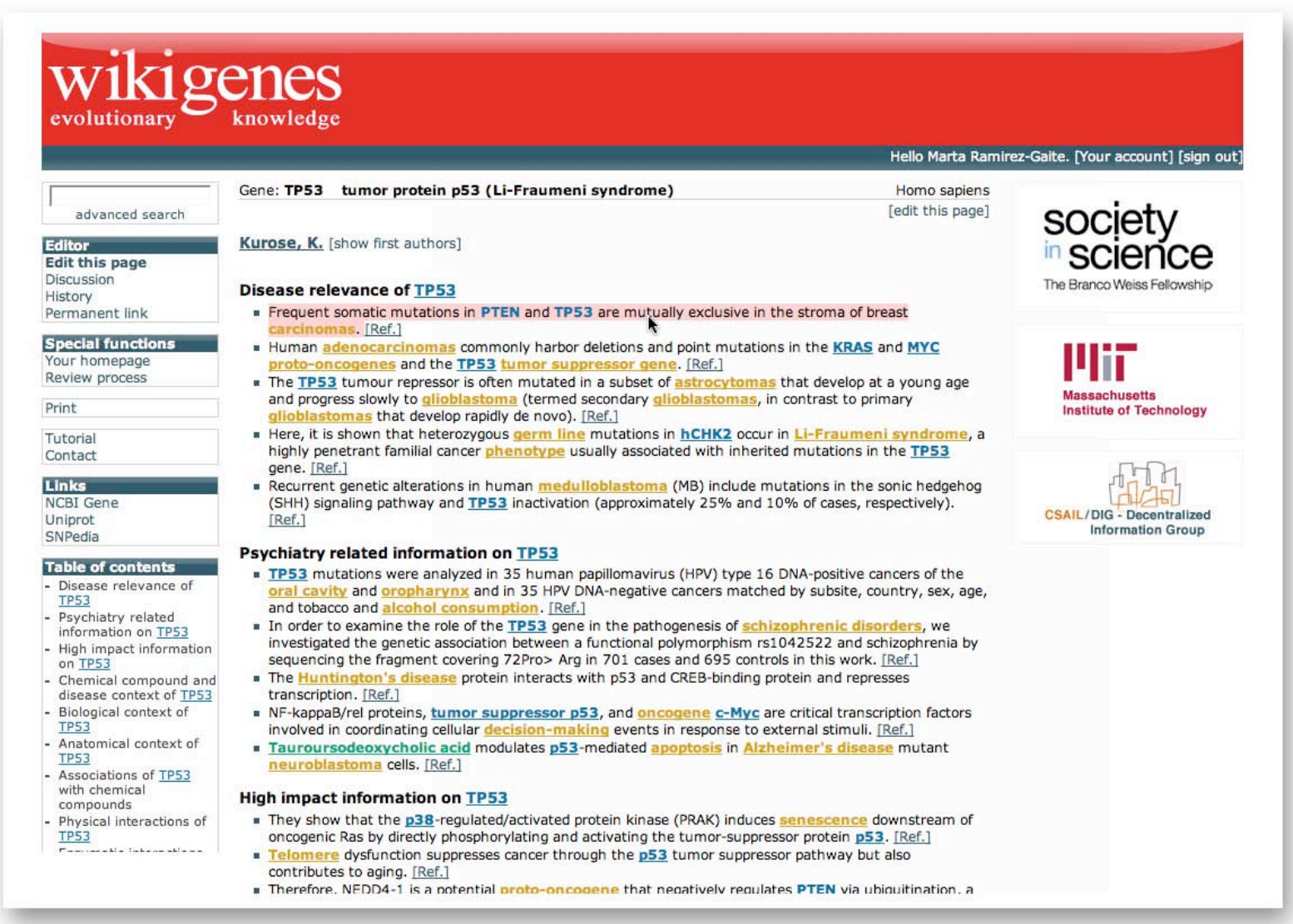
Lists of variations in genomic DNA and their effects have been kept for some time and have been used in diagnostics and research. Although these lists have been carefully gathered and curated, there has been little standardization and coordination, complicating their use. Given the myriad possible variations 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 Heath 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 96 recommendations to act as a foundation for future work worldwide. Here we summarize the background of the project, the meeting and its recommendations.

Towards Collaborative Publishing in Science

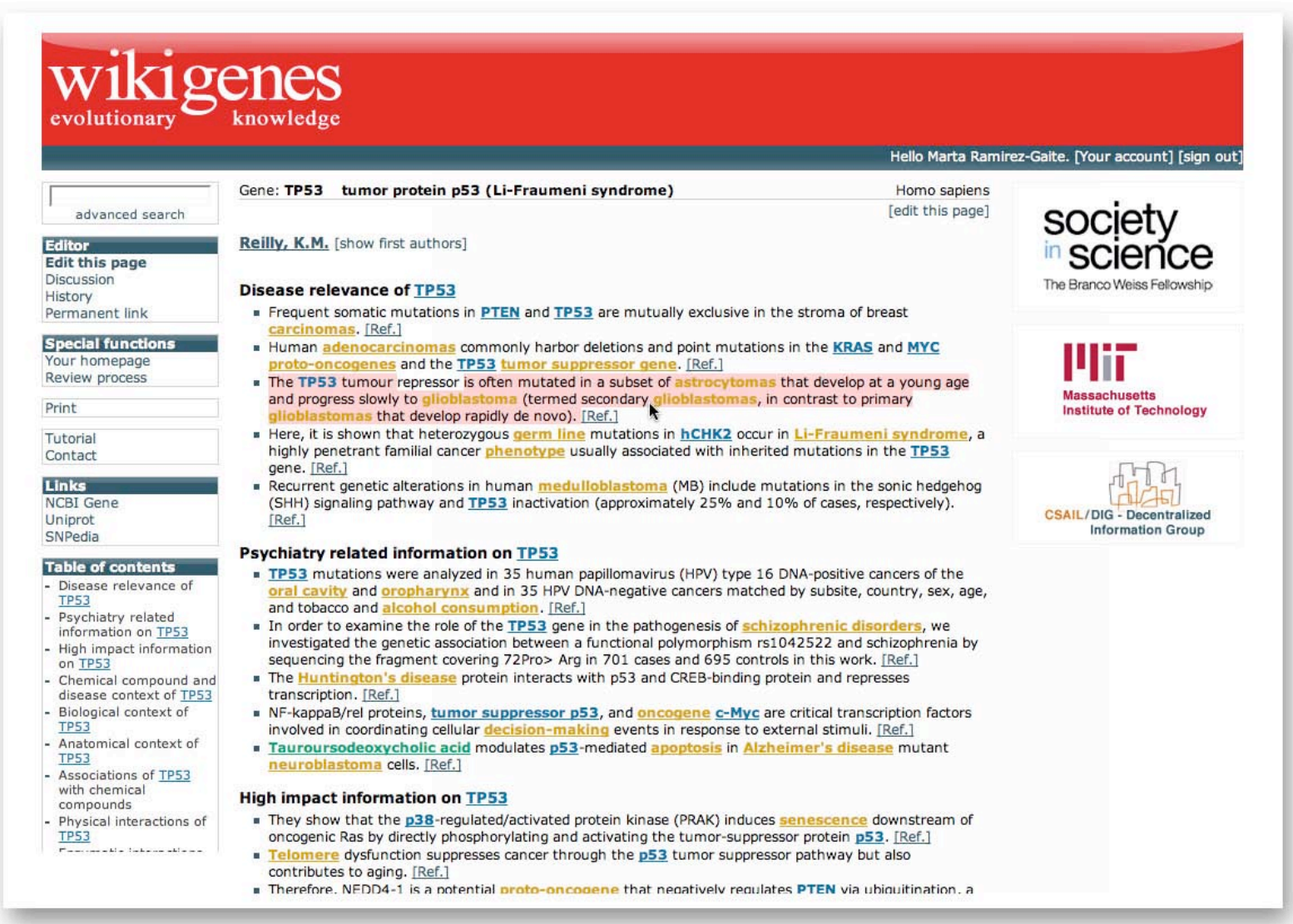


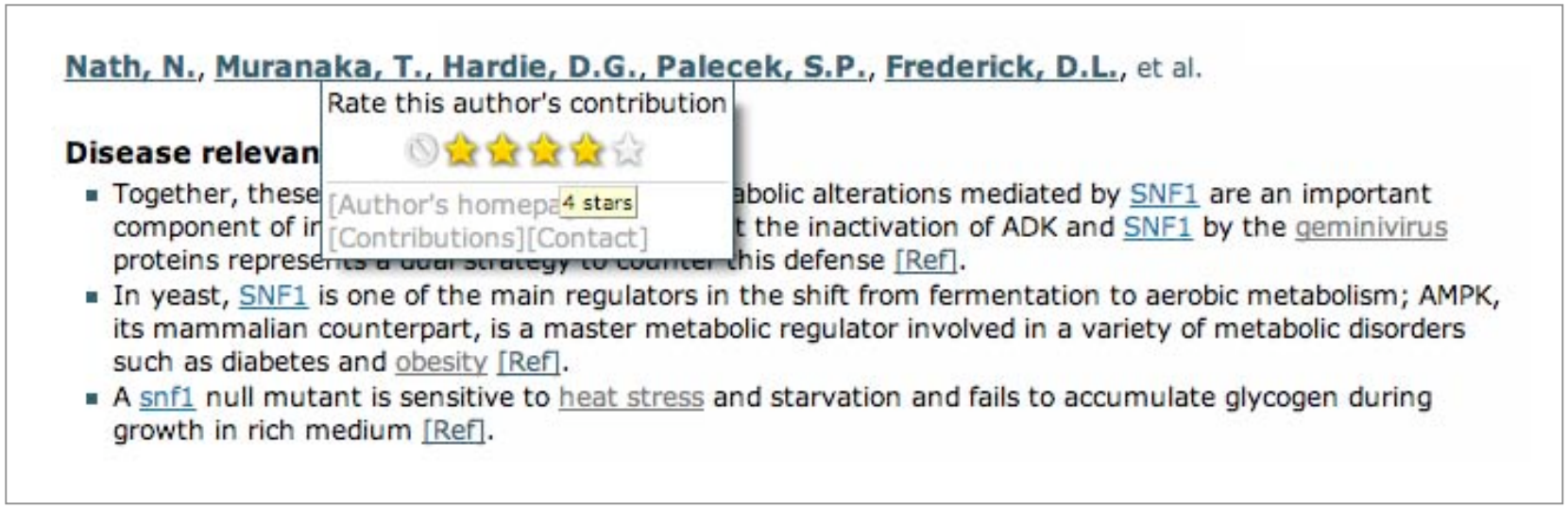
roberth@MIT.edu

Towards Collaborative Publishing in Science

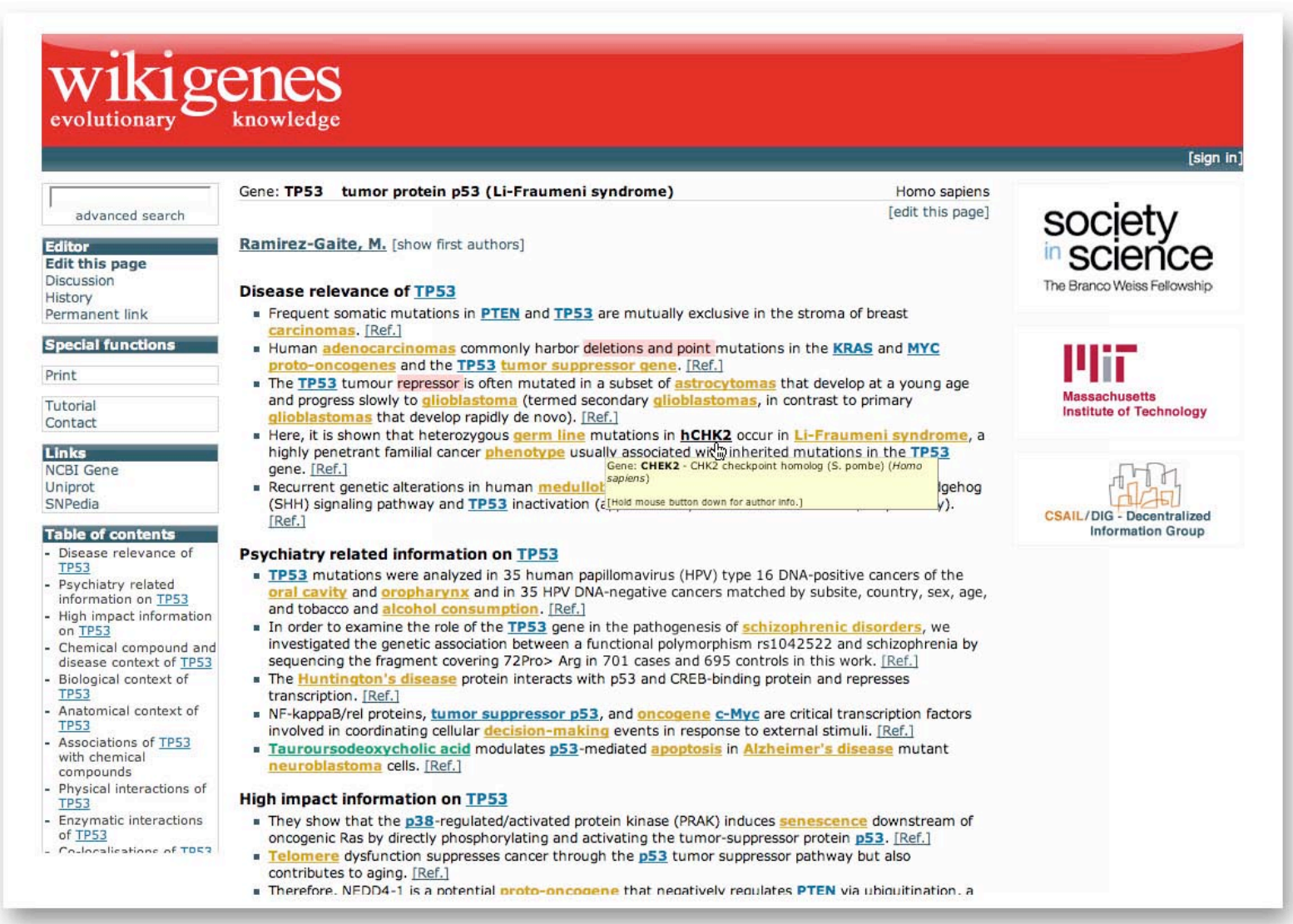


Towards Collaborative Publishing in Science

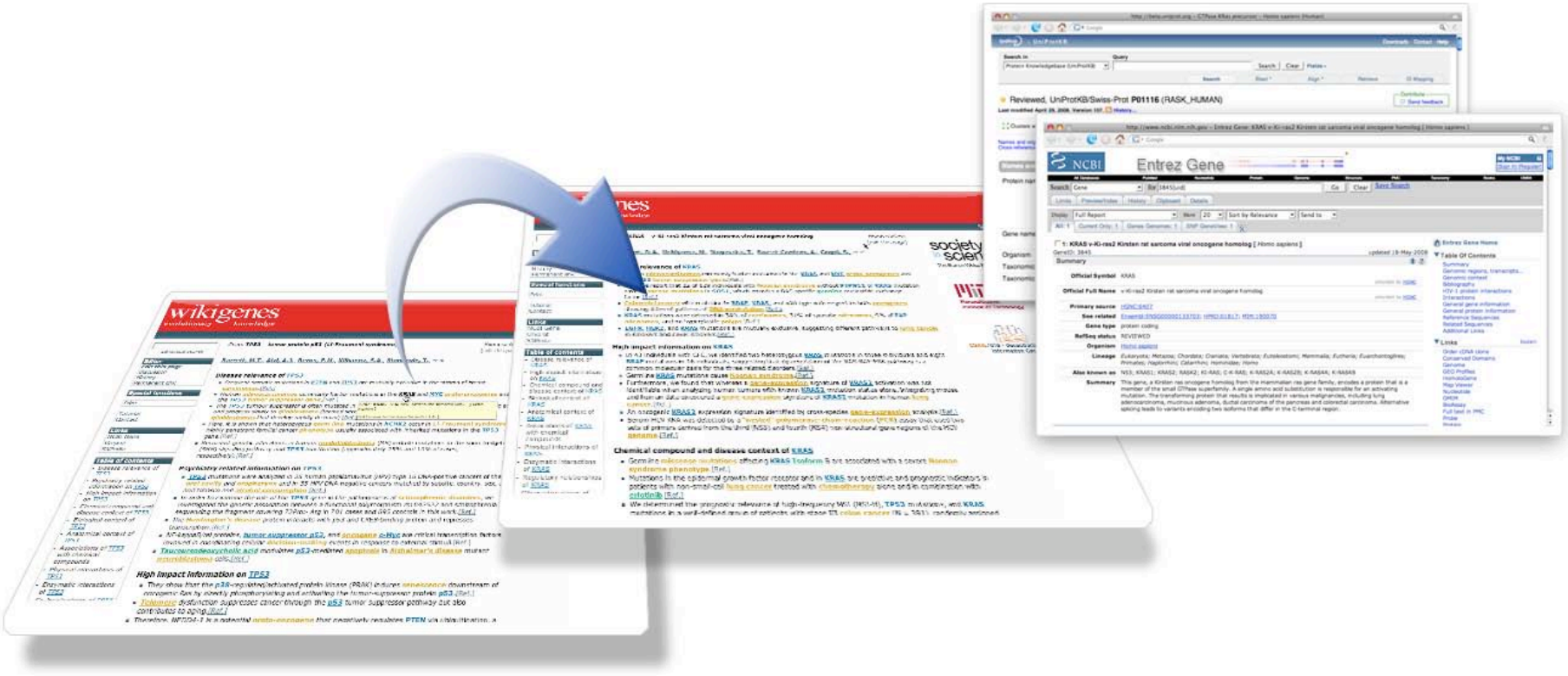



Nath, N., Muranaka, T., Hardie, D.G., Palecek, S.P., Frederick, D.L., et al.
Rate this author's contribution
4 stars
[Author's homepa
[Contributions][Contact]
Disease relevan
Together, these
abolic alterations mediated by SNF1 are an important component of ir
t the inactivation of ADK and SNF1 by the geminivirus proteins represe
his defense [Ref].
In yeast, SNF1 is one of the main regulators in the shift from fermentation to aerobic metabolism; AMPK, its mammalian counterpart, is a master metabolic regulator involved in a variety of metabolic disorders such as diabetes and obesity [Ref].
A snf1 null mutant is sensitive to heat stress and starvation and fails to accumulate glycogen during growth in rich medium [Ref].

Towards Collaborative Publishing in Science

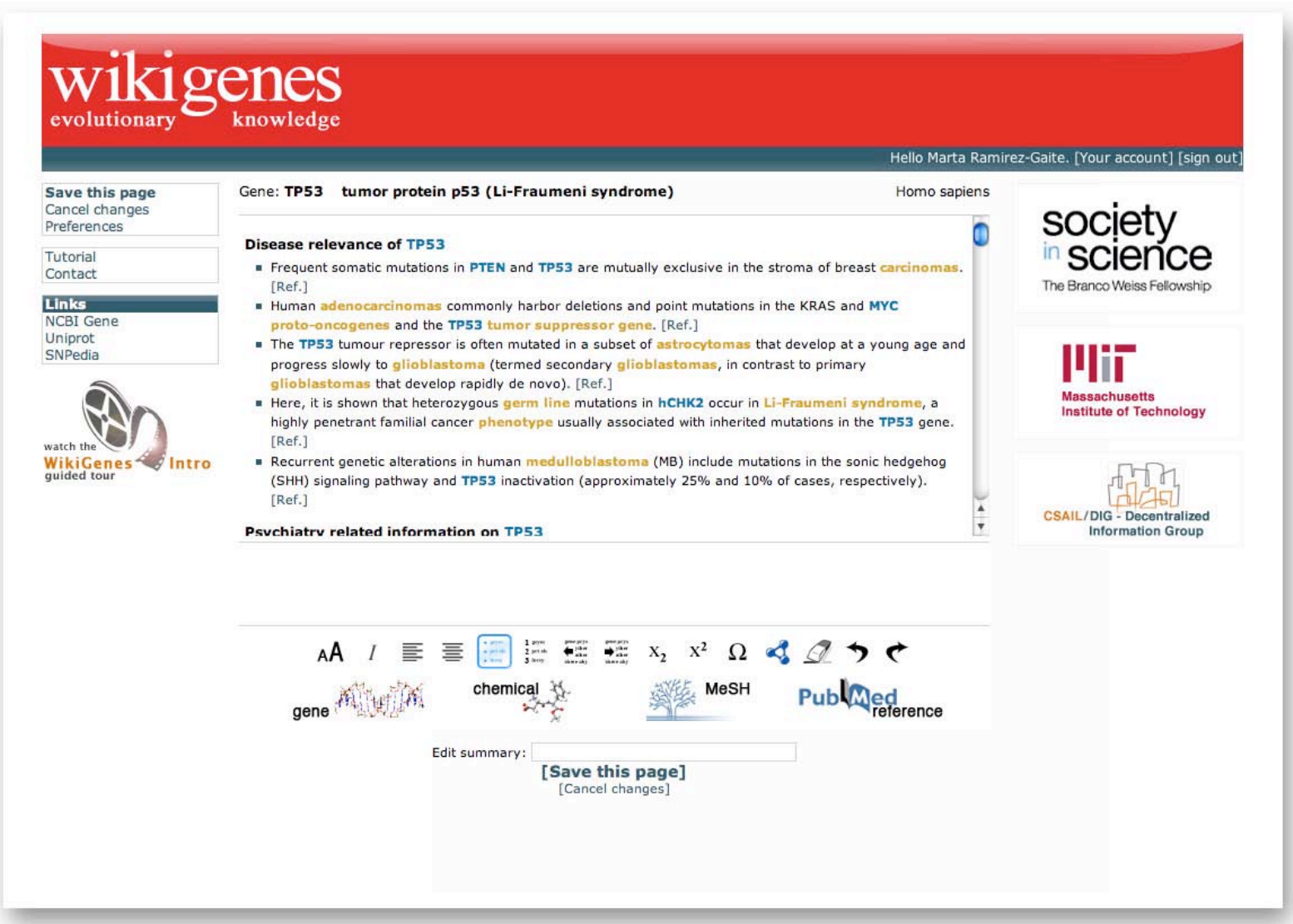


Towards Collaborative Publishing in Science

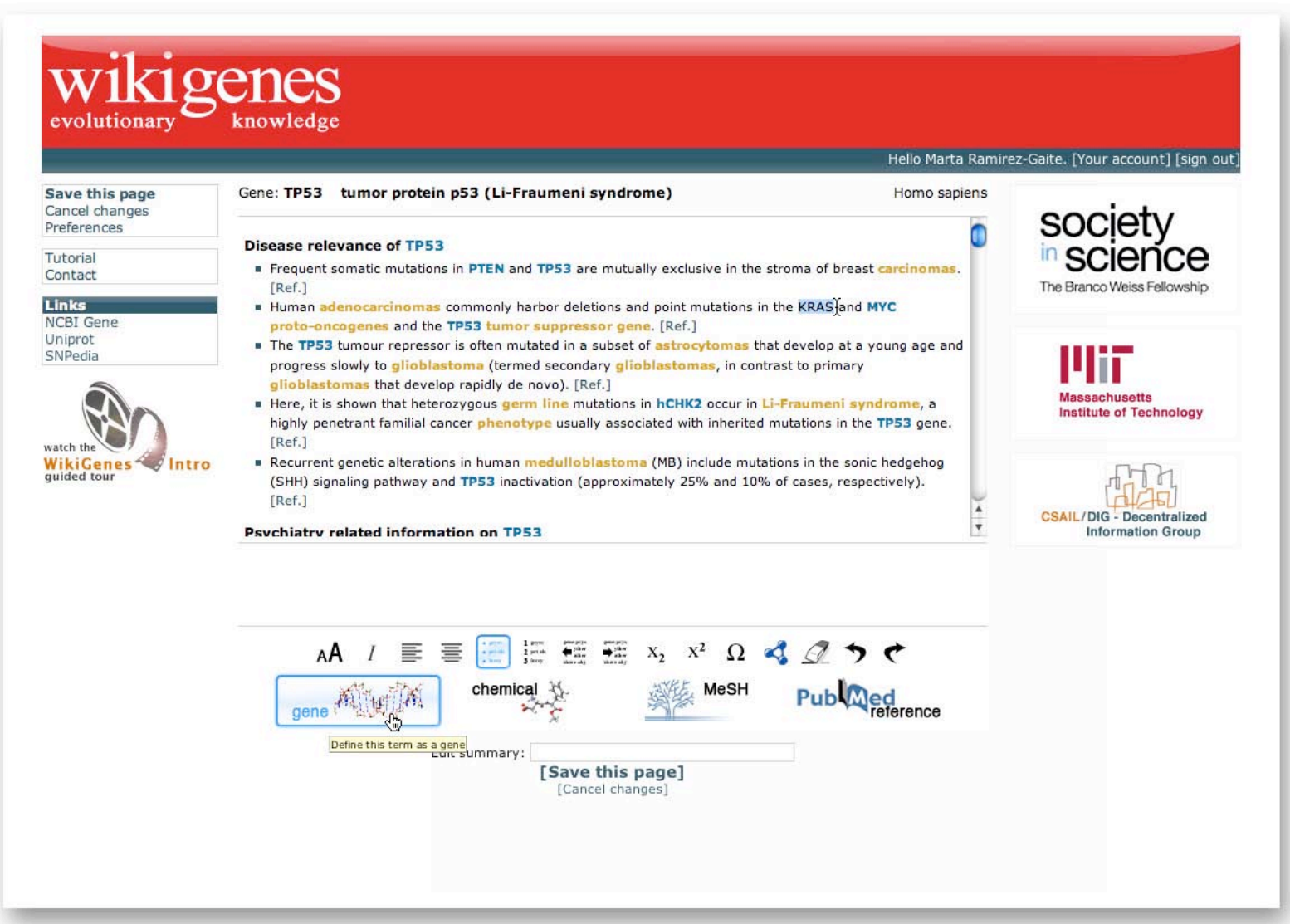


roberth@MIT.edu

Towards Collaborative Publishing in Science

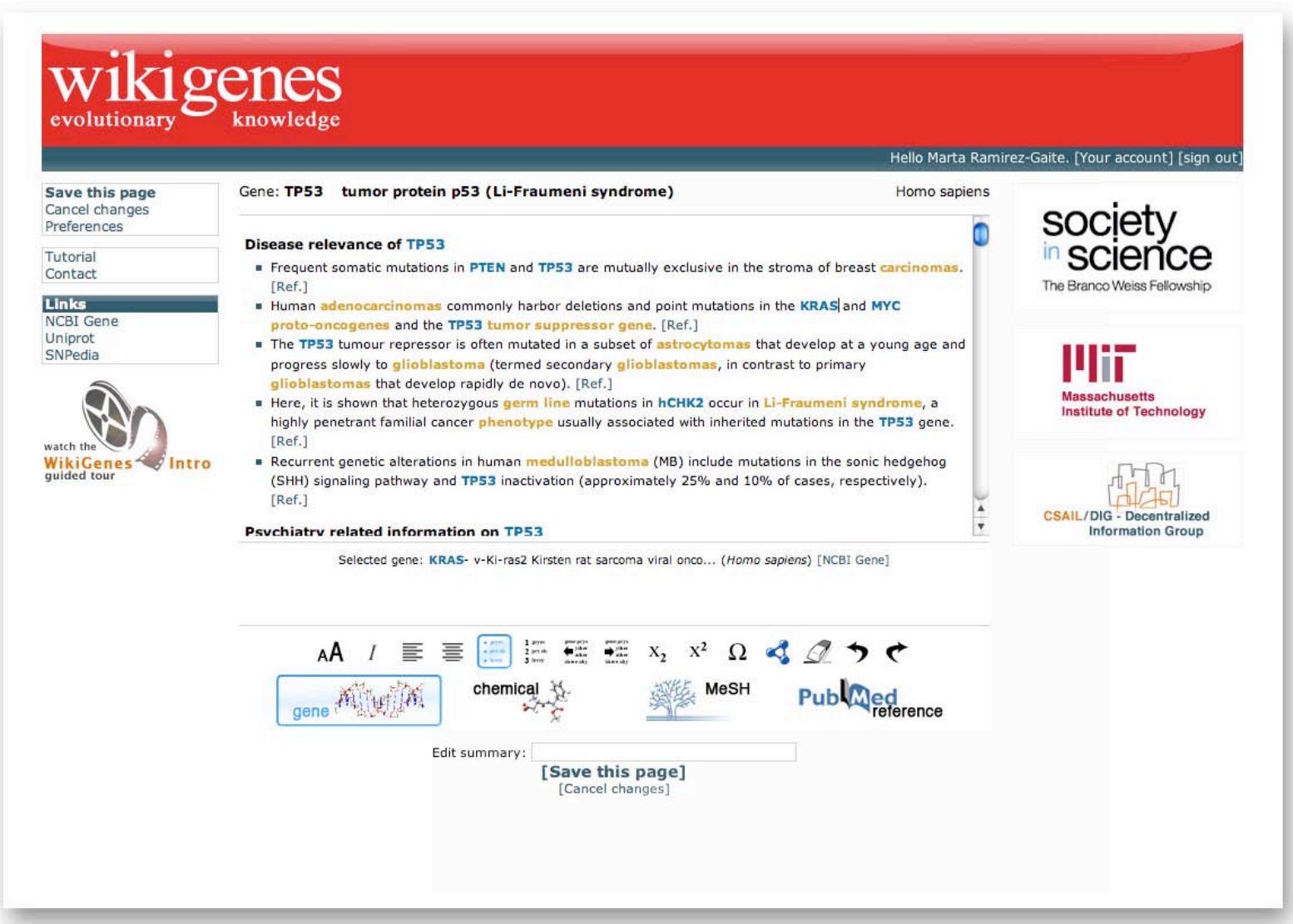


Towards Collaborative Publishing in Science

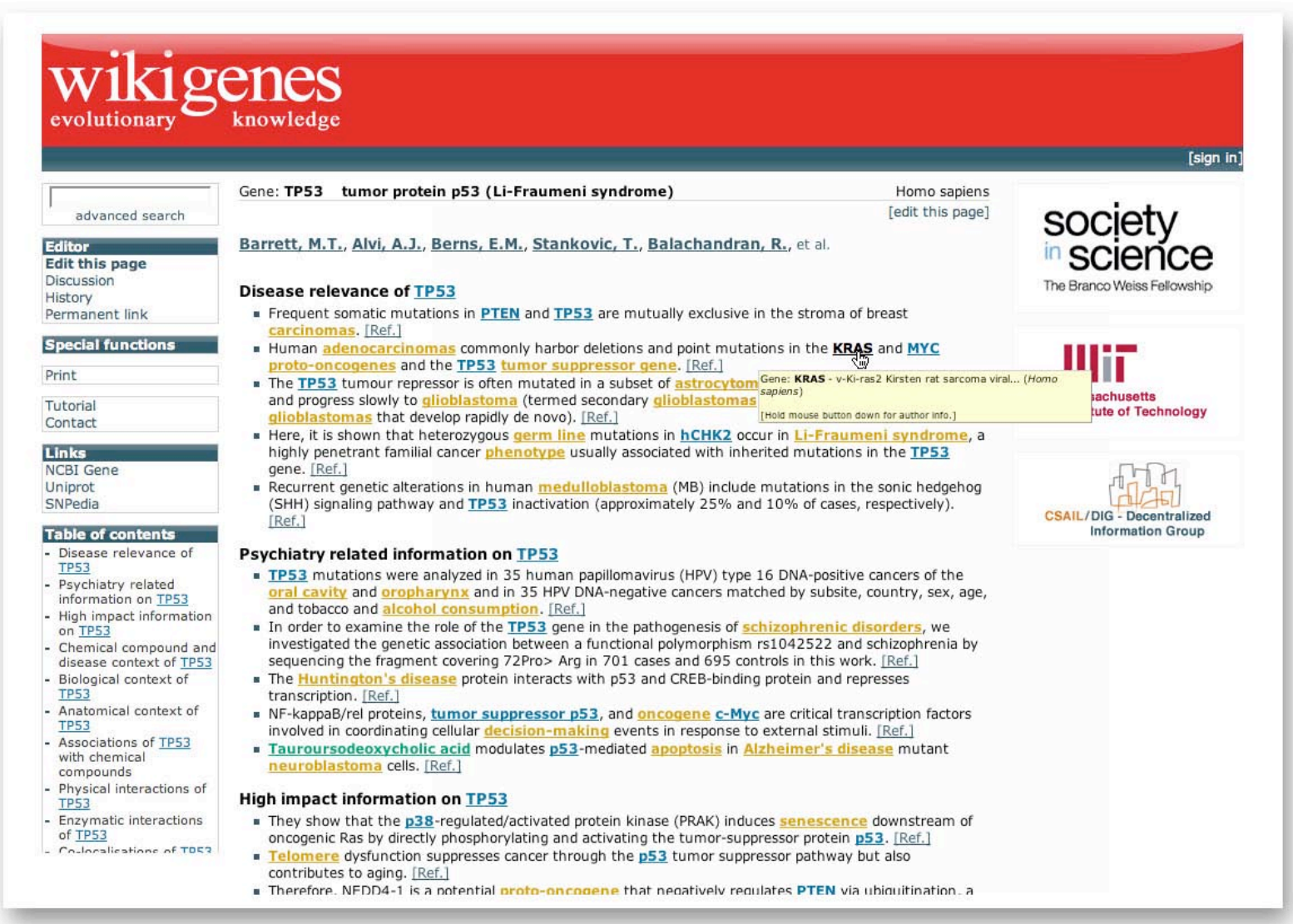


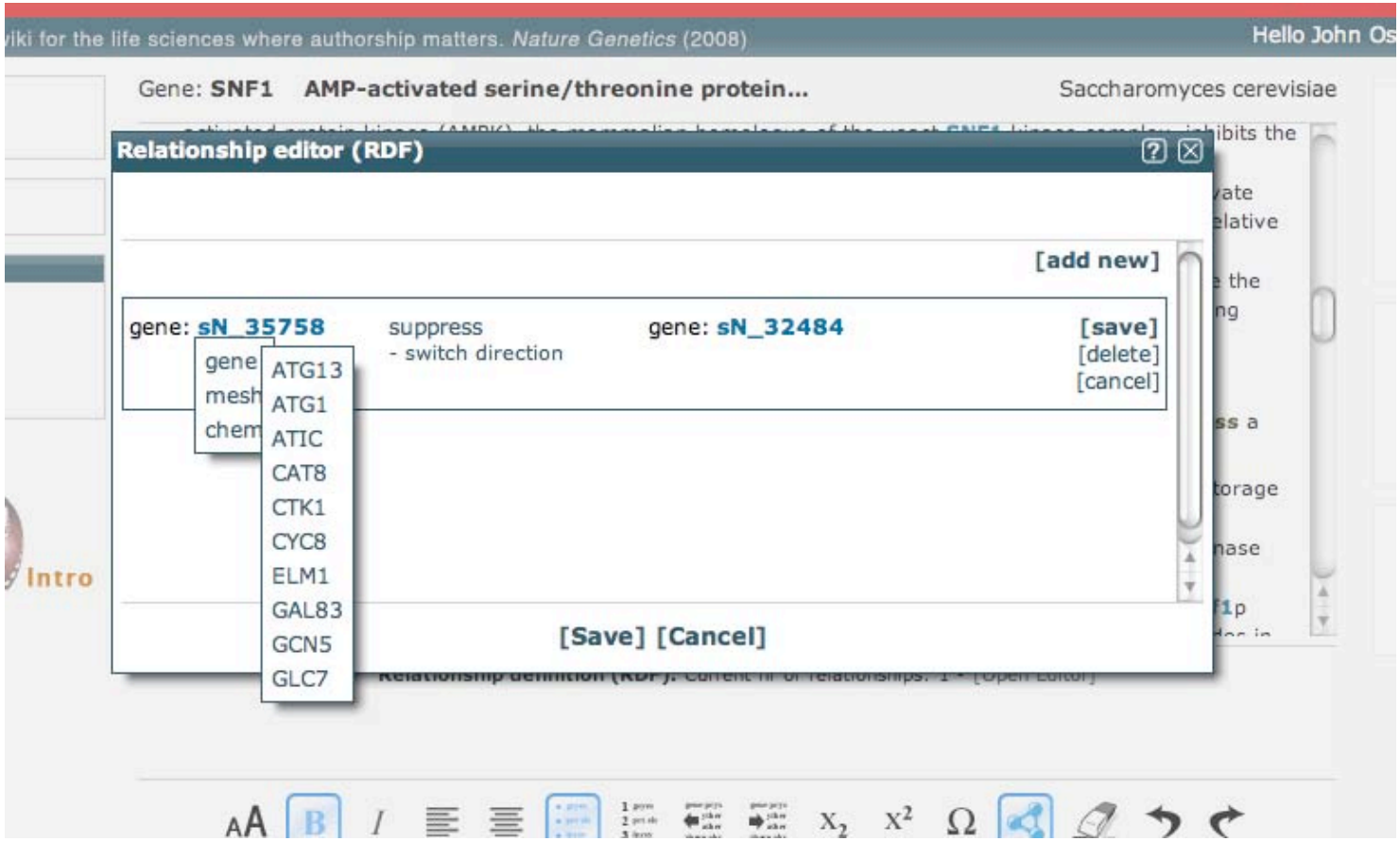
Towards Collaborative Publishing in Science

Towards Collaborative Publishing in Science



Towards Collaborative Publishing in Science





roberth@MIT.edu

Acknowledgements

Branco Weiss, Olaf Kuebler
Tim Berners-Lee, Danny Weitzner

Chris Sander, Ben Gross
Marta Ramirez, Thomas Pfeiffer

Alfonso Valencia
Rolf Apweiler
Peter Schuster

Towards Collaborative Publishing in Science

roberth@MIT.edu