

# Many shades of journal publishing: what colour is peer review in a predatory journal?

Jadranka Stojanovski University of Zadar / Ruđer Bošković Institute

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## Journal quality criteria

- Efficiency identifying and disseminating significant knowledge in timely manner
- Focus the extent to which a journal publishes the most pertinent and meaningful knowledge
- Impact the extent to which its content reflects and inspires the new, relevant knowledge
- Scope reaching audience, potential contributors to knowledge (regional, national, international)
- Selectivity ability to select better knowledge
- Composite rating determined by the journals' efficiency, focus, impact, scope, and selectivity

  Forgionne and Kohli, Information and Management, 2001







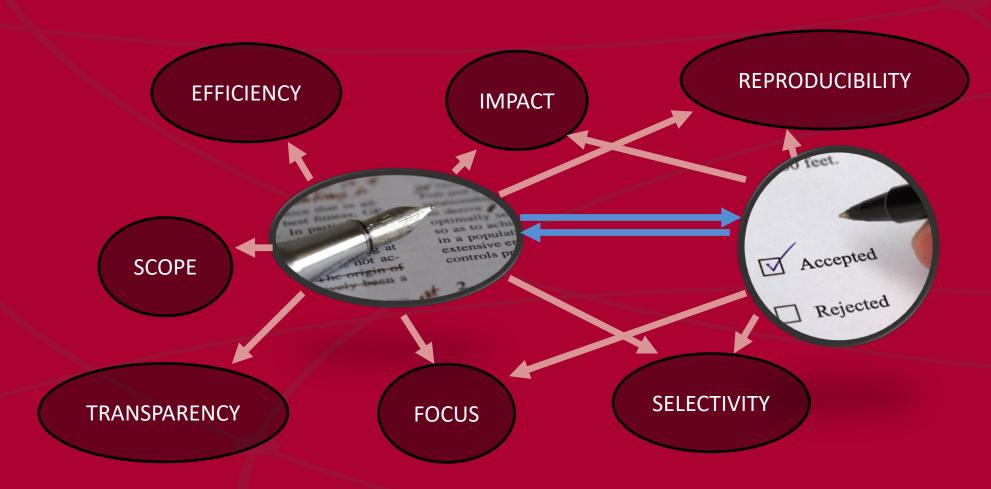
## Journal quality criteria (additional)

- Transparency and openness content, research data, methods, software, editorial policies
- Reproducibility the basic principle of science (repeat, replicate, reproduce and reuse)!
- Licencing article content and research data usage rights





## **Editors and peer reviewers**











### **Peer review**

- "The peer-review process is still the gold standard that will continue to drive scholarly publication" (Mayden, 2012)
- "Peer review involves the unbiased, independent critical assessment of scholarly or research manuscripts submitted to journals by key experts or opinion leaders" (ICMJE)
- "A good reviewer is competent, knowledgeable, unbiased, objective, punctual, consistent, ethically sound, constructive, and maintains confidentiality" (Garmel, 2010; Kumar, 2009)





# WHY "PREDATORY" JOURNALS?









### Possible reasons?

- commercialization of the scholarly publishing
- publish or perish
- bias in acceptance by well-known "western" journals
- language barriers
- not all research is globally relevant
- different funding levels in different countries/research communities
- simplicity of journal still paper-centric







Phil. Trans. R. Soc. Lond. B 326, 535-553 (1990) Printed in Great Britain

Protein structure and function at low temperatures†

#### By R. JAENICKE

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Proteins represent the major components in the living cell that provide the whole repertoire of constituents of cellular organization and metabolism. In the process of evolution, adaptation to extreme conditions mainly referred to temperature, pH and low water activity. With respect to life at low temperatures, effects on protein structure, protein stability and protein folding need consideration.

The sequences and topologies of proteins from psychrophilic, mesophilic and thermophilic organisms are found to be highly homologous. Commonly, adaptive changes refer to multiple alterations of the amino acid sequence, which presently cannot be correlated with specific changes of structure and stability; so far it has not been possible to attribute specific increments in the free energy of stabilization to well-defined amino-acid exchanges in an unambiguous way.

The stability of proteins is limited at high and low temperatures. Their expression and self-organization may be accomplished under conditions strongly deviating from optimum growth conditions. Molecular adaptation to extremes of temperature seems to be accompanied by a flattening of the temperature profile of the free energy of stabilization. In principle, the free energy of stabilization of proteins is small compared to the total molecular energy. As a consequence, molecular adaptation to extremes of physical conditions only requires marginal alterations of the intermolecular interactions and packing density. Careful statistical and structural analyses indicate that altering the number of ion pairs and hydrophobic interactions allows the flexibility of proteins to be adjusted so that full catalytic function is maintained at varying temperatures.

#### 1. Introduction

Proteins as the major components of the living cell provide the basic elements of cellular organization and metabolism. Their structure–function relation is generally assumed to be optimized with respect to the physical conditions characteristic for the natural biotope. Adaptation to extreme conditions during evolution mainly refers to temperature, pH and low water activity (Jaenicke 1981). Low water activity and extremes of pH do not necessarily require molecular adaptation of the cellular inventory as avoidance may take the place of adaptation; for example high salinity or a pH value less than 1 or greater than 11 may be compensated by compatible solutes or proton pumps. In the case of temperature, it is evident that cells are more or less isothermal with respect to their environment. As a consequence, both psychrophiles and thermophiles have to adapt their cell inventory to their respective set of conditions. Strategies promoting thermal stability of proteins have been investigated for many years. The outcome is that in the native state of functional proteins, stabilizing and destabilizing interactions more or less balance each other so that no general mechanism of temperature adaptation can be put forward. Adaptation at the protein level may be

† Dedicated to Professor Hans Neurath on the occasion of his eightieth birthday.

[ 19 ]

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#### **HISTORY OF MEDICINE**

### he Triumph over the Most Terrible of the f Death

1D, and Pere Domingo, MD

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s ago, Edward Jenner performed an the foundation for the eradication of ermed humankind's fight against disted humankind as no other disease ence and diffusion were without parought down at least three empires. ed helplessly as their children suce or were disfigured or blinded by it. e to contain smallpox by isolating its by using variolation with varying devever, the definitive solution was not work was done at the end of the 18th tho had developed cowpox from conrs informed Jenner that they were uman form of the disease; he listened and raised it to the status of scientific discover vaccination, but he was the that this technique offered a reliable Ilpox. It was also a reliable defense is, such as poliomyelitis, measles, and hough this was not known in Jenner's

lable at http://www.acponline.org.

7:635-642.

icia Primària Gràcia, Institut Català de la la Santa Creu i Sant Pau, Barcelona, ior addresses, see end of text.

rays present, filling the churchyard enting with constant fear all whom it ean, leaving on those whose lives it as traces of its power, turning the ling at which the mother shuddered, eyes and cheeks of the betrothed horror to the lover (1).

en one of humankind's greatest time immemorial. Even illnesses plague, cholera, and yellow fever a universal and persistent impact a universal and persistent impact at the time tural settlements in northeastern 000 BC (2). It probably spread dia by means of Egyptian mermillennium BC (3). The earliest sions resembling those of small-faces of mummies from the time h Egyptian Dynasties (1570 to 1085

BC) and in the well-preserved mummy of Ramses V, who died as a young man in 1157 BC (4-6).

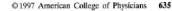
The first recorded smallpox epidemic occurred in 1350 BC during the Egyptian-Hittite war. The illness was passed to the Hittite population by Egyptian prisoners and affected soldiers and civilians alike. The Hittite King Suppiluliumas I and his heir, Arnuwandas, were victims; their civilization fell into sharp decline (2).

During the epidemic in Athens in 430 BC, Thucydides noted that those who survived the disease were later immune to it (7). These observations were reiterated by Rhazes (Abu Bakr Muhammad Ibn Zakariya al-Razi), to whom we owe the first medical description of smallpox, *De variolis et morbillis commentarius*, which was written in about AD 910. Rhazes also noted that the illness was transmitted from person to person (8). His explanation of why survivors of smallpox do not develop the disease a second time is the first theory of acquired immunity.

#### The Fall of Empires: Variola Rex and the Course of History

Smallpox greatly affected the development of western civilization. The first stages of the decline of the Roman Empire, around AD 180, coincided with a large-scale epidemic: the plague of Antonine, which killed between 3.5 and 7 million persons (9, 10). The Arab expansion, the Crusades, and the discovery of the West Indies all contributed to the spread of the illness. Unknown in the New World, smallpox was introduced by Spanish and Portuguese conquistadors. It decimated the local population and was instrumental in the fall of the empires of the Aztecs and the Incas. When the Spanish arrived in 1518, Mexico had about 25 million inhabitants; by 1620, this number had diminished to 1.6 million (11). A similar decrease occurred on the eastern coast of what became the United States, where the advent of smallpox had disastrous consequences for the native population (12), and the disease continued to be spread through the relentless process of European colonization (13). The devastating effect of smallpox gave rise to one of the first examples of biological warfare. In a letter written to Colonel Henry Bouquet in 1763, Sir Jeffrey Amherst, com-





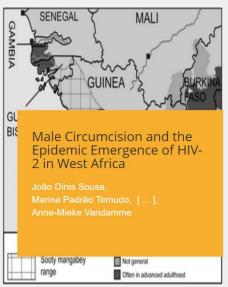
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### The Human Genome Project: Under an International Ethical Microscope

Bartha Maria Knoppers and Ruth Chadwick

At first glance, the Human Genome Project (HGP) seems ungoverned by any explicit ethical or legal norms. However, from its beginnings the HGP has spawned a myriad of international (1-9), regional (10-14), and national (15-38) seports and guidelines and, more recently, some legislation (39-47). A review of the last 5 years (December 1989 to July 1994) reveals several areas of international consensus that could serve to harmonize eventual national regulation. Five basic principles underliethis consensus autonomy, privacy, justice, equity, and quality out of respect for humandignity. Ensuring that these international areas of "commonalty" are seinforced and adopted by the HGP is an ethical and political challenge-a unique opportunity to direct rather than react.

Autonomy, Genetic testing and the resulting information is highly personal. Because this information could be used to discrimmate against individuals on socioeconomic grounds-for example, in selecting employees, immigrants, or insurance applicants-there has been a call for voluntary testing based on autonomous choice, with the participants having full information. The "right" not to know is increasingly raised as a corollary of autonomy. Most genetic information is only predictive and probabilistic-a certain gene may increase the likelihood of developing a disease. Indeed, it is this imprecise nature of genetic information that necessitates further protection against social pressures and a reaffirmation of informed consent procedures. Therefore, counseling has become a prerequnite to the decision to undergo testing-An exception to this principle of individual consent is newborn screening programs for immediately treatable disorders. A recent report from the United States, however, has explicitly recommended that parental consent be obtained (34).

There is consensus limiting genetic testing (including prenatal testing) to tests that are medically therapeutic. Which tests are considered to be therapeutic then re-

B. M. Proppers a si Professor of Law and Senior Researcher, Faculty of Law and Cerete for Public Law Research. Drivensty of Montreat: Canada, and a member of the International Etnics Committee of Music And Unicolo. R. Chadwick is a Professor of Marie Professor, University of Certa's Lancadine, U.K., and Coordinator of EUPCOCRETA.

mains to be decided by individual countries secording to cultural, social, and political norms. Both France (41, 42) and Norway (45) have passed legislation controlising the elaboration of such "therapeutic" criteria in governmental bodies. Adherence to these criteria effectively curtails the use of genetic tests for sex selection or trait enhancement.

Most genetic testing is further limited to individuals at high risk for serious disorders. Furthermore, there is consensus that predisposition testing should be limited to disease that are treatable or preventable. Somatic cell therapy is for the most part considered experimental and thus subject to stringent limitations (used only in serious mortogenic conditions) as well as to additional sufeguards and oversight. Preimplantation embeyo testing remains controversial and severely constrained but not totally probabited, except in Germany (44).

Privacy. Respect for the privacy of the person and for the confidentiality of genetic information is crucial. Although the results of genetic tests could be considered a form of sensitive medical information, genetic testing also reveals information about other family members and is of importance to insurers and employers. Some guidelines would prohibit any communication to all third parties without consent (8, 13, 14, 24, 30). Most guidelines, however, advocase the communication of selevant information to family members at high risk for serious bann without the consent of the patient or of the research participant only when all attempts to elicit voluntary communication have failed. All other disclosures of information-or use of DNA samples (unless anonymous)-would require consent. Furthermore, the collection, storage, and dissemination of genetic information should be subject to special procedures of coding, of removing identifiers, and of obtaining consent for new uses.

In the areas of insurance and employment, the presence or absence of universal health insurance and social security shapes current guidelines. Little is known of the potential discriminatory or stigmastizing effects (or even benefits) of access to genetic information by insurers and employers. Even countries with universal health care recommend rejecting access to or direct testing by employers and insurem for life

and disability insurance. For example, reports from both the Netherlands (28) and the United Kingdom (32) have called for a moratorium on requiring disclosure where life insurance policies are proportionate to income or of moderate size. Only Belgium has specifically included a prohibition on sesting or access to genetic information by inscers in its Civil Code (40). The American NIH-DOE report recommends that "Information about past, present or future health status, including genetic information, should not be used to deny health care coverape or services to anyone" (35). Finally, genetic identity testing confirms either fil ial links (paternity or maternity) or presence at the scene of a crime (forensic testing) and utilizes the same techniques as medical testing [sampling, restriction fragment length polymorphisms (RFLPs), markers, and polymerase chain reaction amplification). Similar privacy concerns arise (38). France has passed legislation requiring court orders for such identity testing (41).

Justice. The international community is united in its concern for vulnerable populations, such as incompetent adults or minors, and for future generations. Although overprotection could make research with these populations impossible, the fact that they cannot decide for themselves and are often in institutions mandates special protection—but not exclusion. Furthermore, in the absence of treatment or prevention, the presymptomatic testing of children for late onset disease has not been recommended. Where possible, both children and incompetent adults should participate in decision-making.

The continuing debate on the desirabil ity of germline modification is sparked by a desire for justice toward future generations and prevention of eugenic uses of the technology. Although most guidelines advocate a total prohibition of germline modification, others have taken a more cautious approach, suggesting continuing discussion of its technical and ethical aspects and the development of adequate safeguards. The 1991 CIOMS Declaration of Ingoma (8) considered continued discussion of its technical and ethical aspects to be essential. Nevertheless, Austria (39), France (41). Germany (44), Norway (45), and Switterland (47) probibit germline alteration by

Egaty. Although not explicitly mentioned as a governing principle, equity is a recurring port of the orgoing discussion. How do we ensure equity of access to genetic research, testing, and information, equal costs; equal resources; and equal sharing of information! There is a potential danger and the accompanying fore of genetic testing increasing social inequality, of access to testing being linked to willingness.



## The defects of peer review

- slow
- expensive
- highly subjective
- "something of a lottery"
- biased against innovative papers, women, non-prestigious institutions, low income countries, language, negative studies...
- easily abused
- inconsistent
- unable to detect errors or fraud







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ward Cadaveric

Andrew Wakefield, Lancet, 1998 – MMR vaccines and autism Haruko Obokata. Nature, 2014 – stem cell research Hwang Woo-suk, Science, 2004 i 2005 – have "succeeded" in creating

Jan Hendrik Schön, Science et al. – semiconductors

Yoshitaka Fujii, anesthesiology – fabricated more than 183 papers! Diederik Stapel, psychology – 54 papers retracted! paper generators – SClgen, Mathgen

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THE LANCET • Vol 351 • February 28, 1998

Early report

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http://dx.doi.org/10.1016/j.athoracsur.2012.05









# Irreproducible research

IS THERE A.—

More than 70% of researchers have tried and failed

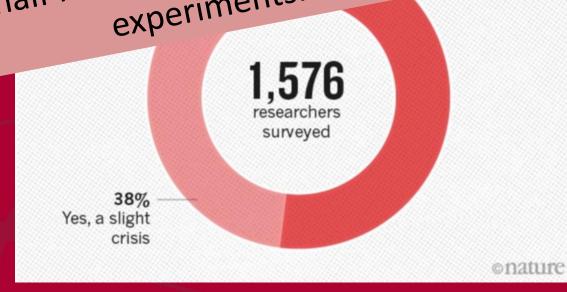
More than 70% of researchers have tried and failed

to reproduce another scientist's experiments, and

to reproduce their own

more than half have failed to reproduce their own

experiments.



Baker, 2016











# "Predatory" journals (J. Beall)

- Editorial bodies editors are not named;
   without affiliations
- Contact missing or fraudulent contact information
- **Fees** costs associated with publishing are hidden or unclear
- Journal name doesn't reflect the scope; imitates name of a prestigious journal; contain the national or international affiliation that does not match information on publisher's location

- Indexing and metrics false information on indexing, false metrics
- Journal scope is to broad
- Peer review missing information on the peer review process, sometimes without peer review
- Spam e-mails journal sends emails requesting submissions or inviting researchers to be members of the editorial board



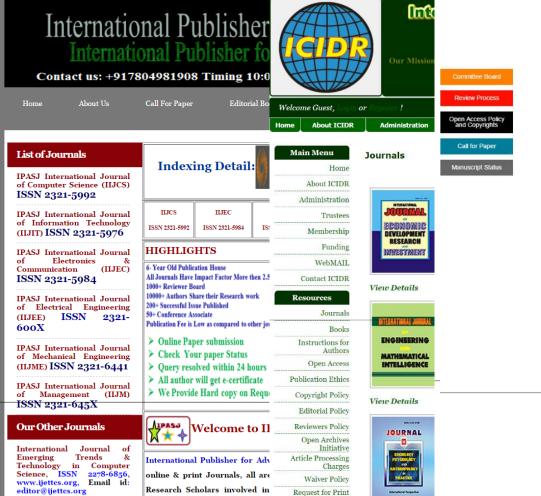




### **Small study**

- 50 "predatory" publishers/journals were analyzed
- publisher name, URL address, note on fees, fees (USD), note on peer review
- author guidelines, reviewer guidelines, ethical policies were collected as a separate files
- screenshot of the publisher web site was captured
- simple content analysis with **peer review** in focus





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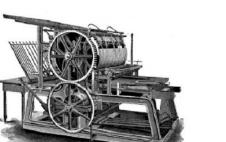


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## Fees (#50)

- 9 journals not charging (new probably will charge in the future)
- 5 journals information on charges not available
- 4 journals price on demand (contact address)
- 32 journals have fees (\$40-\$500) differences between local and foreign authors

- article processing charges, article processing fees, article publishing fee, handling fee, manuscript processing fee, page fee
- upon acceptance





## Ethical policy (#50)

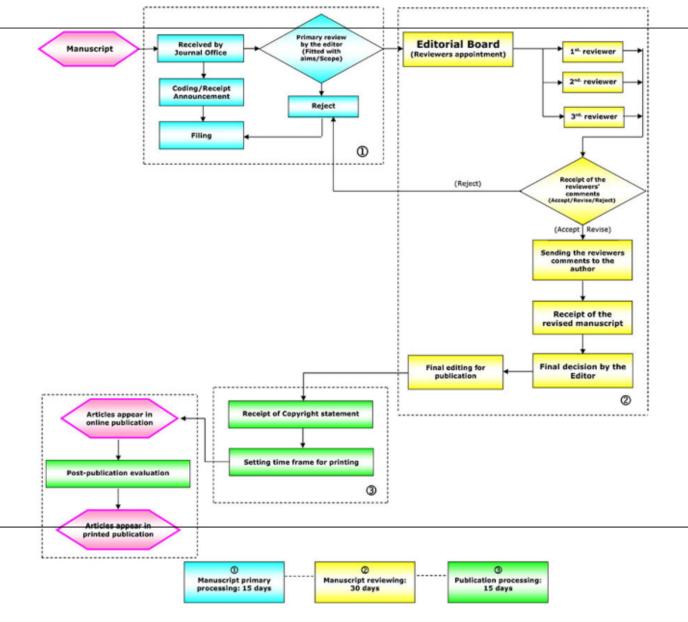
- 31 journals have a kind of ethical policy as a separate document
- all ethical policies have a note on peer review, manuscript originality and plagiarism
- 27 have a note on disclosure and conflict of interest





P

- only 14 journals have guideling document
- 21 jounal have a note on pee
- 37 have information on peer double blind)
- 9 journals have double blind
- ethical issues (plagiarism, fraction confidentiality, conflict of interest





IN SCIENCE AND TECHNOLOGY





## "Predatory" journals

- Editorial bodies editors are named; with affiliations
- **Contact** contact information present
- Fees 80% transparent information on fees
- **Journal name** "international" character

- Indexing and metrics not present
- Journal scope not to broad
- Peer review information on peer review process present

### Limitations of the study:

- small sample
- accuracy of the presented information was not checked







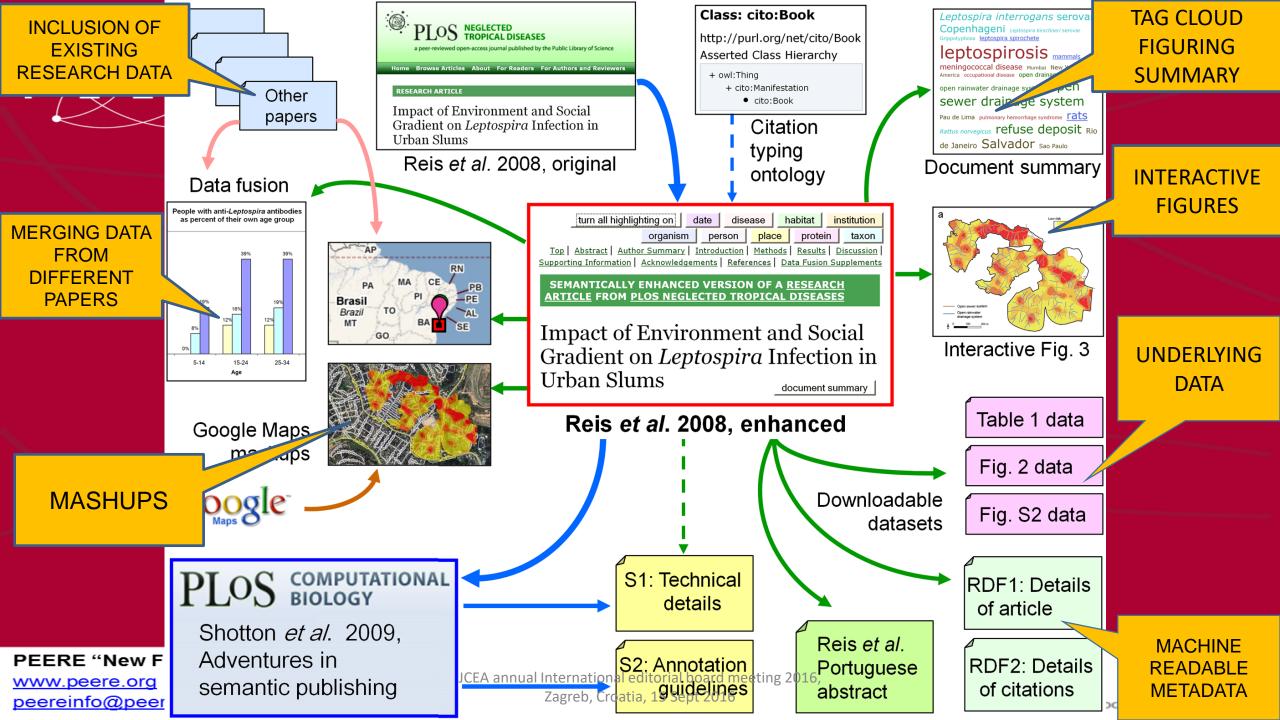


### THE FUTURE OF PREDATORY PUBLISHERS?











### **Semantic enrichment**

turn all highlighting off date disease habitat institution organism person place protein taxon

Top Abstract Author Summary Introduction Methods Results Discussion Supporting Information Acknowledgements References Data Fusion Supplements

#### Introduction

At present, one billion of the world's population resides in slum settlements [1]. This number is expected to double in the next 25 years [1]. The growth of large urban populations which are marginalized from basic services has created a new set of global health challenges [2],[3]. As part of the Millennium Development Goals [4], a major priority has been to address the underlying poor sanitation and environmental degradation in slum communities which, in turn, are the cause of a spectrum of neglected diseases which affect these populations [2],[3],[5].

Leptospirosis is a paradigm for an urban health problem that has emerged due to recent growth of slums [6],[7]. The disease, caused by the Leptospira spirochete, produces life-threatening manifestations, such as Weil's disease and severe pulmonary hemorrhage syndrome for which fatality is more than 10% and 50%, respectively [7]–[9]. Leptospirosis is transmitted during direct contact with animal reservoirs or water and soil contaminated with their urine [8],[9]. Changes in the urban environment due to expanding slum communities has produced conditions for rodent-borne transmission [6],[10]. Urban epidemics of leptospirosis now occur in cities throughout the developing world during seasonal heavy rainfall and flooding [6],[11]–[18]. There is scarce data on the burden of specific diseases that affect slum populations [2], however leptospirosis appears to have become a major infectious disease problem in this population. In Brazil alone, more than 10,000 cases of severe leptospirosis are reported each year due to outbreak in urban centers [19], whereas roughly 3,000, 8,000 and 1,500 cases are reported annually for meningococcal disease, visceral leishmaniasis and dengue hemorrhagic fever, respectively, which are other infectious diseases associated with urban poverty [20]–[22]. Case fatality (10%) from leptospirosis [19] is comparable to that observed for meningococcal disease, visceral leishmaniasis and dengue hemorrhagic fever (20%, 8% and 10%, respectively) in this setting [20],[23],[24]. Furthermore, leptospirosis is associated with extreme weather events, as exemplified by the El Niño-associated outbreak in Guayaquil in 1998 [25]. Leptospirosis is therefore expected to become an increasingly important slum health problem as predicted global climate change [26],[27] and growth of the world's slum population [1] evolves.

Urban leptospirosis is a disease of poor environments since it disproportionately affects communities that lack adequate sewage systems and refuse collection services [6],[10],[11]. In this setting, outbreaks are often due to transmission of a single serovar, L. interrogans serovar Copenhageni, which is associated with the Rattus norvegicus reservoir [6], [28]–[30]. Elucidation of the specific determinants of poverty which have led to the emergence of urban leptospirosis is essential in guiding community-based interventions which, to date, have been uniformly unsuccessful. Herein, we report the findings of a large seroprevalence survey performed in a Brazilian slum community (favela). Geographical Information System (GIS) methods were used to identify sources for Leptospira transmission in the slum environment. Furthermore, we evaluated whether relative differences in socioeconomic status among slum residents contributed to the risk of Leptospira infection, in addition to the attributes of the environment in which they reside.













### Authorship (XML)

Nature Genetics 41, 399 - 406 (2009)

Published online: 22 March 2009 | doi:10.1038/ng.364

### Common variants at ten loci influence QT interval duration in the QTGEN Study

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Rotterdam Study: M.E., K.E., A.H., J.A.K., F.R., B.H.Ch.S., A.G.U., J.C.M.W.

Cardiovascular Health Study: J.C.B., S.R.H., T.L., K.M., C.N.-C., B.M.P., K.M.R., J.I.R., N.L.S., N.S.

**Broad Institute of Harvard and Massachusetts Institute of Technology:** P.I.W.dB., C.N.-C.

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Carninci P, Tomaru Y, Kanamori-Katayama M, Kubosaki A, Akalin A, Ando Y, Arner E, Asada M, Asahara H, Bailey T, Bajic VB, Bauer D, Beckhouse AG, Bertin N, Björkegren

J, Brombacher F, Bulger E, Chalk AM, Chiba J, Cloonan N, Dawe A, Dostie J, Engström PG, Essack M, Faulkner GJ, Fink JL, Fredman D, Fujimori K, Furuno M, Gojobori T,

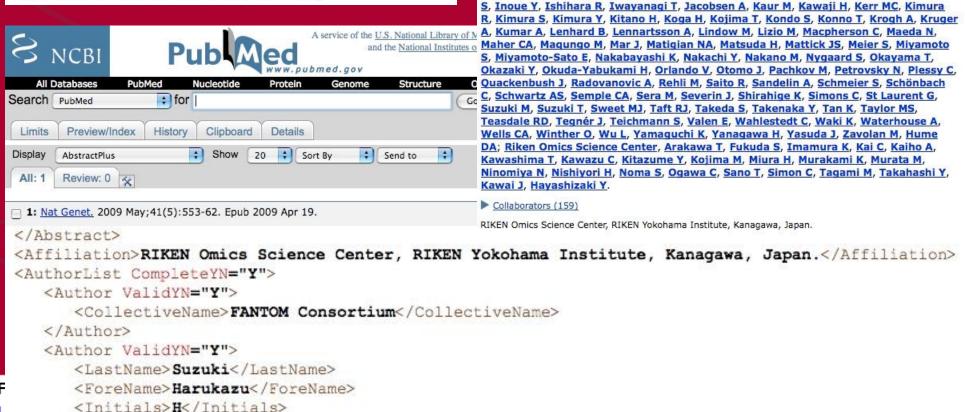
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Gough J, Grimmond SM, Gustafsson M, Hashimoto M, Hashimoto T, Hatakeyama M,

Nature Genetics **41**, 553 - 562 (2009) Published online: 19 April 2009 | doi:10.1038/ng.375

The transcriptional network that controls growth arrest and differentiation in a human myeloid leukemia cell line

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- 2.7. MUC4
- General structure of the apomucins (mucin polypeptides)
  - 3.1. SEA domain
  - 3.2. EGF domain
  - 3.3. NIDO-AMOP-WF-D domains
- 4. Alternative splicing of mucins

References

#### 1. Introduction

Epithelial mucins are heavily Oglycosylated proteins found in the mucus layer or at the cet surface of many epitheliums. They are responsible for the p properties of mucus gels and are involved in epithelial cell protection. There is still no clear definition of a "mucin" and the increasing number of genes with symbol MUC is unfortunately not helping the scientific community ([Dekker et al., 2002] and [Rose and Voynow, 2006]). In a first approach, we can pi the term mucin refers at least to the highly O-glycosylated epithelial molecules mail There are two structurally distinct families of mucins. The first one is made of the fiv Dekker et al., 2002 J. Dekker, J.W. Rossen, H.A. MUC19 that form oligomeric structures (Thornton et al., in press). The other family i Buller and A.W. Einerhand, The MUC family: an membrane-bound mucins are made of at least a mucin-like domain, i.e. a large port obituary, Trends Biochem. Sci. 27 (2002), pp. amino acids that carry the O-glycans. The Ser/Thr/Pro repeat sequences are encoc 126-131. Article | 📆 PDF (72 K) | View Record in typically subject to a VNTR (variable number of TR) polymorphism. This region is ey Scopus | Cited By in Scopus (119) two distinct groups: small mucins and large mucins. Our goal in this review is to give located primarily, but not exclusively, at the cell surface, as their respective genes | EAdditional Information from CSIBS O-glycosylated extracellular portion may be released into the mucus gel by proteolof mucus gels in contrast to small mucin molecules. We will only dwell here on the Concerning the small mucin MUC1 which was the first mucin characterized, several and Muller, 2000], [Patton et al., 1995] and [Taylor-Papadimitriou et al., 1999]). Eve Defining mucins: family values: the large membrane-bound mucins due to sometimes the complexity and repetitive sequence databases can be useful tools to find new genes and to help in the chara to bring new data from database analysis in order to bring some clarification.

#### 2. Domains of the membrane-bound mucins

#### 2.1. The nine gene candidates

To date, several cDNA genomic sequences claming to come from seven putative m ... MUC3 was one of the first MUC proteins found, in MUC12, MUC16 and MUC17, MUC4 was mapped to 3g29, MUC16 has been localize MUC11, MUC12 and MUC17 are organized in a cluster of genes on 7g22.1 (Table

General features of the human and mouse large membrane-bound mucins

Human		Mouse			
Gene	Location	aa/TR <sup>1</sup>	Exons	Gene	Location
MUC3/3A/3B	7q22.1	17	> 11 (13?)		
MUC4	3q29	16	25	Muc4	16B3
MUC11/12	7q22.1		> 11 (13?)		

1990 [4], but it has recently been discovered that there are, in fact, two closely related and adjacent genes (MUC3A and MUC3B) with 98% homology

. There are two approaches to the definition of

defining the relationships of the mucin-encoding

Using this criterion to define mucins would be

modification with lipid mojeties and calling the

similar to conflating all lipoproteins based on their

mucins but both are unsatisfactory when it comes to

#### Conclusions: families and orphans:

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encoding genes ?LIP-number?

 Based on sequence homology, two families of mucins can be distinguished: (1) the mucin genes at locus 11p15, which probably encode mucus-forming mucins; and (2) the mucin genes at loci 7q22, 3q and 1g21, presumably encoding membrane-bound

Dekker et al., 2002 J. Dekker, J.W. Rossen, H.A.

Buller and A.W. Einerhand, The MUC family: an obituary, Trends Biochem. Sci. 27 (2002), pp.

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### Defining mucins: family values:

- There are two approaches to the definition of mucins but both are unsatisfactory when it comes to defining the relationships of the mucin-encoding genes.
- Using this criterion to define mucins would be similar to conflating all lipoproteins based on their modification with lipid moieties and calling the encoding genes ?LIP-number?.

### All in the family?:

 MUC3 was one of the first MUC proteins found, in 1990 [4], but it has recently been discovered that there are, in fact, two closely related and adjacent genes (MUC3A and MUC3B) with 98% homology [26]

### Conclusions: families and orphans:

 Based on sequence homology, two families of mucins can be distinguished: (1) the mucin genes at locus 11p15, which probably encode mucus-forming mucins; and (2) the mucin genes at loci 7q22, 3q and 1q21, presumably encoding membrane-bound mucins.



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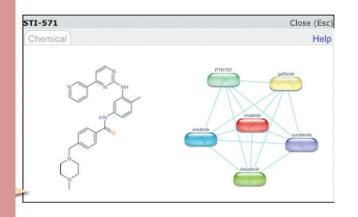




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Influence of synoptic pa

L. Shen<sup>1</sup>, L. J. Mickley<sup>1</sup>, and A. F <sup>1</sup>School of Engineering and Applied Sci

<sup>2</sup>Earth System Science Programme an

Abstract. We investigate the effect

over the eastern United States duri

daily MDA8 JJA ozone shows a bimo

the variability in daily weather. We leading EOF patterns explain 53 %

eastern United States associated w system when its west boundary is

Bermuda High and an enhanced Gr

activity, while the southern peak ap

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Influence of synoptic patterns on surface ozone variability over the eastern United States from 1980 to 2012

L. Shen et al.

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**AR** by Lu Shen on behalf of the Authors (30 Jul 2015) Author's response Manuscript

ED: Referee Nomination & Report Request started (02 Aug 2015) by Steven Brown

the total number of days the jet traverses the Midwest and northeast each summer. In the Midwest and northeast, we find that the correlation coefficient r between detrended mean JJA MDA8 ozone and the polar jet frequency ranges between -0.76 and -0.93 over 1980-2012depending on the time period selected, suggesting that polar jet frequency could provide a simple metric to predict ozone variability in future limate regimes. In the coutheast, the influence of the Bermuda High on mean 114 MDAS exerc depends on the location of its west edge. For



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Severe fever with thrombocytopenia syndrome, an eme borne zoonosis

Quan Liu PhD a b, Biao He PhD b, Si-Yang Huang PhD a, Feng Wei PhD c, Prof Xing-Quan Zhu PhD a PhD

Severe fever with thrombocytopenia syndrome (SFTS) is an emerging haemorrhagic fever that was first described in rural are: of China. The causative agent, SFTS virus (SFTSV), is a novel phlebovirus in the Bunyaviridae family. Since the first report in 2010, SFTS has been found in 11 provinces of China, with about 2500 reported cases, and an average case-fatality rate of 7.3%. The disease was also reported in Japan and Korea in 2012; Heartland virus, another phlebovirus genetically closely related to SFTSV, was isolated from two patients in the USA. The disease has become a substantial risk to public health, not only in China, but also in other parts of the world. The virus could undergo rapid evolution by gene mutation, reassortment, and homologous recombination in tick vectors and vertebrate reservoir hosts. No specific treatment of SFTS is available, and avoiding tick bites is an important measure to prevent the infection and transmission of SFTSV. This Review provides information on the molecular characteristics and ecology of this emerging tick-borne virus and describes the epidemiology, clinical signs, pathogenesis,

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### **INAPPROPRIATELY DELIVERED**

Mutational dynamics and phylogenetic utility of nonchloroplast DNA

Thomas Borsch · Dietmar Quand

Received: 29 April 2009/Accepted: 15 July 2009/Published online: 4 September 2009

Abstract Introns and spacers are a rich and well-appreciated information source for evolutionary studies in plants. Compared to coding sequences, the mutational dynamics of introns and spacers is very different, involving frequent microstructural changes in addition to substitutions of individual nucleotides. An understanding of the biology of sequence change is required for correct application of molecular characters in phylogenetic analyses, including homology assessment, alignment coding, and tree infer ence. The widely used term "indel" is very general, and different kinds of microstructural mutations, such as simple repeats, inversions, inverted repeats, and deletions, need to be distinguished. Noncoding DNA has been indispensable for analyses at the species level because coding sequences isually do not offer sufficient variability. A variety of introns and spacers has been successfully applied for phylogeny inference at deeper levels (major lineages of ingiosperms and land plants) in past years, and phylogenetic structure R in intron and spacer data sets usually outperforms that of coding-sequence data sets. In order to fully utilize their potential, the molecular evolution and applicability of the most important noncoding markers (the trnT-trnF region comprising two spacers and a group I

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group II intron in trnG; the group II introns in petD, rpl16 rps16, and trnK; and the atpB-rbcL and psbA-trnC spacers) are reviewed. The study argues for the use of noncoding DNA in a spectrum of applications from deep level phylogenetics to speciation studies and barcoding and aims at outlining molecular evolutionary principle

Keywords Spacers · Introns · Phylogenetic structure R

The application of noncoding chloroplast DNA sequence data in plant molecular systematics has been steadil increasing over the last decade. Sequencing of rapidly unravelling evolutionary patterns among closely related idea was to use universal amplification primers that anneal to conserved genes and thereby span more variable spacer. and introns. At about the same time, pronounced differ ences in mutational dynamics and consequently in levels o variability between coding and noncoding plastid region: were pointed out by Morton and Clegg (1993), Clegg et al. (1994), and others. As compared to coding genes, the conservation. Introns in particular possess a well-conserved secondary structure that leads to a mosaic of highly conserved and extremely variable parts (Cech 1988; Michel et al. 1989: Cech et al. 1994: Kelchner 2002: Borsch et al

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