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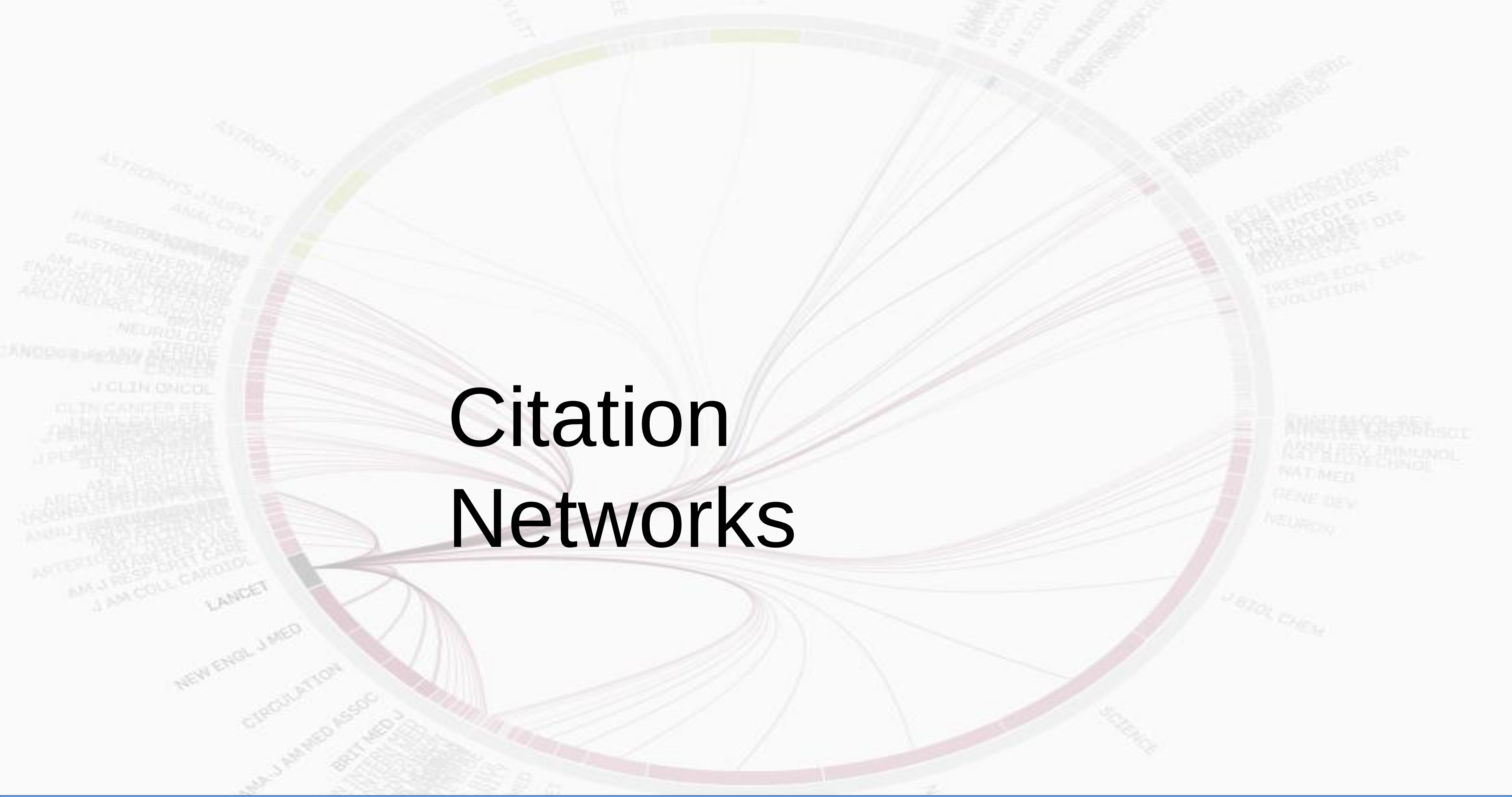

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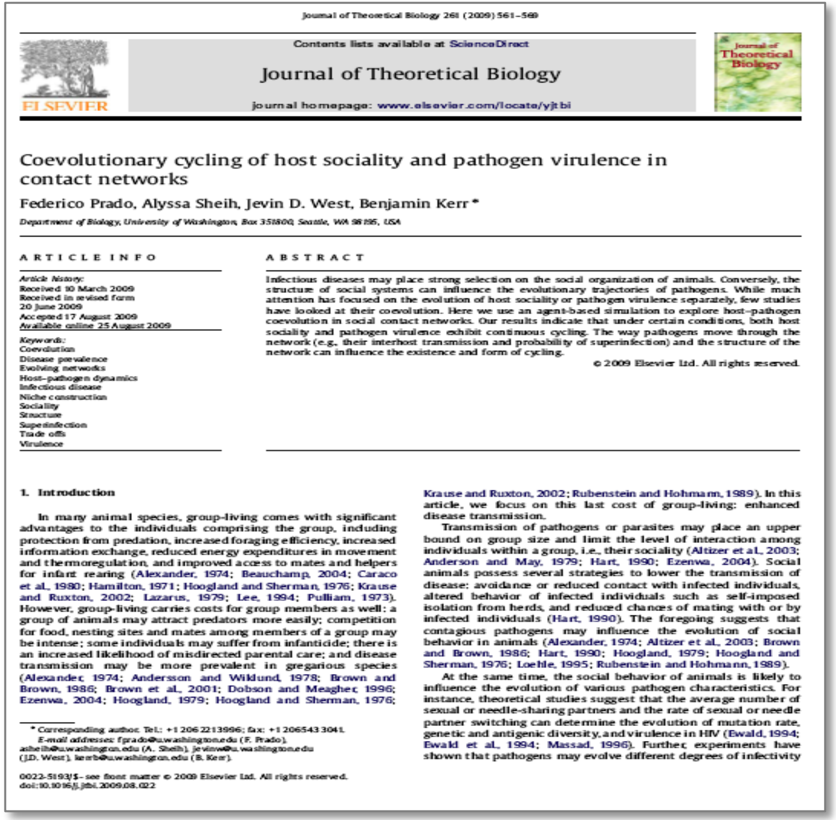
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Coevolutionary cycling of host sociality and pathogen virulence in contact networks

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ABSTRACT

Infectious diseases may place strong selection on the social organization of animals. Conversely, the structure of social systems can influence the evolutionary trajectories of pathogens. While much attention has focused on the evolution of host sociality or pathogen virulence separately, few studies have looked at their coevolution. Here we use an agent-based simulation to explore host–pathogen coevolution in social contact networks. Our results indicate that under certain conditions, both host sociality and pathogen virulence exhibit continuous cycling. The way pathogens move through the network (e.g., their interhost transmission and probability of superinfection) and the structure of the network can influence the existence and form of cycling.

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1. Introduction

In many animal species, group-living comes with significant advantages to the individuals comprising the group, including protection from predation, increased foraging efficiency, increased information exchange, reduced energy expenditures in movement and thermoregulation, and improved access to mates and helpers for infant rearing (Alexander, 1974; Beauchamp, 2004; Caraco et al., 1980; Hamilton, 1971; Hoogland and Sherman, 1976; Krause and Ruxton, 2002; Lazarus, 1979; Lee, 1994; Pulliam, 1973). However, group-living carries costs for group members as well: a group of animals may attract predators more easily; competition for food, nesting sites and mates among members of a group may be intense; some individuals may suffer from infanticide; there is an increased likelihood of misdirected parental care; and disease transmission may be more prevalent in gregarious species (Alexander, 1974; Andersson and Wiklund, 1978; Brown and Brown, 1986; Brown et al., 2001; Dobson and Meagher, 1996; Ezenwa, 2004; Hoogland, 1979; Hoogland and Sherman, 1976; Krause and Ruxton, 2002; Rubenstein and Hohmann, 1989). In this article, we focus on this last cost of group-living: enhanced disease transmission.

Transmission of pathogens or parasites may place an upper bound on group size and limit the level of interaction among individuals within a group, i.e., their sociality (Altizer et al., 2003; Anderson and May, 1979; Hart, 1990; Ezenwa, 2004). Social animals possess several strategies to lower the transmission of disease: avoidance or reduced contact with infected individuals, altered behavior of infected individuals such as self-imposed isolation from herds, and reduced chances of mating with or by infected individuals (Hart, 1990). The foregoing suggests that contagious pathogens may influence the evolution of social behavior in animals (Alexander, 1974; Altizer et al., 2003; Brown and Brown, 1986; Hart, 1990; Hoogland, 1979; Hoogland and Sherman, 1976; Loehle, 1995; Rubenstein and Hohmann, 1989).

At the same time, the social behavior of animals is likely to influence the evolution of various pathogen characteristics. For instance, theoretical studies suggest that the average number of sexual or needle-sharing partners and the rate of sexual or needle partner switching can determine the evolution of mutation rate, genetic and antigenic diversity, and virulence in HIV (Ewald, 1994; Ewald et al., 1994; Massad, 1996). Further, experiments have shown that pathogens may evolve different degrees of infectivity

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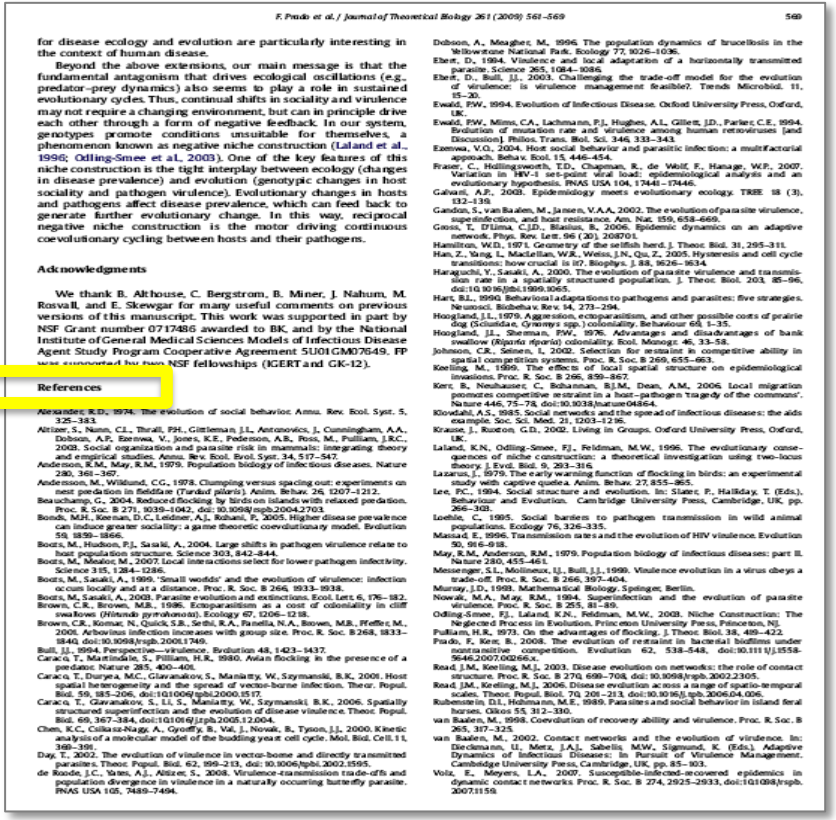
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for disease ecology and evolution are particularly interesting in the context of human disease.

Beyond the above extensions, our main message is that the fundamental antagonism that drives ecological oscillations (e.g., predator–prey dynamics) also seems to play a role in sustained evolutionary cycles. Thus, continual shifts in sociality and virulence may not require a changing environment, but can in principle drive each other through a form of negative feedback. In our system, genotypes promote conditions unsuitable for themselves, a phenomenon known as negative niche construction (Laland et al., 1996; Odling-Smee et al., 2003). One of the key features of this niche construction is the tight interplay between ecology (changes in disease prevalence) and evolution (genotypic change in host sociality and pathogen virulence). Evolutionary changes in hosts and pathogens affect disease prevalence, which can feed back to generate further evolutionary change. In this way, reciprocal negative niche construction is the motor driving continuous coevolutionary cycling between hosts and their pathogens.

Acknowledgments

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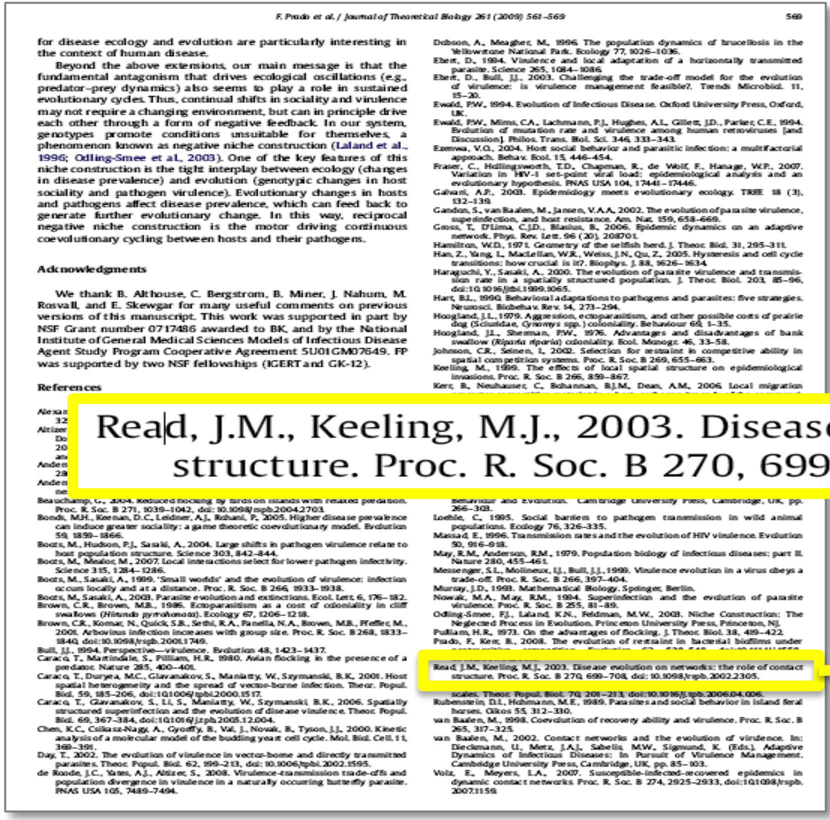

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As this paper exceeds the maximum length normally permitted, the authors have agreed to contribute to production costs.

Evolution of Virulence: a Unified Framework for Coinfection and Superinfection

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Models of the evolution of parasite virulence have focused on computing the evolutionarily stable level of virulence favored by tradeoffs within a host and by competition for hosts, and deriving conditions under which strains with different virulence levels can coexist. The results depend on the type of interaction between disease strains, such as single infection (immunity of infected individuals to other strains), coinfection (simultaneous infection by two strains), and superinfection (instantaneous takeover of hosts by the more virulent strain). We present a coinfection model with two strains and derive the superinfection model as the limit where individuals are rapidly removed from the doubly-infected class. When derived in this way, the superinfection model includes not only the takeover of hosts infected by the less virulent strain, but new terms which take into account the possibility of increased mortality of doubly-infected individuals. Coinfection tends to favor higher virulence and support more coexistence than the single infection model, but the detailed results depend sensitively on two factors: (1) whether and how the model is near the superinfection limit, and (2) the shape of the coinfection function (the function describing the rate at which a more virulent strain can infect a host). If the superinfection limit arises due to rapid mortality of doubly-infected hosts, there is a region of uninvadable virulence levels rather than coexistence. When the coinfection function is discontinuous, as in many previous models, neither the coinfection model nor the superinfection limit can support an evolutionarily stable virulence level. Piecewise differentiable and differentiable coinfection functions produce qualitatively different results, and we propose that these more general cases should be used to study evolution of virulence when other mechanisms like space, population dynamics, and stochasticity interact.

1. Introduction

Disease models provide an ecological and evolutionary theatre where a small troupe of actors act out dramas of life and growth before an ever-increasing audience. The actors are the host (often reduced to the status of a stage prop to be destroyed during the course of the performance) and one or more diseases seeking to exploit the host to further their own reproduction. When can several such diseases live together, either in the same host or the same population of hosts? Under what circumstances should the diseases reduce the degree of harm caused to the host? The conclusions of these

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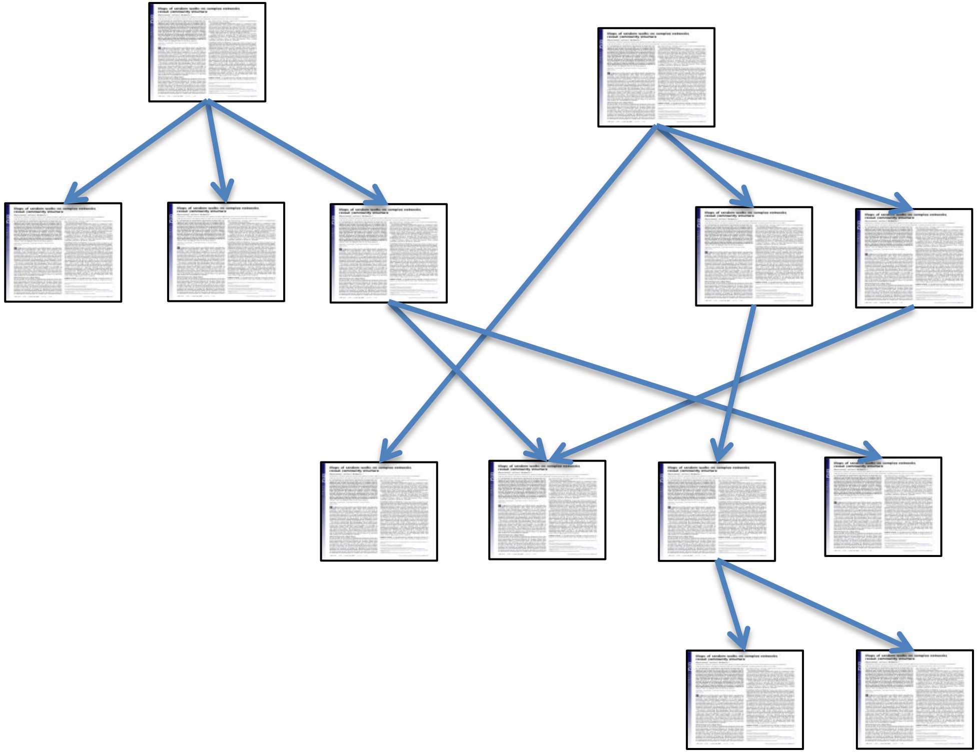

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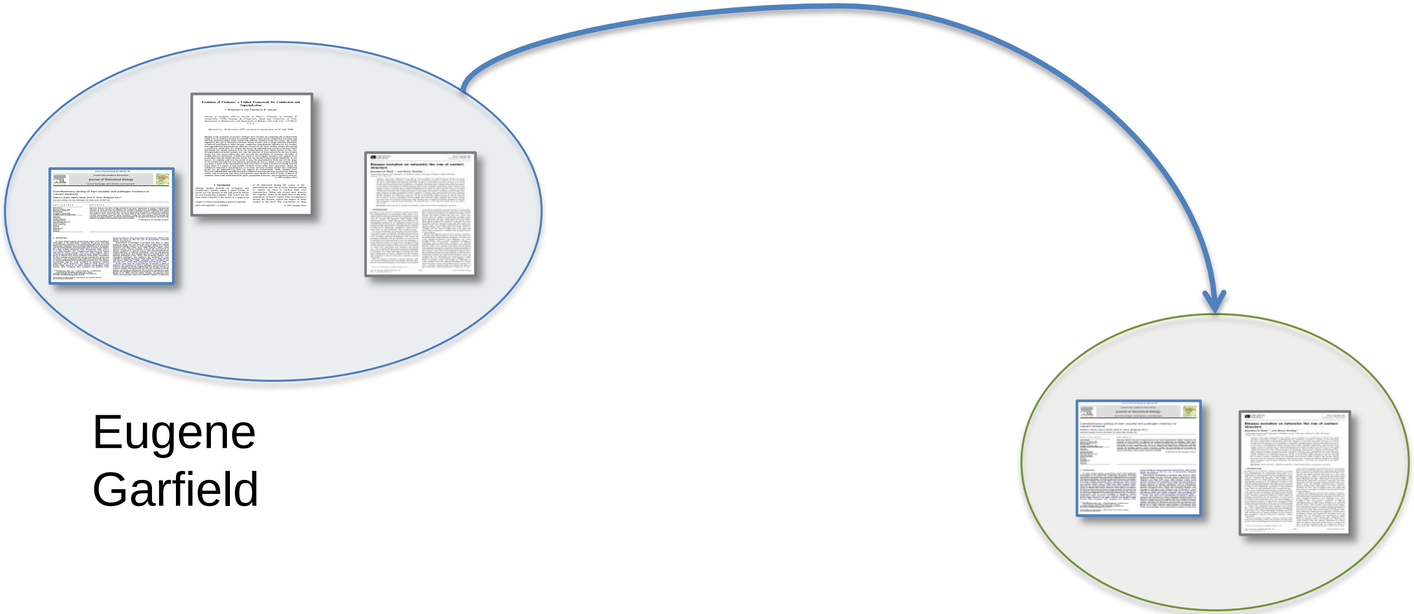

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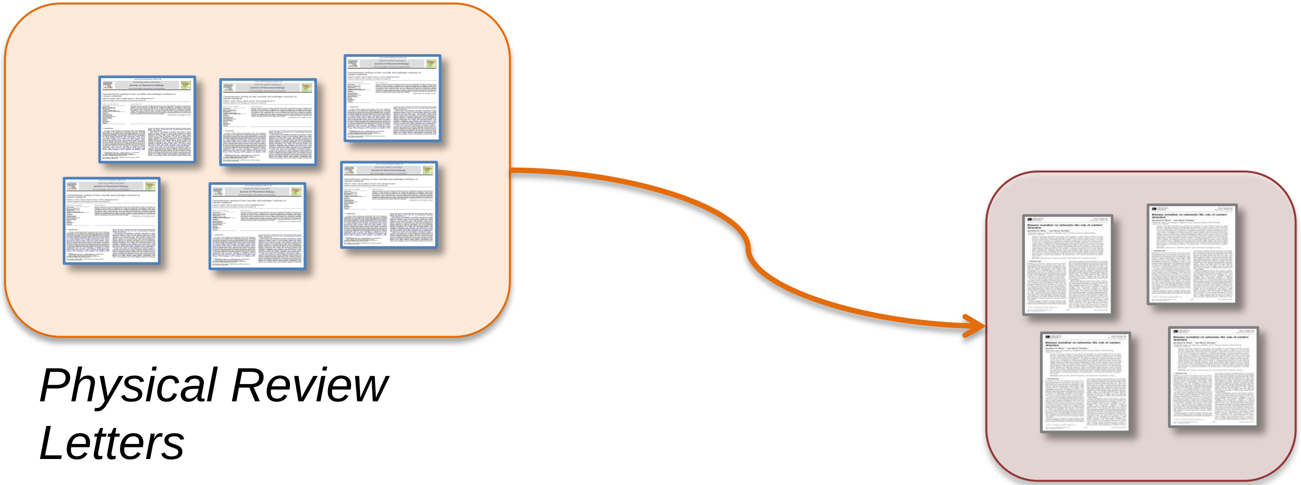

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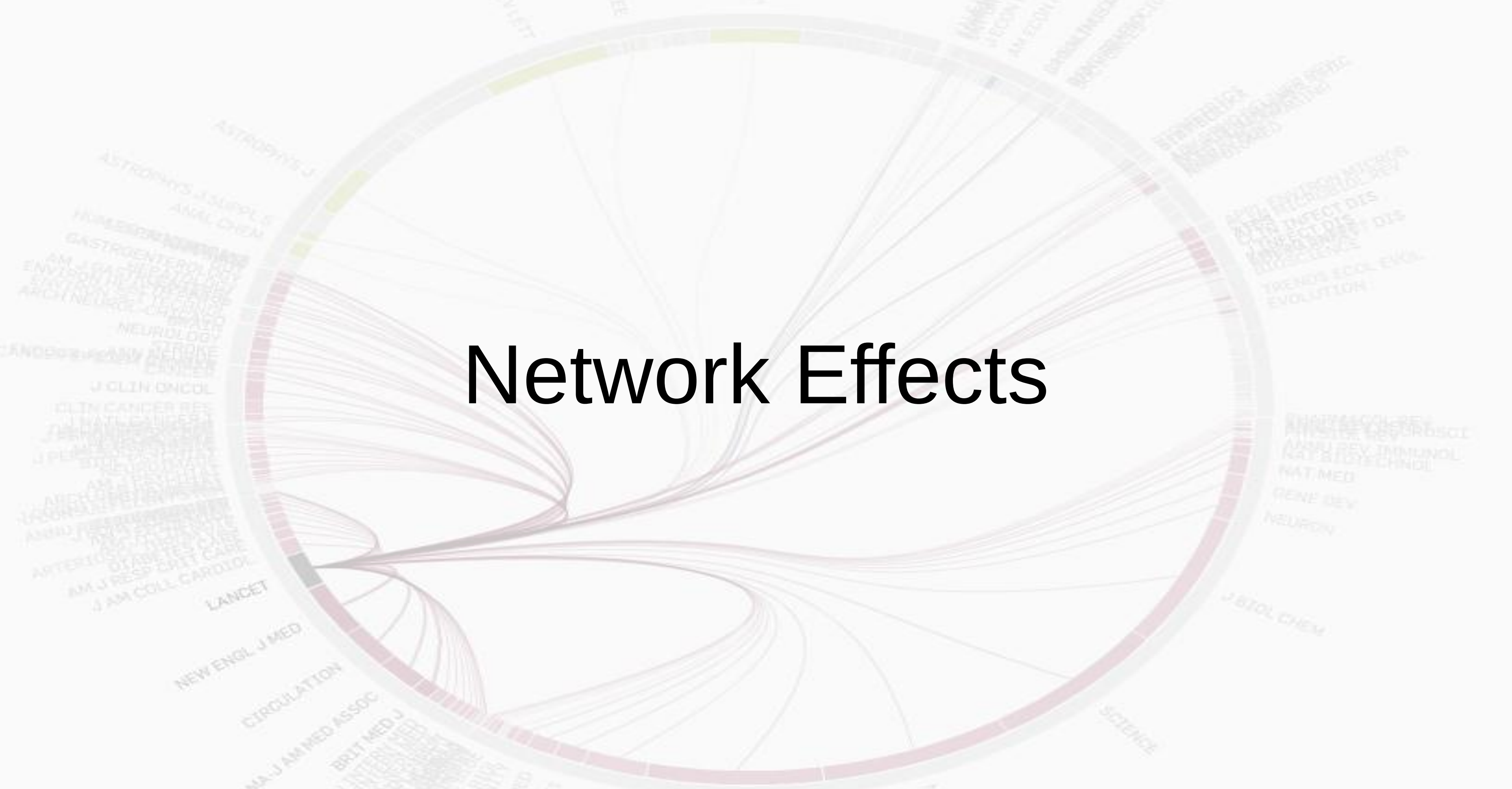

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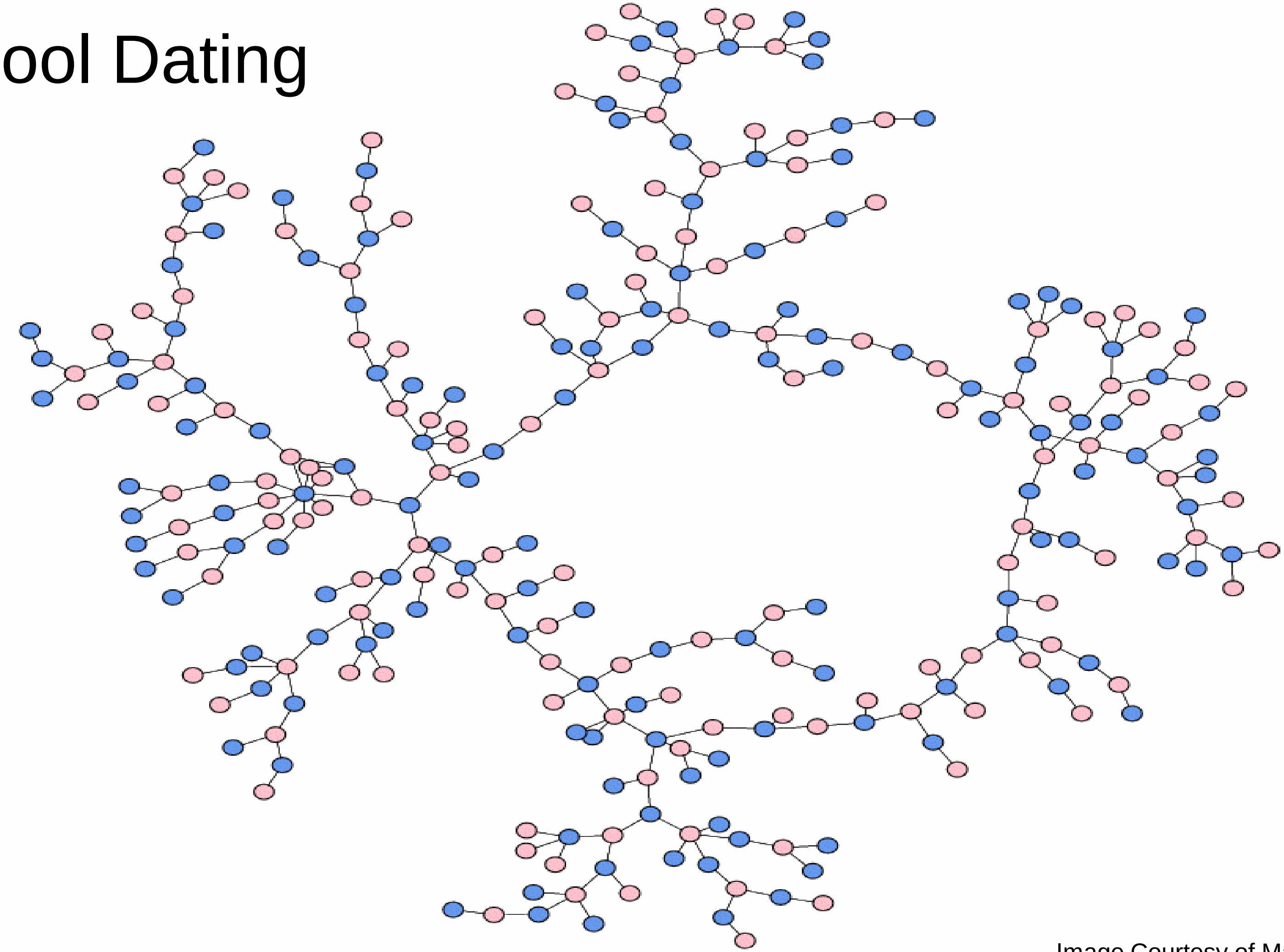

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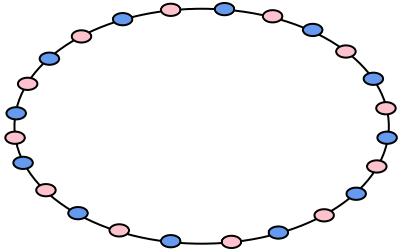
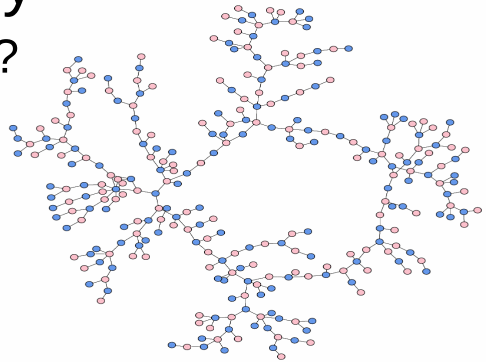
High School Dating Network

System Scale: Network Topology Matters

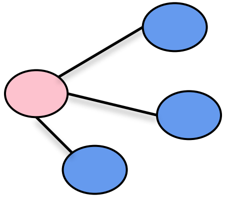
Q: Which network would *MONO* prefer?

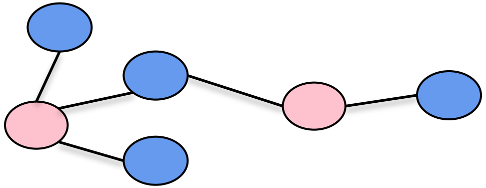




Individual Scale: Connection Source Matters

Q: Which girl/boy should I date if I don’t want *MONO*?





Who is the most ‘influential’ high school student?

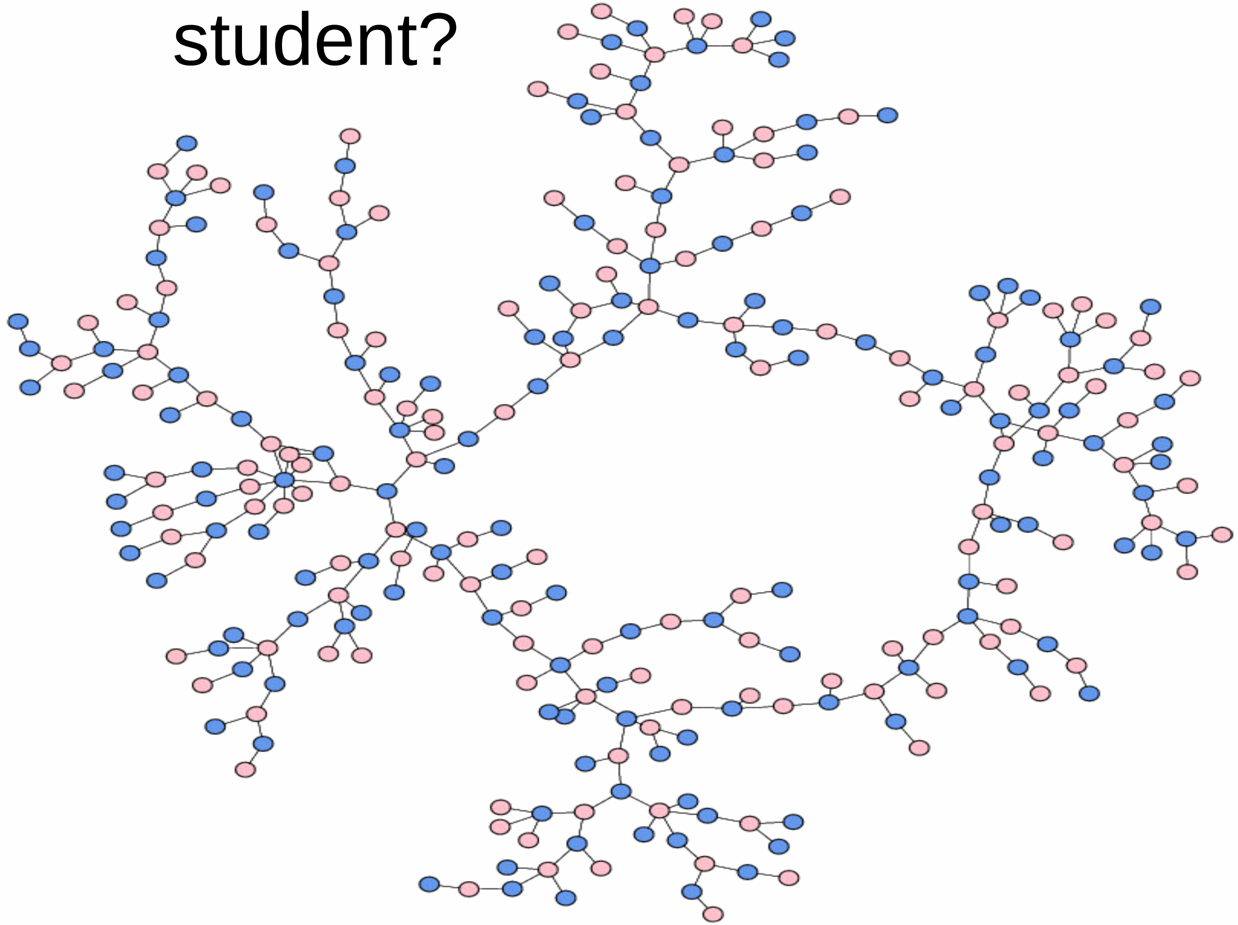
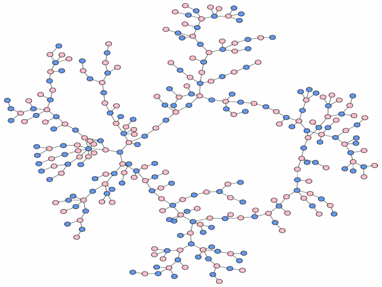
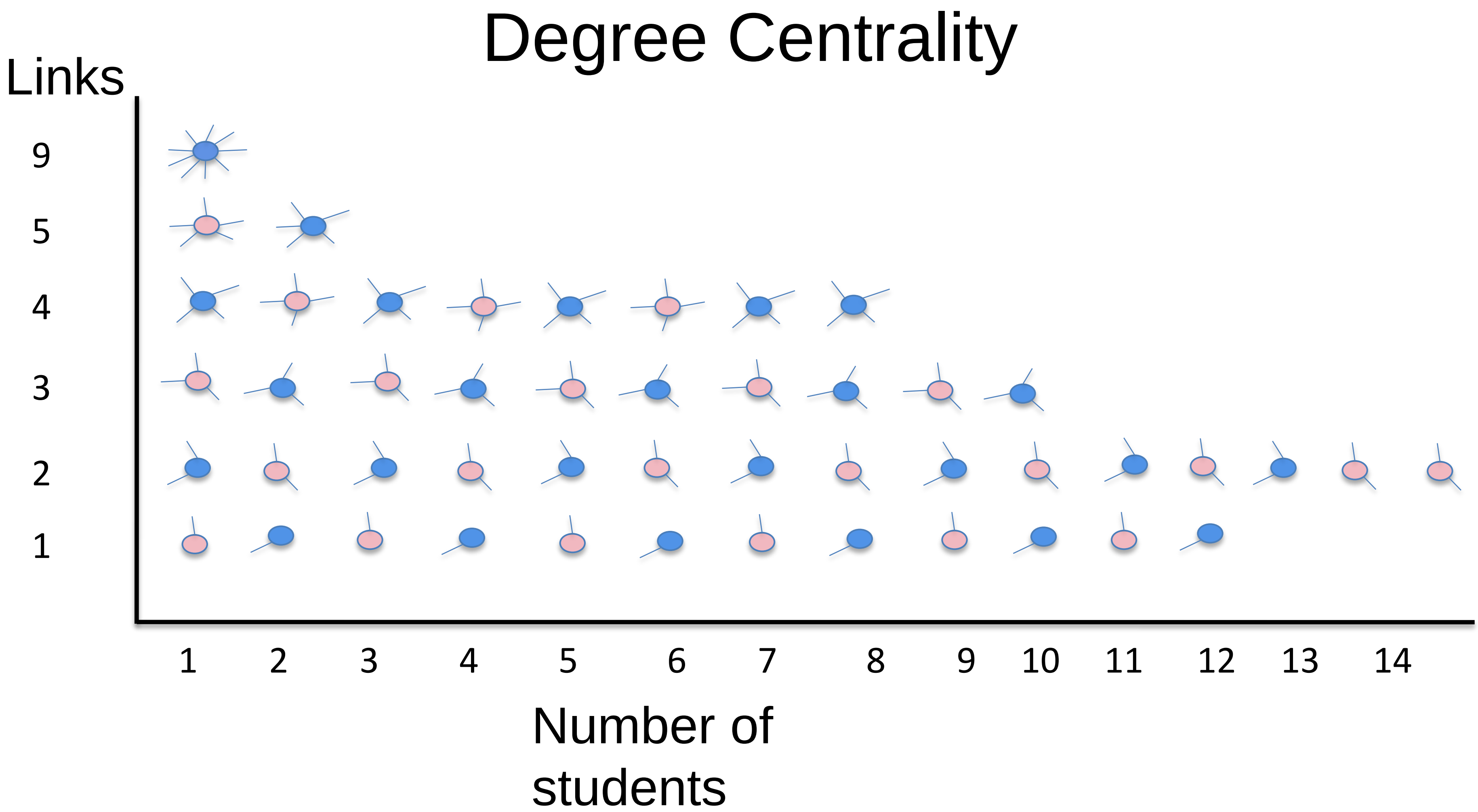


Image Courtesy of Mark

Degree Centrality

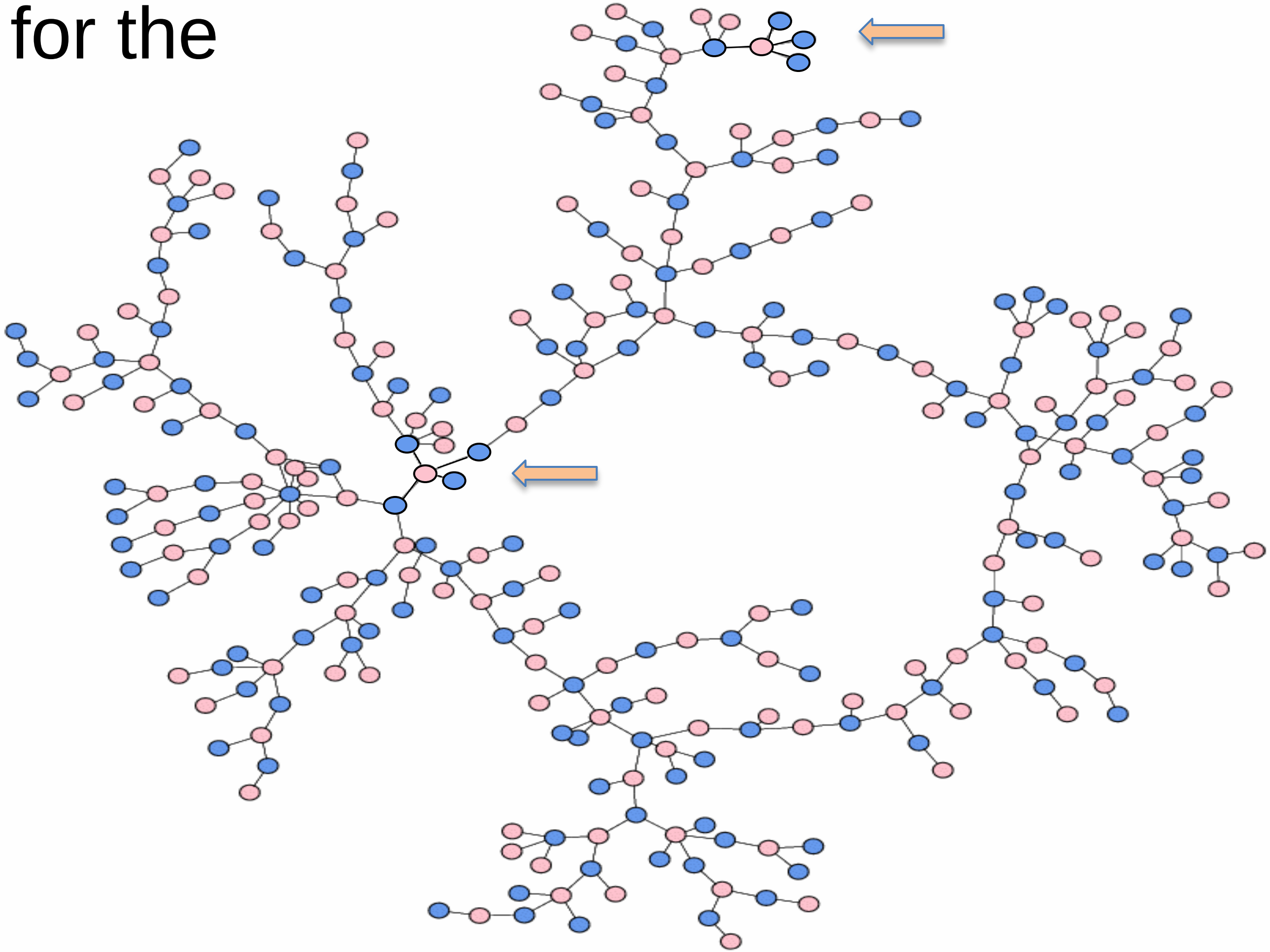
Eigenvector Centra





Degree Centrality
Links
9
5
4
3
2
1
1
2
3
4
5
6
7
8
9
10
11
12
13
14
Number of students

Accounting for the Network

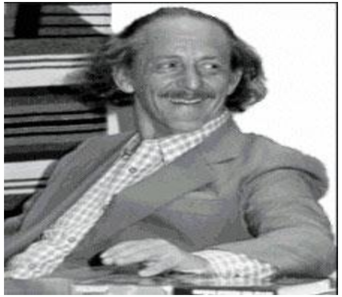


The Network Matters

This network property of the scholarly literature was largely ignored over the first century of scholarly evaluation.

How can we extract this information in order to better measure information *flow*?

Impact factor



Garfield, *Science* (1955)

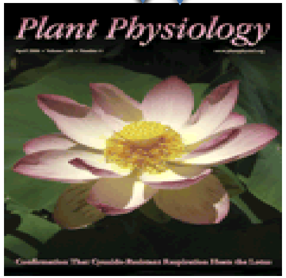
$$\frac{\text{Cites in 2010 to articles in 2009 or 2008}}{\text{Number of articles in 2009 and 2008}}$$

Impact Factor

2010

3 citations

2008 - 2009

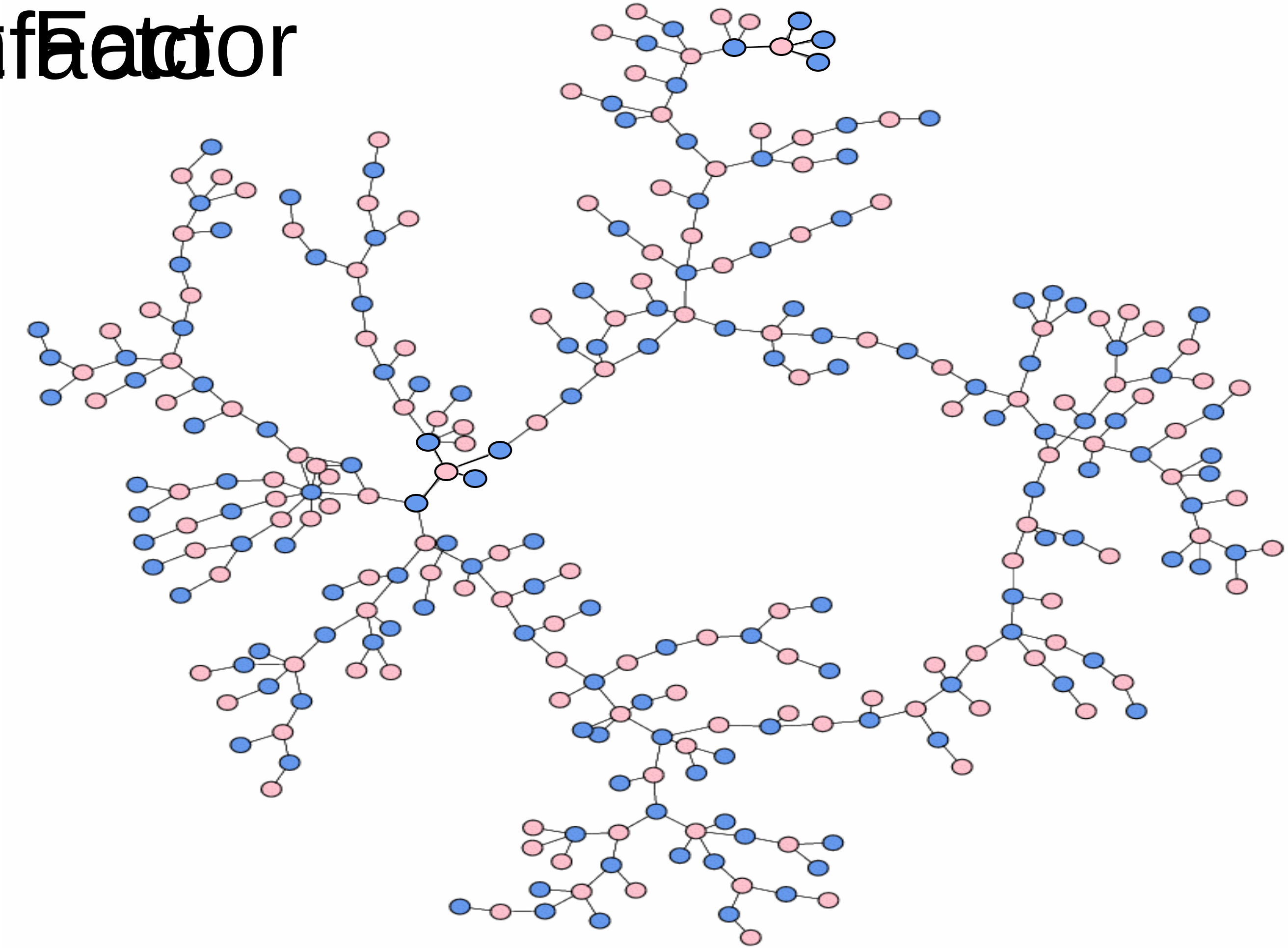


2
articles

$$IF_{2010} = \frac{3}{2}$$

Impact Factor

Eigenfactor



Citation Networks, Scaling Up

Network Effects, Centrality

Eigenfactor, Article Influence

Mapping, Future Directions

Eigenfactor algorithm

$$P = \alpha H + (1 - \alpha)\, a.e^{T}$$

Matrix representing the random walk over citations

Probability of not teleporting

Cross-citation Matrix dictating the structure of the citation network

Probability of teleporting to completely new journal weighted by the number of articles in that journal

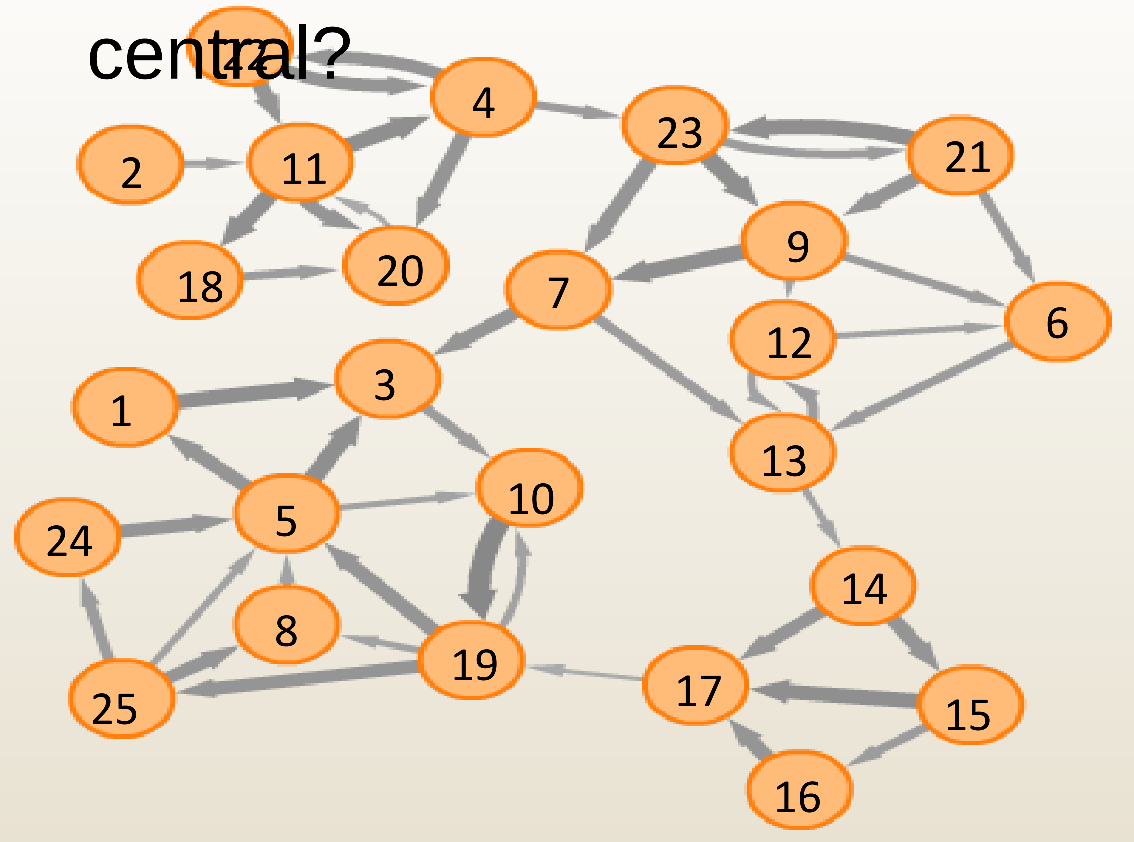
$$EF = 100 \frac{Hp}{\sum_i [Hp]_i}$$

Leading eigenvector of the random walk matrix P.

Normalization

Bergstrom (2007); West et al (2010)

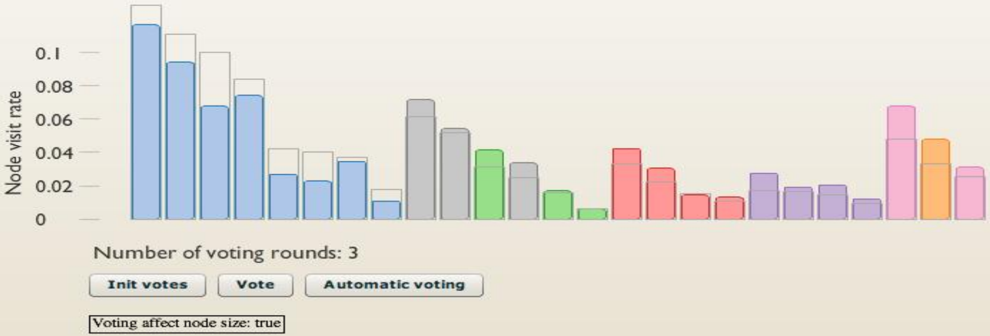
Which node is the most central?



25 nodes and 42 **weighted, directed** links

Deterministic: iterative voting

Stochastic: random walker



Applet: Daniel Edler

Eigenfactor – Top Ten Journals in Science ([illegible])

Rank	Journal	Eigenfactor
1	Nature	1.76
2	PNAS	1.70
3	Science	1.58
4	Journal of Biological Chemistry	1.33
5	Physical Review Letters	1.28
6	Journal of the American Chemical Society	0.95
7	Physical Review B	0.77
8	Applied Physics Letters	0.72
9	New England Journal of Medicine	0.68
10	Cell	0.67

Total number of journals ranked = [illegible]

Eigenfactor – Top Ten Journals in Science (2008)

Rank	Journal	Eigenfactor
1	Nature	1.76 %
2	PNAS	1.70 %
3	Science	1.58 %
4	Journal of Biological Chemistry	1.33 %
5	Physical Review Letters	1.28 %
6	Journal of the American Chemical Society	0.95 %
7	Physical Review B	0.77 %
8	Applied Physics Letters	0.72 %
9	New England Journal of Medicine	0.68 %
10	Cell	0.67 %

Total number of journals ranked = 8,222

$$\text{Article Influence} = \frac{\text{Eigenfactor}}{\text{\# articles}}$$

Article Influence – Top Ten Journals in Science

Rank	Journal	Article Influence
1	Review of Modern Physics	24.9
2	Annual Review of Immunology	24.7
3	Annual Review of Biochemistry	20.9
4	Nature Reviews Molecular Cell Biology	20.0
5	Annual Review of Neuroscience	18.9
6	Cell	18.9
7	New England Journal of Medicine	18.8
8	CA – A Cancer Journal for Clinicians	17.5
9	Nature	17.3
10	Science	16.3

Total number of journals ranked =

eigenFACTOR.org™

RANKING AND MAPPING SCIENTIFIC KNOWLEDGE

start a search

search by

journal name

Eigenfactor™ subject category — All

year — 2007

Search

Use advanced search to search by Thomson JCR subject categories, publisher, and other fields.

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2008 JCR Science Edition

Journal Summary List

Journal Title Changes

Journals from: **subject categories EVOLUTIONARY BIOLOGY** VIEW CATEGORY SUMMARY LIST

Sorted by: ArticleInfluence(TM) Score SORT AGAIN

Journals 1 - 20 (of 39) [1 | 2] **Page 1 of 2**

MARK ALL | UPDATE MARKED LIST

Ranking is based on your journal and sort selections.

Mark	Rank	Abbreviated Journal Title *(linked to journal information)*	ISSN	JCR Data						*Eigenfactor*™ Metrics	
				Total Cites	Impact Factor	5-Year Impact Factor	Immediacy Index	Articles	Cited Half-life	*Eigenfactor*™ Score	*Article Influence*™ Score
	1	ANNU REV ECOL EVOL S	1543-592X	10653	10.161	17.176	0.133	30	>10.0	0.02416	8.322
	2	TRENDS ECOL EVOL	0169-5347	16830	11.904	17.188	1.913	92	7.4	0.06469	7.846
	3	SYST BIOL	1063-5157	7636	7.833	12.743	0.946	74	5.9	0.03104	4.668
	4	MOL BIOL EVOL	0737-4038	20661	7.280	6.846	1.259	263	7.0	0.08141	2.951
	5	AM NAT	0003-0147	21622	4.670	5.508	0.784	190	>10.0	0.04815	2.586
	6	EVOLUTION	0014-3820	25826	4.737	5.427	0.798	247	>10.0	0.06063	2.197
	7	BMC EVOL BIOL	1471-2148	2562	4.050	4.373	0.508	331	2.2	0.02196	1.888
	8	MOL ECOL	0962-1083	19960	5.325	5.966	1.506	403	5.5	0.06926	1.811
	9	PALEOBIOLOGY	0094-8373	2761	2.800	3.063	0.688	32	>10.0	0.00728	1.739
	10	CLADISTICS	0748-3007	2966	3.515	4.881	0.647	51	>10.0	0.00576	1.625
	11	J EVOLUTION BIOL	1010-061X	5691	3.471	3.757	0.767	180	5.0	0.02980	1.556
	12	EVOL DEV	1520-541X	1424	3.627	3.679	0.634	71	4.5	0.00879	1.513

Done

Mapping

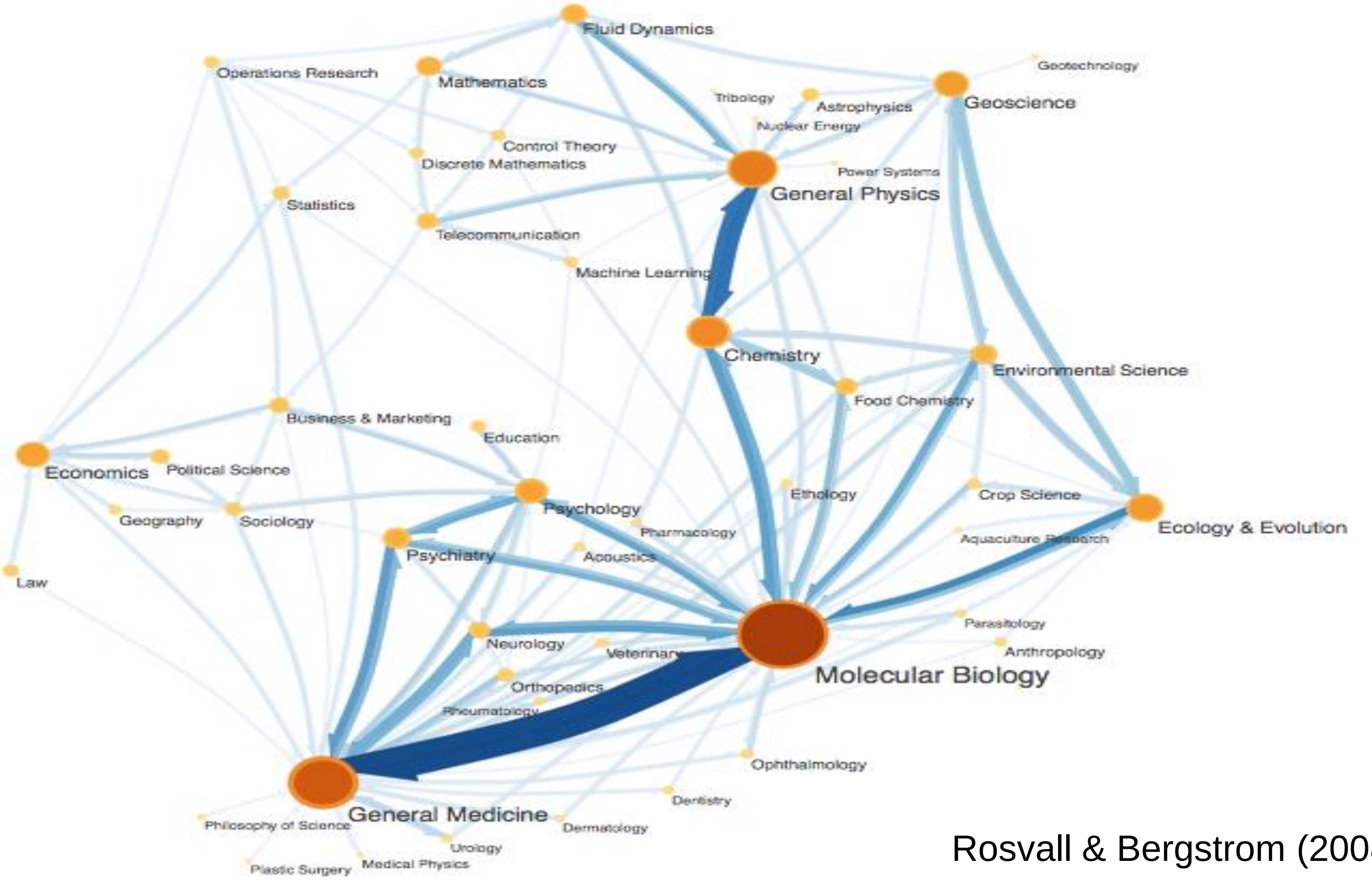

ASTROPHYS J
ANAL CHEM
NEUROLOGY
STROKE
J CLIN ONCOL
LANCET
NEW ENGL J MED
CIRCULATION
BRIT MED J
SCIENCE
J BIOL CHEM
NEURON
GENE DEV
NAT MED
TRENDS ECOL EVOL
EVOLUTION

good maps simplify
and highlight
relevant
structures

The Map Equation

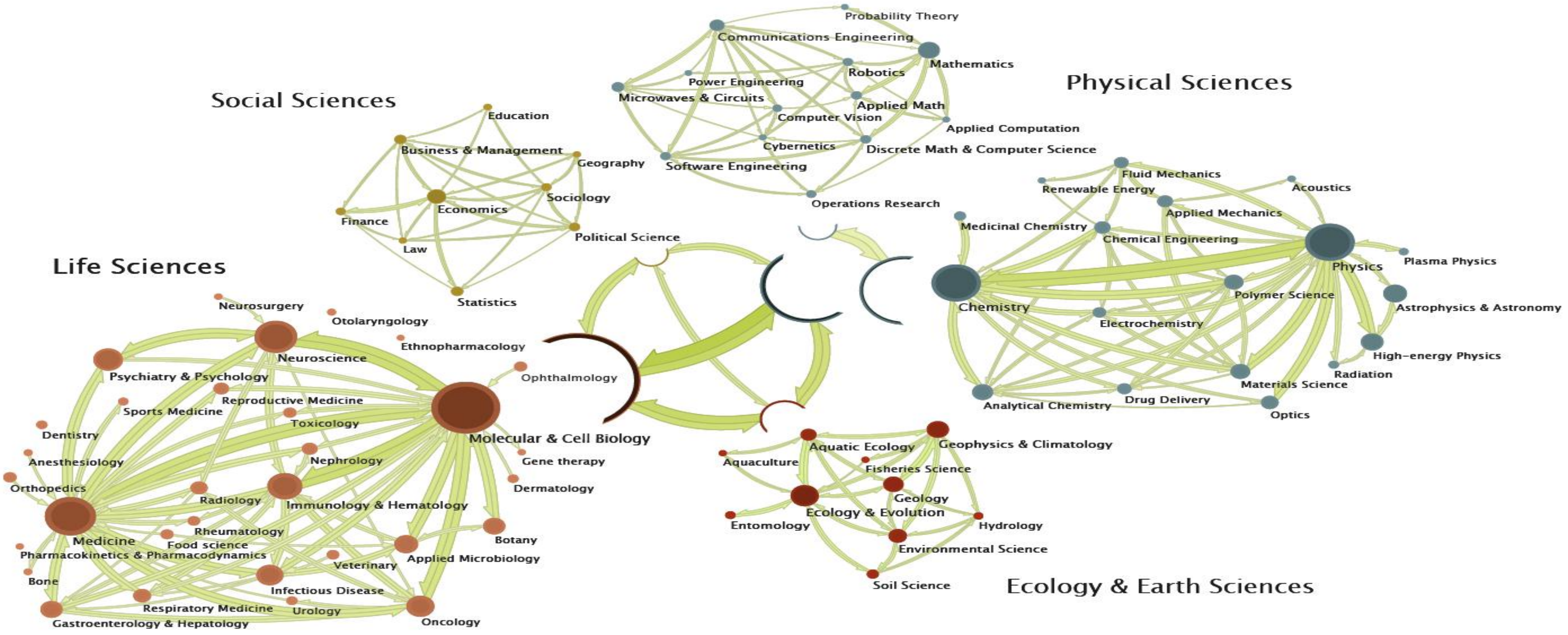
$$L(\mathsf{M}) = q_{\curvearrowright} H(\mathcal{Q}) + \sum_{i=1}^{m} p^{i}_{\circlearrowright} H(\mathcal{P}^{i})$$

The map equation tells us the description length for a particular modular structure



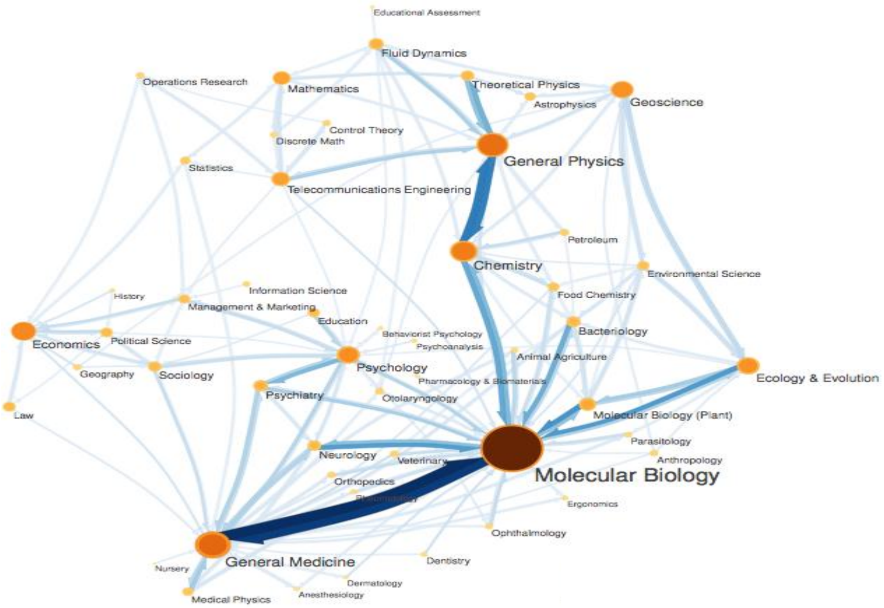
Rosvall & Bergstrom (2008)

Hierarchical Maps



Rosvall & Bergstrom (2011)

1995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Control Theory
Discrete Math
General Physics
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Telecommunications Engineering
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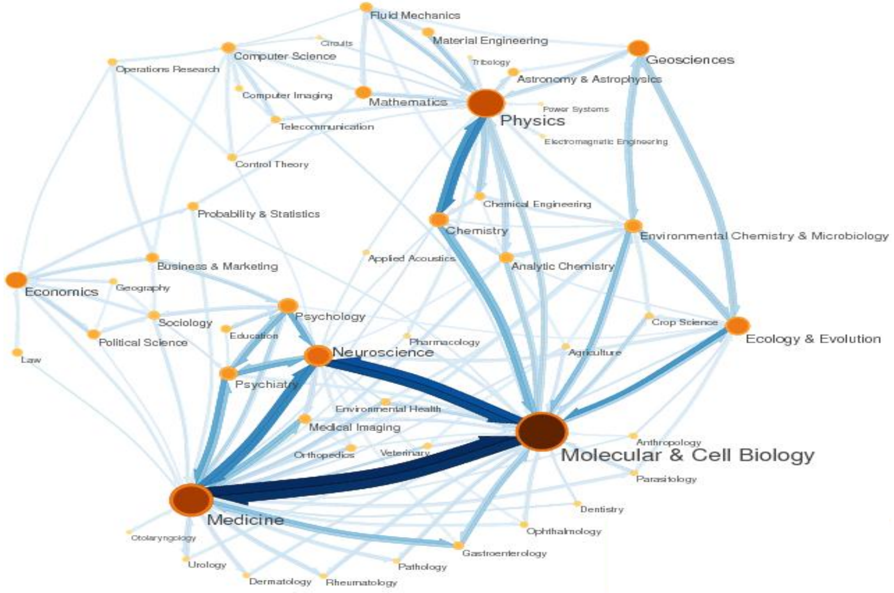
History
Information Science
Food Chemistry
Management & Marketing
Education
Bacteriology
Behaviorist Psychology
Psychoanalysis
Economics
Political Science
Animal Agriculture
Psychology
Geography
Sociology
Pharmacology & Biomaterials
Ecology & Evolution
Psychiatry
Otolaryngology
Law
Molecular Biology (Plant)
Parasitology
Neurology
Veterinary
Anthropology
Molecular Biology
Orthopedics
Ergonomics
Ophthalmology
Dentistry
Nursery
General Medicine
Dermatology
Medical Physics
Anesthesiology

2004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Electromagnetic Engineering
Control Theory
Chemical Engineering
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Economics
Geography
Psychology
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Crop Science
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Neuroscience
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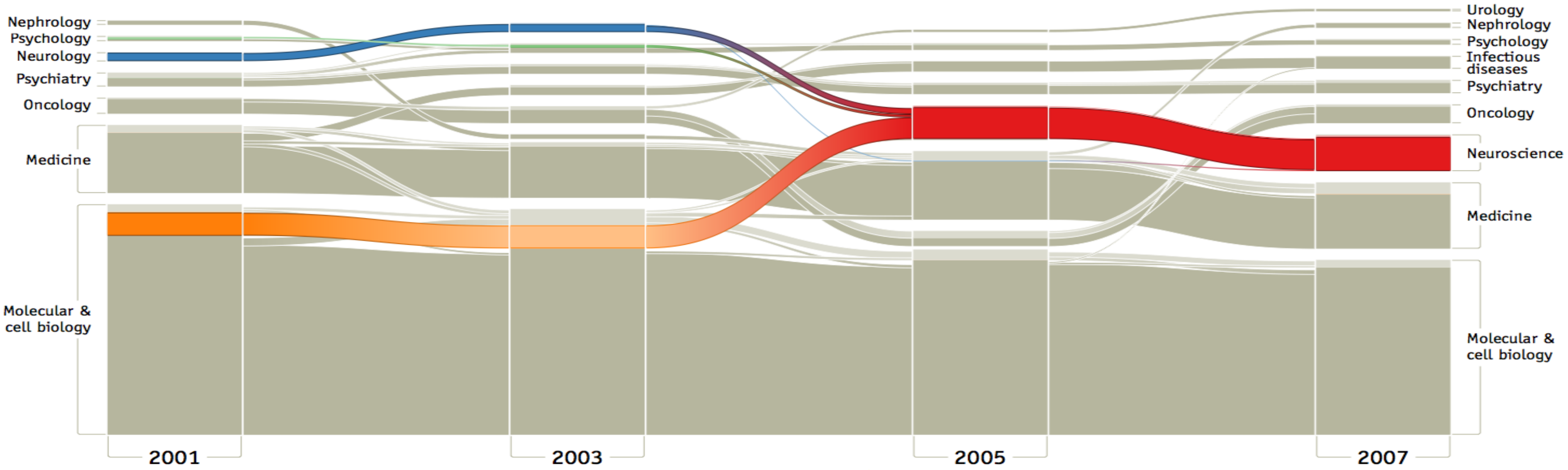
Gastroenterology
Urology
Pathology
Dermatology
Rheumatology



Rosvall & Bergstrom (2010)

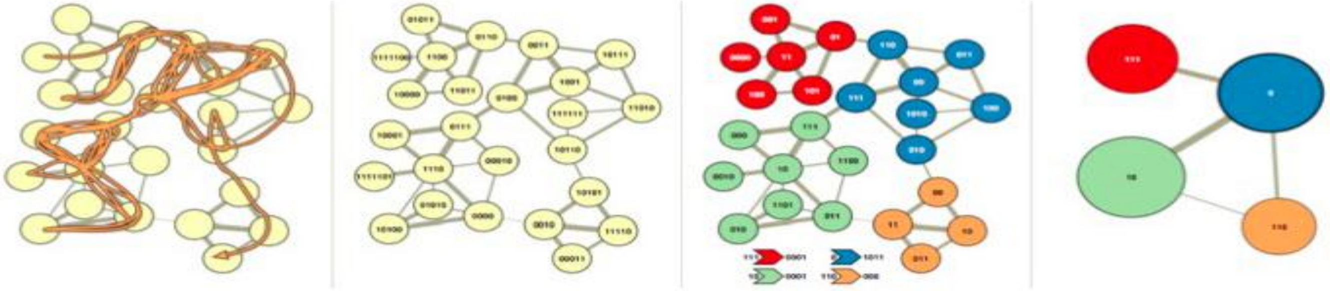
Future Directions

How can we better
evaluate the scholarly literature?

How can we better
navigate the scholarly literature?

Eigenfactor and Microsoft Academic Research

Home | Recommend | Map: Journals | Map: Papers | Explore | Rank | Categorize | About



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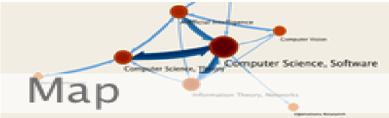
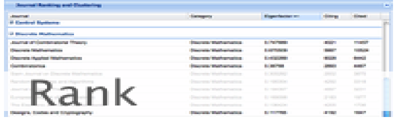
Microsoft Academic Search

By uncovering the hierarchical structure of scholarly citation, we can identify key papers pertaining to any search query. For a reader new to the field we can find the classic and foundational papers; for an expert we can find the latest innovations.



From patterns of scholarly citation, we use Rosvall and Bergstrom's map equation to chart the topography of science and the relations among fields and subfields.
[journal map] [paper map]

By integrating a hierarchical clustering of citation networks with semantic analysis, we develop a scalable map of scientific fields and the key research terms and topics therein.



Scientific influence is often quantified using simple citation counts, but the structure of a citation network provides far more information than can be revealed by these simple counts. This is principle behind the Eigenfactor metrics; we can better rank the importance of scientific journals or papers by viewing them in the context of the full citation network.

Three things to remember…

1. The scholarly literature forms a vast network
2. There is a wealth of information in the *structure* of network
3. Eigenfactor is a *network* metric that ranks scholarly journals

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