THE BIOLOGICAL PHYSICIST

The Newsletter of the Division of Biological Physics of the American Physical Society

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In this Issue

It's Not Too Late!

A call was recently sent out asking for proposals for Focus Sessions and Symposia for the March 2004 APS meeting, with a deadline of June 5th. Many DBP members found themselves scrambling to put proposals together at what they thought was the eleventh hour.

However, it turns out that this was not, in fact, the eleventh hour! APS's deadline for receiving Symposium Proposals from the Division is October 10th. With nearly five months to go, the DBP Program Chair has decided to extend the deadline for Symposium submission to September 12th. A formal announcement extending the deadline should be sent out to the membership soon.

SB

ICAM Conference Announcement: Frontiers in Biological Physics

Greg Boebinger and David Pines

Dear Colleagues,

We write to bring to your attention the forthcoming Institute for Complex Adaptive Matter (ICAM) Symposium, **"Frontiers in Biological Physics: Signaling Complexes, Membranes, and the Cytoskeleton,"** that will take place in Snowmass and Aspen, Colorado, July 25-28, 2003. We encourage you to attend and to bring the attached program of the Symposium and registration information to the attention of those of your colleagues in both correlated matter and biological physics who might be interested in attending.

The Symposium, supported in part by a Grant to ICAM from the National Science Foundation, will begin at 9:00 a.m. on Saturday, July 26, 2002, and conclude by 1:00 p.m. on July 28. There will be a registration fee of $175.00 that will cover the opening reception and dinner on Friday, July 25, breakfast and lunch on July 26 and July 27, the Symposium banquet on July 26, and breakfast on July 28. We have secured a block of hotel rooms for participants for the nights of July 25 through July 27. Registration and hotel information are given below.

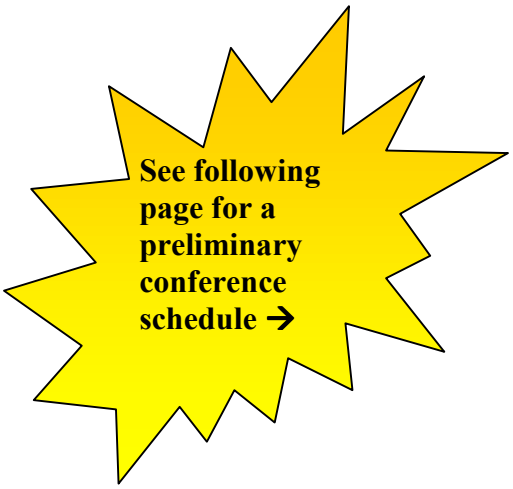
The talks at the Symposium will be either lectures on a particular area, or a more general review, followed by extensive discussions. The most important aspect of this Symposium that differentiates it from traditional meetings is that discussions will be allotted about the same time as the formal lectures.

We have reserved rooms at the Silvertree Hotel in Snowmass at the rate of $89.00 single, and $89.00 double. Check-in time is 4:00 p.m., and check-out time is 10:00 a.m. You can make your reservations by calling 1-800-525-9402 or by fax at 970-923-5494. When making your reservations, please refer to "Frontiers in Biological Physics Symposium." **Please note the cut off date for making your reservations is July 1, 2003.**

We very much hope that you can join us for what promises to be an exciting and stimulating symposium, one that provides not only a reading of the way biologists and the biological physics community look at the search for organizing principles behind emergent behavior, but gives us a sense of exciting and promising paths to pursue in that search. To register, please complete the attached registration form and return to Rose Romero, the ICAM conference coordinator, at rbromero@lanl.gov.

We look forward to your response to this invitation at your early convenience. With all good wishes,

Yours sincerely,
Greg Boebinger and David Pines



2003 ICAM Symposium on Frontiers in Biological Physics: Signaling Complexes, Membranes, and the Cytoskeleton

Aspen and Snowmass, Colorado

July 25-28, 2003

Program as of 5-14-03

Friday, July 25, 2003
Silvertree Lodge, Snowmass
6:00pm — **Welcome Reception and Dinner**

Saturday, July 26, 2003
Silvertree Lodge, Snowmass
9:00am-5:30pm, — **Signaling (A. Ruckenstein, chairman)**
J. Stock, Princeton,
J. Groves, UC Berkeley
D. Bray, Cambridge
A.McCammon, UCSD
Discussion leader: P.Wolynes, UCSD

7:00pm — **Symposium Banquet**

Sunday, July 27, 2003
Aspen Center for Physics, Aspen
9:00am-3:30pm — **Cytoskeleton Dynamics (S. Gross, chairman)**
T. Pollard, Yale
J. Kas, Leipzig
A. Mogilner, UC Davis
D. Purich, U Florida, Gainesville
Discussion leader: F. Julischer, MPI for Complex Systems, Dresden

4:00pm — **MAA Concert, Aspen**

Monday, July 28, 2003
Silvertree Lodge, Snowmass
9:00am-1:00pm — **Membranes (H. Levine, chairman)**
C. Safinya,UCSB
R. Bruinsma,UCLA
P. Nelson, Penn
Discussion leader: K-Y Lee, Chicago

Lewis Wolpert, Univ. College, London - conference summary and prognosis

Systems Biology at Harvard's Bauer Center for Genomics Research

Laura Garwin

The Bauer Center for Genomics Research, at Harvard University, is a new interdepartmental initiative whose goal is to identify general principles underlying the structure, behavior and evolution of cells and organisms. Although we carry the "genomics" label, what we do could just as well be described as "systems biology". The unifying themes of our research are a system-level approach to biology, and close interactions among experiment, theory and computation. Our scientists come to biology from many disciplines, including physics, mathematics and computer science.

Research at the Bauer Center is done by Fellows — young scientists appointed for up to five years, who lead their own small research groups. The Fellows form a truly collaborative group of scientists; at last count, there were ten pairwise collaborations among the eight groups currently in the center. Interactions among the Fellows are promoted both by what they share (an interest in uncovering general principles in biology, and a commitment to interdisciplinary research) and by their differing backgrounds and expertise, allowing them to tackle problems together in ways that none of them would have devised separately.

Physicists and other quantitatively inclined scientists can contribute to molecular and cell biology in various ways — for example, by formulating mathematical and computer models and analytical tools, developing sensitive and accurate techniques for data collection, and bringing to the subject a predilection for reducing complex problems to their essentials. The eleven Bauer Center Fellows include two mathematicians, a biophysicist, and a computational biologist who are already at the center, and two physicists who will be arriving over the next eight months. Their work exemplifies these types of contribution.

Lani Wu and Steven Altschuler, pure mathematicians by training who spent six years at Microsoft working on problems ranging from video compression to speech recognition before being drawn into biology, have a number of collaborations with experimental biologists both inside and outside the center. In one, with Professor Rong Li of Harvard Medical School, they are investigating the self-organizing processes that underlie the establishment and maintenance of cell polarity in budding yeast. Experiments had suggested the existence of a positive feedback loop involving an activated form of the protein Cdc42, required for cell polarization, and the Cdc42-dependent assembly of actin cables,

The Bauer Center for Genomics Research.

which deliver Cdc-42 to the cell membrane. In this picture, a stochastic increase in the local concentration of activated Cdc42 on the membrane increases the probability of actin polymerization, and hence further promotes the accumulation of Cdc42 at the site. Wu and Altschuler simulated this process mathematically, and were able to predict

Mathematicians Steven Altschuler and Lani Wu.

conditions (subsequently verified experimentally) in which yeast cells would develop one, two or more polar caps of Cdc42 (ref. 1). In nature, cell polarization is usually controlled by spatial signals, but the intrinsic polarization mechanism modelled by Wu and Altschuler may be used to amplify a small initial asymmetry.

Roy Kishony, a postdoctoral fellow with Stanislas Leibler at Rockefeller University, will be joining the center as a Fellow in August. Kishony has a Ph.D. in theoretical physics (his thesis was about the ignition criterion in inertial confinement fusion), but for the past four years he has been working as an experimental biologist. Kishony is addressing fundamental questions in evolutionary genetics, using new techniques for obtaining quantitative physiological data. In particular, he has developed a bioluminescence technique that allows accurate, automated measurements of bacterial growth rates at very low cell densities, and has used the technique to study the interaction between environmental stresses and deleterious mutations in the bacterium *E. coli* (ref. 2). At the Bauer Center, Kishony will extend this work to budding yeast, using his growth rate assay to perform a comprehensive and quantitative perturbation analysis of fitness with respect to both internal (genetic) and external (environmental) perturbations. He will use these data to reconstruct genetic circuits in yeast, and to generate testable hypotheses regarding the nature of interactions between pairs of genes.

Another physicist making the transition into biology is Sharad Ramanathan, a theoretical physicist on the technical staff at Bell Labs, who will be starting as a Bauer Center Fellow early next year. Ramanathan hopes to transfer insights from work he has done on electronic communication networks to the problem of signal transduction in biology. In particular, he is interested in questions

Meeting of minds: the Bauer Center café.

related to fidelity and cross-talk in the mitogen-activated protein (MAP) kinase cascades in yeast. Another Fellow, biophysicist Kurt Thorn, is approaching signaling in yeast from a different angle, developing fluorescence-based techniques such as fluorescence resonance energy transfer (FRET) to monitor the association of signaling proteins in living yeast cells. His new techniques should allow many proteins to be monitored simultaneously in real time, providing data to which computational tools can be applied to decipher the structure of signaling pathways.

Many of the Fellows share an interest in reconstructing biological networks, and will benefit from collaborating with the center's latest arrival, computational biologist Aviv Regev. Regev is using computational approaches to look for modular organization (ref. 3) in biological networks, and to characterize the behavior of modules (ref. 4).

It will not have escaped the notice of DBP members that there is a new influx of physicists and other physical and computational scientists into biology (see http://www.aps.org/apsnews/1102/110204.html). The Bauer Center's Systems Biology program (http://cgr.harvard.edu/research/systems_biology.html) aims to facilitate such career transitions, with jointly mentored postdoctoral fellowships designed to integrate quantitative scientists into biology, and a two-week summer school featuring lectures and laboratory experiments that will introduce postdocs and advanced graduate students from physics, mathematics, computer science and engineering to experimental biology. One of the center's main aims is to promote an intimate

Biophysicist Kurt Thorn.

symbiosis between theory and experiment, of the kind that is normal in physics, but has been all too rare in molecular and cell biology. In addition to welcoming theorists into the Systems Biology program, we also encourage visits, ranging from one month to two years, from theorists who are interested in interacting strongly with the experimental biologists in the center.

There are of course many types of barrier — caused by differences of language, culture, assumptions and philosophy — that need to be surmounted when scientists from different disciplines start to work together. By bringing Fellows from many fields into the same building to work closely together, we are learning how to lower these barriers. But we will not be content merely to do successful interdisciplinary research within the center's walls. Instead, our aim is to catalyze fruitful interactions between the center's Fellows and faculty in the surrounding departments, and among faculty in different departments. To this end, we hold a weekly series of "Genomics Talks", at which the speakers are asked to make themselves intelligible to a mixed audience, and where there is no such thing as a stupid question. Although we have a long way to go, we consider it a sign of success that two of the most dependable audience members at these talks are condensed-matter theorists from Harvard's physics department. An equally important venue for interdisciplinary interactions is the Bauer Center's café — after all, scientists from all disciplines have to eat!

References

1. Wedlich-Soldner, R., Altschuler, S., Wu, L. & Li, R. *Science* **299**, 1231-1235 (2003).
2. Kishony, R. & Leibler, S. *J. Biol.* **2**: 14 (2003).
3. Hartwell, L. H., Hopfield, J. J., Leibler, S. & Murray, A. W. *Nature* **402**, C47-52 (1999).
4. Segal, E., Shapira, M., Regev, A., Pe'er, D., Botstein, D., Koller, D. & Friedman, N. *Nature Genetics* 10.1038/ng1165 (2003).

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Bauer Center website: http://cgr.harvard.edu

An Introduction to the Evolutionary Epidemiology of Ideas

Aaron Lynch

Aaron Lynch is the author of Thought Contagion: How Belief Spreads Through Society. New York: Basic Books, 1996. In the following essay, he discusses the concept of the epidemiology of ideas.

Much as molecules catalyzing their own reproduction warrant special attention in biological physics, ideas catalyzing their own reproduction deserve special attention in the study of mass ideologies. Such self-replicating ideas can participate in a process of evolution by variation and natural selection, giving them special power over human affairs. The process has numerous practical implications that are not readily apparent using other theoretical paradigms. These include mechanisms amplifying the spread of AIDS, ideologies leading to war and terrorism, beliefs that interfere with embryonic stem cell research, ideological opposition to biological evolution theory, ideas that cause stock market bubbles and crashes, factors affecting the creativity of populations, and many others.

To date, the most widely familiar expression of the concept of evolution in self-replicating ideas stems from an analogy to evolution in genetic replicators drawn by Richard Dawkins at the end of his popular book *The Selfish Gene*. There, Dawkins introduced the term "meme", to parallel the biological concept of the gene. He also presented a number of good examples of replicating cultural phenomena. But unfortunately, Dawkins did not formally define the term "meme." This has led to great confusion and controversy over whether it refers to an idea, an artifact, a behavior, a perspective, or some combination of these. Radically contrary and staunchly promoted definitions of the word "meme" evolved and spread in recent years. The resulting confusion has thus created some reasons for setting aside the word "meme" in favor of more specific and better-defined terminology.

The more clearly defined term that I use in evolutionary replicator theory of ideas is *thought contagion*. A *thought contagion* is a memory item, or portion of an organism's neurally-stored information, identified using the abstraction system of the observer, whose instantiation depended critically on causation by prior instantiation of the same memory item in one or more other organisms' nervous systems. "Sameness" of memory items is determined with respect to the abstraction system used by the observer. Depending on context, the term "thought contagion" can also refer to the *process* of repeated causation of new instantiations of memory items in which the causation of those new instantiations depended critically upon the prior instantiation of the same memory item in one or more other organisms' nervous systems.

It is worth noting that the term "thought contagion" is more specific than the broad term "cultural replicator." For example, a computer virus is not itself a thought contagion. But the propagated *belief* in some fictitious computer virus is a thought contagion. A paper copy of a chain letter is not itself a thought contagion, but the (often fleeting) belief that it is wise to send out copies of it is a thought contagion. In other words, some cultural replicators are not thought contagions, but all thought contagions are cultural replicators.

Furthermore, many ideas are thought contagions but many others are not. Instances of ideas that are either original or independently formed are among the ideas that are not thought contagions. Among those ideas that are thought contagions, some show sufficient effects from replication iterating on a population level to usefully warrant evolutionary replicator analysis.

Ideas about social ostracism in school children (and for that matter, in adults) exemplify evolution in self-propagating ideas. When one child decides she does not like another, she can simply drop that person as a friend. But if she also has the idea of ostracism, she may launch an effort to convince all of her friends to also reject the person she does not like. In doing so, she incidentally spreads the idea of ostracism to the classmates she is trying to recruit into the social shunning. Those who ostracize also spread ideas about what sorts of things call for ostracism. Here too, selection is at work. Those who ostracize only for major transgressions do not spread their ostracism criteria very often, if at all. Yet those who ostracize for facile reasons are the ones who ostracize peers more often. They therefore end up expressing their ostracism criteria to more peers, who in turn also re-transmit the ostracism criteria more often. This favors an evolution toward relatively facile reasons for imposing the severe social sanction of ostracism on classmates. Details of hair style, clothing, skin color, academic interests, parents' financial status, athletic performance, and body shape thus become prevailing causes for ostracism among school children. Some children are even ostracized for taking a strong interest in science. When that happens, the social "example" of what happens to them can deter others from showing great interest in science.

This evolutionary replicator argument is equivalent to an evolutionary epidemiology of ideas argument. That is, one can equivalently view ideas as being copied into new people or as being transmitted to and retained by new people. Thus, I use the terms "evolutionary replicator theory of ideas" and "evolutionary epidemiology of ideas" interchangeably. Less formally, I also call the subject "thought contagion theory."

The term "thought contagion" is neutral with respect to truth or falsity, as well as good or bad. False beliefs can spread as thought contagions, but so too can true beliefs. Similarly, harmful ideas can spread as a thought contagions, but so too can beneficial ideas. For example, ideas that cause war to break out can spread as thought contagions, but so too can ideas that cause peace to break out. Thought contagion analysis concerns itself primarily with the mechanisms by which ideas spread through a population. Whether an idea is true, false, helpful, or harmful are considered mainly for the effects they have on transmission rates.

The replication/transmission processes can be represented as discrete events, using symbols to represent ideas. So if the letter A represents the general idea of rejecting a peer through ostracism, one kind of transmission event would be A + ~A→ 2A (host of idea A plus nonhost of memory idea A yields two hosts of A). A type of parent to child transmission can be represented as 2A → 2A + ~A → 3A (two hosts of A have a baby nonhost of A who is subsequently inculcated with A by the parents). Spontaneous (non-replicated) dropout can be represented as A → ~A (host of A drops out), and death can be represented as A → 0A (host of A dies). Transmission to 10 peers can be represented as A + 10(~A) → 11A, or it can be represented as 10 separate instances of the event A + ~A → 2A. Events involving

combinations of ideas can also be represented. So if B represents the idea of rejecting those who do not wear a certain type of clothing, we can have an event such as A*B + A*~B → 2A*B, where the * indicates conjunction (host of A and B). The notation provides a way of tracking the effects of multiple types of transmission events happening for just one or two ideas. Doing so indicates just part of the complexity of an idea transmission process. For example, someone may spend years of trial and error efforts to impart idea A into a best friend. That friend may even rely on his or her own creativity during the process of becoming a new host of A. Yet if the idea transition depended critically upon the prior person having idea A, then all the talking, interaction, and even creative components are still summarized in the abstract form A + ~A → 2A and counted as a replication event.

Event diagrams and associated methods of quantitative analysis provide a non-metaphoric method of discussing the evolutionary replication of ideas. While analogies and metaphors to genes, plasmids, prions, viruses, bacteria, computer viruses, etc., stimulate creativity and facilitate pedagogy, the analogies and metaphors do have their limitations. This is reflected in the way that idea transition event diagrams are not all isomorphic to replication, reproduction, and transmission diagrams from other fields.

Because idea transition event diagrams summarize numbers of hosts and non-hosts before and after, they are amenable to mathematical analysis. Rates for different types of transmission events can be modeled with systems of differential equations. They can also be quantitatively analyzed using agent-based computer simulation programs, such as the SWARM system developed at the Santa Fe Institute. A paper called "Units, Events, and Dynamics in the Evolutionary Epidemiology of Ideas" discusses such quantitative analysis, along with other technical issues in the evolutionary contagion of ideas. It is also often useful to summarize propagation parameters in the broad terms of *transmissivity*, *receptivity*, and *longevity* of beliefs. *Transmissivity* is the rate at which adherents of a belief express the belief to others. *Receptivity* is the rate at which people being exposed to the expression of a belief go on to adopt that belief. *Longevity* is the measure of how long an adherent remains an adherent before dropping out or dying.

The novel or independent creation of new ideas from precursor ideas can also be expressed in terms of event diagrams. For example, suppose that a combination of 6 precursor ideas A, B, C, D, E, and F play a causal role in the new formation of idea Z. This can be represented as A*B*C*D*E*F*~Z → A*B*C*D*E*F*Z.

Now suppose that 0.1% of the hosts of A*B*C*D*E*F have enough interest, effort, and talent to generate idea Z, perhaps by noticing and proving that Z is logically implied by the combination A*B*C*D*E*F. Suppose also, for simplicity, that the precursors A, B, C, D, E, and F propagate independently, so that the probability of a person having all of them is equal to the product of the probabilities of a person having each one separately. When each of the precursor ideas is rare, the probability that any person on earth will form Z is very low. For example, if only one person in a million has each of A, B, C, D, E, and F, then only one in 10^{36} has the combination needed to form Z. But if each of A, B, C, D, E, and F spread from one in a million to one in 100,000 to 1 in 10,000, and so forth, the chances of someone having the complete combination rise. If each of A, B, C, D, E, and F reach 10% prevalence, then one in a

million humans has the combination A*B*C*D*E*F. This means that 1 in a billion humans will actually form Z independently, after taking account of the hypothetical 0.1% rate for forming Z from the A*B*C*D*E*F combination. That is, about 6 people out of 6 billion humans will form Z independently during a relatively short time span during which the precursors are nearing 10% prevalence. The time span is short: the prevalence at which one person is expected to for Z is 7.4%, and the prevalence at which 6 people are expected to for Z is 10%. If each of A, B, C, D, E, and F propagate as uniform exponentials from the time that they have their first hosts, then the first 6 instances of Z are all expected to happen during the last 1.3% of the time it takes for A, B, C, D, E, and F to each reach 10% prevalence. This effect, and its many non-idealized variations, might account for how newly discovered ideas are often co-invented at nearly the same time by different people who may be widely separated.

For some sets of replicators, propagation of combinations is more vigorous than it is for the replicators separately, and propagation of combinations of more of the replicators is more vigorous than propagation of combinations of fewer of the replicators. The proliferation of these replicators spreading separately can thus lead them to propagate into a combination of two. But the combination of two of them then spreads more rapidly. As a result, the combination of two can more quickly propagate until it combines with a third symbiotic replicator, and so on. This means that combinations of large numbers of mutually symbiotic replicators tend to emerge very quickly compared to their overall history of separate propagation. Gradualistic replication can thereby exhibit a self-punctuating effect, in which new mutually adaptive combinations emerge quickly and then spread explosively. This argument is extended further in the paper "Units, Events, and Dynamics in the Evolutionary Epidemiology of Ideas." The principle behind it is very general. Applied to replicating ideas, it can cause rapid emergence of large ideologies or belief systems from previously separated component ideas. Applied to replicating genes, it might lead to the rapid combination and proliferation of mutually adapted systems of genes, and thereby even species of organisms. It may thus be possible to reconcile some famously contested positions taken by proponents of the punctuated equilibrium and gradualistic paradigms in evolutionary biology.

From these lines of reasoning, it appears that both the creative formation of new ideas and the development of co-adapted combinations of ideas are affected by the epidemiology of precursors and combinations. This suggests that creativity can be studied not only as an individual phenomenon, but also as a population phenomenon.

Some ideas develop and spread mostly through centrally planned propaganda or marketing campaigns. Their propagation can structurally resemble the centrally planned propagation of vaccinia virus (cowpox virus) by networks and alliances of smallpox vaccine programs – though centrally planned idea dissemination can range from helpful to harmful to the public. In any case, thought contagion theory does not assume that centrally propagated ideas follow the same replicator dynamics and mechanisms of ideas that spread on a non-centralized basis. Natural selection still has ways of manifesting itself in centralized communication phenomena, but it can be quite complex.

The evolutionary replication of ideas involves not just transmission by simple imitation, but

also transmission by inculcation or by mixtures of imitation and inculcation. For instance, ancient people who believed in wrathful gods that needed to be appeased had an incentive to inculcate their idea into others. Believers felt a need to persuade others to join in the appeasement of a god so as not to receive part of the god's collective punishment. So they inculcated friends, offspring, and others with their religious beliefs. A wide variety of other inculcation and imitation processes contributed to the overall evolution of religious belief systems.

While natural selection in ideas replicating by inculcation can explain many phenomena, the concept can also stir some emotional resistance. This is because recognizing the importance of inculcation can threaten one's sense of free will. When inculcation is done at an early age or by people who are very close to us, we often have little or no choice in whether to be inculcated or with what ideas. It can also be more comfortable to think that such inculcation happens entirely by intelligent design, and for our own good.

This can make the concept of natural selection specifically by imitative learning more palatable. People generally have a sense (rightly or wrongly) that they can always choose whether to imitate someone, and what to imitate. Meanwhile, theories that what we imitate is controlled by our genes have a certain appeal as well. Such theories at least allow one to think that genetic evolution ensures our genes are well served by what we imitate. But the concept of replicating inculcation more clearly raises the unsettling specter that some core cultural ideas may spread virally – despite harming adherents or their genes. Whether for this or other reasons, an emphasis on imitation has emerged in much of the literature on evolutionary cultural replicator theory, particularly for phenomena not driven by centrally planned inculcation campaigns. Some definitions of the word "meme," for instance, now place a distinct emphasis on imitation. In contrast, the term "thought contagion" is neutral on the relative importance of imitation and inculcation. The importance of imitation and inculcation is left as matter to be investigated for ideas on a case by case basis.

Fundamental to the concept of replication is the notion of calling two things "the same," or "of the same kind." In biology and physics, this is often taken for granted. There now exist clear procedures for deciding whether to call two strands of DNA "the same." If they are, and if the one caused the existence of the other, then we say that "replication" has happened. Yet as routine as this may become, the sameness of the molecules exists only at some level of abstraction. It does not mean that the two molecules have the same placement of isotopes, that they have the same tertiary structures, the same vibrational states, etc.

With ideas, one must pay more careful attention to the fact that two people only have "the same" idea with respect to an observer's abstraction system. We might classify two people as both having the belief that "abortion is wrong," even though one of them has a more extreme idea than the other. One of them might think that abortion is merely unethical, while another thinks that aborting an embryo is murder. They have "the same" idea with respect to the abstraction represented by the statement "abortion is wrong," but different ideas with respect to the abstraction represented by the statement "aborting an embryo is murder." With ideas, there is no one, absolute, or uniquely "right" system of abstractions – though some are more useful than others. Hence, evolutionary replicator analysis can be done in multiple ways by applying different systems of sameness criteria to the range of ideas on a

given subject. In some respects, it resembles the way physics allows for different spatial coordinate systems, or location abstractions, to be applied to a given phenomenon. Much as simultaneity of physical events occurs only with respect to a coordinate system, replication of an idea happens only with respect to a system of sameness criteria. Technical papers on the evolutionary epidemiology of ideas go into these fundamental points in greater detail.

The idea that abortion is wrong may spread by leading adherents to have and raise more children, an effect that is compounded each generation. The idea that abortion is murder can spread not only this way, but also by motivating adherents to persuade peers in order to stop what they see as murder. Both versions can also spread as indirect efforts to enforce conservative sexual mores. With a variety of transmission mechanisms, the choice of abstractions one uses makes a difference in how the analysis proceeds.

Thought contagions may play significant roles in important biological phenomena quite apart from abortion and embryos. For instance, the evolutionary epidemiology of ideas may interact with the evolutionary epidemiology of the HIV virus in a way that worsens the AIDS epidemic. Like other viruses, HIV virus spreads largely by the way it manipulates its hosts. Yet with this virus, much of that manipulation is apparently psychological. When an infected person develops terrifying and sexually disabling symptoms, any steady mate they have may be frightened into leaving. But by the time of breakup, that partner has a good chance of being also infected. After the breakup, the first person to show symptoms usually goes into episodes of remission in which they can become sexually active again and take on new partners. Thus, the virus spreads by its ability to shatter relationships. Indeed, the more virulent strains of the virus would have been most effective at shattering relationships and thus inducing more virus transmission. That could partly account for the evolution of virulence in HIV and its precursors. Any steady or semi-steady relationship ranging from marriage to prostitutes' returning clientele can be shattered this way, thereby spreading the virus to more people.

Yet thought contagions about the virus also came into play. As people began to informally recognize the disease in Africa, they began talking about it with others and warning people of the signs. As a result, people could be more quickly and easily terrified into leaving steady relationships by seeing symptoms that resembled AIDS. This would have increased the ability of HIV to spread by shattering relationships.

Stigmas against AIDS also spread as thought contagions. Those who hold stigmatizing ideas want to make sure that all their friends and family also hold the same stigmatizing ideas in order to avoid having friends and family bring them into contact with AIDS victims. It becomes another kind of replicating ostracism idea.

The contagious ostracism and stigma for AIDS further terrifies people, who flee anyone who they think may be infected. Yet many of the people who move away from stigmatized victims are themselves asymptomatically infected. Stigmas even lead intermittently symptomatic victims to move to other communities to escape the stigmas suffered when their symptoms were conspicuous and known. Stigmas do cause people to avoid sex with those who have overt AIDS, but victims thus avoided are often too sick to have sex or attract partners anyway. Thus, the spread of stigmas may accelerate the epidemic rather than slowing it down. This is consistent with a pattern that

regions with especially severe stigmas against AIDS also have rampant infection rates.

When whole towns become devastated by AIDS, the disease and the ideas about the disease terrify many members of communities to leave and move to areas with low prevalence. This happened, for example, in people who believed that enemies had placed curses over some hard-hit towns in Africa. But virus-induced migrations from areas of high prevalence to areas of lower prevalence also happened in major cities of the United States during the 1980s. So the virus and ideas about AIDS may induce more biological contagion and thought contagion by causing people to move from high incidence areas to low incidence areas that have more people susceptible to infection.

By manipulating "ordinary" people to spread HIV, the virus and its co-propagating thought contagions insure that eventually enough people are infected so that the infected population includes some super spreaders – for example, people who have many partners in many different places. Hence, the way the virus and thought contagions manipulate "ordinary" people plays a major role in spreading the epidemic internationally.

To make matters worse, sexually motivated belief transmission enters the picture and helps spread AIDS denial ideas. People have sexual motives for adopting the belief that AIDS is not sexually transmitted. It makes people feel freer to have sex. They also have sexual motives for telling potential sex partners that the disease is not sexually transmitted – even if they do not know their own infection status. This could make potential partners more willing to have sex.

Adding to the sexual motives for spreading these misbeliefs are social motives. Spreading the AIDS denial ideas is a way of protecting oneself from stigma by implying that there is really nothing sexual to stigmatize. Likewise, the idea that HIV does not cause AIDS leads people who know or suspect they have the virus to spread their belief to others. They have both sexual motives and stigma-avoidance motives for doing so.

With various ways that thought contagions and biological contagions can interact, understanding the mechanisms may help in developing strategies to curb the epidemic at a societal level. A variety of other health problems might also be more effectively addressed by taking account of the evolutionary epidemiology of ideas.

Perhaps the most important application of the evolutionary epidemiology of ideas is to the ideologies that lead to war and to weapons proliferation. This includes issues of nuclear weapons proliferation, a phenomenon where physics, biophysics and the evolutionary epidemiology of ideas all intersect. A recent paper that elaborates on evolutionary epidemiology of ideas aspects is "Thought Contagion in the Dynamics of Mass Conflict," presented at the Swedish Defence Research Agency in 2002.

With numerous and widely varied applications, the evolutionary epidemiology of ideas warrants further investigation on both the theoretical and practical levels. It has implications of potential interest across many disciplines, including physics, biology, and biophysics.

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PRE HIGHLIGHTS

APRIL 2003
Biological Physics Articles from Physical Review E
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Erratum to "Biological Physics in the Physical Review"
[THE BIOLOGICAL PHYSICIST, April (2003)]
Margaret Foster, Margaret Malloy, Gary Grest, and Frederick MacKintosh

We reported that the median time from receipt to acceptance for Rapid Communications submitted directly to PRE in 2002 was 71 days. This was, in fact, the median time for Rapid Communications published in PRE in 2002, following direct submission to the journal. The median time reported for receipt to acceptance for regular articles and Brief Reports submitted directly to PRE in 2002 was based on papers submitted from January through June 2002. We now report that the median time for receipt to acceptance for regular articles and Brief Reports published in 2002 in PRE, following direct submission to PRE, was 99 days.