

# Modeling Causal Interaction Between Human Systems and Natural Systems

By Sara Friedman

9 August, 2002

Santa Fe Institute Research Experience for Undergraduates

University of California, Berkeley

Applied Mathematics & Interdisciplinary Social Sciences

## **Introduction**

In order to model any aspect of natural systems, the fundamentally cyclic character of natural systems must be attended to. Nutrient cycling, the water cycle, the carbon cycle, the nitrogen cycle... the most basic of natural processes are named “cycles.” In order to maintain topsoil, for example, cycles from water, phosphorus, nitrogen and organic matter (among others) must be understood and accounted for. Thus, an attempt to model causation in natural systems, as well as our interventions and their effects, will be strikingly unrealistic if it fails to encompass these cyclic natural processes. Dealing with cyclic causation is not necessarily easy, though.

Regardless of the level of difficulty, to inform our interaction with systems and especially to determine the best course of action to take to fulfill our ends, we always (either implicitly or explicitly) use causal models.

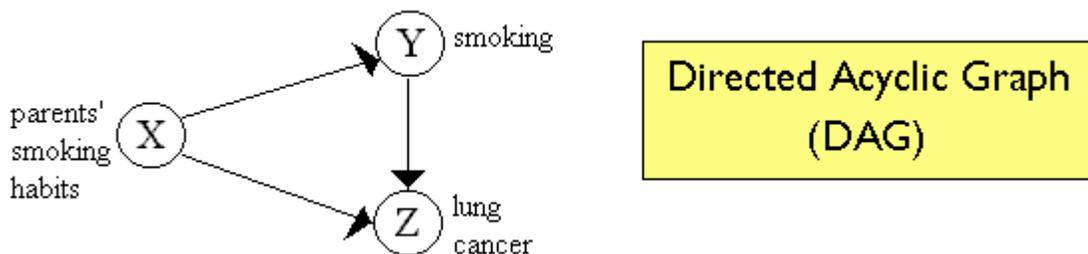
Mathematical causal models have been a focus of much research, ever since some statisticians started to break the mold and dared to speak of inferring causation from data. (Previously, statistics had been dogmatic about never making causal statements, but instead simply finding correlations.) The very structure of the scientific method is set up to test causal hypotheses (if I change A, then B will become such-and-such, all else remaining constant). Now, with modern dilemmas such as whether tobacco companies can be held responsible for widespread lung cancer, science has been struggling to mathematically represent causal networks, rather than the just the short causal chains of the traditional scientific method. In causal networks, things may be jointly caused by different factors, and factors causally lead one to the other in long causal chains. In order to keep the problem tractable, the mathematics almost universally assumes that the networks are acyclic, so that Bayesian inferences are possible. Otherwise, it is unclear whether one could update the network structure according to increasing information about conditional probabilities.

Now, our task should be clear. Mathematical causal models are overwhelmingly acyclic, but our causal interaction with natural systems definitely has a cyclic character. How can we represent cyclic causal chains, and with such models, predict effects of different interventions?

## Existing Mathematical Causal Theories:

### Graphical Models and Causal State Theory

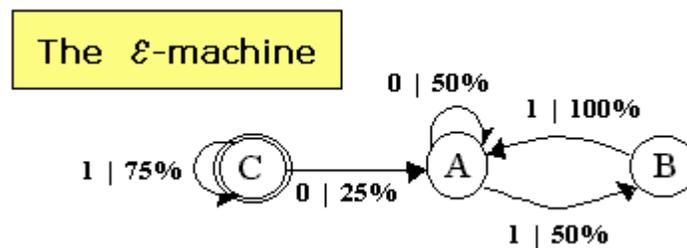
Graphical models generally rely on Directed Acyclic Graphs (DAGs) as their underlying structure. DAGs, by definition, do not display information about recursive behavior or feedback loops (see fig.1). This limitation constrains the ability of conventional graphical model theory to aid us in our quest to understand cyclic causal chains. The field and its literature is vast, however, and there are snippets of research (mostly from over ten years ago) that do attempt to deal with cyclic graphs. The field is underunified in general, and for cyclic models in particular, there is no definitive presentation of the theory. The main problem is the strength of existing statistical tools at uncovering a lot of information from conditional probabilities, which leads the theory to look at causal influence as a purely probabilistic relation. Once you look at it this way, inferring mutual causation and recursion is intractable. Perhaps it would be more feasible if the focus was on time series and causation as a temporal phenomenon (i.e. something is said to cause something else by invariably preceding it).



**Fig. 1:** A Generic Graphical Model: no recursion or cycles

In fact, Shalizi and Crutchfield use this temporal notion of causality in their presentation of Causal State Theory. The concept of a causal state is fundamentally different from the conventional view of causation in term of causal factors. A causal state is a set of “histories” of a process, such that every history in the causal state has the same distribution of future observables, conditional on being in the causal state. They are

discovered along with the transition probability matrices that govern movement between causal states, and together the causal states and their transitions constitute the  $\epsilon$ -machine (see fig. 2). An  $\epsilon$ -machine is the mathematical embodiment of Occam's Razor, in that it is the minimally statistically complex representation of a process subject to the constraint of maximal predictive capacity. Once you're in a causal state, you can forget about how you got there, and you know where you're likely to go next. "Factors" (it rained yesterday, your parents were smokers, you've taken linear algebra) don't matter, the only thing that matters is that you're in causal state A, which means that you'll either emit a 1 and go to causal state B or you'll emit a zero and go back to A.



**Fig. 2:** Causal States with the emitted symbols and transition probabilities between them

Causal state theory has a lot of potential to deal with very complex processes. The trouble is that it needs a very long time series of symbols from a finite alphabet in order to do  $\epsilon$ -machine reconstruction, and most real world data is not in that form – the theory has mostly been applied to machine learning. However, the field is still in infancy, and the fact that it easily captures mutual causation and recursive causal cycles means it holds some promise for our quest, at least in the future.

$\epsilon$ -machines do not help us figure out causal problems of public interest, such as how we could decrease cancer rates. Graphical models have major analytical weaknesses (incoherent theory, inability to deal with causal cycles). In the present circumstances, it seems practically impossible to combine the two approaches in a Unified Causal Theory that lets us tackle many kinds of relevant policy issues with analytical rigor and clarity. Although we now have some language and ideas to bring to our task at hand, the mathematical causal theories have not resolved the issue: How can we represent cyclic causal chains, and with such models, predict effects of different interventions?

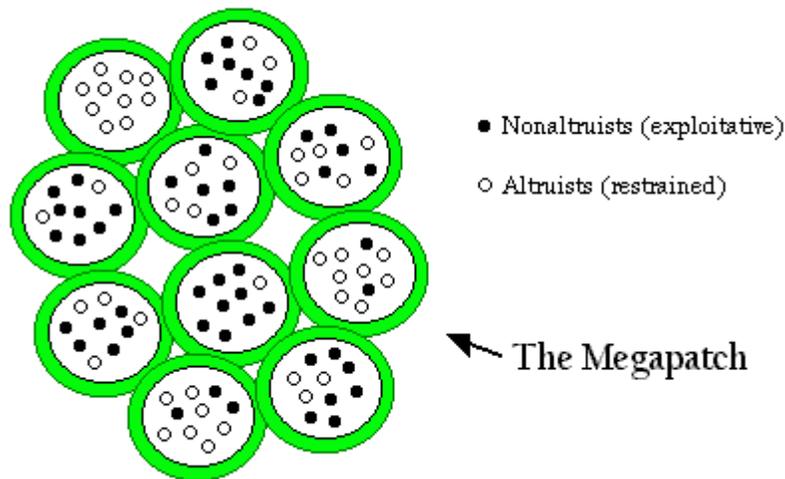
## Simulating a Mutual Causal Process:

### Feedback Between Human Behavior and Environmental Quality

If deducing mutual causation from real world data is problematic, perhaps we can “grow” a process through simulation, to test implications of our mutual causal hypotheses and make some generalized predictions about effects of system interventions. This is what I tried to do for the simulation I created with Sam Bowles and programmed in R.

The story goes like this:

There is an ecological megapatch, equally divided into separate patches. The patches can grow up to a maximum of 400% of their original productivity, or they can die off and have their productivity go to zero. Each patch has one group of individuals living on it. All groups have the same number of individuals, and individuals are of two cultural types: A’s and N’s, altruists (restrained people) and nonaltruists (exploitative people), respectively. The patches all start out at the same initial productivity, and the groups are seeded with random frequencies of A’s. Here is a visual representation:

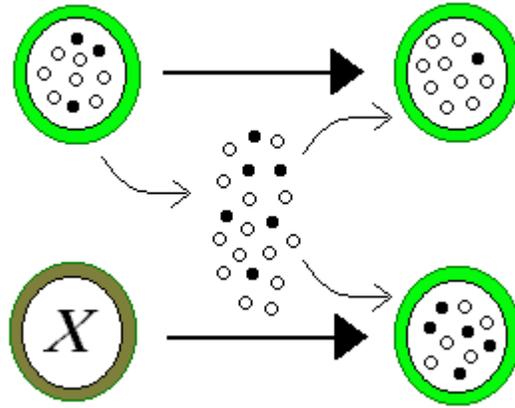


Then come the dynamics, which occur over discrete time steps. Individuals have a payoff that depends directly on the productivity of the patch they live on, as well as their type. N’s get an extra X-percentage payoff from the productivity, since they are exploiters. On the other side of the coin, the patch’s productivity depends on the X factor, times the number of nonaltruists in the group living on it, as well as its productivity in the last time step, and the growth increment, which is a parameter constant for all patches. Patches whose groups have sufficiently low frequencies of A’s

can decline quite rapidly; one period of all N's will bring the patch productivity down 85% (with default parameters). However, repeated predominantly-A periods will allow the patch to grow exponentially to its maximum productivity.

The replicator dynamic governs strategy updating – updating happens within-group  $(1-g)\%$  of the time, and payoffs from individuals in other groups are compared (global updating)  $g\%$  of the time. Global updating occurs probabilistically group-by-group, and since everybody in a group does global updating or not en masse, it can be thought of as an institution. Within-group updating can only decrease the frequency of A's, since on any one patch, the N's are better off than the A's. Global updating can increase the frequency of A's, especially when most A's are in predominantly A, well-off groups. A group doing global updating picks a random A from the pool of all A's in the megapatch, then picks a random N from the pool of all N's in the megapatch. They then compare the payoff of that A and the payoff of that N, relative to the average overall, global payoff. Each group looks for themselves and might have different results in the global updating process, or as the chips fall, might not do global updating at all if  $g < 1$ .

The more the megapatch (summed total) productivity goes down since the beginning of time, the more likely there will be a group extinction/colonization event in any time period. This is best thought of as a genetic extinction with an ecological recovery, and it is probabilistic. A patch's likelihood of going extinct in any time period is based on how much the megapatch productivity has gone down, and on the patch's "rank" relative to the other patches. The patch of lowest productivity is most likely to go extinct, while the patch of highest productivity is least likely. Once a group "dies", its patch's frequency of A's is wiped out, its patch productivity revitalizes to the prevailing average patch productivity in the megapatch, and another group colonizes it. A group is probabilistically chosen to be the colonizer based on the average payoff to individuals in it, relative to the total average payoff among all groups. The colonizer group reproduces itself and A's and N's are assigned to the two patches with random assortment. The random assortment has the potential to increase between-group variability, since the distribution of A's in the colonized patch and the original colonizing patch could differ. Here is a visual representation of the process:



random assortment with colonization

So: if the megapatch is degrading, patches of lowest productivity “die out”, get their productivity revitalized through ecological diffusion from the megapatch, and have their group’s A vs N distribution replaced by types from a group where individuals have relatively high payoffs.

Also, in every period, with a certain probability, there is idiosyncratic updating in a group. If it occurs in some group in some time period, the group’s number of A’s goes either up by 1 or down by 1 with equal likelihood, before updating occurs.

To summarize: this is a simple model describing feedback effects between human cultural evolution (prosociality/restraint of resource-getting) and ecological evolution (productivity in group-monopolized patches which together constitute a larger ecosystem), incorporating some stochastic and group-level effects.

Definitions, parameters and equations of the model:

Patch Productivity:	K
Number of A’s:	A
Number of N’s:	N
Frequency of A’s:	P
Average Payoff:	W

Initial Patch Productivity:	$K_{zero} = 100$
Direct Dependence of Fitness on K:	$B = 1$
Exploitation Increment of Nonaltruist:	$x = 0.05$

Growth Increment of Patches:  $k = 0.3$   
 Number of Patches/Groups:  $m = 10$   
 Number of Individuals in Each Group:  $n = 10$   
 Global Updating Percentage:  $g = 0.8$   
 Idiosyncrasy Rate:  $mut = 0.5$   
 Number of Time Steps:  $time = 200$

Payoff to Altruist on Patch j:  $W_{Aj} = B * K_j * (1+0)$   
 Payoff to Nonaltruist on Patch j:  $W_{Nj} = B * K_j * (1+X)$

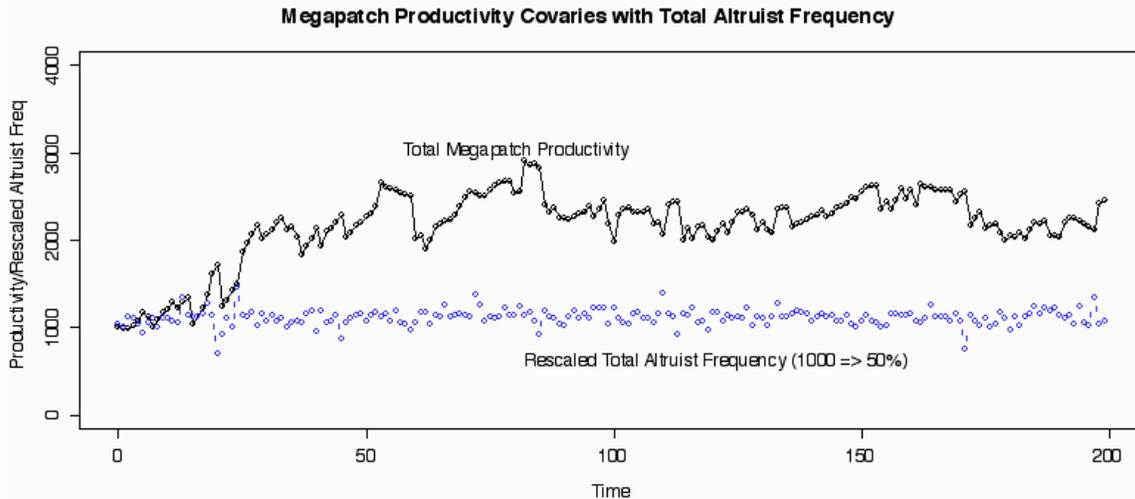
Productivity of Patch j at Time t:  $K_j^{(t)} = K_j^{(t-1)} * (1 - (N_j^{(t-1)} * X) / (1+k))$

Replicator Dynamic for  $t \rightarrow t+1$ :  $\Delta P_j = P_j * (1 - P_j) * (W_{Aj} - W_{Nj}) / W_j$

Note: in global updating, the replicator works the same way, but with different  $W_A$ ,  $W_N$  and  $W$

The model takes time-dependent, recursive feedback loops (cycles), as well as a large degree of stochasticity for various events (mutation, extinction/colonization, global updating, plus initial conditions), as its general dynamic. Analytical closed-form solution is impossible. Thus, simulation is the best way to explore the implications of this mutual causal hypothesis for interaction between human systems and natural systems.

The results showed that stochasticity played a very large role in outcomes, and different runs with the same parameter values looked very different. Also, the feedback process was often successful in correcting deviations. With default parameters, many runs led to a dynamic equilibrium similar to the one displayed in this graph:



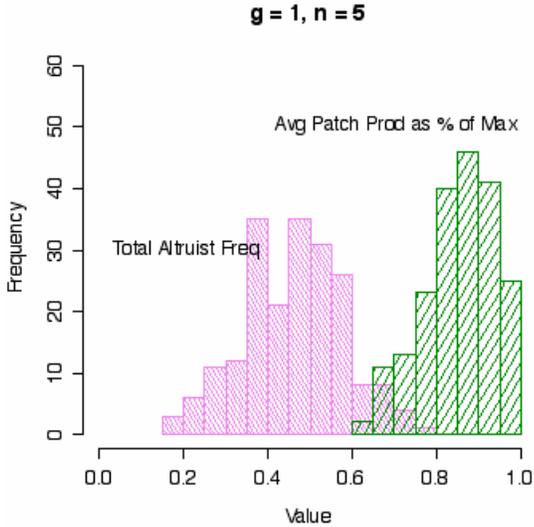
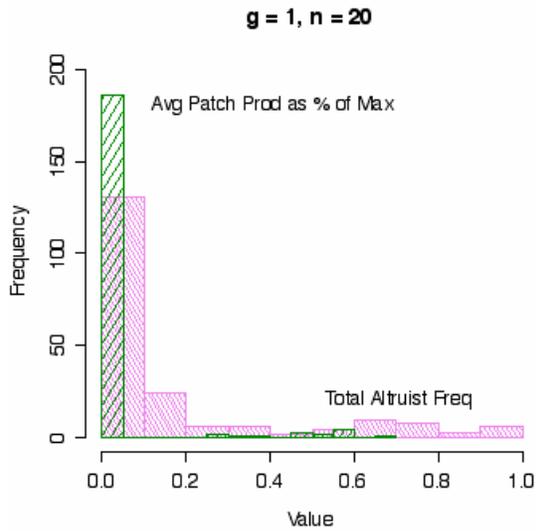
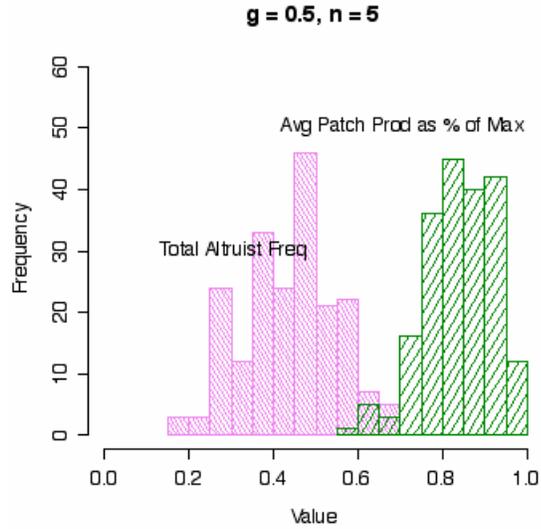
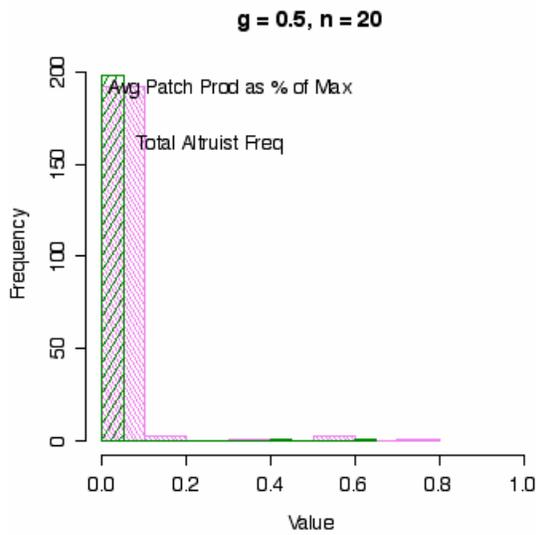
A main observation was that group size and global updating were key parameters, with the power to create no-dieoff conditions as well as nearly-certain dieoff conditions.

Small  $n$  increased between-group variability relative to within-group variability, augmenting the influence of group-level effects. Particularly, the random assortment (with colonization) had a good shot at creating all-A or mostly-A groups when  $n$  was very small.

Global updating worked especially well when there were “ideal patches” to copy, i.e. when there were a groups with all or nearly all A’s whose patches were extremely productive. Additionally, most A’s had to be in idea patch groups so that the random selection of an A to copy would probably be in an ideal patch. This depended largely on the initial distribution of A’s in the megapatch, and was a result of high between-group variance relative to within-group variance.

Without any global updating, it was still possible for A’s to do well, but it would be a result of the megapatch degrading and many extinctions occurring because of that. The patches wouldn’t revitalize as much in such cases, since the average productivity of the megapatch was not as high.

The following histograms show the results of varying  $n$  and  $g$  under default parameters. As you can see, small  $n$  had a huge effect; high  $g$  had only a subtle effect by itself, but significantly aided the possibility of altruism when  $n$  was large and made very high  $K$  more likely when  $n$  was small.



From these results, we can conclude (given the feedbacks in this model) that small groups, who can see how other groups are doing, will protect their resource base more than if there are large groups who don't look at other groups as often. Also, when differences between groups are dramatic compared to differences within a group, and group-level effects occur, it is easier for altruism to become a common strategy.

The model is in many ways similar to other models of the cultural evolution of cooperation in groups. However, in this model, the benefits of cooperation are slightly (1 period) time-lagged and are mediated indirectly through the entity of the patch, so there is

no “pairing noise” in individual updating. This is for ecological realism in the attempt to model, not just cultural evolution, but coevolution of culture and the environment.

The archaeological record shows that, during the Pleistocene and especially the Holocene, human behavior could cause significant ecological damage to natural resource bases, probably through various combinations of imprudent hunting practices and habitat destruction (often with fire and introduction of invasive exotic species). However, some ethnographic and anthropological case studies indicate that premodern societies have been able to evolve cultures which respect the species around them and protect, even increase, the productivity of their natural environment. There is no unified theory of the interactions of human cultures with their natural resources to explain this range of cases. The literature often supports two alternative views: one, a romanticized vision of “primitive harmony” and the other, a pessimistic view of societies consistently leaving ecological death and destruction in their wake.

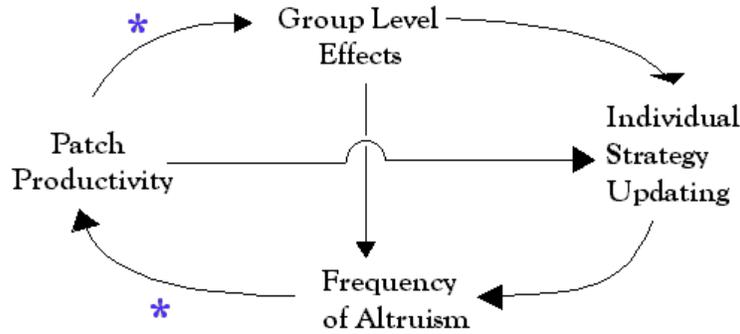
This model provides another perspective, that humans are neither “good” nor “evil” in how they treat nature, but that their behavior is context-dependent. Institutions and other conditions determining local versus global socio-ecological interactions, strategy updating, group size, etc, could be fundamental to the ecological outcome. Also, the simulation results suggest that, with global updating to any significant degree, the existence of “ideals” – productive patches with predominantly altruistic groups – for struggling non-altruistic groups to copy, have a large impact on the end productivity of the megapatch. Finally, since many of the feedback effects on human groups from the environment are stochastic in nature, the outcome really depends a lot on luck. Things work out well when different groups are doing really different things, and people can see people in other groups experiencing different effects from that.

In this model, when the patch people live on goes extinct, they do too. In real life, however, people could simply move on to another patch. This would be really bad for the patches and the A’s if there was no global updating, and there was an unfavorable initial distribution of altruists. Thus, it makes sense that in terms of human evolution, we see a pattern of extinction/migrations, especially on islands where there is effectively no megapatch and thus no between-group variability or global updating. However, in relatively harsh lands (not too productive or resilient, but not too unproductive or fragile)

where there were many connected patches and small groups monopolizing them, and especially if the groups were able to observe each other, we would expect a higher likelihood of sustainable societies developing. One could make many conjectures regarding the nature of this initial “altruism” (was it just inefficiency?) and how the cultural information transmission could rationalize it over time, in the form of myths, superstitions, and taboos.

The model has many limitations, which come from its simplicity. It is not spatial or agent-based, and so fails to capture more realistic dynamics, such as varying patch boundaries, spatial diffusion, and importantly, group-size dynamics. It would be much more realistic in terms of capturing hunter-gatherer dynamics if groups could be of variable size, and small groups were likely to die out during random climactic events. Also, it would be realistic to make the patch productivities somewhat stochastic to reflect significant climate variations, which ice core data is now showing to have occurred throughout the Pleistocene.

Regardless of these limitations, however, the process of making the simulation brought out some heuristic insights into dealing with causal cycles. When I first started simulating, there was what I called a “winners and losers” effect. The patch either shot off to infinity, or died out completely, never to recover. This was a result of some feedback effects that exaggerated relatively short-term or small differences. One was that my first equation for patch productivity depended directly on the frequency of altruists; one period of all N’s would kill the patch. Because of the logistic growth curve, the patch could never recover on its own. Also, I didn’t allow for patches to “revitalize” after becoming extinct. So, patches were dying like crazy, and A-dominant groups were attempting to colonize dead patches and not being able to. Correcting these feedbacks led to a more stable, homeostatic type of dynamic. Here is a cybernetic view of the model’s feedbacks:



\* exaggeration of patch differences at these links led to runaway (autocatalytic) "winners vs. losers" effect; making model less reactive to differences created homeostatic effect instead

So we have “grown” our own cyclic causal process. Some results and rules of thumb can be concluded, relating both to this process in particular, and to causal cycles in general. More importantly, we can now attempt to answer our original question.

### Conclusion

Back to the original question: How can we represent cyclic causal chains, and with such models, predict effects of different interventions? If we can do so, we can prevent maladaptive practices from becoming autocatalytically entrenched, as well as detect and ameliorate such effects that exist already. Our question is really asking another, purposive question: how can we create sustainable causal cycles between human and natural systems?

The first main point is that we can’t directly apply acyclic models to the question. Interventions in cyclic causal chains have different effects over time (the  $n^{\text{th}}$  time around) than they do initially. If we simply deduce interventions from acyclic models and apply them to cyclic socio-ecological processes, we can potentiate maladaptive decisions that initially seem to satisfy our ends.

What we really need to watch out for is autocatalytic cycles, because they are difficult to manage once they get going, and that can be a very bad thing. Bayesian reasoning and thought experiment will not necessarily be able to recognize the potential for such effects. Take the use of pesticides, which has been going on for at least half a

century. Using acyclic reasoning, we see that pests eat crops, and say “If I intervene to kill the pests, then the crops won’t get eaten by the pests.” So we start applying pesticides, and for some time, it works extremely well. Then it stops working, because of the effects that are now taught in introductory environmental science classes: secondary pest outbreaks, resulting from the niche vacuum and/or the death of pest predator insects, and resurgence, as the pests genetically evolve to tolerate the poison being used. The reasonable solution with an acyclic causal model is to develop a stronger, more fatal pesticide and use that. However, this ingrains the causal cycle commonly known as the pesticide treadmill – farmers input costs rise, more and more biocides make their way into neighboring habitat and waterways, and the pests get more relentless. Cyclic causal reasoning, the recognition of mutual causation and especially of autocatalysis, is essential to understand this and other important interactions between our human systems and the natural systems we depend on.

It is important to recognize that conceptual causal models are, in fact, culturally transmitted norms. These conceptual models of how causation operates in the world strongly affect the way people deal with ecosystems and resource getting. For those who missed Alison Gopnik’s recent talk here at SFI, developmental psychologists and cognitive scientists have shown that 3-year-old children can recognize direct one-way cause and effect (the red blanket makes the music come on, etc). The graphical models infer this type of causality from data, as well. Causal models, at least acyclic ones, seem to be hardwired into the human brain. But what about cyclic models? Cyclic causation is difficult to infer statistically and people don't necessarily develop conceptual models for it on their own. Western science has trouble with mutual causation, and so do many people in western societies – consider the rampant problems with addiction, everything from caffeine and refined sugar to crack, tranquilizers and marijuana. Eastern traditions tend to be more cognizant of mutual causation. Buddha had the idea of "dependent arising" (part of Right View, the first fold of his eightfold path), which says everything in the world is jointly caused by everything else, and so all causation is mutual. In addition, he taught that we could find a point in a causal cycle where our interventions are likely to have the best effect. At the time, the spiritual community in India knew that craving was the source of suffering, and that suffering caused more craving. Buddha noticed that the

best way to cut the cycle was not to just stop craving (which was the status quo opinion), but to work on moderating our reaction to suffering so that it does not lead to craving. Such an approach to causation is extremely useful for breaking harmful autocatalytic cycles: find the links where driving deviations are being amplified, and try to dampen them instead. Our science would do well to come up with ways to infer and represent mutual causal processes in nature. Especially, it would be powerful to understand the effects of autocatalytic processes involving human and ecological interaction and mutual degradation, and find their key linkages. That way we can make rational decisions on things like climate change, fishery management and other environmental policy issues, to develop a coevolutionary symbiosis with the ecosystems we live in.

Another question: These things are so hard to measure, anyway. Why not be content with our qualitative mutual causal notions? My answer is that we definitely need a scientific approach to modeling causality: we need something clearly stated and falsifiable, that people with different spiritual belief systems can agree on. One of the main sources of maladaptive cultural traits (according to Edgerton in Sick Societies) is mistaken causal attribution in a belief system. An especially prevalent and maladaptive one is the idea that witchcraft and sorcery are responsible for all problems; people with such beliefs tend to live in fear and resolve their helplessness by scapegoating and often killing members of their own or neighboring groups. If we stick with Bayes' nets, both in our minds and our science, then the more we try to account for, the more we introduce "unobservable" factors. The human mind, however, does not enjoy dealing with mysterious "unobservables" and tends end up attributing causal influence to some intentional agent. Hence we postulate God, as well as the abundance of conspiracy theories in the Western modern world, such as the idea that the global political economy is orchestrated by shape-changing reptilian aliens who have been slowly taking over for the past thousand years. The theory is internally consistent, makes basically no falsifiable statements and is impossible to disprove. Occam's razor would point out that the global elite could have evolved from preexisting social tendencies. As societies get more complex and large, they are prone to increasing levels of elitism and division between rich and poor, and in general the elites then attempt to increase their power through expansion. Such an understanding requires some recognition of cyclic causal

chains, whereas the reptilian alien hypothesis makes no such demands. So, why not believe that reptilian aliens run the world? Because it doesn't help us figure out how to intervene in the system and improve it. Routinely ascribing causal influence to "unobservables" does not help us see the kinds of cyclic causal relations in natural systems that we need to see in order to deal with real policy issues. In short, the reptilian alien theory may be appealing to our desire for simple acyclic causal relations, but it's just not very practical if the point is to understand and change the system in question. People's desire for simple acyclic causal relations, and the harmful potential consequences of that, should not be underestimated. In the absence of a unified scientific theory that captures cyclic causal relations, people's belief systems will tend to construct conspiracy theories and witchcraft hypotheses, science will lead to interventions which are self-destructive in the long term, and we will lack definite and coherent knowledge of how to create sustainable causal cycles between human societies and ecosystems.

## Acknowledgements

Many people guided this project in different ways.

My mini-project for the Dynamical Learning Group, under mentorship from Cosma Shalizi and Jim Crutchfield, involved comparing and contrasting their Causal State Theory with graphical models; obviously, the information I picked up there aided me significantly in this project. I'd like to thank them for giving me that project and for helping me learn so much about mathematical causal models.

Jeff Brantingham has been unwaveringly supportive all summer long, even during the weeks he was in the field on the Tibetan Plateau. He gave me a lot of good direction and readings from anthropology and biogeography, always seemed to understand the main point of what I'm trying to do, and strongly encouraged me to follow my own ideas. Thank you Jeff!

Sam Bowles really made the simulation possible, and was very generous with his time in working with me on the equations and group effects. He also gave me some great reading, both indirectly by authoring or co-authoring some very good papers I read before he came back from Europe, and directly by recommending Sick Societies and Jung-Kyoo's paper, among others. My project and I greatly benefited from his clarity and understanding of human cultural evolution. He was very good at expecting a lot from me and pushing me to make the model better and better, with the right amount of cheering me on, and I thank him for that especially.

Paolo Patelli helped me figure out some really basic aspects of programming functions in R that I wasn't picking up from the manuals I downloaded. He was totally my hero when he worked with me for an hour and got the base model simulation running. Thanks so much, Paolo.

I talked to Bae Smith a lot. She was very supportive of my ideas, especially when I was feeling discouraged, and suggested books, readings and concepts to chase down. She also let me practice my presentation for her.

Dave Krakauer spent a good chunk of time with me towards the beginning of the summer, when my approach to my project was very unclear, and the experience was invaluable. I've had no formal training in biology, and had no real exposure to the basic mindset of mathematical biology. My conversations with Dave, and the readings and concepts he suggested to me, gave me some important grounding in the ideas of the field relevant to my quest. Although I didn't end up doing the tritrophic predator-prey-resource model he suggested, his conviction that I "had something there" helped me focus on actually trying to figure something out, instead of going out to convince people of my intuitive notions. I thank him for his time and energy.

The other REU's (Lauren, Jeremy, Jacob and Alex, also Gina and Carl) were very patient in at least pretending to listen to my incessant ranting about my project. Alex, as my housemate, heard the most and probably listened the most. I'd like to thank them along with everybody else at SFI who had a willing ear and whose reactions helped me figure out the clearest way to present my information.

Finally, I'd like to thank my dad, Dan Friedman, who told me about this place and encouraged me to apply for the REU. He also talked to me about my project on the phone regularly, and gave me lots of fatherly advice, like "you'd enjoy working with Sam Bowles" and "just don't get up there and start ranting."

## **Bibliography**

I'm not even going to try to write down everything I've read this summer, or even everything I read that was directly relevant to the project, because there's too much. The stack of papers on my desk is at least three inches high. I'm just going to mention here the most important of the books and papers I read.

### BOOKS

Causation, Prediction, and Search. Second Edition. Peter Spirtes, Clark Glymour, and Richard Scheines. Cambridge, MA: The MIT Press, 2000.

Causality: Models, Reasoning, and Inference. Judea Pearl. New York: Cambridge University Press, 2000.

Fragile Dominion: Complexity and the Commons. Simon Levin. Reading, MA: Perseus Books, 1999.

Sick Societies: Challenging the Myth of Primitive Harmony. Robert Edgerton. New York: The Free Press, 1992.

### WORKING PAPERS

*The Coevolution of Individual Behaviors and Social Institutions*. Sam Bowles and Astrid Hopfensitz. September 2, 2000.

*Play Locally, Learn Globally: The Structural Basis of Cooperation*. Jung-Kyoo Choi. University of Massachusetts, Amherst. April 2002.

### ARTICLES

"The Archaeological Record of Human Impacts on Animal Populations." Donald Grayson. *Journal of World Prehistory*, Vol. 15, No. 1, 2001.

"Gene-Culture Coevolutionary Theory." Marcus Feldman and Kevin Laland. *Trends in Evolution and Ecology*, Vol. 11, No. 11, 1996.

"Computational Mechanics: Pattern and Prediction, Structure and Simplicity." Cosma Shalizi and Jim Crutchfield. *Journal of Statistical Physics*, 104:819-881, 2001.