SECOND DRAFT
PREDICTING HEALTH IMPACTS OF THE WORLD TRADE CENTER DISASTER: 1. Cognitive condensation, halogenated hydrocarbons, and traumatic perturbation

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Abstract

The recent attack on the World Trade Center, in addition to direct injury and psychological trauma, appears to have exposed an exceedingly large population to dioxins, dibenzo-furans, related endocrine disruptors, and a multitude of other physiologically active chemicals arising from the decomposition of the massive quantities of halogenated hydrocarbons and other plastics within the affected buildings. Combining

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recent theoretical perspectives on immune, CNS, and socio-cultural cognition with empirical studies of those affected by past large toxic fire incidents suggests the appearance of complex, developing spectra of synergistically linked social, psychosocial, psychological and physical symptoms among the 100,000 or so persons directly affected by the attack. The expected pattern greatly transcends a simple 'Post Traumatic Stress Disorder' model, and may come to resemble particularly acute forms of Gulf War Syndrome.

Introduction

The fiery collapse of New York City's World Trade Center has, at this writing, killed over six thousand, driven nearly ten thousand to seek medical attention, and caused the hospitalization of hundreds. It has directly and profoundly affected well over a hundred thousand people. Impacts range from extremities of psychological and psychosocial stress, to physical trauma, burns, and significant inhalation and skin-dose exposure to the highly and often persistently toxic combustion products from the thousand or so of tons of halogenated and nitrogenated plastics and plastics additives in the affected buildings. These exceedingly combustion-toxic materials continue to smolder three weeks after the event, often covering lower Manhattan with an eye-burning, cloud of stinking smoke. Combinations of smoke inhalation, burns and trauma are reported to be the most common factors in the immediate hospitalizations, and, very significantly, large numbers of persons with allergic skin and anaphylactic reactions were seen immediately after the incident (Larkin, 2001). If recent case histories of multi-fatal and other toxic plastics fires provide guidance, psychological and psychosocial trauma, burns and other physical injuries, and toxic exposure will interact synergistically to produce complex patterns of symptoms on several timescales in a path-dependent manner at the individual and group levels.

We first review recent theoretical developments which cross link processes of central nervous system (CNS), immune, and sociocultural cognition. This allows analysis of the effects of a sudden, traumatic, perturbation – characteristic combinations of physical, social and chemical assault – on such 'condensed' cognitive systems at different timescales.

Next, we review the observed effects of two recent large toxic fire events on injured survivors from this perspective, explore some important results from studies of Gulf War Syndrome, and then combine the analyses to try

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and predict the developing spectra of symptoms to be expected from the WTC disaster as at both individual and group levels.

Genes, cognition, and culture

Recently, interactions between the central nervous system (CNS) and the immune system, and between the genetic heritage and the immune system have become officially recognized and academically codified through journals with titles such as *Neuroimmunology* and *Immunogenetics*. Here and elsewhere we argue that a cognitive socioculture – a social network embodying culture – in which individuals are embedded, and through which they are both acculturated and function to meet collective challenges of threat and opportunity, may interact strongly with individual immune function to produce a composite entity which might well be labeled an *Immunocultural Condensation* (ICC). Thus, we propose that ultimately CNS, immune system and embedding socioculture can all become jointly convoluted to form a composite entity which will respond coherently, but in a complex manner, to sudden 'delta function' external perturbation. Elsewhere we have explored the response of such systems to long-term 'structured' systems of perturbation, for example embedding within an Apartheid system (R Wallace, 2001a, b).

Increasingly, biologists are roundly excoriating simple genetic reductionism which neglects the role of environment. Lewontin (2000), for example, explains that genomes are not 'blueprints,' a favorite public relations metaphor, as genes do not 'encode' for phenotypes. Organisms are instead outgrowths of fluid, conditional interactions between genes and their environments, as well as developmental 'noise.' Organisms, in turn, shape their environments, generating what Lewontin terms a triple helix of cause and effect. Such interpenetration of causal factors may be embodied by an array of organismal phenomena, including, as we shall discuss, culture's relationships with the brain and the immune system.

The current vision of human biology among evolutionary anthropologists is consistent with Lewontin's analysis and is summarized by Durham (1991) as follows:

> "...[G]enes and culture constitute two distinct but interacting systems of inheritance within human populations... [and] information of both kinds has influence, actual or potential, over ...

behaviors [which] creates a real and unambiguous symmetry between genes and phenotypes on the one hand, and culture and phenotypes on the other...

[G]enes and culture are best represented as two parallel lines or 'tracks' of hereditary influence on phenotypes..."

With regard to such melding, over hominid evolution genes came to encode for increasing hypersociality, learning, and language skills, so the complex cultural structures which better aid in buffering the local environment became widespread in successful populations (Bonner, 1980).

Every successful human population seems to have a core of tool usage, sophisticated language, oral tradition, mythology and music, focused on relatively small family/extended family groupings of various forms. More complex social structures are build on the periphery of this basic genetic/cultural object (Richerson and Boyd, 1995, 1998).

At the level of the individual human, the genetic-cultural object appears to be mediated by what evolutionary psychologists postulate are cognitive modules within the human mind. Each module was shaped by natural selection in response to specific environmental and social conundrums Pleistocene hunter-gatherers faced. One set of such domain-specific cognitive adaptations addresses problems of social interchange (Barkow et al., 1992; Cosmides and Tooby, 1992). The human species' very identity may rest, in part, on its unique evolved capacities for social mediation and cultural transmission. Anthropologist Robert Boyd has remarked that culture is as much a part of human biology as the enamel on our teeth.

Indeed, a brain-and-culture condensation has been adopted as a kind of new orthodoxy in recent studies of human cognition. For example Nisbett et al. (2001) review an extensive literature on empirical studies of basic cognitive differences between individuals raised in what they call 'East Asian' and 'Western' cultural heritages. They view Western-based pattern cognition as 'analytic' and East-Asian as 'holistic.' Nisbett et al. (2001) find that

1. Social organization directs attention to some aspects of the perceptual field at the expense of others.

2. What is attended to influences metaphysics.

3. Metaphysics guides tacit epistemology, that is, beliefs about the nature of the world and causality.

4. Epistemology dictates the development and application of some cognitive processes at the expense of others.

5. Social organization can directly affect the plausibility of metaphysical assumptions, such as whether causality should be regarded as residing in the field vs. in the object.

6. Social organization and social practices can directly influence the development and use of cognitive processes such as dialectical vs. logical ones.

Nisbett et al. conclude that tools of thought embody a culture's intellectual history, that tools have theories build into them, and that users accept these theories, albeit unknowingly, when they use these tools.

We may assume, then, the existence of both gene-culture and brain-culture condensations.

Immune cognition

Recently H Atlan and IR Cohen (1998) have proposed an information-theoretic adaptation of IR Cohen's (1992) 'cognitive principle' model of immune function and process, a paradigm incorporating pattern recognition behaviors analogous to those of the central nervous system.

We paraphrase Atlan and Cohen's description of immune system cognitive pattern recognition-and-response behavior as follows:

The meaning of an antigen can be reduced to the type of response the antigen generates. That is, the meaning of an antigen is functionally defined by the response of the immune system. The meaning of an antigen to the system is discernible in the type of immune response produced, not merely whether or not the antigen is perceived by the receptor repertoire. Because the meaning is defined by the type of response there is indeed a response repertoire and not only a receptor repertoire.

To account for immune interpretation IR Cohen has proposed a cognitive paradigm for the immune system. The immune system can respond to a given antigen in various ways, it has 'options.' Thus the particular response we observe is the outcome of internal processes of weighing and integrating information about the antigen.

In contrast to Burnet's view of the immune response as a simple reflex, it is seen to exercise cognition by the interpolation of a level of information processing between the antigen stimulus and the immune response. A cognitive immune system organizes the information borne by the antigen stimulus within a given context and creates a format suitable for internal processing; the antigen and its context are transcribed internally into the 'chemical language' of the immune system.

IR Cohen's (2000) cognitive paradigm suggests a language metaphor to describe immune communication by a string of chemical signals. This metaphor is apt because the human and immune languages can be seen to manifest several similarities such as syntax and abstraction. Syntax, for example, enhances both linguistic and immune meaning.

Although individual words and even letters can have their own meanings, an unconnected subject or an unconnected predicate will tend to mean less than does the sentence generated by their connection.

The immune system, in Atlan and Cohen's view, creates a 'language' by linking two ontogenetically different classes of molecules in a syntactical fashion. One class of molecules are the T and B cell receptors for antigens. These molecules are not inherited, but are somatically generated in each individual. The other class of molecules responsible for internal information processing is encoded in the individual's germline.

Meaning, the chosen type of immune response, is the outcome of the concrete connection between the antigen subject and the germline predicate signals.

The transcription of the antigens into processed peptides embedded in a context of germline ancillary signals constitutes the functional 'language' of the immune system. Despite the logic of clonal selection, the immune system does not respond to antigens as they are, but to abstractions of antigens-in-context.

Immune and sociocultural cognition

As we show at length in the Appendix, it is possible to give Atlan and Cohen's language metaphor of meaning-from-response a precise information-theoretic characterization, and to place that characterization within a context of recent developments which propose the 'coevolutionary' mutual entrainment – in a large sense – of different information sources to create larger meta-languages containing the original as subdialects. This work, a highly natural extension of formalism based on the Large Deviations Program of applied probability, also permits treating gene-culture and brain-culture condensations using a similar, unified, conceptual framework of information source 'coevolutionary condensation'.

Cohen's immune cognition model suggests, then, the possibility that culture, in the sense of a local cognitive sociocultural network by which individuals are acculturated and within which they participate in collective response

to patterns of threat and opportunity, and the individual immune system may be jointly convoluted. That is, there would appear to be, in the sense of the gene-culture and brain-culture condensations of the previous section, an immune-culture condensation as well: To 'neuroimmunology' and 'immunogenetics' we add 'immunocultural condensation.' Ultimately, however, these arguments suggest that CNS, immune and sociocultural cognition are strongly linked into a single composite entity by various kinds of 'crosstalk' – hormones and cytokines are neurologically active, while neurotransmitters are well known to have impact on the immune system. Contact with our fellows affects both.

Sudden perturbation

The WTC disaster constitutes significant and highly multifactorial perturbation of individual and collective life for a very large population, including both commuters and local residents of lower Manhattan. The modeling exercise of the Appendix suggests that the response to perturbation of an 'information source' made up of the convolution of CNS, immune and sociocultural cognition will be in terms of characteristic but 'nonorthogonal' eigenmodes of mixed patterns of pathology, so that a variable degree of mixing of different identifiable patterns of pathology – comorbidity of syndromes – will occur in proportion to the magnitude of the imposed perturbation. Equation (42) in particular suggests that, depending on the 'time constants' and 'lag times' of the elements of the syndrome, different aspects of pathology will 'kick in' at different times following the disaster. That is, although the 'incident' occurred at a single discrete time and place, the social, physiological, and psychological sequelae will not all appear simultaneously, but rather will develop according to their own internal 'time clocks' after delays which may themselves be highly, if systematically, variable. The perturbation itself will, further, interact with individual and group histories so that different individuals or groups may embark on different 'paths' of response.

The response may be multiply comorbid: At low levels of applied stress, be it physical, psychosocial or chemical trauma or their mix, a relatively small number of distinct syndromes of behavioral, psychological, social, or immune pathology should be observed, according to the model of the Appendix. As the 'magnitude' of perturbation experienced increases, however, these syndromes should become mixed, i.e. increasingly comorbid. Again, this effect should be complicated, as the 'time of integration' increases af-

ter perturbation, by the sequential appearance of symptoms and symptom patterns with differing time constants or lag times, in a broad sense.

Equation (43) suggests that 'eigenpatterns' of mixed pathology appear as synergistically amplified internal representations of external perturbation. If $\sigma(y)$ represents the standard deviation of pathology, and $\sigma(w)$ that of perturbation, then they will be related as

$$\sigma(y) \propto \frac{\sigma(w)}{|1 - \lambda|},$$

where λ is a kind of multiple correlation coefficient, so that, for 'tightly coupled' systems, $|\lambda| \to 1$, and this expression implies that applied perturbations will be greatly amplified by mixed pathologies of physiological, psychological and social response.

Note particularly that, in this formulation, symptom patterns may express themselves within embedding social networks and associated social cognition as well as simply within individuals, that is, collective, as well as individual, 'eigenpatterns' of pathology.

A brief introduction to the 'new' combustion toxicology

Fumes and smoke from modern office and construction materials are not like those from natural materials. Although wood, paper, natural fabrics, etc., can emit irritating fumes and soot in a fire, the intensity and irritancy is much less than most synthetic materials now commonly used as substitutes for them. Rigid polyvinyl chloride (PVC) emits 60 % of its weight as hydrogen chloride, HCl, in the early stages of a fire, which coats soot particles. These are small, smaller than those given off by, say, wood or cotton. They get into the lung and deliver a huge dose in a short time, destroying lung tissue by sheer corrosion.

Because of the flame retardant properties of chlorine, much more mass of the PVC goes off as soot and the smoke is extremely dense. People cannot see to escape. Plasticized PVC, as is used in communication cable, also produces highly acidic soot and fumes of dense smoke, but also gives off masses of phthalate anhydride, very irritating and explosive. Both forms of PVC produce large quantities of benzene, xylene, and toluene which are narcotic, and may give rise to secondary explosions.

A variety of halogenated species are present both as gases and in the soot. Large quantities of dioxins and dibenzofurans – notoriously powerful

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endocrine disruptors – form during the cooling phase, as they do in incinerators. PVC fumes and soot may contain as many as 300 species, as reported in the literature, ranging from formaldehyde to the polyaromatics, with and without chlorine. Most PVCs also contains a variety of additives which may include lead stearate as a stabilizer, phthalate plasticizers, and metal-based colorants.

The brominated fire retardants can be present in other plastics and in fabrics at more than 10 % by weight and form similar pyrolysis and combustion products. Other halogenated plastics and highly brominated-flame retarded plastics and fabrics behave analogously to PVC. It is our understanding that the WTC replaced PVC in its communication cable banks with Teflon which emits hydrogen fluoride, HF, and fluorinated organics.

Nitrogen-based polymers such as urethane foam, acrylonitile-butadyene-styrene (ABS) and some of the acrylics will likely have played a considerable role. Hydrogen cyanide, HCN, is emitted massively from these materials shortly before ignition. Cyanide also delivers a skin dose, besides a lung dose. Nitrogenated organics also form, of course. These should contain large quantities of flame retardants of different kinds, including brominated ethers or metal salts.

The WTC fuel load, between the two planes and the several buildings which burned, collapsed, and then smoldered copiously for weeks, would make the 1975 New York Telephone Exchange fire look like a minor event. That fire involved over 200,000 pounds of plasticized PVC (D Wallace, 1981, 1982, 1990, 1992).

The 1975 NY Telephone firefighters, and survivors of the 1980 MGM Grand Hotel plastics fire, suffered a spectrum of subsequent symptoms which we examine in some detail below.

Two toxic fire case histories

The New York Telephone Company and MGM Grand Hotel fires, and their effects on survivors, are described at great length in D Wallace (1981, 1982, 1990, 1992). We summarize that work briefly here.

As described above, the Telephone Company fire involved about 100 tons of heavily plasticized PVC cable burning over a 14 hour period. Some 700 firefighters responded to the blaze, 239 of them reporting themselves as injured. 113 of these latter subsequently participated in a 1980 survey sponsored by the Uniformed Firefighters Association, the firefighters union.

The MGM Grand Hotel fire involved a very large, mixed load of various common plastics burning at ground level – PVC, ABS, etc. Rising smoke trapped hotel patrons in their rooms, killing 85 of them at a considerable distance from the fire itself. 54 injured survivors participated in a subsequent detailed study sponsored by the Plaintiffs Legal Committee, which oversaw the resulting lawsuit.

Firefighters and civilians constitute separately socialized populations – different and quite distinct 'sociocultures' – in their response to the fire environment. To the former, it is the 'normal' working condition, for which they are rigorously trained. To the latter, it is a horror and a gross and traumatic aberration from normal life. Very large or multifatal fires, or persistent episodes of contagious urban decay which devastate communities, however, will breach even the paramilitary cultural discipline of the fire service.

Tables 1-3, below, describe the pattern of reported firefighter symptoms from the NY Telephone Company fire on short, intermediate and long time scales, respectively at the fire, two weeks to three months after, and six months or more after the fire. 64 % of the respondents complained of persistent symptoms. It is of some note that the pattern differed significantly between those with long and short work histories. Those over thirty-five years of age and those who had served more than ten years as firefighters tended to have permanent damage. Indeed, of those who had served more than 15 years, 80 % reported permanent injury.

Tables 4 and 5, by contrast, examine the respiratory and non-respiratory complaints of the sample of injured survivors of the MGM Grand Hotel fire. Reproductive, neurological, and psychological dysfunction, in contrast to the firefighter sample, dominate.

Besides the group pattern shown in Tables 4 and 5, certain individuals experienced special disabilities from the smoke, for example muscle spasms which show that certain parts of the nervous system that cause involuntary movement of the normally voluntary skeletal muscles were affected. Both Parkinsonism and epilepsy are based on these parts of the nervous system. In addition, sufferers of Parkinsonism and epilepsy have low levels of one or more catecholamines, as do depressed individuals. On injured survivor had Parkinsonism even before the fire, but required a much greater amount of medication to control his condition after the fire. A second person became epileptic after the fire and suffered grand mal seizures. These two individuals are index cases of the neurotoxic nature of the smoke.

Besides the physical injuries and individual psychological distress, social

interactions appear to have been affected by the experience of the MGM fire, possibly synergistic with exposure to neurotoxins. Marriages became troubled and were dissolved. The divorce rate among the survivors was very high. This high rate of divorce is also seen in other disasters besides fires, and reflects personality changes and relational changes that often result from having risked death and knowing that others died in the same event.

In addition to divorce, another socioeconomic consequence of the MGM fire was the decline of several businesses that had sent key people to conventions or seminars that were held at the hotel. These people stayed overnight in the hotel, and several within the same office may have suffered personality changes. Individual productivity typically declined, but group interactions also suffered so that decisions could not be made, consensus could not be reached, and actions could not be taken. At least two mid-sized companies suffered greatly and became much smaller when several key managers could not perform.

The MGM Grand Hotel fire in particular shows precisely the inextricably mixed pattern of physical, neurological, psychological, and social pathology to be expected from the 'perturbation' of a traumatic toxic exposure incident.

Traumatic toxic incident of sufficient magnitude should be expected to produce a similar pattern among affected emergency services personnel, in spite of both discipline and experience. 'Post Traumatic Stress Disorder' is a grossly inadequate characterization.

These two examples appear to be fully consistent with the theoretical development we have presented as an introductory context.

Table 1: Immediate symptoms of Firefighters

INJURY	PERCENT AFFECTED
Sore throat, irritated eyes, dizziness, aching nostrils, confusion, weakness, and exhaustion	Over 50
Chest pains, nausea, chest congestion, and headache	35-50
Irritated skin and faintness	20-30
Loss of control of arms and/or legs	10-20

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Table 2: Intermediate time symptoms of Firefighers

SYMPTOMS                                    PERCENT AFFECTED
------------------------------------------------------------
RESPIRATORY:
Chest congestion                            51.2
Chronic cough                               22.1
Sore throat                                 18.5
Sore chest                                   9.3
Hoarseness, wheezing,
  allergy to smoke, difficulty
  breathing, irritated nasal
  membranes, shortness of breath    Less than 5
------------------------------------------------------------
NEUROLOGICAL:
Muscular weakness                           19.8
Impaired smell/taste                        16.3
Increased irritability                      10.5
Headaches                                   10.5
Perception difficulty                        7.0
Confusion, anxiety,
  numbness of extremities           Less than 5
------------------------------------------------------------
MISCELLANEOUS SYMPTOMS:
Heart trouble, irritated eyes,       2 complaints each
  irritated skin
Chills, sinus trouble, weight loss,
  bowel problem, nausea, head
  congestion                         1 complaint each
GENERAL WELL-BEING
Fatigue                                      5.8
Impaired endurance                           3.5
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Table 3: Long-Term Effects among Firefighters

INJURY                                           PERCENT AFFECTED
----------------------------------------------------------------
RESPIRATORY:
Impaired disease resistance                           37.5
Coughing                                              33.3
Hoarseness                                            23.6
Shortness of breath                                    9.7
'Lung function' or pain                               15.8
Chest congestion                                       9.7
Sensitivity to smoke                                  11.1
Sinus or nasal drip                                    6.9
Repetitive bronchitis                                  8.3
Sore throat                                            8.3
Asthma                                                 6.9
Allergy, unspecified upper
  respiratory problem                            Less than 5%
----------------------------------------------------------------
GROWTHS (epidermal or membrane
lining)                                               13.9
----------------------------------------------------------------
HEART:
Palpitations, acute myocardial
  infarction, prolapsed mitral valve,
  enlarged heart, unspecified damage                   8.3
----------------------------------------------------------------
Headaches, perception difficulty         4 individual complaints
Fatigue, kidney-urinary tract            3 individual complaints
Weakness, pancreatitis/diabetes          2 individual complaints
Elevated blood count, elevated bilirubin,
  high pressure, gall bladder
  deterioration, irritation of
  hemorrhoid, irritated eyes,
  convulsive seizures                     1 individual complaint
----------------------------------------------------------------
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Table 4: Respiratory Complaints of 58 MGM Survivors

COMPLAINT	NUMBER	PERCENT
Frequent sore throat	30	51.7
Hoarseness	26	44.8
Sinusitis	23	39.7
Sensitive to dust/smoke	44	75.9
Coughs	27	46.6
Wheezing	26	44.8
Phlegm production	29	50.0
Shortness of breath	38	65.5
Bronchitis	21	36.2
Frequent upper respiratory infection	25	43.1

Table 5: Non-Respiratory Symptoms of 57 MGM Survivors

SYMPTOMS	NUMBER	PERCENT
CIRCULATORY SYSTEM:		
Developed heart problem	12	21.1
Hands/feet fall asleep	39	68.4
Hands/feet easily cold	22	38.6
Mottling or bulue hands/feet	8	14.0
Blood pressure change	20	35.1
NEUROLOGICAL/PSYCHONEUROLOGICAL:		
Headaches	34	59.6
Change in sleep pattern	42	73.7
Memory lapses	32	56.1
Irritable	43	75.4
Difficulty learning	14	24.6
Change in perception abilities	31	54.4
Coordination decline	15	26.3

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Dizziness                                27          47.4
Disorientation                           20          35.1
----------------------------------------------------------------
KIDNEY/BLADDER:
Frequent urination                       15          26.3
Pain/burning during urination            12          21.1
Lower back pains                         27          47.4
----------------------------------------------------------------
SKIN CHANGES:
Acne-like breakouts                      13          22.8
Rashes                                   12          21.1
Burns from the smoke                      4           7.0
Infections                                6          10.5
Skin growths (warts, moles)               3           5.3
Skin dry, sensitive                      14          24.6
----------------------------------------------------------------
REPRODUCTIVE:*
New menstrual difficulties               11          84.6*
Miscarriage                               2          15.4*
Hysterectomy                              2          15.4*
Dilation and curettage                    1           7.7*
----------------------------------------------------------------
PSYCHOLOGICAL:
Nightmares                               42          73.7
Depression                               40          70.2
Guilt                                    15          26.3
Anger                                    17          29.8
Change in relationships                  24          42.1
----------------------------------------------------------------
GENERAL WELL-BEING:
Change in appetite                       16          28.1
Less endurance                           31          54.4
General weakness                         24          42.1
Ceased/decreased activity                17          29.8
Lack of sexual desire                    14          24.6
----------------------------------------------------------------
OTHER COMPLAINTS:
Sensitized eyes                          11          19.3
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Dizziness with position change              8              14.0
Heavy sweating                              7              12.3
Drink more water                            3               5.3
Tremors, spasms, clenching                  6              10.5
----------------------------------------------------------------
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* Percentage is that of the women of reproductive age
in the data base, not of all 57 people.

The following affected less than 5 percent:
liver dysfunction, nausia, neck pains, impotence,
pigmentation of skin, loss of hair, ears hurt,
swollen glands, onset of epilepsy,
exacerbation of Parkinsonism,
return of menses to elderly women,
swollen testes, prostate infections.

Gulf War Syndrome

Recently a number of researchers have begun to critically examine characteristic patterns of 'medically unexplained somatic symptoms', including multiple chemical sensitivity (MCS), chronic fatigue syndrome (CFS), fibromyalgia (FM), irritable bowel syndrome (IBS) and Gulf War Syndrome (GWS). Kipen and Fiedler (1999) find that psychiatric explanations and nomenclature have less than a perfect fit for many cases of unexplained sysmtoms, although there is often very great comorbidity among them, often as much as 50 % between MCS and CFS, for example. In the case of Gulf War symptoms, careful epidemiology has shown that, compared with soldiers who did not deploy to the Persian Gulf, those who deployed had two- to threefold increases in symptoms, without apparent medical explanations (Kipen and Fieldler, 1999; The Iowa Group, 1997; Fukuda et al., 1998).

Haley et al. (1997) used multivariate methods to examine symptom patterns among about 250 Gulf War veterans in a construction battalion, and found three particularly distinct and characteristic clusters, roughly characterized as 'impaired cognition', 'confusion-ataxia', and 'arthro-myo-neuropathy'. Veterans with the second pattern were found to be 12.5 times more likely to be unemployed than those with no health problems. Haley et

al. conclude that clusters of symptoms of many Gulf War veterans represent discrete factor-analysis derived syndromes that appear to reflect a spectrum of neurologic injury involving the central, perhipheral, and autonomic nervous systems. Subsequent work by Haley et al. (2001) extends these studies.

Knoke et al. (2000) responded directly to the report of Haley et al. (1997) with their own multivariate study which compared Gulf War-era veterans who had and had not been deployed to the Persian Gulf. Closely similar clusters of syndromes were identified within both groups, although the deployed veterans showed these clusters with greater prevalence than the non-deployed, and the strongest clusters matched fairly well with the observations of Haley et al. (1997).

Doebbeling et al. (2000) conducted a similar deployed/non-deployed veterans study, and again found similar clusters of symptoms within both groups, with the deployed again having very much greater prevalence than the non-deployed.

Doebbeling et al. (2000) conclude, however, that the markedly increased prevalance of nearly every symptom assessed from all bodily organs among the Gulf War veterans is difficult to explain pathophysiologically as a single condition. Identification of the same patterns of symptoms among the deployed veterans and the nondeployed controls suggests that the health complaints of Gulf War veterans are similar to those of the general military population and are not, in their view, consistent with the existence of a unique Gulf War syndrome.

The modeling exercise of the Appendix, leading to equation (43), however, suggests a more consistent explanation of these results. Deployed and non-deployed veterans were embedded within very similar – and highly rigid and nonresilient – sociocultural structures, and seemed to represent very similar cross-sections of the US population. This suggests that the 'multiple correlation coefficient' representing the linking of internal mechanisms would satisfy the condition $\lambda \to 1$, giving a very large amplification pattern for perturbations, $\sigma(w)$. Combat experience is indeed a significant perturbation at both individual and social levels, and amplified 'eigenmodes' of characteristic patterns of mixed pathology are, from this model, the expected result at both scales. Some degree of comorbidity is inherent, since the response 'eigenmodes' are nonorthogonal.

Predicting the spectrum of WTC pathologies

On the basis of these case histories, it is not difficult to construct an expected pattern of symptoms for both civilians and emergency workers having significant exposures to the WTC disaster, and this is left as an exercise for the reader. The great prevalence of uterine dysfunction among women exposed in the MGM Grand Hotel fire suggests, however, particularly nasty consequences for those in the WTC disaster: the presence of massive quantities of endocrine disruptors and other physiologically active chemical species in the aftermath of the WTC attack takes the matter far beyond either Post Traumatic Stress Disorder or Gulf War Syndrome.

Less obvious, however, are the inevitable consequences of the couplings of cognitive process across scale. As one example of what are likely to be many subtle but critical cross-system impacts we examine in more detail the possible convolution of immune function into the more general pathology.

As is well known, dioxins can cause severe immunosuppression (e.g. Tonn et al., 1996), an effect possibly complicated by an inverted-U dose-response effect (e.g Fang et al., 1996). This is to be contrasted to the standard dose-response effect on the muco-cilliary tract which is expected from exposure to irritants such as HF, HCl and HBr. As is increasingly understood, however, psychosocial stress may itself have an immunosuppressive effect which is indeed sufficient to interfere with vaccine efficacy.

Recent work by Kiecolt-Glaser and Glaser (1996, 1998, 2000), for example, has examined the effect of 'chronic stress' on the efficacy of influenza, hepatitis-B, and pneumococcal pneumonia vaccine among elderly caregivers of dementia patients, and among medical students.

They found, for influenza, that the caregivers showed a poorer antibody response following vaccination relative to control subjects, as assessed by ELISA and hemagglutination inhibition. Caregivers also had lower levels of *in vivo* virus-specific-induced interleukin 2 levels and interleukin 1β. The data demonstrate that down-regulation of the immune response to influenza virus vaccination is associated with a chronic stressor in the elderly.

Similar effects were found among the elderly caregivers for response to pneumoccocal pneumonia vaccination, leading to the conclusion that chronic stress can inhibit the stability of the IgG antibody response to a bacterial vaccine.

Medical students who reported greater social support and lower anxiety and stress demonstrated a higher antibody response to HEP-B surface antigen at the end of the study period.

Glaser et al. (2000) conclude that

"The differences in antibody and T-cell responses to HEP-B and influenza virus vaccinations provide a demonstration of how stress may be able to alter both the cellular and humoral immune responses to vaccines and novel pathogens in both younger and older adults."

In addition a vast body of animal model studies involving socially structured populations shows clear impacts of acute and chronic social and other stressors on immune competence (e.g. deGroot et al., 2001; Gryazeva et al., 2001). Elenkov and Chrousos (1999) in particular suggest that glucocorticoids and catecholamines, the end-products of the stress system at the individual level, might selectively suppress cellular immunity, Th1 phenotype, in favor of humoral response – again at the individual level.

We suggest, however, that the particular role of cognitive socioculture in human biology takes matters considerably beyond such individual-level stress models, and into realms for which, to paraphrase Robert Boyd's aphorism, culture is as much a part of the human immune system as are T-cells.

These considerations suggest in particular that, in addition to the characteristic mix of symptoms which can be inferred from both theory and past case histories, it seems likely that those exposed to both stress and toxic combustion products at the WTC disaster will enter the coming influenza season during the peak 3-9 month period of 'normal' grieving, and may thus be both markedly more susceptible to infection and markedly less responsive to the available vaccine than unexposed populations. This effect may be complicated by persisting muco-cilliary damage consequent on exposure to smoke irritants. Vaccine strategy for the coming season, and probably for many seasons to come, must be significantly altered in the New York metropolitan region if a large number of excess flu/pneumonia deaths are to be averted.

Analogous interacting mechanisms at both individual and larger social scales will become clear as matters unfold, particularly involving carcinogenisis, as dioxins are among the most potent of cancer-causing substances. 'Post Traumatic Stress Disorder' is a grossly inadequate characterization of the likely consequences of this attack for individuals, families, social networks, and larger organizational structures. The role of the massive quantities of highly combustion-toxic halogenated hydrocarbons within the affected buildings appears to constitute a serious, and continuing, exacerbation of the disaster.

Medical practitioners treating victims of the WTC disaster and their families over the next several years should be aware that, on both empirical and theoretical grounds, there is good reason to expect complex patterns of respiratory, immune, reproductive, neurological, psychological, social and other pathology to emerge, persist, and interact for the foreseeable future. Further, this is not the first, nor is it likely the last, such incident: repeated exposures seem increasingly likely, and will be synergistic.

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MATHEMATICAL APPENDIX

The essence of the modeling approach is to, first, express the cognitive pattern recognition-and-response described as characterizing immune cognition by Atlan and Cohen (1998) in terms of a 'language,' in a broad sense, and then to show how that language can interact and coalesce with similar cognitive languages at lager scales – central nervous system (CNS) and the embedding local sociocultural network.

The next step is to model the way such a coalesced system may, in turn, interact with sudden external 'perturbation.' The 'nonorthogonal eigenmode' solution emerges from this discussion after a certain amount of development.

We begin with a summary of relevant information theory formalism.

Information theory preliminaries

Suppose we have an ordered set of random variables, X_k, at 'times' $k = 1, 2, ...$ – which we call $\mathbf{X}$ – that emits sequences taken from some fixed alphabet of possible outcomes. Thus an output sequence of length n, x_n, termed a path, will have the form

$$x_n = (\alpha_0, \alpha_1, ..., \alpha_{n-1})$$

where α_k is the value at step k of the stochastic variate X_k,

$$X_k = \alpha_k.$$

A particular sequence x_n will have the probability

$$P(X_0 = \alpha_0, X_1 = \alpha_1, ..., X_{n-1} = \alpha_{n-1}),$$

(1)

with associated conditional probabilities

$$P(X_n = \alpha_n | X_{n-1} = \alpha_{n-1}, ..., X_0 = \alpha_0).$$

(2)

Thus substrings of x_n are not, in general, stochastically independent. That is, there may be powerful serial correlations along the x_n. We call $\mathbf{X}$ an information source, and are particularly interested in sources for which the long run frequencies of strings converge stochastically to their time-independent probabilities, generalizing the law of large numbers. These we call *ergodic* (Ash, 1990, Cover and Thomas, 1992, Khinchine, 1957. We will refer to these in the future as ACTK). If the probabilities of strings do not change in time, the source is called *memoryless*.

We shall be interested in sources which can be parametized and that are, with respect to that parameter, *piecewise memoryless*, i.e. probabilities do not change markedly within a 'piece,' but may do so between pieces. This allows us to apply the simplest results from information theory, and to use renormalization methods to examine transitions between 'pieces.' Learning plateaus represent regions where, with respect to the parameter, the system is, to first approximation, memoryless in this sense. In what follows we use the term 'ergodic,' to mean 'piecewise memoryless ergodic.'

For any ergodic information source it is possible to divide all possible sequences of output, in the limit of large n, into two sets, S_1 and S_2, having, respectively, very high and very low probabilities of occurrence. Sequences in S_1 we call *meaningful*.

The content of information theory's Shannon-McMillan Theorem is twofold:

First, if there are $N(n)$ meaningful sequences of length n, where $N(n) \ll$ than the number of all possible sequences of length n, then, for each ergodic information source $\mathbf{X}$, there is a unique, path-independent number $H[\mathbf{X}]$ such that

$$\lim_{n \to \infty} \frac{\log[N(n)]}{n} = H[\mathbf{X}].$$

(3)

See ACTK for details.

Thus, for large n, the probability of *any* meaningful path of length $n \gg 1$ – independent of path – is approximately

$$P(x_n \in S_1) \propto \exp(-nH[\mathbf{X}]) \propto 1/N(n).$$

(3)

This is the *asymptotic equipartition property* and the Shannon-McMillan Theorem is often called the Asymptotic Equipartition Theorem (AEPT).

$H[\mathbf{X}]$ is the *splitting criterion* between the two sets S_1 and S_2, and the second part of the Shannon-McMillan Theorem involves its calculation. This requires introduction of some nomenclature.

Suppose we have stochastic variables X and Y which take the values x_j and y_k with probability distributions

$$P(X = x_j) = P_j$$

$$P(Y = y_k) = P_k$$

Let the joint and conditional probability distributions of X and Y be given, respectively, as

$$P(X = x_j, Y = y_k) = P_{j,k}$$

$$P(Y = y_k | X = x_j) = P(y_k | x_j)$$

The *Shannon uncertainties* of X and of Y are, respectively

$$H(X) = -\sum_j P_j \log(P_j)$$

$$H(Y) = -\sum_k P_k \log(P_j)$$

(4)

The *joint uncertainty* of X and Y is defined as

$$H(X,Y) = -\sum_{j,k} P_{j,k} \log(P_{j,k}).$$

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(5)

The *conditional uncertainty* of Y given X is defined as

$$H(Y|X) = -\sum_{j,k} P_{j,k} \log[P(y_k|x_j)].$$

(6)

Note that by expanding $P(y_k|x_j)$ we obtain

$$H(X|Y) = H(X,Y) - H(Y).$$

The second part of the Shannon-McMillan Theorem states that the – path independent – splitting criterion, $H[\mathbf{X}]$, of the ergodic information source $\mathbf{X}$, which divides high from low probability paths, is given in terms of the sequence probabilities of equations (1) and (2) as

$$H[\mathbf{X}] = \lim_{n \to \infty} H(X_n|X_0, X_1, ..., X_{n-1}) =$$

$$\lim_{n \to \infty} \frac{H(X_0, ..., X_n)}{n+1}.$$

(7)

The AEPT is one of the most unexpected and profound results of 20th Century applied mathematics.

Ash (1990) describes the uncertainty of an ergodic information source as follows;

"...[W]e may regard a portion of text in a particular language as being produced by an information source. the probabilities $P[X_n = \alpha_n | X_0 = \alpha_0, ..., X_{n-1} = \alpha_{n-1})$ may be estimated from the available data about the language. A large uncertainty means, by the AEPT, a large number of 'meaningful' sequences. Thus given two languages with uncertainties H_1 and H_2 respectively, if $H_1 > H_2$, then in the absence of noise it is easier to communicate in the first language; more can be said in the same amount of time. On the other hand, it will be easier to reconstruct a scrambled portion of text in the second language, since fewer of the possible sequences of length n are meaningful."

Languages can affect each other, or, equivalently, systems can translate from one language to another, usually with error. The Rate Distortion Theorem, which is one generalization of the SMT, describes how this can take place. As IR Cohen (2001) has put it, in the context of the cognitive immune system,

"An immune response is like a key to a particular lock; each immune response amounts to a functional image of the stimulus that elicited the response. Just as a key encodes a functional image of its lock, an effective [immune] response encodes a functional image of its stimulus; the stimulus and the response fit each other. The immune system, for example, has to deploy different types of inflammation to heal a broken bone, repair an infarction, effect neuroprotection, cure hepatitis, or contain tuberculosis. Each aspect of the response is a functional representation of the challenge.

Self-organization allows a system to adapt, to update itself in the image of the world it must respond to... The immune system, like the brain... aim[s] at representing a part of the world."

These considerations suggest that the degree of possible back-translation between the world and its image within a cognitive system represents the profound and systematic coupling between a biological system and its environment, a coupling which may particularly express the way in which the system has 'learned' the environment. We attempt a formal treatment, from which it will appear that both cognition and response to systematic patterns

of selection pressure are – almost inevitably – highly punctuated by 'learning plateaus' in which the two processes can become inextricably intertwined.

Suppose we have a ergodic information source $\mathbf{Y}$, a generalized language having grammar and syntax, with a source uncertainty $H[\mathbf{Y}]$ that 'perturbs' a system of interest. A chain of length n, a path of perturbations, has the form

$$y^n = y_1, ..., y_n.$$

Suppose that chain elicits a corresponding chain of responses from the system of interest, producing another path $b^n = (b_1, ..., b_n)$, which has some 'natural' translation into the language of the perturbations, although not, generally, in a one-to-one manner. The image is of a continuous analog audio signal which has been 'digitized' into a discrete set of voltage values. Thus, there may well be several different y^n corresponding to a given 'digitized' b^n. Consequently, in translating back from the b-language into the y-language, there will generally be information loss.

Suppose, however, that with each path b^n we specify an inverse code which identifies exactly one path $\hat{y}^n$. We assume further there is a measure of distortion which compares the real path y^n with the inferred inverse $\hat{y}^n$. Below we follow the nomenclature of Cover and Thomas (1992).

The *Hamming distortion* is defined as

$$d(y, \hat{y}) = 1, y \neq \hat{y}$$

$$d(y, \hat{y}) = 0, y = \hat{y}.$$

(8)

For continuous variates the *Squared error distortion* is defined as

$$d(y, \hat{y}) = (y - \hat{y})^2.$$

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(9)

Possibilities abound.

The distortion between paths y^n and $\hat{y}^n$ is defined as

$$d(y^n, \hat{y}^n) = (1/n) \sum_{j=1}^{n} d(y_j, \hat{y}_j)$$

(10)

We suppose that with each path y^n and b^n-path translation into the y-language, denoted $\hat{y}^n$, there are associated individual, joint and conditional probability distributions $p(y^n), p(\hat{y}^n), p(y^n, \hat{y}^n)$ and $p(y^n|\hat{y}^n)$.

The *average distortion* is defined as

$$D = \sum_{y^n} p(y^n) d(y^n, \hat{y}^n)$$

(11)

It is possible, using the distributions given above, to define the information transmitted from the incoming Y to the outgoing $\hat{Y}$ process in the usual manner, using the appropriate Shannon uncertainties:

$$I(Y, \hat{Y}) \equiv H(Y) - H(Y|\hat{Y}) = H(Y) + H(\hat{Y}) - H(Y, \hat{Y})$$

(12)

If there is no uncertainty in Y given $\hat{Y}$, then no information is lost. In general, this will not be true.

The *information rate distortion* function $R(D)$ for a source Y with a distortion measure $d(y, \hat{y})$ is defined as

$$R(D) = \min_{p(y|\hat{y}); \sum_{(y,\hat{y})} p(y)p(y|\hat{y})d(y,\hat{y}) \leq D} I(Y, \hat{Y})$$

(13)

where the minimization is over all conditional distributions $p(y|\hat{y})$ for which the joint distribution $p(y, \hat{y}) = p(y)p(y|\hat{y})$ satisfies the average distortion constraint.

The Rate Distortion Theorem states that $R(D)$, as we have defined it, is the maximum achievable rate of information transmission which does not exceed distortion D. Note that the result is *independent of the exact form of the distortion measure $d(y, \hat{y})$*.

More to the point, however, is the following: Pairs of sequences $(y^n, \hat{y}^n)$ can be defined as *distortion typical*, that is, for a given average distortion D, pairs of sequences can be divided into two sets, a high probability one containing a relatively small number of (matched) pairs with $d(y^n, \hat{y}^n) \leq D$, and a low probability one containing most pairs. As $n \to \infty$ the smaller set approaches unit probability, and we have for those pairs the condition

$$p(\hat{y}^n) \geq p(\hat{y}^n|y^n) \exp[-nI(Y, \hat{Y})].$$

(14)

Thus, roughly speaking, $I(Y, \hat{Y})$ embodies the splitting criterion between high and low probability pairs of paths. These pairs are, again, the input 'training' paths and corresponding output path.

Note that, in the absence of a distortion measure, this result remains true for two interacting information sources, the principal content of the *joint asymptotic equipartition theorem*, [Cover and Thomas, 1992, Theorem 8.6.1].

Thus the imposition of a distortion measure results in a limitation in the number of possible jointly typical sequences to those satisfying the distortion criterion.

For the theory we will explore later – of pairwise interacting information sources – $I(Y, \hat{Y})$ (or $I(Y_1, Y_2)$ without the distortion restriction), can play the role of H in the critical development of the next section.

The RDT is a generalization of the Shannon-McMillan Theorem which examines the interaction of two information sources under the constraint of a fixed average distortion. For our development we will require one more iteration, studying the interaction of three 'languages' under particular conditions, and require a similar generalization of the SMT in terms of the splitting criterion for triplets as opposed to single or double stranded patterns. The tool for this is at the core of what is termed *network information theory* [Cover and Thomas, 1992, Theorem 14.2.3]. Suppose we have (piecewise memoryless) ergodic information sources Y_1, Y_2 and Y_3. We assume Y_3 constitutes a critical embedding context for Y_1 and Y_2 so that, given three sequences of length n, the probability of a particular triplet of sequences is determined by *conditional probabilities with respect to* Y_3:

$$P(Y_1 = y_1, Y_2 = y_2, Y_3 = y_3) =$$

$$\Pi_{i=1}^{n} p(y_{1i}|y_{3i}) p(y_{2i}|y_{3i}) p(y_{3i}).$$

(15)

That is, Y_1 and Y_2 are, in some measure, driven by their interaction with Y_3

Then, in analogy with the previous two cases, triplets of sequences can be divided by a splitting criterion into two sets, having high and low probabilities respectively. For large n the number of triplet sequences in the high probability set will be determined by the relation [Cover and Thomas, 1992, p. 387]

$$N(n) \propto \exp[nI(Y_1; Y_2|Y_3)],$$

(16)

where splitting criterion is given by

$$I(Y_1; Y_2|Y_3) \equiv$$

$$H(Y_3) + H(Y_1|Y_3) + H(Y_2|Y_3) - H(Y_1, Y_2, Y_3)$$

Below we examine phase transitions in the splitting criteria H, which we will generalize to both $I(Y_1, Y_2)$ and $I(Y_1, Y_2|Y_3)$. The former will produce punctuated cognitive and non-cognitive learning plateaus, while the latter characterizes the interaction between selection pressure and sociocultural cognition.

Phase transition and coevolutionary condensation

The essential homology relating information theory to statistical mechanics and nonlinear dynamics is twofold (Wallace and Wallace, 1998, 1999):

(1) A 'linguistic' equipartition of probable paths consistent with the Shannon-McMillan and Rate Distortion Theorems serves as the formal connection with nonlinear mechanics and fluctuation theory – a matter we will not fully explore here, and

(2) A correspondence between information source uncertainty and statistical mechanical free energy density, rather than entropy. See Wallace and Wallace (1998, 1999) and Rojdestvenski and Cottam, (2000) for a fuller discussion of the formal justification for this assumption, described by Bennett (1988) as follows:

"...[T]he value of a message is the amount of mathematical or other work plausibly done by the originator, which the receiver is saved from having to repeat."

This is a central insight to which we will return at some length below. In sum, we will generally impose invariance under renormalization symmetry on the 'splitting criterion' between high and low probability states from the Large Deviations Program of applied probability (e.g. Dembo and Zeitouni, 1998). Free energy density (which can be reexpressed as an 'entropy' in microscopic systems) is the splitting criterion for statistical mechanics, and information source uncertainty is the criterion for 'language' systems. Imposition of renormalization on free energy density gives phase transition in a physical system. For information systems it gives interactive condensation.

This analogy is indeed a mathematical homology:

The definition of the free energy density for a parametized physical system is

$$F(K_1, ...K_m) = \lim_{V \to \infty} \frac{\log[Z(K_1, .., K_m)]}{V}$$

(17)

where the K_j are parameters, V is the system volume and Z is the 'partition function' defined from the energy function, the Hamiltonian, of the system.

For an ergodic information source the equivalent relation associates source uncertainty with the number of 'meaningful' sequences $N(n)$ of length n, in the limit

$$H[\mathbf{X}] = \lim_{n \to \infty} \frac{\log[N(n)]}{n}.$$

We will *parametize* the information source to obtain the crucial expression on which our version of information dynamics will be constructed;

$$H[K_1, ..., K_m, \mathbf{X}] = \lim_{n \to \infty} \frac{\log[N(K_1, ..., K_m)]}{n}.$$

(18)

The essential point is that while information systems do not have 'Hamiltonians' allowing definition of a 'partition function' and a free energy density, they may have a source uncertainty obeying a limiting relation like that of free energy density. Importing 'renormalization' symmetry gives phase transitions at critical points (or surfaces), and importing a Legendre transform in a 'natural' manner gives dynamic behavior far from criticality, a matter leading to our analysis of the effects of sudden perturbation on coalesced cognitive systems.

As neural networks demonstrate so well, it is possible to build larger pattern recognition systems from assemblages of smaller ones. We abstract this process in terms of a generalized linked array of subcomponents which 'talk' to each other in two different ways. These we take to be 'strong' and 'weak' ties between subassemblies. 'Strong' ties are, following arguments from sociology (Granovetter, 1973), those which permit disjoint partition of the system into equivalence classes. Thus the strong ties are associated with some reflexive, symmetric, and transitive relation between components. 'Weak' ties do not permit such disjoint partition. In a physical system these might be viewed, respectively, as 'local' and 'mean field' coupling.

We fix the magnitude of strong ties, but vary the index of weak ties between components, which we call P, taking $K = 1/P$.

We assume the array, sensory activity and ongoing activity depend on three parameters, two explicit and one implicit. The explicit are K as above and an 'external field strength' analog J, which gives a 'direction' to the system. We may, in the limit, set $J = 0$.

The implicit parameter, which we call r, is an inherent generalized 'length' on which the phenomenon, including J and K, are defined. That is, we can write J and K as functions of averages of the parameter r, which may be quite complex, having nothing at all to do with conventional ideas of space, for example degree of niche partitioning in ecosystems.

Rather than specify complicated patterns of individual dependence or interaction for sensory activity, ongoing activity and array components, we

follow the direction suggested above and instead work entirely within the domain of the uncertainty of the ergodic information source dual to the large-scale pattern recognition process, which we write as

$$H[K, J, \mathbf{X}]$$

Imposition of invariance of H under a renormalization transform in the implicit parameter r leads to expectation of both a critical point in K, which we call K_C, reflecting a phase transition to or from collective behavior across the entire array, and of power laws for system behavior near K_C. Addition of other parameters to the system, e.g. some Q, results in a 'critical line' or surface $K_C(Q)$.

Let $\kappa = (K_C - K)/K_C$ and take χ as the 'correlation length' defining the average domain in r-space for which the dual information source is primarily dominated by 'strong' ties. We begin by averaging across r-space in terms of 'clumps' of length R, defining J_R, K_R as J, K for $R = 1$. Then, following Wilson's [53] physical analog, we choose the renormalization relations as

$$H[K_R, J_R, \mathbf{X}] = R^{\mathcal{D}} H[K, J, \mathbf{X}]$$

$$\chi(K_R, J_R) = \frac{\chi(K, J)}{R}$$

(19)

where $\mathcal{D}$ is a non-negative real constant, possibly reflecting fractal network structure. The first of these equations states that 'processing capacity,' as indexed by the source uncertainty of the system which represents the 'richness' of the inherent language, grows as $R^{\mathcal{D}}$, while the second just states that the correlation length simply scales as R.

Other, very subtle, symmetry relations – not necessarily based on elementary physical analogs – may well be possible. For example McCauley, (1993, p.168) describes the counterintuitive renormalization relations needed to understand phase transition in simple 'chaotic' systems.

For K near K_C, if $J \to 0$, a simple series expansion and some clever algebra (e.g. Wilson, 1971, the argument leading from his eqs. 4 and 5 to his eqs. 22 and 23) gives

$$H = H_0 \kappa^{s\mathcal{D}}$$

$$\chi = \chi_0 \kappa^{-s}$$

(20)

where s is a positive constant. Some rearrangement produces, near K_C,

$$H \propto \frac{1}{\chi^{\mathcal{D}}}$$

(21)

This suggests that the 'richness' of the pattern recognition language is inversely related to the domain dominated by disjointly partitioning strong ties near criticality. As the nondisjunctive weak ties coupling declines, the efficiency of the coupled system as an information channel declines precipitously near the transition point: see ACTK for discussion of the relation between channel capacity and information source uncertainty.

Far from the critical point matters are considerably more complicated, apparently driven by appropriate (and usually counterintuitive) generalizations of a physical system's 'Onsager relations,' described below in terms of a 'thermodynamics' of information sources.

The essential insight is that *regardless of the particular renormalization symmetries involved, sudden critical point transition is possible in the opposite direction for this model*, that is, from a number of independent, isolated and fragmented pattern recognition systems operating individually and more

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or less at random, into a single large, interlocked, coherent pattern recognition system, once the parameter K, the inverse strength of weak ties, falls below threshold, or, conversely, once the strength of weak ties parameter $P = 1/K$ becomes large enough.

Thus, increasing weak ties between them can bind several different pattern recognition or other 'language' processes into a single, embedding hierarchical metalanguage which contains the different languages as linked sub-dialects.

This heuristic insight can be made exact using a rate distortion argument:

Suppose that two ergodic information sources $\mathbf{Y}$ and $\mathbf{B}$ begin to interact, to 'talk' to each other, i.e. to influence each other in some way so that it is possible, for example, to look at the output of $\mathbf{B}$ – strings b – and infer something about the behavior of $\mathbf{Y}$ from it – strings y. We suppose it possible to define a retranslation from the B-language into the Y-language through a deterministic code book, and call $\hat{\mathbf{Y}}$ the translated information source, as mirrored by $\mathbf{B}$.

Take some distortion measure d comparing paths y to paths $\hat{y}$, defining $d(y, \hat{y})$. We invoke the Rate Distortion Theorem's mutual information $I(Y, \hat{Y})$, which is a splitting criterion between high and low probability pairs of paths. Impose, now, a parametization by an inverse coupling strength K, and a renormalization symmetry representing the global structure of the system coupling. This may be much different from the renormalization behavior of the individual components. If $K < K_C$, where K_C is a critical point (or surface), the two information sources will be closely coupled enough to be characterized as condensed.

We will make much of this below; cultural and genetic heritages are generalized languages, as are neural, immune, and sociocultural pattern recognition.

Pattern recognition as language

The task of this section is to express cognitive pattern recognition-and-response in terms of a ergodic information source constrained by the AEPT. This general approach would then apply to the immune system, the CNS and sociocultural networks. Pattern recognition, as we will characterize it here, proceeds by convoluting an incoming 'sensory' signal with an internal 'ongoing activity' and, at some point, triggering an appropriate action based on a decision that the pattern of the sensory input requires a response. For

the purposes of this work we do not need to model in any particular detail the manner in which the pattern recognition system is 'trained,' and thus adopt a 'weak' model which may have considerable generality, regardless of the system's particular learning paradigm, which can be more formally described using the RDT.

We will, fulfilling Atlan and Cohen's (1998) criterion of meaning-from-response, define a language's contextual meaning entirely in terms of system output.

The model is as follows: A pattern of sensory input is convoluted with a pattern of internal 'ongoing activity' to create a path

$$x = (a_0, a_1, ..., a_n, ...).$$

This is fed into a (highly nonlinear) 'decision oscillator' which generates an output $h(x)$ that is an element of one of two (presumably) disjoint sets B_0 and B_1.

We take

$$B_0 = b_0, ..., b_k$$

$$B_1 = b_{k+1}, ..., b_m.$$

Thus we permit a graded response, supposing that if

$$h(x) \in B_0$$

the pattern is not recognized, and

$$h(x) \in B_1$$

that the pattern is recognized and some action $b_j, k + 1 \leq j \leq m$ takes place.

We are interested in paths which trigger pattern recognition exactly once. That is, given a fixed initial state a_0 such that $h(a_0) \in B_0$, we examine all possible subsequent paths x beginning with a_0 and leading exactly once to the event $h(x) \in B_1$. Thus $h(a_0, a_1, ...a_j) \in B_0$ for all $j < m$ but $h(a_0, ..., a_m) \in B_1$.

For each positive integer n, let $N(n)$ be the number of paths of length n which begin with some particular a_0 having $h(a_0) \in B_0$, and lead to the

condition $h(x) \in B_1$. We shall call such paths 'meaningful' and assume $N(n)$ to be considerably less than the number of all possible paths of length n – pattern recognition is comparatively rare – and in particular assume that the finite limit

$$H = \lim_{n \to \infty} \frac{\log[N(n)]}{n}$$

exists and is independent of the path x. We will – not surprisingly – call such a pattern recognition process ergodic.

We may thus define a ergodic information source $\mathbf{X}$ associated with stochastic variates X_j having joint and conditional probabilities $P(a_0, ... a_n)$ and

$P(a_n | a_0, ..., a_{n-1})$ such that appropriate joint and conditional Shannon uncertainties satisfy the relations

$$H[\mathbf{X}] = \lim_{n \to \infty} \frac{\log[N(n)]}{n}$$

$$= \lim_{n \to \infty} H(X_n | X_0, ..., X_{n-1})$$

$$= \lim_{n \to \infty} \frac{H(X_0, ... X_n)}{n + 1}$$

We say this ergodic information source is *dual* to the pattern recognition process.

Different 'languages' will, of course, be defined by different divisions of the total universe of possible responses into different pairs of sets B_0 and B_1, or perhaps even by requiring more than one response in B_1 along a path. Like the use of different distortion measures in the RDT, however, it seems obvious that the underlying dynamics will all be qualitatively similar.

Meaningful paths – creating an inherent grammar and syntax – are defined entirely in terms of system response, as Atlan (1983, 1987, 1998) and Atlan and Cohen (1998) propose, quoting Atlan (1987)

"...[T]he perception of a pattern does not result from a two-step process with first perception of a pattern of signals and then processing by application of a rule of representation. Rather, a

given pattern in the environment is perceived at the time when signals are received by a kind of resonance between a given structure of the environment – not necessarily obvious to the eyes of an observer – and an internal structure of the cognitive system. It is the latter which defines a possible functional meaning – for the system itself – of the environmental structure."

Elsewhere (R Wallace, 2000b, 2001) we have termed this process an 'information resonance.'

Although we do not pursue the matter here, the 'space' of the a_j can be partitioned into disjoint equivalence classes according to whether states can be connected by meaningful paths. This is analogous to a partition into domains of attraction for a nonlinear or chaotic system, and imposes a 'natural' algebraic structure which can, among other things, enable multitasking (R Wallace, 2000b, 2001).

We can apply this formalism to the stochastic neuron: A series of inputs $y_i^j, i = 1...m$ from m nearby neurons at time j is convoluted with 'weights' $w_i^j, i = 1...m$, using an inner product

$$a_j = \mathbf{y}^j \cdot \mathbf{w}^j = \sum_{i=1}^{m} y_i^j w_i^j$$

(22)

in the context of a 'transfer function' $f(\mathbf{y}^j \cdot \mathbf{w}^j)$ such that the probability of the neuron firing and having a discrete output $z^j = 1$ is $P(z^j = 1) = f(\mathbf{y}^j \cdot \mathbf{w}^j)$. Thus the probability that the neuron does not fire at time j is $1 - f(\mathbf{y}^j \cdot \mathbf{w}^j)$.

In the terminology of this section the m values y_i^j constitute 'sensory activity' and the m weights w_i^j the 'ongoing activity' at time j, with $a_j = \mathbf{y}^j \cdot \mathbf{w}^j$ and $x = a_0, a_1, ...a_n, ...$

A little more work, described below, leads to a fairly standard neural network model in which the network is trained by appropriately varying the $\mathbf{w}$ through least squares or other error minimization feedback. This can be shown to, essentially, replicate rate distortion arguments, as we can use the

error definition to define a distortion function $d(y, \hat{y})$ which measures the difference between the training pattern y and the network output $\hat{y}$ as a function of, for example, the inverse number of training cycles, K. As we will discuss in some detail, 'learning plateau' behavior follows as a phase transition on the parameter K in the mutual information $I(Y, \hat{Y})$.

Park et al. (2000) treat the stochastic neural network in terms of a space of related probability density functions $[p(\mathbf{x}, \mathbf{y}; \mathbf{w})|\mathbf{w} \in \mathcal{R}^m]$, where $\mathbf{x}$ is the input, $\mathbf{y}$ the output and $\mathbf{w}$ the parameter vector. The goal of learning is to find an optimum $\mathbf{w}^*$ which maximizes the log likelihood function. They define a loss function of learning as

$$L(\mathbf{x}, \mathbf{y}; \mathbf{w}) \equiv -\log p(\mathbf{x}, \mathbf{y}; \mathbf{w}),$$

and one can take as a learning paradigm the gradient relation

$$\mathbf{w}_{t+1} = \mathbf{w}_t - \eta_t \partial L(\mathbf{x}, \mathbf{y}; \mathbf{w})/\partial \mathbf{w},$$

where η_t is a learning rate.

Park et al. (2000) attack this optimization problem by recognizing that the space of $p(\mathbf{x}, \mathbf{y}; \mathbf{w})$ is Riemannian with a metric given by the Fisher information matrix

$$G(\mathbf{w}) = \int \int \partial \log p/\partial \mathbf{w}[\partial \log p/\partial \mathbf{w}]^T p(\mathbf{x}, \mathbf{y}; \mathbf{w})d\mathbf{y}d\mathbf{x}$$

where T is the transpose operation. A Fisher-efficient on-line estimator is then obtained by using the 'natural' gradient algorithm

$$\mathbf{w}_{t+1} = \mathbf{w}_t - \eta_t G^{-1} \partial L(\mathbf{x}, \mathbf{y}; \mathbf{w})/\partial \mathbf{w}.$$

Again, through the synergistic family of probability distributions $p(\mathbf{x}, \mathbf{y}; \mathbf{w})$, this can be viewed as a special case – a 'representation', to use physics jargon – of the general 'convolution argument' given above.

Again, it seems that a rate distortion argument between training language and network response language will nonetheless produce learning plateaus, even in this rather elegant special case.

The foundation of our mathematical modeling exercise is to assume that both the immune system and a sociocultural network's pattern recognition behavior, like that of other pattern recognition systems, can also be represented by the language arguments given above, and is thus dual to a ergodic

information source, a context-defining language in Atlan and Cohen's sense (1998), having a grammar and syntax such that meaning is explicitly defined in terms of system response.

Sociogeographic or sociocultural networks – social networks embedded place and embodying culture – serve a number of functions, including acting as the local tools for teaching cultural norms and processes to individuals. Thus, for the purposes of this work, a person's social network – family and friends, workgroup, church, etc. – becomes the immediate agency of cultural dynamics, and provides the foundation for analysis of the 'brain/culture condensation'.

Sociocultural networks serve also, however, as instruments for collective decision-making, a cognitive phenomenon. Such networks serve as hosts to a political, in the large sense, process by which a community recognizes and responds to patterns of threat and opportunity. To treat pattern recognition on sociocultural networks we impose a version of the structure and general formalism relating pattern recognition to a dual information source:

We envision problem recognition by a local sociocultural network as follows: A 'real problem,' in some sense, becomes convoluted with a community's internal sociocultural 'ongoing activity' to create the path of a 'perceived problem' at times $0, 1, ...$, producing a path of the usual form $x = a_0, a_1, ..., a_n, ...$ That serially correlated path is then subject to a decision process across the sociocultural network, designated $h(x)$ which produces output in two sets B_0 and B_1, as before. The problem is officially recognized and resources committed to if and only if $h(x) \in B_1$, a rare event made even more rare if resources must then be diverted from previously recognized problems.

For the purposes of this work, then, we will view 'culture' as, in fact, a sociocultural cognitive process which can entrain individual CNS and immune cognition.

To reiterate, we have, following the earlier discussion of Atlan and Cohen's work (1998), implicitly assumed that the immune cognition can likewise be expressed as a pattern recognition-and-response language characterized by an information source uncertainty.

Toward a 'thermodynamics' of information source uncertainty

We suppose a cognitive system – more generally a linked and broadly coevolutionary condensation of several such systems – is exposed to a sudden

perturbation of sensory and other activity – trauma, toxic chemicals, and the like – and wish to estimate the response of that system. This requires some considerable development.

Since the source uncertainty of a (coevolutionarily condensed) behavioral language, $H[\mathbf{X}]$ is the average uncertainty per symbol, we must have $H[\mathbf{X}] \leq C$, where C is the capacity of the underlying geographically-focused social network as an information channel. We suppose that capacity is a function of system-wide average variables, K, J, Q which represent the *ensemble* indices – associated with the entire individual-and-group.

Thus we may write

$$C = C(K, J, Q)$$

$$H = H[K, J, Q, \mathbf{X}].$$

(23)

We assume that as K, J, Q increase, that $C(K, J, Q)$ and H decrease monotonically.

The essential trick – which is highly counterintuitive – is to recognize that, in its definition,

$$H[K, J, Q, \mathbf{X}] = \lim_{n \to \infty} \frac{\log[N(K, J, Q, n)]}{n}$$

(24)

is the analog of the *free energy density* of statistical mechanics: Take a physical system of volume V which can be characterized by an inverse temperature parameter $K = 1/T$. The 'partition function' for the system is (K. Wilson, 1971)

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$$Z(K) = \sum_j \exp[-KE_j]$$

(25)

where E_j represents the energy of the individual state j. The probability of the state j is then

$$P_j = \frac{\exp[-KE_j]}{Z(K)}.$$

Then the 'free energy density' of the *entire* system is defined as

$$F(K) = \lim_{V \to \infty} \frac{\log[Z(K)]}{V}.$$

(26)

The relation between information and thermodynamic free energy has long been recognized. As Elitzur (1996, p. 179) puts it

"Recall... the lesson of Maxwell's Demon: Information, when applied under appropriate circumstances, can save work."

Again, Bennett (1988, p. 230) finds that the value of a message is the amount of mathematical or other work plausibly done by its originator, which the receiver is saved from having to repeat.

Equation (24) expresses one of the central theorems of information theory in a form similar to equation (26). It is this similarity which suggests that, for some systems under proper circumstances, there may be a 'duality' which maps Shannon's ergodic source uncertainty onto thermodynamic free energy density, and perhaps vice versa.

The method we propose here, based entirely on equation (24), the Shannon-McMillan Theorem for ergodic information sources, may prove to be more generally applicable to information systems, not requiring the explicit identification a 'duality' in each and every case.

The formal analogy – the thermodynamic duality – between free energy density for a physical system and ergodic source uncertainty, based on equations (24) and (26), suggests that we impose a *thermodynamics* on source uncertainty.

By a thermodynamics we mean, in the sense of Feigenbaum (1988, p. 530),

> "...[T]he deduction of variables and their relations to one another that in some well-defined sense are averages of exponential quantities defined microscopically on a set."

In our context the relation between the number of meaningful sequences on length n, $N(n)$, for (fixed) large n and the source uncertainty, i.e.

$$N(n) \approx \exp(nH[\mathbf{X}])$$

for large n provides the exponential dependence exactly analogous to performing statistical mechanics. We are, to reiterate, going to express $H[\mathbf{X}]$ in terms of a number of parameters which characterize the underlying community which carries the behavioral language.

We suppose that H, representing the information source of our coevolutionary condensation of CNS, immune and sociocultural cognition, is allowed to depends on a number of observable parameters, which we will not fully specify here.

If source uncertainty H is the analog to free energy density in a physical system, K is the analog to inverse temperature, the next 'natural' step is to apply a *Legendre transformation* to H so as to define a generalized 'entropy,' S, and other (very) rough analogs to classical thermodynamic entities, depending on the parameters.

Courant and Hilbert (1989, p.32) characterize the Legendre transformation as defining a surface as the envelope of its tangent planes, rather than as the set of points satisfying a particular equation.

Their development shows the Legendre transformation of a well-behaved function $f(Z_1, Z_2, ...Z_w)$, denoted g, is

$$g = f - \sum_{i=1}^{w} Z_i \partial f / \partial Z_i \equiv f - \sum_{i=1}^{w} Z_i V_i.$$

(27)

with, clearly, $V_i \equiv \partial f / \partial Z_i$.

This expression is assumed to be invertible, hence the 'duality:'

$$f = g - \sum_{i=1}^{w} V_i \partial g / \partial V_i.$$

Transformation from the 'Lagrangian' to the 'Hamiltonian' formulation of classical mechanics (Landau and Lifshitz, 1959) is via a Legendre transformation.

The generalization when f is not well-behaved is via a variational principle (Beck and Schlogl, 1993) rather than a tangent plane argument. Then

$$g(V) = \min_{Z}[f(Z) - VZ]$$

$$f(Z) = \min_{V}[g(V) - VZ].$$

(28)

In the first expression the variation is taken with respect to Z, in the second with respect to V.

For a badly behaved function it is usually possible to fix up a reasonable invertible structure since the singularities of f or g will usually belong to 'a set of measure zero,' for example a finite number of points on a line or lines on a surface where we may designate inverse values by fiat.

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We first consider a very simple system in which the ergodic source uncertainty H depends *only* on the inverse strength of weak ties K, giving an analog to the 'canonical ensemble' of statistical mechanics which depends only on temperature. We define S, an entropy-analog which we term the 'disorder,' as the Legendre transform of the Shannon uncertainty $H[K, \mathbf{X}]$:

$$S = H - KdH/dK \equiv H - KU$$

(29)

where we take $U = dH/dK$ as an analog to the 'internal energy' of a system. Note that S and H have the same physical dimensionality.

Since

$$dS/dK = dH/dK - U - KdU/dK = -KdU/dK$$

we have

$$dS/dU = -K$$

and

$$dU \propto PdS.$$

This is the analog to the classic thermodynamic relation $dQ = TdS$ for physical systems. Thus what we have defined here as the disorder S is indeed a generalized entropy.

Note that since $dS/dU = -K$ we have

$$S = H - KU = H + UdS/dU$$

or

$$H = S - UdS/dU$$

which explicitly shows the dual relation between H and S.

Again let $N(n)$ represent the number of meaningful sequences of length n emitted by the source $\mathbf{X}$. Since

$$H[\mathbf{X}] = \lim_{n \to \infty} \frac{\log[N(n)]}{n}$$

for large n, we have

$$U = dH/dK = \lim_{n \to \infty} \frac{1}{nN} dN/dK.$$

(30)

For fixed (large) n, U is thus the *proportionate* rate of change in number of meaningful sequences of length n with change in K. This is something like the rate of change of mass per unit mass for a person losing weight: A small value will not be much noticed, while a large one may represent a rigorous starvation causing considerable distress.

Some rearrangement gives

$$I \equiv (S - H) = -K\,dH/dK = U\,dS/dU$$

(31)

We define $I = S - H$ as the *instability* of the system.

If $-dH/dK$ is approximately constant – something like a heat capacity in a physical system – then we have the approximate linear relation

$$I \approx b_K K$$

with

$$b_K \equiv -\partial H/\partial K.$$

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We generalize this as follows:

Allow H to depend on a number of parameters, for example average probability of weak ties, the inverse level of community resources, and or other factors which we call $Z_i, i = 1....$ Then, taking $H = H[Z_i, \mathbf{X}]$, we obtain the equations of state

$$S = H - \sum_{j=1}^{s} Z_j \partial H / \partial Z_j = H - \sum_{j=1}^{s} Z_j V_j.$$

$$V_i \equiv \partial H / \partial Z_i$$

(32)

and the *instability relation*

$$I = S - H = -\sum_{j=1}^{s} Z_j \partial H / \partial Z_j = \sum_{j=1}^{s} V_j \partial S / \partial V_j,$$

$$= -\sum_{j=1}^{s} V_j Z_j = -\mathbf{Z} \cdot \nabla|_{\mathbf{z}} H,$$

(33)

taking $\mathbf{Z} = (Z_1, Z_2, ..., Z_s)$.

I represents the degree of disorder above and beyond that which inherent to the ergodic information source itself.

We suggest that risk behaviors, patterns of anxiety, and of depression and demoralization, or other indicators of distress, are primary environmental indices of community instability.

Instability, as we have defined it, is driven by the declining capacity of behavioral language to convey messages across a community's sociogeographic networks.

We suppose that the capacity, C, of the underlying communication channel defined by the sociogeographic networks linked across the community declines with increasing K, so that $C = C(K)$ is monotonically decreasing in K. An ergodic information source can be transmitted without error by a channel only if $H[K, \mathbf{X}] < C(K)$ – again see ACTK – so that declining C will inevitably result in rising I.

I is, according to this development, driven by parameters characterizing public policy and underlying economic and social constraints – the Z_j and V_j.

For a social system, equation 33 is interpreted as stating that the rate of intentional violent deaths, depression or other indices of distress, is proportional a community's experience of instability.

It may be possible to generalize the development to include temporal effects if we suppose that H depends on $d\mathbf{Z}/dt \equiv \dot{\mathbf{Z}}$ as well as on $\mathbf{Z}$. Note that terms of the form $\partial H/\partial t$ would violate ergodicity. Then we would take

$$ I = -(\mathbf{Z}, \dot{\mathbf{Z}}) \cdot \nabla|_{(\mathbf{Z}, \dot{\mathbf{Z}})} H \equiv $$

$$ \sum_j -Z_j \partial H/\partial Z_j - \dot{Z}_j \partial H/\partial \dot{Z}_j. $$

(34)

This suggests that both parameter gradients and their rates of change can be globally destabilizing.

In linear approximation, assuming $-\partial H/\partial Z_i = -V_i = b_{Z_i} \approx$ constant, equation (22) can be rewritten as

$$ I \approx b_K K + b_J Q + b_M M. $$

The use of environmental index variates for critical system parameters will generally result in a nonzero intercept, giving the final equation

$$I \approx b_K K + b_J Q + b_M M + b_0.$$

(35)

Note that the intercept b_0 may, in fact, be quite complex, perhaps incorporating other parameters not explicitly included in the model. But it may include as well an 'error term' representing stochastic fluctuations not entirely damped by large population effects, or even some 'nonlinear' structure when the b_{Z_i} are not quite constant.

Most importantly for our analysis here, if the 'potentials' $V_i = \partial H/\partial Z_i$ cannot be approximated as constants, then simple linear regression will fail entirely, and equations (32) and (35) will represent an appropriate generic model – possibly with 'error terms' – however *the system will be both nonlinear and nonmonotonic*, hence representing signal transduction in physiological systems.

In sum, we claim the instability relation derived from a fairly simple quasi-thermodynamic argument applied to an ergodic information source parametized by various significant indices, (as well as, perhaps, their rates of change), explains the high degree to which simple regression models based on those parameters account for observed patterns of physiological, psychological, psychosocial, or immune response to perturbation. We further claim that extension of this model into nonlinear and nonmonotonic realms is quite direct, a matter we will not further treat here.

Ecological resilience of cognitive condensations

In reality, matters are significantly more complex than we have described so far: physiological and psychological responses are indeed produced by sudden deterioration of an individual's underlying circumstances, but may, in turn, affect these as well, basically through nonlinear, feedback loops.

Thus the inherently nonlinear model for response as produced by increasing stimulation, $I = -\sum_j V_j Z_j$ is replaced by an even more nonlinear structure:

$$I(t) = -\sum_j V_j(I(t))Z_j(I(t)).$$

(36)

In a first iteration using linear approximation, we can replace this equation with a series for which *each* of s variates – 'independent' as well as 'dependent' – is expressed in terms of s linear regressions on all the others:

$$x_i(t) = \sum_{j \neq i}^{s} b_{i,j} x_j(t) + b_{i,0} + \epsilon(t, x_1(t)...x_s(t)).$$

(37)

Here the $x_j, j = 1...s$ are both 'independent' and 'dependent' variates involved in the feedback, $b_{i,0}$ is the intercept constant, and the ϵ terms are 'error' terms which may not be small, in this approximation.

In matrix notation this set of equations becomes

$$X(t) = \mathbf{B}X(t) + U(t)$$

(38)

where $X(t)$ is a s-dimensional vector, $\mathbf{B}$ is an $s \times s$ matrix of regression coefficients having a zero diagonal and U is an s-dimensional vector containing the constant and 'error' terms. *'Error' terms are taken as including public and economic policy changes which are external to the internal feedback loops.*

We suggest that, on the timescale of applied perturbations and of initial responses, the **B**-matrix remains relatively constant. Following the analysis of Ives (1995) this structure has a number of interesting properties which permit estimation of the effects of external perturbation on the likelihood of triggering phase change in marginalized communities.

We begin by rewriting the matrix equation as

$$[\mathbf{I} - \mathbf{B}]X(t) = U(t)$$

(39)

where **I** is the $s \times s$ identity matrix and, to reiterate, **B** has a zero diagonal. We reexpress matter *in terms of the eigenstructure of* **B**.

Let **Q** be the matrix of eigenvectors which diagonalizes **B**. Take $\mathbf{Q}Y(t) = X(t)$ and $\mathbf{Q}W(t) = U(t)$. Let **J** be the diagonal matrix of eigenvalues of **B** so that $\mathbf{B} = \mathbf{Q}\mathbf{J}\mathbf{Q}^{-1}$. In R. Wallace, Y Huang, P Gould and D Wallace (1997) we show the eigenvalues of **B** are all real. Then, for the eigenvectors Y_k of **B**, corresponding to the eigenvalues λ_k,

$$Y_k(t) = \mathbf{J}Y_k(t) + W_k(t).$$

(40)

Using a term-by-term shorthand for the components of Y_k, this becomes

$$y_k(t) = \lambda_k y_k(t) + w_k(t).$$

(41)

Define the mean $E[f]$ of a time-dependent function $f(t)$ over the time interval $0 \to \Delta T$ as

$$E[f] = \frac{1}{\Delta T} \int_0^{\Delta T} f(t)dt.$$

(42)

We assume an appropriately 'rational' structure as $\Delta T \to \infty$, probably some kind of 'ergodic' hypothesis.

Note that this form of expectation does not include the effects of differing timescales or lag times. Under such circumstances, increasing ΔT will begin to 'pick up' new effects, in a path-dependent manner: The mathematics of equation (42) suddenly becomes extremely complicated.

The variance $V[f]$ over the same interval is defined as $E[f - E[f]^2]$.

Again taking matters term-by-term, we take the variance of the $y_k(t)$ from the development above to obtain

$$V[(1 - \lambda_k)y_k(t)] = V[w_k(t)]$$

so that

$$V[y_k] = \frac{V[w_k]}{(1 - \lambda_k)^2}$$

or

$$\sigma(y_k) = \frac{\sigma(w_k)}{|1 - \lambda_k|}.$$

(43)

The y_k are the components of the eigentransformed pathology, income, crowding, community size etc. variates x_i and the w_k are the similarly transformed variates of the driving externalities u_i.

The eigenvector components y_i are characteristic but non-orthogonal combinations of the original variates x_i whose standard deviation is that of the (transformed) externalities w_i, but *synergistically amplified* by the term $1/|1-\lambda_i|$, a function of the eigenvalues of the matrix of regression coefficients **B**. If $\lambda_i \to 1$ then any change in driving factors external to the community, like public policy or economic practice, will cause great instability within the affected community.

A simple example suffices. Suppose we have the two empirical regression equations

$$x_1 = b_1 x_2 + b_{01}$$

and

$$x_2 = b_2 x_1 + b_{02}$$

where x_1 is, for example, an index of violent crime and x_2 is an index of the 'strength of weak ties.' These equations say that weak ties affect violence and violence affects weak ties. Then, after normalizing x_1 and x_2 to zero mean and unit variance, the **B**-matrix becomes

$$\mathbf{B} = \begin{pmatrix} 0 & R \\ R & 0 \end{pmatrix}$$

where $R = b_1 = b_2$ is simply the correlation between x_1 and x_2.

This matrix has eigenvalues $\pm|R|$ and eigenvectors $[\pm 1/\sqrt{2}, 1/\sqrt{2}]$. As the variates become more closely correlated, $R \to 1$ and the ratio of the standard deviation of the eigenvector with positive components and that of the external perturbations, $1/[1-R]$, diverges.

There is a kind of physical picture for this model. Imagine a violin strung with limp, wet cotton twine. Then $R \approx 0$ and no amount of bowing – an external perturbation – will excite any sound from the instrument. Now restring that violin with finely tuned catgut (to be somewhat old fashioned).

Then $R \approx 1$ and external perturbation – bowing – will now excite loud and brisant eigenresonances.

Ives (1995) defines an ecosystem for which $\lambda \approx 0$, so that $1/|1 - \lambda| \approx 1$, as *resilient* in the sense that applied perturbations will have no more effect than their own standard deviation.

As a brief digression, in consonance with our previous arguments on phase transition, we might suppose that there are domains of quasi-stability marked by different possible relations of the form

$$X(t) = \mathbf{B}_k X(t) + U_k(t)$$

and that a sufficiently perturbed system will suddenly change between them. Suppose there to be a threshold value for an eigenresonance Y, which we call Y_C, such that if it is exceeded, then the system 'falls off the table' into a more pathological one. If Y has a normal distribution, then the probability that the system exceeds the critical limit Y_C, which we write Q_C, will be given by

$$Q_C = \frac{1}{\sqrt{2\pi}\sigma(Y)} \int_{Y_C}^{\infty} \exp[-Y/2\sigma^2(Y)]dY$$

$$= \frac{1}{2}[1 - ERF(\frac{Y_C}{\sqrt{2}\sigma(Y)})]$$

(44)

where ERF is the error function.

For large Y_C and $\lambda \to 1$ this becomes

$$Q_C \approx \frac{1}{Y_C|1 - \lambda|}\sqrt{V[w]/2\pi} \propto \frac{1}{Y_C|1 - \lambda|}\sigma(w).$$

(45)

A nonlinear treatment of these matters is clearly necessary, but will be difficult. Such treatments are becoming routine in population and community ecology (e.g. Higgins et al., 1997), but are still rare in epidemiological or physiological studies. The essential point is that a nonlinear deterministic 'backbone' serves to amplify external perturbations (Higgins et al., 1997):

> "...[R]elatively small environmental perturbations can markedly alter the dynamics of deterministic biological mechanisms, producing very large fluctuations..."

This, then, is our version of Holling's (1992) mechanism for the loss of ecological resilience by which the small can affect the large.