

COMMUNITY LYNCHING AND THE US ASTHMA EPIDEMIC

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Abstract

We explore the implications of I.R. Cohen's work on immune cognition for understanding rising rates of asthma morbidity and mortality in the US. Immune cognition is inherently linked with central nervous system cognition, and with the cognitive function of the embedding sociocultural networks by which individuals are acculturated and through which they work with others to meet challenges of threat and opportunity. Externally-imposed patterns of 'structured stress' can, through their effect on a child's socioculture, become synergistic with the development of immune cognition, triggering the persistence of an atopic Th2 phenotype, a necessary precursor to asthma and other immune disease. Structured stress in the US particularly includes the cross sectional and longitudinal effects of a systematic destruction of minority urban communities occurring since the end of World War II which we characterize as community lynching. Reversal of the rising tide of asthma and related chronic diseases in the US thus seems unlikely without a 21st Century version of the earlier Great Urban Reforms which ended the scourge of infectious diseases, in particular an end to the de-facto ethnic cleansing of minority neighborhoods.

Key words: American Apartheid, asthma, atopy, community lynching, immune cognition, information theory, renormalization, stress.

Running head: Community lynching and asthma

Introduction

Karlsen and Nazroo [1] open a recent discussion of the relation between social class and health among ethnic minority groups by arguing

"...that the contemporary equivalent of [individual] lynching... may have an important effect on the health experience of ethnic minorities in industrialized countries. The way in which this has been ignored in research in general and health research in particular, means that an important element of social disadvantage has been inadequately explored."

Karlsen and Nazroo [1], in the context of the United Kingdom, focus on the individual experience of personal and institutional racism as that equivalent, and indeed show clear impacts of individual-level discrimination.

We have, elsewhere and at length, examined population-level effects of public policies of 'urban renewal' and 'planned shrinkage' directed against ethnic minority communities in the US. In

some contrast to [1], we find that the contemporary equivalent of lynching in the US is not a matter of individually directed verbal or physical attacks, or even of individually-experienced institutional racism, which do indeed exist. It is, rather, the persistent, systematic, large scale dispersal and dismemberment of urban ethnic minority concentrations of political, social, and economic capital [e.g. 2-4]. Late twentieth century lynching in the US was not done one-by-one using a rope, but rather, we claim, with wholesale efficiency using bulldozers and the systematic withdrawal of fire, sanitation, and other housing-related services [2-4]. These tools triggered vast outbreaks of contagious urban decay which have left virtually every urban ghetto in the US looking like Dresden or Hiroshima after World War II.

The consequences for health and illness at every level of scale and organization have been dire indeed: For example, a study by McCord and Freeman [5] of New York City's Central Harlem found that, by 1980, the men of that community had lower life expectancy than the men of Bangladesh. Our own studies have shown profound effects on local and regional tuberculosis [6-8], local, regional, and national patterns of AIDS [8-11], local and regional deadly violence [8, 11-14], and the increasing susceptibility of the US as a whole to the growing risk of deadly emerging infection [15].

Within the context of the US political system, blame-the-victim genetic and behavioral 'explanations' of the resulting patterns of dysfunction and illness have proliferated (e.g. [16]). The already large literature contesting such 'explanations' is rapidly growing. Indeed, an entire recent issue of The American Journal of Public Health [17], including the Karlsen and Nazroo article, has been dedicated to related topics, and we refer the reader to that volume for an overview.

Here we present a new theoretical perspective addressing the cascading impacts of coherent large-scale patterns of externally-imposed discrimination across levels of organization, from the social, to the individual, to the cellular. Since the analysis is based on recent elaborate mathematical models of the immune system [18], we are, in the sense of Pielou [19], raising questions for subsequent empirical test rather than answering them, work which remains to be done.

Our focus is on community lynching and our contention is that policies causing destruction of urban minority communities in the US, after 1980, began to literally write themselves onto the developing immune systems of children in the affected areas, triggering a pattern of atopy deeply implicated in the subsequent development of the US 'asthma epidemic'. We will outline how this might be possible. For the sake of brevity we will not, however, recapitulate the usual 'genetic susceptibility' or 'air-pollution-and-rat-feces' explanations, and refer to [18] for mathematical detail. In spite of these simplifications, the argument remains fairly arduous.

The US 'asthma epidemic'

Morbidity and mortality from asthma have risen nearly 50 % in the US since 1980 [20, 21]. In 1994 asthma-related demand for medical care accounted for one sixth of all emergency room visits and one out of eleven doctors' office visits [21]. By 1994, nearly 15 % of all urban children were afflicted with asthma, compared with 7 % of the entire US population. Among children one to four years of age, asthma hospital discharge rates increased 57 % between 1980 and 1992, nationally, and African-American children in this age range were six times more likely to die of asthma than Caucasian children [20].

Carr et al. [22] described the late 1980's geography of asthma in New York City: Minority neighborhoods such as Harlem, the South Bronx, Bedford-Stuyvesant, North Crown Heights and

Washington Heights showed roughly five times the asthma mortality incidence of that found in affluent neighborhoods such as the Upper East Side, South Staten Island, and Forest Hills. Carr et al. [22] found that "Household income, percentage of population Black, and percentage of population Hispanic were significant predictors of area hospitalization rates (adjusted $R^2=0.75$)."

This pattern, which was later confirmed by DePalo et al. [23], is typical for other large US cities as well [24, 25], and suggests, prima facie, that the highly structured psychosocial stressors of the system of American Apartheid have very recently become entrained into the developing immune systems of urban minority children.

We postulate mechanisms by which such a 'phase transition' can take place, and, at the population level, produce widespread precursor conditions for a subsequent outbreak of asthma and related atopic diseases.

Asthma is necessarily associated with failure of the child's developing immune system to switch from the Th2 'humoral' phenotype thought necessary to prevent maternal rejection in utero to a predominantly Th1 'cellular' phenotype more suited to the functioning of acquired immunity [26]. Biochemical feedback mechanisms tend to fix one or the other mode once it becomes developmentally predominant, although they tend to overlap somewhat, and are not 'orthogonal' [26]. This mechanism will be the focus of our modeling exercise.

According to current theory, five factors affect the 'decision' as to which phenotype will emerge from a newly-developed T cell, the 'naive' Th0 cell [26]. These include:

1. Local cytokine milieu; mainly IL-4 for Th2 and IL-12 for Th1.
2. Presence of immunologically active hormones, e.g., glucocorticoids stimulate Th2 and inhibit Th1. Similarly, catecholamines inhibit type 1 cytokine production and stimulate type 2 cytokines.
3. Dose and route of antigen presentation. High antigen doses suppress cell-mediated immunity, Th1, apparently a protective effect against self-tissue destruction.
4. The type of presenting cell stimulating the T cell.
5. The 'strength of signal' which is an ill-defined summation of the affinity of the T-cell receptor for the major histocompatibility complex (MHC), combined with the timing and density of receptor ligation.

Of these five, the cytokine milieu surrounding the newly-activated T cell is thought to be the most important, but this is profoundly influenced by the other four. That is, there appears to be a complicated 'grammar' and 'syntax' to a meaningful 'statement' which results in the binary outcome of the Th1/Th2 polarization. The nature of these 'statements' is profoundly affected by 'stress.'

As Elenkov and Chrousos [27] put the matter,

"Recent evidence indicates that glucocorticoids and catecholamines, the end-products of the stress system... might selectively suppress cellular immunity [Th1], and favor humoral immune responses [Th2]. This is mediated by a differential effect of stress hormones and histamine on [Th1/Th2] patterns and type 1/type 2-cytokine production. Thus, systemically, stress might induce a Th2 shift, while, locally, under certain conditions, it might induce pro-inflammatory activities through neural activation of the peripheral corticotrophin-releasing factor-mast cell-histamine axis. Through the above mechanisms, stress may influence the onset and/or course of infectious, autoimmune/inflammatory, allergic and neoplastic diseases."

Clearly, then, the gestational and neonatal environment of the developing immune system will be critical in the 'decision' as to whether Th1 or Th2 immune phenotypes will predominate. Wright et al. [28] state the essential hypothesis as follows:

"Prospective seroepidemiological studies have shown that the newborn period is dominated by Th2 reactivity in response to allergens, and it is also evident that the Th1 memory cells selectively develop shortly after birth (at 3-6 months of age) and persist into adulthood in non-atopic subjects. For most children who become allergic or asthmatic, the polarization of their immune systems into an atopic phenotype probably occurs during early childhood.

These findings have sparked off vigorous investigation into the potential influence of early life environmental risk factors for asthma and allergy on the maturation of the immune system, in the hopes of understanding which factors will potentiate (or protect from) this polarization...

Although there is no direct evidence for the influence of stress on Th phenotype differentiation in the developing immune system, there is evidence that parental reports of life stress are associated with subsequent onset of wheezing in children between birth and one year. It has been speculated that stress triggers hormones in the early months of life which may influence Th2 cell predominance, perhaps through a direct influence of stress hormones on the production of cytokines that are thought to modulate the direction of differentiation."

The spatiotemporal pattern of the asthma increases among US children, in its exact match with patterns of residential segregation and community disintegration, suggests, however, that 'stress' is itself very highly structured. In this paper we will invoke Irun Cohen's theory of immune cognition to argue that the developing immune system interacts with, and is affected by, structured patterns of external stress through the intermediate medium of a local embedding -- and cognitive -- sociocultural network, of necessity including immediate family. Our development will further suggest that the internally coherent grammar and syntax, in a large sense, of that stress 'signal' are no less important than its 'magnitude'.

This is not a particularly new vision of the world. Interactions between the central nervous system (CNS) and the immune system, and between the genetic heritage and the immune system have long been officially recognized and academically codified through journals with titles such as Neuroimmunology and Immunogenetics. Here we will 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).

We first examine current visions of the interaction between genes and culture, and between the CNS and culture, and follow with a summary of Cohen's view of immune cognition. Next we argue that immune cognition and cognitive socioculture can become fused into a composite entity -- the ICC -- and that this composite, in turn, can be profoundly influenced by embedding systems of highly structured psychosocial and socioeconomic stressors. In particular, we argue that the internal structure of the stress -- its 'grammar' and 'syntax' -- are important in defining the coupling with the ICC.

Wallace [18] presents a detailed mathematical model of the ICC and its linkage with structured patterns of psychosocial or socioeconomic stress which is based on adapting renormalization techniques from statistical mechanics to information theory, in the spirit of the

Large Deviations Program of applied probability. The necessity of such an approach will emerge from examination of Cohen's theory of immune cognition.

Genes, cognition, and culture

Increasingly, biologists are roundly excoriating simple genetic reductionism which neglects the role of environment. Lewontin [29], 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. We propose reinterpreting immune function in this light, in particular the coupling of the individual immune system with larger, embedding structures.

The current vision of human biology among evolutionary anthropologists is consistent with Lewontin's analysis and is summarized by Durham [30] 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 [31].

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 built on the periphery of this basic genetic/cultural object [32].

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 [33]. 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 [34]. 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. [35] 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. [35] 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. [35] 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 gene-culture and brain-culture condensations.

Immune cognition

Recently Atlan and I.R. Cohen [36] have proposed an information-theoretic adaptation of Cohen's [37, 38] 'cognitive principle' model of immune function and process, a paradigm incorporating pattern recognition behaviors analogous to those of the central nervous system.

Atlan and Cohen [36] describe immune system behaviors of cognitive pattern recognition-and-response 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 Cohen [37, 38] 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.

Cohen's 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 is 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 cognition and culture

As shown at length in [18, 39-43], 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 metalanguages 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 human culture and the human 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.'

The evolutionary anthropologists' vision of the world, as we have interpreted it, sees language, culture, gene pool, and individual CNS and immune cognition as intrinsically melded and synergistic. We propose, then, that culture, as embodied in a local cognitive sociocultural network, and individual immune cognition may become a joint entity whose observation may be 'confounded' -- and even perhaps masked -- by the distinct population genetics associated with linguistic and cultural isolation.

The 'decision' of the developing immune system to switch or not switch from a Th2 to a Th1 phenotype is significantly different from the minute-to-minute or day-to-day 'immediate' function mode of the immune system which Atlan and Cohen describe above. Rather, it takes place on a considerably longer time scale, over much of the first year of life. The 'chemical language' of immediate function must, then, be collapsed -- 'integrated' -- in some manner to form a sequence of chemical signals having a non-uniquely 'renormalized' grammar and syntax. That is, many different functional patterns of signal on a short time scale can give the same integral. The simplest hypothesis is that the integration or renormalization period, like so much else, is determined by the 24-hour human activity pattern, which suggests, for example, linkage of the child's developing immune system with the parental or familial cortisol-leptin cycle, which alternates over the day. Voice patterns, facial expression, pheromone emission, expressed emotion, and so on, may all play an immediate role.

The cortisol-leptin cycle is worthy of some comment. Leptin, the newly-discovered 'fat hormone', increases Th1 and suppresses Th2 cytokine production [44] and also stimulates proliferation and activation of circulating monocytes, and may play a direct role in inflammatory processes [45]. Leptin and cortisol have, however, a complex relation. Cortisol, an adrenal stress hormone, and leptin alternate their plasma peaks as part of the normal circadian cycle [46]. Cortisol

increases can trigger answer leptin increases [47]. Glucocorticoid levels also influence plasma leptin levels [48]. Thus leptin and the adrenal hormones regulate each other: patterns of stress thus influence weight change, disease resistance, and inflammatory response. Th1/Th2 balance may be heavily influenced, in turn, by the adrenal hormone/leptin balance. Stress imposed on pregnant women may result in changes fetal immune and metabolic processes, with implications for birth weight, fat metabolism and risk for cardiovascular disease and allergenic susceptibility over the life course.

We suggest that the interplay of these factors over a day, and the correlational relations of renormalized or 'rate distorted' signals between sequences of days, constitutes no small part of the sociocultural milieu in which the child's developing immune system reaches its decision as to Th phenotype. The sociocultural network which envelops the child -- including but not limited to parent or parents -- in turn, engages in cognitive process to meet the structured challenges of threat and opportunity imposed upon it by the embedding socioeconomic system. Those challenges, to reiterate, have their own logical structure, their own grammar and syntax and, as [18] suggests, their powerful organization can impose itself down the nested hierarchy of interaction, to be translated, with some distortion, into the internal language of the child's developing immune system. As the next section indicates, this is not exactly a new thought.

Why neighborhoods count for individual health

Holling [49] has argued, since most ecologies are nested hierarchies, that a relatively few processes, having distinct frequencies in space and time, structure ecosystems, entrain other variables, and set the rhythm of ecosystem dynamics at other scales. A critical feature of such hierarchies is the asymmetric interactions between levels of organization. In particular, the larger, slower levels maintain constraints within which the faster levels operate. In that sense, then, slower levels control faster ones, but, in the context of a loss of ecological resilience at the larger scale, faster processes can affect slower ones by means of their 'brittleness,' a concept explored at more length for human populations below and in several other papers of this series (e.g. [50]).

In Holling's view [49], ecosystems are structured hierarchically by a small number of underlying processes into a small number of levels, each characterized by a distinct scale of 'architectural' texture and of temporal speed of variables.

Each of the small number of processes influencing structure does so over limited scale ranges. The temporal and architectural structure of discrete ecosystem components are determined by three broad groups of processes, each dominant over different ranges of scale: micro, meso and macro.

According to Holling [49], the meso level of organization plays a particularly critical role. There, distinct disturbance phenomena are triggered at thresholds of tens of meters to kilometers. These driving variables of disturbance form the kind and amount of structure found at mesoscales by causing local events to cascade upward in scale to affect much larger landscape patterns. Analysis of the function of mesoscale process and structure thus provides the bridge between individual and global dynamics.

The mesoscale of human ecosystems is the neighborhood. Elsewhere we have explored how vulnerable neighborhoods of marginalized communities in the largest US cities constitute 'keystone populations,' in Holling's sense, for the national ecology of emerging and re-emerging infection (e.g. [51]), and more recently, in the spread of contagious behaviors associated with chronic disease [52]. Here we attempt to extend this analysis downward in scale to examine how

systematic neighborhood disruption might affect the decision of a developing immune system to adopt a predominant Th1 or Th2 phenotype.

The Stage/State model of induced community collapse

By 1980, large US urban minority neighborhoods -- the keystones for public health at both larger and smaller scales -- had begun to reflect a relentless siege of community lynching by a combination of policy-related forces ranging from 'urban renewal', 'planned shrinkage,' and deliberately triggered contagious urban decay, whose effects were compounded by the impacts of widespread deindustrialization driven by the diversion of technical resources from civilian enterprise into the Cold War. We recapitulate something of the history leading up to those events, focusing on New York City's Harlem neighborhood.

Urbanization of African Americans began at the start of the 20th Century. Small numbers migrated to Northern cities, and established themselves as "succession" communities in neighborhoods that had housed other ethnic groups seeking entry into American urban life. Gradually these urban communities expanded, incorporating later waves of immigrants, among them many who were forced out of agricultural work because of the mechanization of the farms. The integration of these newcomers into urban life was slow, yet the communities grew in complexity and organization, gaining political and economic power along the way [53].

As noted earlier, a series of policies hostile to poor urban neighborhoods undermined their physical and social infrastructures. In the most serious cases, urban renewal obliterated whole communities, which simply ceased to exist. Later policies of systematic disinvestment, including planned shrinkage, led to gradual destruction of individual buildings, and the outbreak of widespread contagious urban decay which undermined wholesale the built environment. In either event, the functioning of social groups was fatally compromised by the alteration of the built environment within which they were embedded.

For Harlem, New York, an important African American community, a stage/state model has been proposed to describe community disintegration [54]. The model assumes a range of possible states of community organization, from the highly integrated "model community" at one end, to the very disintegrated "collection of individuals" at the other. The stage/state model postulates that communities are not statically set at a single point on this range. Rather, they are dynamic entities, constantly working to maintain internal organization. External stressors, such as a war, loss of employment in the community, outbreaks of contagious urban decay or epidemic disease, can destabilize a community and trigger its decomposition. The Stage/State Model postulates that the transformation from "model" to "collection" follows a spiral pathway, in which each turn of the spiral is triggered by a destabilizing event.

Three turns of the spiral are proposed for Harlem. The first stage follows the initial loss of housing and is characterized by confusion in the population. Efforts were undertaken to reorganize the community, but the failure to rebuild the urban infrastructure created a barrier to full recovery. In the case of Harlem, further destabilization was caused by several factors, including the loss of manufacturing jobs and further loss of housing. This second stage was characterized by increasing disorder in the population. Along with the increase in disorder was an increase in the use and sale of psychoactive substances, and a decline in social controls on violence and related behaviors.

The growing use of drugs, licit and illicit, was accompanied by a shift in social relationships. Drug behaviors, which had been confined to designated areas, were able to occupy more and more territory. The effects on family life were magnified. The likelihood of family

trauma, family dysfunction and family separation increased dramatically. Although in previous eras the 'home' and the 'street' had been carefully separated from each other, with increasing community disintegration, the 'street' was able to invade the territory of the 'home.'

This prepared the way for the incursion of crack cocaine in the mid-1980's [55-58]. The crack epidemic was extremely violent. Addiction to crack was very disabling, and involved a very large number of women. Both the noxious effects of crack, and the loss of the community-building efforts of women, contributed to further destabilization of the community, a stage characterized by 'non-sense' in the population. In this stage, it was common to observe scenes that were frankly unreal by any measure of life prior to the arrival of crack cocaine.

At each scale and level of organization -- the personal, the family, the small group and the community -- sharp changes in attitudes and behaviors have been documented. The exposure to trauma and violence has left psychological scars on a large portion of the population [59, 60]. The collapse of social relationships has increased the weight on individuals, as well as the sense of 'individualism.' The failure of group institutions has eroded the group power to contain and order behavior in public places. Further, behavior in public places has overwhelmed the interdiction to enter the home. The atomization of the population is one factor contributing to a marked loss of political participation and political consciousness among the population. The increasing political weakness of the group leaves it more vulnerable to majority decisions that are not in the best interest of the community. The siting of noxious facilities in vulnerable, disintegrating communities is one example. Closing firehouses in high fire incidence neighborhoods is another.

The argument presented here suggests that the experience of history has altered the functioning of the socio-geographic community of Harlem, and created heavy social, political and emotional burdens for all residents. The future course of individual and group life will be build from the experience of disintegration: the downward spiral towards non-sense thus sets the pathways to the next stage.

The story of Harlem illustrates the experience of virtually every large African-American and Puerto-Rican urban community since the end of World War II. By 1980 this disruption had contributed materially to the milieu of 'structured stress' which, we claim, has entrained immune development and function for many, if not most, community residents.

Discussion and conclusions

We have extended the mathematical model of [18] to encompass the cascading effects of the highly structured stress of community lynching on the inner workings of the developing immune system, and some explicit methodological caution is appropriate. Mathematical models of the immune system do not have a good record of success. 'Theoretical immunology' based on naïve technology transfer of reaction rate arguments, or of equally simplistic predator-prey models adapted uncritically from population dynamics, has made little or no contribution to immunology, particularly in the case of chronic disease [61]. The mathematical ecologist E.C. Pielou has relegated such exercises as ours to a very limited role [19, p. 106]:

"...[M]athematical models are easy to devise; even though the assumptions of which they are constructed may be hard to justify, the magic phrase 'let us assume that...' overrides objections temporarily. One is then confronted with a much harder task: How is such a model to be tested? The correspondence between a model's predictions and observed events is sometimes gratifyingly

close but this cannot be taken to imply the model's simplifying assumptions are reasonable in the sense that neglected complications are indeed negligible in their effects...

In my opinion the usefulness of models is great... [however] it consists not in answering questions but in raising them. Models can be used to inspire new field investigations and these are the only source of new knowledge as opposed to new speculation."

Very much in Pielou's sense, we have extended the mathematical model of [18] to speculate that the practice and policy of community lynching in the US is deeply implicated in the development of that nation's current asthma epidemic. 'Urban renewal' and 'planned shrinkage' of urban minority communities had, by 1980, interacted with the outfalls of a massive deindustrialization driven largely by the diversion of engineering and scientific resources into the Cold War [e.g. 9]. This synergism greatly affected community sociocultural networks, triggering a literal phase transition in their function, so that externally-imposed structured stress became part of the 'higher order' immune cognition associated with development, and rapidly expressed itself in a rising proportion of ghetto children having the atopic phenotype.

Our approach expands Cohen's vision of immune cognition one step by allowing day-to-day immune function to be 'renormalized' or 'integrated' into a longer-term developmental process, resulting in a 'higher order' cognitive decision regarding the binary shift from Th2 to Th1 immune phenotype. This is, we contend, the 'initiating event' in the chain of causality resulting in asthma. A second iteration of our theory would be required to understand the 'promoting process' by which children with atopic phenotype are driven to asthma. Such work remains to be done, but we might speculate that, once a Th2 phenotype has been expressed, a second iteration could involve a leptin-cortisol-modulated or driven switch between phenotypes expressive of specific immunoglobulin G (IgG) vs. specific immunoglobulin E (IgE) antibodies, IgE being the most characteristic 'asthma antibody'.

In sum, we propose that an immunocultural condensation affects 'higher order' processes as well, so that the cognitive functioning of the embedding sociocultural network becomes coevolutionarily condensed, in a large sense, with immune developmental cognition, and with the subsequent 'promotion' to explicit asthma. Although some mathematical details of such 'renormalized cognition' are discussed in [18, 39-43], further theoretical development, coupled with empirical study to clarify pathways, will clearly be necessary.

A structured pattern of externally-imposed sociocultural or socioeconomic driving stressors has, at the population level, clear grammar and syntax: certain intercorrelated patterns of abuse and injustice are recognizable and 'make sense' within American Apartheid at a particular time, others do not. For example, by the late 20th Century, we had moved from the individual killings and slave auctions of the 19th Century to population-level policies of community lynching, i.e. de-facto ethnic cleansing.

As [18] implies, the population-level 'language' of externally-imposed stress may very suddenly become closely coupled with the 'language' of local community sociocultural cognition in a kind of 'phase transition' to form a coevolutionary condensation. It is this joint object which we believe forms the embedding milieu for a child's developing immune system. Our model suggests that sociocultural phase transitions, like many such, are likely to be difficult to reverse, subject to a path-dependent 'hysteresis' requiring far more than the simple removal of the triggering structured stress for amelioration [43]. Information-theoretic phase transitions may, then, tend to persist even when the conditions which triggered them are changed, essentially because the internal grammar

and syntax of an information source 'makes sense,' and the making-of-sense tends to be conserved in time and resist change. Elsewhere we have made a quantitative argument regarding such matters, in the character of 'generalized Onsager relations' [18, 43]. Change, when it occurs, might well, according to our model, be very sudden. Thus corrective interventions against certain classes of problem -- like the rise of asthma in minority urban communities -- may not seem to work for a very long time, and will likely need to be highly proactive. This, too, is subject to empirical study.

The foremost intervention, of course, would be to significantly change the system of 'structured stress' which we believe to be the driving force behind the 'asthma epidemic.' Programs, policies and practices which further destabilize urban minority communities -- i.e. continuing the practice of community lynching -- will, in our view, exacerbate asthma and other patterns of chronic and infectious disease and behavioral pathology.

The multiscale, hierarchical, and interactive nature of the urban ecosystem means that interventions against asthma and other pathologies associated with community lynching need to be similarly multiscale, hierarchical and interactive, with the considerable effort focused, not just on the individual, but rather at the keystone neighborhood level.

We are suggesting, then, the necessity of a 21st Century chronic disease version of the Great Urban Reform Movement of the late 19th and early 20th Centuries which brought a virtual end to the scourges of infectious disease -- cholera, yellow fever, and the 'Captain of all the Men of Death', tuberculosis.

Recent empirical studies show that, as with the earlier contagious scourges, the conditions leading to increases in chronic disease cannot be confined within marginalized urban communities. A large and growing body of work implies that Holling's vision of keystone communities is indeed correct, and that the social disintegration of urban minority neighborhoods is acting upward in scale as well as downward, entraining even suburban communities [4, 51]. In particular the rising tide of asthma deaths has become regionalized in a very precise sense about certain US urban epicenters, as the growing decay indexed by the collapse of the inner-city itself became contagious, spreading into surrounding metropolitan areas along pathways defined by the daily journey-to-work [52]. 'Structured stress', once it emerged as a concentrated and proliferating social force within the largest central cities, could not be contained, and spread its deadly effect beyond the marginalized, dragging even the affluent down a slippery slope of chronic disease.

The structural reforms needed to reverse the asthma epidemic within US central cities in the 21st Century will benefit all, in much the same sense as did the bringing of clean water to the poor in the 19th.

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