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The cultural psychology of obesity: diffusion of pathological norms from Western to East Asian societies

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Abstract

We examine the accelerating worldwide obesity epidemic using a mathematical model relating a cognitive hypothalamic-pituitary-adrenal axis tuned by embedding cultural context to a signal of chronic, structured, psychosocial threat. The obesity epidemic emerges as a distorted physiological image of ratcheting social pathology involving massive, policy-driven, economic and social 'structural adjustment' causing increasing individual, family, and community insecurity. The resulting, broadly developmental, disorder, while stratified by expected divisions of class, ethnicity, and culture, is nonetheless relentlessly engulfing even affluent majority populations across the globe. The progression of analogous epidemics in affluent Western and East Asian societies is particularly noteworthy since these enjoy markedly different cultural structures, known to influence even such fundamental psychophysical phenomena as change blindness. Indeed, until recently population patterns of obesity were quite different for these cultures. We attribute the entrainment of East Asian societies into the obesity epidemic to the diffusion of Western socioeconomic practices whose imposed resource uncertainties and exacerbation of social and economic divisions constitute powerful threat signals. We find that individual-oriented 'therapeutic' interventions against obesity will be largely ineffective since the therapeutic process itself (e.g. reliance on drug treatments) embodies the very threats causing the epidemic.

Key Words culture, deindustrialization, deurbanization, globalization, hierarchy, information theory, obesity, phase transition, social dominance.

Introduction

Obesity is epidemic across the globe. Current rates of overweight and obesity in the United States are 61% and 14% in adults and children respectively. Obesity in adults has nearly doubled since 1980, from 15% in 1980 to 27% by 1999 (e.g. Wellman and Friedberg, 2002). Childhood overweight is rapidly rising in the US, particularly among African Americans and Hispanics. By 1998 prevalence increased to 21.5% in African Americans, 21.8% among Hispanics, and 12.3% among non-Hispanic whites aged 4 to 12 years (Strauss and Pollack, 2001).

Japan (e.g. Yoshiike et al., 2002; Isshiki et al., 2002; Matsushita et al., 2004) and China (e.g. Yu, 2006), although starting from significantly lower baselines, now show rapid increases in the prevalence of the problem. Yoshiike et al. (2002), for example, find that

> "Obesity has become a public health problem in Japan... By 2000 prevalence of preobese (bmi 25-29.9 kg/m^2) and obesity (bmi $\geq 30 kg/m^2$) was 24.5% and 2.3% in males, and 17.8% and 3.4% respectively in females aged 20 years and older. Although the fraction of the population of obese adults is quite low compared with western societies and some other Asian countries, the prevalence of overweight... reached one-fifth to one-quarter of Japanese adults."

This rapid change in previously non-obese, East Asian populations will be a particular focus of our analysis.

The obesity epidemic is associated with serious health conditions including type 2 diabetes, heart disease, high blood pressure and stroke, certain types of cancer, hypoxia, sleep apnea, hernia, and arthritis. It is a major cause of economic loss, death, and suffering which shows no indications of abatement.

Within the US obesity is unevenly distributed geographically, ethnically, and by socioeconomic class. Urban people of color (Allan, 1998), poor Southern states (Mokdad et al., 1999), and poor neighborhoods within cities (Ginsberg-Fellner et al, 1981) have higher prevalences. The Southern states form the epicenter of the geographically spreading epidemic (Mokdad et al., 1999), a picture of contagion between populations.

The famous Whitehall Studies of British civil servants (Brunner et al., 1997) found that coronary heart disease and

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central abdominal fat deposition incidences were strongly associated with the occupational hierarchy. Locus of work control was a major factor in both central abdominal fat deposition and coronary heart disease. Power relations in the workplace imposed a particular structure of stress.

Furthermore, stress which causes sleep deficits shifts metabolism toward fat accumulation and central abdominal deposition (Spiegel et al, 1999). The Hypothalamic-pituitary-adrenal (HPA) axis is central to the mechanisms (Chrousos, 2000; Bjorntorp, 2001). So the stress involves adrenal reactions to serious threats.

Our overall hypothesis is that large numbers of people across the globe now feel seriously threatened. The obesity epidemic embodies the consequences of public policies: economic insecurity from local deindustrialization and the related globalization of industrial work, social upheaval from policy-driven destruction of cities in the US, (e.g. Wallace and Wallace, 1998), wealth increasingly concentrated in fewer and fewer hands, and a voting ritual which, as recent US presidential elections show clearly, does not seem to matter. The US obesity epidemic embodies, in our view, a worsening crisis of democratic locus-of-control which will not be addressed by platitudes about 'eating less and exercising more'. The global epidemic, we will argue, represents the diffusion of a grossly exploitative system of socioeconomic structure and practice which has begun to threaten previously stable cultures across the world. Individual-oriented medicalized interventions such as obesity drugs and 'just-say-no' admonitions to eat less and exercise more will not serve at the population level to significantly reduce what is essentially an addictive behavior driven by powerful threat signals.

Stress and the HPA axis

Abdominal obesity and visceral fat accumulation are particularly associated with disease, and have become the focus of much research on 'stress' and its relation to the 'fight-or-flight' responses of the HPA axis. We paraphrase Bjorntorp (2001), who extensively summarizes the role of the HPA axis in physiological responses to stress.

When the input of noxious signals is prolonged, the HPA axis reactivity changes from normal and relatively transient attempts to maintain homeostasis or allostasis with temporary peaks of cortisol secretion, first, to a state of sensitization, which reacts with exaggerated cortisol secretion after a given input. This occurs during the most active phase of the HPA axis, which is the early morning in humans. When repeated too often and with sufficient strength of the input, the first sign of malfunction is a delayed down-winding, so that cortisol secretion stays elevated for a prolonged period of time. Subsequently, the normal diurnal pattern is disrupted, and morning values tend to be lower. This subsequently develops into a low, steady, rigid diurnal cortisol secretion with little reactivity, a 'burned out' HPA.

In parallel, the controlling, central glucocorticoid receptors become less efficient, and down-regulated. Further challenges are followed by atrophy of the entire system, often found after long-term, severe hypercortisolaemia as in Cushing's syndrome, melancholic depression, post traumatic stress disorder (PTSD), and the aftermath of war.

Bjorntorp (2001) describes how elevation of cortisol is followed by visceral fat accumulation. Much research shows consequent lowered sex steroid and growth hormone secretions have the same consequence, because of the insufficient counteraction against cortisol effects, and the combination of these abnormalities powerfully directs a larger than normal fraction of total body fat to visceral deposits.

In sum, increased activity of the HPA axis triggers both inhibition of both the pituitary gonadal and growth hormone axes. Stress may, then, synergistically cause accumulation of visceral fat, via elevated cortisol secretion and decrease of sex steroid and growth hormones.

Bjorntorp concludes in particular that the deposit of central body fat, which is closely correlated with general measures of obesity, can serve as a reasonable approximation to the long-term endocrine abnormalities associated with stress and often-repeated or chronic activation of the HPA axis.

That is, stress literally writes an image of itself onto the body as visceral fat accumulation, first having written an image of itself onto the HPA axis. The phenomenon can be interpreted as the transmission of a structured signal between communicating systems, in a large sense, i.e. from embedding psychosocial structure to individual HPA axis.

Recent studies using a primate animal model have uncovered possible mechanisms, showing a determining relation between food insecurity, early life stress, HPA axis dysregulation and subsequent metabolic syndrome and obesity (Coplan et al., 1996). Bonnet macaque mother-child pairs were subjected to variable foraging demand (VFD) for 12 weeks beginning when infants were 17 weeks old. Alternatively they had to search through a bin with food pellets mixed with wood chips (high foraging demand) or merely picked up food pellets out in the open (low foraging demand). Control mothers were exclusively subjected to either to low or high demand. VFD mothers were distracted and showed their infants much less attention and affection than control mothers. Persistent elevations of cerebrospinal fluid cortocotropin releasing factor (CSF-CRF) concentrentations and HPA axis suppression were evident in macaques whose mothers were subjected to VFD but not to control mothers. Moreover these CSF-CRF elevations in VFD endured from childhood into adulthood (Coplan et al., 2001).

Other changes that endured into adulthood included lower gregariousness, altered disturbance in response to fear stimulus, and greater social timidity (Rosenblum et al., 2001). Even brain structure showed change with alterations in teh same brain areas implicated in trauma-related psychiatric disorders (Matthew et al., 2003). Evidence from this primate model thus suggests maternal foraging unpredictability is stressful to developing infants. Both mothers subject to VFD and their infants show synchronized elevations of CSF-CRF (Coplan et al., 2005) Recent data indicate that VFD offspring exhibit early and accelerated onset of metabolic syncrome (Banerji et al., 2005).

In addition, juvenile CSF-CRF concentrations positively

predict adult BMI (Gohil et al., 2001). Thus these primate studies found that infants exposed to maternal food insecurity showed long-term behavioral, hormonal, brain structural and metabolic effects originating from the early life experience of maternal-infant bond disruption. The offspring responded to early food uncertainty in a developmental manner, with long term dysregulation of the HPA axis and subsequent elevations of BMI.

Here we will adapt recent developments involving a rate distortion manifold analysis of cognitive process to the question of how the communication of the embedding psychosocial structure and the HPA axis might be constrained by certain of the asymptotic limit theorems of probability. We know that, regardless of the distribution of a particular stochastic process, the Central Limit Theorem ensures that long sums of independent realizations of that process will follow a Normal distribution. Similar constraints exist on the behavior of communicating structures, and are described by the limit theorems of information theory. Importation of phase transition methods from statistical physics, done much in the spirit of the Large Deviations Program of applied probability, permits concise and unified description of evolutionary and cognitive learning plateaus which, in the evolutionary case, are interpreted as evolutionary punctuation (e.g. Wallace, 2002 b, 2005a, b, 2006, 2007; Wallace and Wallace, 1998, 1999).

'Stress' is seldom random in human societies, nor is it undifferentiated like pressure under water. It is usually highly structured, a language of sorts, having both a grammar and a syntax, so that certain stressors are meaningful in a particular cultural context, and others are not, having little or no long-term physiological effect. We will argue that the HPA axis is, in fact, a cognitive system itself associated with a 'dual information source' which may also be expressed as a kind of language. It is the punctuated interaction of these two language-analogs which we will find critical to an understanding of how psychosocial stress affects the HPA axis, which is itself tuned by embedding culture. Structured stress, ultimately, writes a distorted image of itself on the human body as visceral fat deposition, triggering what becomes a broadly developmental disorder.

This analysis has profound implications for intervention policy.

Cultural psychology

Recent studies in cultural psychology indicate that even the most basic psychophysical and physiological phenomena can be culturally modulated.

The necessity for the inclusion of culture in any analysis of the HPA axis perception of, and response to, 'threat' lies in the observations of Nisbett et al. (2001), and others, following the tradition of Markus and Kitayama (1991), regarding fundamental differences in perception between test subjects of Southeast Asian and Western cultural heritage across an broad realm of experiments. East Asian perspectives are characterized as holistic and Western as analytic. Nisbett et al. (2001) find:

(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 practice can directly influence the development and use of cognitive processes such as dialectical vs. logical ones.

Nisbett et al. (2001) 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.

Heine (2001) states the underlying case as follows:

> "Cultural psychology does not view culture as a superficial wrapping of the self, of as a framework within which selves interact, but as something that is intrinsic to the self. It assumes that without culture there is no self, only a biological entity deprived of its potential... Cultural psychology maintains that the process of becoming a self is contingent on individuals interacting with and seizing meanings from the cultural environment..."

More recently Masuda and Nisbett (2006) examined cultural variations in change blindness, a basic psychophysical phenomenon related to inattentional blindness, and found striking differences between Western and East Asian subjects:

> "We presented participants with still photos and with animated vignettes having changes in focal object information and contextual information. Compared to Americans, East Asians were more sensitive to contextual changes than to focal object changes. These results suggest that there can be cultural variation in what may seem to be basic perceptual processes."

The central focus of Wallace (2007) was on how culture can affect basic perceptual process, in essence creating a topological structure which constrains individual consciousness. Here we simplify that analysis and apply it to a cognitive, but not conscious, phenomenon, namely the manner in which the HPA axis perceives and respond to patterns of threat.

The onset of an obesity epidemic in East Asian societies, taking this perspective, is thus particularly notable, and will be explained in terms of the market selection-driven diffusion of pathological practices from Western culture.

HPA axis cognition

Atlan and Cohen (1998) argue that the essence of cognition is comparison of a perceived external signal with an internal,

learned picture of the world, and then, upon that comparison, the choice of a response from a much larger repertoire of possible responses. Clearly, from this perspective, the HPA axis, the 'flight-or-fight' reflex, is cognitive. Upon recognition of a new perturbation in the surrounding environment, emotional and/or conscious cognition (Wallace, 2004, 2005a,b, 2006, 2007) evaluate and choose from several possible responses: no action necessary, flight, fight, helplessness (i.e. flight or fight needed, but not possible). Upon appropriate conditioning, the HPA axis is able to accelerate the decision process, much as the immune system has a more efficient response to second pathogenic challenge once the initial infection has become encoded in immune memory. Certainly hyperreactivity in the context of PTSD is a well known example.

Following the approach of Wallace (2000, 2002a, 2004, 2005a, b, 2006, 2007), we make a very general model of that process.

Pattern recognition-and-response, as we characterize it, proceeds by algorithmically mixing (and comparing) an incoming external 'sensory' signal with an internal 'ongoing activity' – the 'learned picture of the world' – and, at some point, triggering an appropriate action based on a decision that the pattern of sensory activity requires a response. We need not, at this point, model how the pattern recognition system is tuned, and hence adopt a weak model regardless of learning paradigm, which can be more formally described by the Rate Distortion Theorem. 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. A subsequent section will examine cultural tuning of the HPA axis.

The basic model is as follows.

A pattern of sensory input is mixed in an unspecified but systematic manner with an internal ongoing activity to create a path of convoluted signal $x = (a_0, a_1, ..., a_n, ...)$. Each a_k thus represents some algorithmic or functional composition of internal and external signals. This path is fed into a highly nonlinear, but otherwise similarly unspecified highly nonlinear decision oscillator which generates an output $h(x)$ that is an element of one of two disjoint sets B_0 and B_1 of possible system responses. Take

$$B_0 \equiv b_0, ..., b_k,$$

$$B_1 \equiv b_{k+1}, ..., b_m,$$

and permit a graded response, supposing that if

$$h(x) \in B_0$$

the pattern is not recognized, and if

$$h(x) \in B_1$$

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

We are interested in paths x which trigger pattern recognition-and-response 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 $h(a_0, ..., a_j) \in B_0$ for all $j < m$, but $h(a_0, ..., a_m) \in B_1$. More complete discussion, leading to an elaborate groupoid formulation, is given in Wallace (2007).

For each positive integer n let $N(n)$ be the number of high probability 'grammatical' paths of length n which begin with some particular a_0 having $h(a_0) \in B_0$ and lead to the condition $h(x) \in B_1$. Call such paths 'meaningful' and assume, not unreasonably, that $N(n)$ is considerably less than the number of all possible paths of length n. While convolution algorithm, the form of the nonlinear oscillator, and the details of grammar and syntax, may all be unspecified in this model, the critical assumption which permits inference on necessary conditions is that the finite limit

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

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

It is thus possible to define a stationary, 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 may be defined which 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 information source is *dual* to the ergodic cognitive process.

The H-functions are cross-sectional sums of terms typically having the form

$$-\sum_k P_k \log[P_k]$$

where the P_k are joint or conditional probabilities, so that equation (1) represents a kind of law-of-large-numbers for 'language' systems. See Ash (1990) or Cover and Thomas (1991) for details, and Wallace (2007) for a more formal perspective.

Different language-analogs 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 by requiring more than one response in B_1 along a path. Like the use of different distortion measures in the Rate Distortion Theorem (e.g. Cover and Thomas, 1991), however, it seems obvious that the underlying dynamics will all be qualitatively similar. Dividing the full set of possible responses into the sets B_0 and B_1 may itself, however, require higher order cognitive decisions by other modules. This is an important matter to which we will return below, and serves as the foundation for speculations regarding obesity as a developmental disorder.

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

Wallace (2005a) applies this formalism to the stochastic neuron in a neural network.

We will eventually need to parametize the information source uncertainty of the dual information source to a cognitive pattern recognition-and-response with respect to one or more variates, writing, e.g. $H[\mathbf{K}]$, where $\mathbf{K} \equiv (K_1, ..., K_s)$ represents a vector in a parameter space. Let the vector $\mathbf{K}$ follow some path in time, i.e. trace out a generalized line or surface $\mathbf{K}(t)$. We will, following the argument of Wallace (2005a), assume that the probabilities defining H, for the most part, closely track changes in $\mathbf{K}(t)$, so that along a particular 'piece' of a path in parameter space the information source remains as close to stationary and ergodic as is needed for the mathematics to work. Between pieces we impose phase transition characterized by a renormalization symmetry, in the sense of Wilson (1971). See Binney, et al. (1986) for a more complete discussion.

We will call such an information source adiabatically piecewise stationary ergodic (APSE). The ergodic nature of the information sources is a generalization of the 'law of large numbers' and implies that the long-time averages we will need to calculate can, in fact, be closely approximated by averages across the probability spaces of those sources. This is no small matter.

This argument is consistent with what Adams (2003) has called the informational turn in philosophy. Our innovation is to return attention from the information content of individual symbols to the asymptotic properties of long streams of symbols, a kind of thermodynamic limit allowing importation of phase transition and quasi-thermodynamic methods from statistical physics via a homology between information source uncertainty and free energy density.

Note that our treatment does not preclude the existence of cognitive processes or submodules which may not have appropriately simple dual information sources. We cannot, however, address them in any obvious way, although some generalization may be possible to a subset of non-ergodic sources (e.g. Wallace, 2005a).

Interacting information sources: punctuated crosstalk

Suppose that the behavior of the HPA axis can be represented by a sequence of states in time, the path $x \equiv x_0, x_1, ...$ Similarly, we assume an external signal of structured psychosocial stress can be similarly represented by a path $y \equiv y_0, y_1, ...$ These paths are, however, both very highly structured and, within themselves, are serially correlated and can, in fact, be represented by information sources $\mathbf{X}$ and $\mathbf{Y}$. We assume the HPA axis and the external stressors interact, so that these sequences of states are not independent, but are jointly serially correlated. We can, then, define a path of sequential pairs as $z \equiv (x_0, y_0), (x_1, y_1), ...$

The essential content of the Joint Asymptotic Equipartition Theorem is that the set of joint paths z can be partitioned into a relatively small set of high probability which is termed *jointly typical*, and a much larger set of vanishingly small probability. Further, according to the JAEPT, the *splitting criterion* between high and low probability sets of pairs is the mutual information

$$I(X,Y) = H(X) - H(X|Y) = H(X) + H(Y) - H(X,Y) \tag{2}$$

where $H(X), H(Y), H(X|Y)$ and $H(X,Y)$ are, respectively, the Shannon uncertainties of X and Y, their conditional uncertainty, and their joint uncertainty. See Cover and Thomas (1991) for mathematical details. Similar approaches to neural process have been recently adopted by Dimitrov and Miller (2001).

The high probability pairs of paths are, in this formulation, all equiprobable, and if $N(n)$ is the number of jointly typical pairs of length n, then

$$I(X,Y) = \lim_{n \to \infty} \frac{\log[N(n)]}{n}. \tag{3}$$

Generalizing the earlier language-on-a-network models of Wallace and Wallace (1998, 1999), we suppose there is a 'coupling parameter' P representing the degree of linkage between the HPA axis and the system of external signals and stressors, and set $K = 1/P$, following the development of those earlier studies. Then

$$I[K] = \lim_{n \to \infty} \frac{\log[N(K,n)]}{n}.$$

The essential homology between information theory and statistical mechanics lies in the similarity of this expression with the infinite volume limit of the free energy density. If $Z(K)$ is the statistical mechanics partition function derived

from the system's Hamiltonian, then the free energy density is determined by the relation

$$F[K] = \lim_{V \to \infty} \frac{\log[Z(K)]}{V}. \quad (4)$$

F is the free energy density, V the system volume and $K = 1/T$, where T is the system temperature.

We and others argue at some length (e.g. Wallace, 2005a; Wallace and Wallace, 1998, 1999; Rojdestvensky and Cottam, 2000; Feynman, 1996) that this is indeed a systematic mathematical homology which, we contend, permits importation of renormalization symmetry into information theory. Imposition of invariance under renormalization on the mutual information splitting criterion $I(X, Y)$ implies the existence of phase transitions analogous to learning plateaus or punctuated evolutionary equilibria in the relations between the cognitive mechanism of the HPA axis and the system of external perturbations. An extensive mathematical treatment of these ideas is presented elsewhere (e.g. Wallace, 2000, 2002a, b, 2005a, b, 2007; Wallace et al., 2003).

Elaborate developments are possible. From a the more limited perspective of the Rate Distortion Theorem the onset of a punctuated interaction between the cognitive mechanism of the HPA and external stressors is a distorted image of those stressors within the HPA axis:

Suppose that two (piecewise, adiabatically stationary) ergodic information sources $\mathbf{Y}$ and $\mathbf{B}$ begin to interact, talking to each other, i.e. influencing 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 $\mathbf{B}$.

Define some distortion measure comparing paths y to paths $\hat{y}$, $d(y, \hat{y})$ (Cover and Thomas, 1991). We invoke the Rate Distortion Theorem's mutual information $I(Y, \hat{Y})$, which is the 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.

Extending the analysis, 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)

$$N(n) \propto \exp[nI(Y_1; Y_2|Y_3)], \quad (5)$$

where splitting criterion is given by

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

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

We can then examine mixed cognitive/adaptive phase transitions analogous to learning plateaus (Wallace, 2002b) in the splitting criterion $I(Y_1, Y_2|Y_3)$. These results are almost exactly parallel to the Eldredge/Gould model of evolutionary punctuated equilibrium (Eldredge, 1985; Gould, 2002).

Note that the expression above can be generalized to a number of interacting information sources, Y_j, embedded in a larger, generally slower, context, Z, as

$$I(Y_1, ..., Y_s|Z) = H(Z) + \sum_{j=1}^{s} H(Y_j|Z) - H(Y_1, ..., Y_s, Z) \quad (6)$$

If we impose another, even slower, embedding context-information source beyond this, say U, mathematical induction on equation (6) gives a very complicated expression which we write as

$$I(Y|Z|U) \quad (7)$$

This idea will prove important to subsequent development.

A simple HPA axis model

Stress is not a random sequence of perturbations, and is not independent of its perception. Rather, it involves a highly correlated, grammatical, syntactical process by which an embedding psychosocial environment communicates with an individual, particularly with that individual's HPA axis, in the context of social hierarchy and cultural interpretation. We view the stress experienced by an individual as APSE information source, interacting with a similar dual information source defined by HPA axis cognition.

Again, the ergodic nature of the language of stress is essentially a generalization of the law of large numbers, so

that long-time averages can be well approximated by cross-sectional expectations. Languages do not have simple autocorrelation patterns, in distinct contrast with the usual assumption of random perturbations by 'white noise' in the standard formulation of stochastic differential equations.

Let us suppose we cannot measure stress, but can determine the concentrations of HPA axis hormones and other biochemicals according to some natural time frame, the inherent period of the system. Suppose, in the absence of extraordinary meaningful psychosocial stress, we measure a series of n concentrations at time t which we represent as an n-dimensional vector X_t. Suppose we conduct a number of experiments, and create a regression model so that we can, in the absence of perturbation, write, to first order, the concentration of biomarkers at time $t+1$ in terms of that at time t using a matrix equation of the form

$$X_{t+1} \approx \mathbf{R}_0 X_t + b_0, \tag{8}$$

where $\mathbf{R}_0$ is the matrix of regression coefficients and b_0 a vector of constant terms. $\mathbf{R}_0$, we will argue below, is culturally determined and culturally tuned. Cultural changes can thus have profound effects on HPA axis behavior in the presence of stress signals.

Suppose that, in the presence of a perturbation by structured stress

$$X_{t+1} = (\mathbf{R}_0 + \delta\mathbf{R}_{t+1})X_t + b_0$$

$$\equiv \mathbf{R}_0 X_t + \epsilon_{t+1}, \tag{9}$$

where both b_0 and $\delta\mathbf{R}_{t+1}X_t$ are absorbed into a vector ϵ_{t+1} of error terms which are not necessarily small in this formulation. Note that this is not a population process whose continuous analog is exponential growth. Rather it is akin to the passage of a signal – structured psychosocial stress – through a distorting physiological filter.

If the matrix of regression coefficients $\mathbf{R}_0$ is sufficiently regular, we can (Jordan block) diagonalize it using the matrix of its column eigenvectors $\mathbf{Q}$, writing

$$\mathbf{Q}X_{t+1} = (\mathbf{Q}\mathbf{R}_0\mathbf{Q}^{-1})\mathbf{Q}X_t + \mathbf{Q}\epsilon_{t+1}, \tag{10}$$

or equivalently as

$$Y_{t+1} = \mathbf{J}Y_t + W_{t+1}, \tag{11}$$

where $Y_t \equiv \mathbf{Q}X_t, W_{t+1} \equiv \mathbf{Q}\epsilon_{t+1}$, and $\mathbf{J} \equiv \mathbf{Q}\mathbf{R}_0\mathbf{Q}^{-1}$ is a (block) diagonal matrix in terms of the eigenvalues of $\mathbf{R}_0$.

Thus the (rate distorted) writing of structured stress on the HPA axis through $\delta\mathbf{R}_{t+1}$ is reexpressed in terms of the vector W_{t+1}.

The sequence of W_{t+1} is the rate-distorted image of the information source defined by the system of external structured psychosocial stress. This formulation permits estimation of the long-term steady-state effects of that image on the HPA axis. The essential trick is to recognize that because everything is (APS) ergodic, we can either time or ensemble average both sides of equation (11), so that the one-period offset is absorbed in the averaging, giving an equilibrium relation

$$< Y >=< \mathbf{J} >< Y > + < W >$$

or

$$< Y >= (\mathbf{I} - < \mathbf{J} >)^{-1} < W >, \tag{12}$$

where $\mathbf{I}$ is the $n \times n$ identity matrix. We henceforth drop the brackets.

Now reverse the argument: Suppose that Y_k is chosen to be some fixed eigenvector of $\mathbf{R}_0$. Using the diagonalization of $\mathbf{J}$ in terms of its eigenvalues, we obtain the average excitation of the HPA axis in terms of some eigentransformed pattern of exciting perturbations as

$$Y_k = \frac{1}{1-\lambda_k} W_k \tag{13}$$

where λ_k is the eigenvalue of Y_k, and W_k is some appropriately transformed set of ongoing perturbations by structured psychosocial stress.

The essence of this result is that *there will be a characteristic form of perturbation by structured psychosocial stress – the W_k – which will resonantly excite a particular eigenmode of the HPA axis.* Conversely, by cultural or other tuning the eigenmodes of $\mathbf{R}_0$, the HPA axis can be trained to galvanized response in the presence of particular forms of perturbation.

This is because, if $\mathbf{R}_0$ has been appropriately determined from regression relations, then the λ_k will be a kind of multiple correlation coefficient (e.g. Wallace and Wallace, 2000), so that particular eigenpatterns of perturbation will have greatly amplified impact on the behavior of the HPA axis. If $\lambda = 0$ then perturbation has no more effect than its own magnitude. If, however, $\lambda \to 1$, then the written image of a perturbing psychosocial stressor will have very great effect on the HPA axis. Following Ives (1995), we call a system with $\lambda \approx 0$ *resilient* since its response is no greater than the perturbation itself.

We suggest, then, that learning by the HPA axis is, in fact, the process of tuning response to perturbation. The regression matrix $\mathbf{R}_0$ is tunable by both individual experience and by the effect of embedding cultural context. If individual experience or embedding culture change, then $\mathbf{R}_0$ will change, producing a different eigenpattern of physiological responses to imposed patterns of stress.

Suppose we require that λ itself be a function of the magnitude of excitation, i.e.

$$\lambda = f(|W|)$$

where $|W|$ is the vector length of W. We can, for example, require the amplification factor $1/(1-\lambda)$ to have a signal transduction form, an inverted-U-shaped curve, for example the signal-to-noise ratio of a stochastic resonance, so that

$$\frac{1}{1-\lambda} = \frac{1/|W|^2}{1 + b\exp[1/(2|W|)]}. \tag{14}$$

This places particular constraints on the behavior of the learned average $\mathbf{R}_0$, and gives precisely the typical HPA axis pattern of initial hypersensitization, followed by anergy or burnout with increasing average stress, a behavior that might well be characterized as pathological resilience, and may also have evolutionary significance.

Variants of this model permit imposition of cycles of different length, for example hormonal on top of circadian. Typically this is done by requiring a cyclic structure in matrix multiplication, with a new matrix $\mathbf{S}$ defined in terms of a sequential set of the $\mathbf{R}$, having period m, so that

$$\mathbf{S}_t \equiv \mathbf{R}_{t+m}\mathbf{R}_{t+m-1}...\mathbf{R}_t.$$

Essentially one does matrix algebra modulo m, in a sense.

In general, while the eigenvalues of such a cyclic system may remain the same, its eigenvectors depend on the choice of phase, i.e. where you start in the cycle. This is a complexity of no small note, and could represent a source of contrast in HPA axis behavior between men and women, beyond that driven by the ten-fold difference in testosterone levels. See Caswell (2001) for mathematical details.

Cultural topology of the HPA axis

The matrix of regression coefficients $\mathbf{R}_0$, and hence its eigenstructure, was taken as given and fixed in the development above. Here we will argue that it is not fixed, but can undergo something crudely analogous to the 'stream of consciousness' whose topological constraints were the subject of much attention in Wallace (2007). The essential point is to impose something like Bernard Baars' (1988) 'contexts' onto the development of an individual's HPA axis. These would typically be seen as relatively slowly varying boundary conditions for the more rapid HPA responses. Thus instead of writing a simple fixed $\mathbf{R}_0$ we suppose, in the usual sense, that the HPA axis tunes itself according to some dual information source, $\mathbf{X}$, so that there is, heuristically speaking, a signal path of the form

$$x = (\mathbf{R}_0^0, \mathbf{R}_0^1, ..., \mathbf{R}_0^n, ...). \tag{15}$$

which operates on the most rapid timescale of the system. Note, however, that we are averaging out perturbations on the timescale of the $\delta\mathbf{R}_t$ used above.

X is embedded within two nested contexts: individual experience and development, characterized by an intermediate rate information source Y, and both X and Y are embedded in a slowly-varying cultural matrix represented by an information source Z. These are all cross-linked by the tripartite mutual information measure of equation (7), which we, again, schematically write as $I(X|Y|Z)$. This too will partake of the homology between equations (3) and (4), giving, in the sense of Wallace (2005a), a punctuated linkage between X, and its two embedding contexts: individual and cultural development. Thus both individual experience, symbolized by the information source Y, and cultural context, symbolized by the information source Z, can write images of themselves, in a Rate Distortion manner, on the basic structure of the HPA axis, represented by the 'stream of consciousness' analog, the signal path x of equation (15).

Post traumatic stress disorder – PTSD – in this formulation results when brutal personal experience writes a distorted image of itself on the HPA axis through crosstalk between the dual information source X and the embedding context Y. Wallace (2007) provides detailed topological arguments why this should occur as a pathological resilience domain shift, in

which sets of paths x can be classified into an equivalence class topological groupoid. Different equivalence classes represent differing modes of HPA axis response to stress, with external forcing mechanisms needed to cause shifts between them.

An exactly similar argument leads to expectation that changes in cultural context, the information source Z, should write permanent images of themselves onto HPA axis behavior in a similarly punctuated, and quasi-equilibrium manner as a resilience domain shift.

Topological arguments regarding equivalence class groupoids of possible information source paths like x above are very powerful, and are pursued in Wallace (2007).

Obesity as a developmental disorder

Hirsch (2003) suggests, in effect, that obesity is a developmental disorder with roots in utero or early childhood. He and others have developed a set point or homeostatic theory of body weight, finding that it is the process which determines that 'set point' which needs examination, rather than the homeostasis itself, which is now fairly well understood. He concludes that the truly relevant question is not why obese people fail treatment, it is how their level of fat storage became elevated, a matter, he concludes, is probably rooted in infancy and childhood, when strong genetic determinants are shaping a still-plastic organism. The argument directly above provides a punctuated, topological resilience model of the phenomenon.

The question we raised earlier regarding the division of sets of possible responses of a cognitive HPA axis into the sets B_0 and B_1 has special significance in this matter.

Recall that the essential characteristic of cognition in our formalism involves a function h which maps a (convolutional) path $x = a_0, a_1, ..., a_n, ...$ onto a member of one of two disjoint sets, B_0 or B_1. Thus respectively, either (1) $h(x) \in B_0$, implying no action taken, or (2), $h(x) \in B_1$, and some particular response is chosen from a large repertoire of possible responses. We discussed briefly the problem of defining these two disjoint sets, and suggested that some 'higher order cognitive module' might be needed to identify what constituted B_0, the set of 'normal' states. This is because there is no low energy mode for information systems: virtually all states are more or less high energy states involving high rates of information transfer, and there is no way to identify a ground state using the physicist's favorite variational or other extremal arguments.

Suppose that higher order cognitive module, which we might well characterize as a Zero Mode Identification (ZMI), interacts with an embedding language of structured psychosocial stress and, instantiating a Rate Distortion image of that embedding stress, begins to include one or more members of the set B_1 into the set B_0. If that element of B_1 involves a particular mode of HPA axis cortisol/leptin cycle interaction, then the bodymass set point may be reset to a higher value: onset of obesity, and a new topology for paths of the HPA axis.

Work by Barker and colleagues suggests that those who develop coronary heart disease (CHD) grow differently from others, both in utero and during childhood. Slow growth during fetal life and infancy is followed by accelerated weight gain in childhood, setting a life history trajectory for CHD, type II diabetes, hypertension, and, of course, obesity. Barker (2002) concludes that slow fetal growth might also heighten the body's stress responses and increase vulnerability to poor living conditions later in life. Thus faulty ZMI function at critical periods in growth could lead to a permanently high body mass set point as a developmental disorder.

Empirical tests of our 'higher order' hypothesis, however, quickly lead into real-world regression models involving the interrelations of measurable biomarkers, behaviors, and so on, requiring formalism much like that used in the section above.

Therapeutic failure

Intervention against obesity, or other HPA axis pathologies, at the individual level is complicated by the manner in which pathological context can impose images of itself onto the therapeutic process. That is, pathological context, which is often responsible for HPA axis disorders of various kinds, can become convoluted with the intervention itself, resulting in therapeutic failure. It is possible to model this in more detail.

Recall that the essential characteristic of cognition in this formalism involves a mapping, $h(x)$, of a (convolutional) path $x = a_0, a_1, ..., a_n, ...$ onto a member of one of two disjoint sets, B_0 or B_1. Thus respectively, either (1) $h(x) \in B_0$, implying no action taken, or (2), $h(x) \in B_1$, and some particular response is chosen from a large repertoire of possible responses. We reiterate that there is, as discussed earlier, an evident problem in defining these two disjoint sets, suggesting that some higher order, i.e. executive, cognitive module is needed to determine what constitutes B_0, the set of normal actions and procedures, those not constituting explicit intervention. Again, this is because there is no low energy mode for information systems. That is, virtually all states are more or less high energy, high information content or transmission, states. Thus there is no natural way to identify a ground state using the physicist's favorite variational or other minimization arguments.

Suppose that a higher order executive cognitive module, which we have called Zero Mode Identification, interacts with an embedding, highly structured quasi-language of systemic perturbation – personal or cultural context. Instantiating a Rate Distortion image of that embedding stress, the ZMI begins to include one or more members of the set B_1 into the set B_0, or vice versa, when a circumstance requiring action is ignored. Again, recurrent hits on that aberrant state would be experienced as episodes of pathology, over or under reaction, for example resetting the bodymass setpoint.

Different eigenmodes Y_k of the regression model characterized by the zero mode matrix $\mathbf{R}_0$ can be taken to represent the shifting-of-gears between different languages defining the sets B_0 and B_1. That is, different eigenmodes of $\mathbf{R}_0$ would correspond to different required (and possibly mixed) characteristic systemic responses.

If there is a state (or set of states) Y_1 such that $\mathbf{R}_0 Y_1 = Y_1$, then the unitary kernel Y_1 corresponds to the condition 'no response required', i.e. the set B_0.

Suppose pathology becomes manifest, i.e.

$$\mathbf{R}_0 \rightarrow \mathbf{R}_0 + \delta\mathbf{R} \equiv \hat{\mathbf{R}}_0,$$

so that some chronic 'excited state' becomes the new unitary kernel, and

$$Y_1 \rightarrow \hat{Y}_1 \neq Y_1$$

$$\hat{\mathbf{R}}_0 \hat{Y}_1 = \hat{Y}_1.$$

Next, assume other, embedding, cognitive workspaces induce a sequence of therapeutic counterperturbations – deliberate therapeutic interventions – $\delta\mathbf{T}_k$ according to the pattern

$$[\hat{\mathbf{R}}_0 + \delta\mathbf{T}_1]\hat{Y}_1 = Y^1,$$

$$\hat{\mathbf{R}}_1 \equiv \hat{\mathbf{R}}_0 + \delta\mathbf{T}_1,$$

$$[\hat{\mathbf{R}}_1 + \delta\mathbf{T}_2]Y^1 = Y^2$$

...

(16)

so that, in some sense,

$$Y^j \rightarrow Y_1.$$

(17)

That is, the system is driven to some approximation of its original condition.

It may or may not be possible to have $\hat{\mathbf{R}}_0 \rightarrow \mathbf{R}_0$. That is, actual remediation may not be possible, in which case palliation or control is the therapeutic aim.

The essential point is that the pathological state represented by $\hat{\mathbf{R}}_0$ and the sequence of therapeutic interventions $\delta\mathbf{T}_k, k = 1, 2, ...$ are interactive and reflective, depending on the regression of the set of vectors Y^j to the desired state Y_1, much in the same spirit as Jerne's immunological idiotypic hall of mirrors.

The therapeutic problem revolves around minimizing the difference between Y^k and Y_1 over the course of treatment. That difference represents the inextricable convolution of treatment failure with adverse reactions to the course of treatment itself, and failure of compliance, often attributed through social construction by 'provider' to 'patient', i.e. failure of the therapeutic alliance.

It should be obvious that the treatment sequence $\delta\mathbf{T}_k$ itself is a cognitive path of interventions which has, in turn, a dual information source in the sense previously invoked.

Treatment may, then, interact in the usual Rate Distortion manner with the pathogenic patterns of structured perturbation - market pressures, failures, disasters, resource limitations, embedding channel capacity limits, structured 'red' noise, the burdens of history, and the like – which are, themselves, signals from an embedding information source. Thus treatment failure, adverse 'drug' reactions, and noncompliance will, of necessity, embody a distorted image of embedding structured perturbations which may indeed be responsible for the primary misfunction, in this case the resetting of the bodymass set point.

This coupling would most likely occur in a highly punctuated manner, depending in a quantitative way on the degree of interlinking of the three-fold system of affected individual, therapeutic interaction, and treatment mode, with that perturbation.

Clearly this is only one example of a much larger spectrum of possibilities. Empirical study would seem necessary at this point to prune down the search tree, as it were, making further analysis practical.

One disturbing implication of this analysis is the apparent difficulty of correcting a bodymass set point once it becomes overtly pathological.

In particular, contexts of social disintegration and resource limitation, individually and likely synergistically, severely limit the possibilities of therapeutic intervention to correct pathologies of bodymass set point, in the context of continuing patterns of social and cultural pathology.

Discussion and conclusions

Two powerful and intertwining phenomena of socioeconomic disintegration – deurbanization and deindustrialization – have combined to profoundly damage many US communities, dispersing historic accumulations of economic, political, and social capital. These losses have had manifold and persisting impacts on both institutions and individuals (e.g. Pappas, 1989; Ullmann, 1988; D. Wallace and R. Wallace, 1998; Wallace and Wallace, 2005). Elsewhere we examined the effect of these policy-driven phenomena on the hierarchical diffusion of AIDS in the US (Wallace et al., 1999). Wallace and Wallace (2005) extend that work to the association with obesity, in the context of a causal biological model.

By 1980, not a single African-American urban community established before or during World War II remained intact. Many Hispanic urban neighborhoods established after the war suffered similar fates. Virtually all lost considerable housing, population, and economic and social capital either to programs of 'urban renewal' in the 1950's or to policy-related contagious urban decay from the late 1960's through the late 1970's (e.g. Wallace and Wallace, 1998; M. Fullilove, 2004).

Wallace and Wallace (2005) examined the relation between obesity, obesity disorders, and both deindustrialization and deurbanization in the US, concluding that cultural change has become a pervasive threat signal, triggering chronic responses in the HPA axis across broad sectors of the population, with effects extending even into affluent and powerful subgroups.

The very forces which have caused the collapse of the US industrial base, i.e. the Cold War and its aftermath, have served as market selection pressures on East Asian nations. The overwhelming military power of the US empire, self destructive as it may be in the long term, serves as both example and threat to these ancient and traditional societies, placing them under increasingly acute pressure to adopt employment practices causing widespread resource insecurity.

That is, US economic power and imperial military power serve as engines to drive diffusion of pathogenic economic structure and practice into East Asian and other societies.

Such stress is critical to the etiology of visceral obesity, the metabolic syndrome, and their pathological sequelae, mediated by the HPA axis and several other physiological subsystems.

Both animal and human studies, however, have indicated that not all stressors are equal in their effect: particular forms of domination in animals and lack of control over work activities in humans are well-known to be especially effective in triggering metabolic syndrome and chronic inflammatory coronary lipid deposition.

Our analysis has been in terms of a cognitive HPA axis responding to a highly structured quasi-language of psychosocial stress, which we see as literally writing a distorted image of itself onto the behavior of the HPA axis in a manner analogous to learning plateaus in a neural network or to punctuated equilibrium in a simple evolutionary process. The first form of phase transition might be regarded as representing the progression of a normally staged disease. The other could describe certain pathologies characterized by stasis or only slight change, with staging a rare (and perhaps fatal) event.

Psychosocial stress is a cultural artifact, one of many such which interact intimately with human physiology. Indeed, much current theory in evolutionary anthropology focuses on the essential (but not unique) role culture plays in human biology (e.g. Durham, 1991; Avital and Jablonka, 2000).

If, as the evolutionary anthropologist Robert Boyd has suggested, "Culture is as much a part of human biology as the enamel on our teeth", what does the rising tide of obesity in the US suggest about American culture and the American system? About 22% of both African-American and Hispanic children are overweight, as compared to about 12% of non-Hispanic whites, and that prevalence is rising across the board (Strauss and Pollack, 2001). This suggests that, while the effects of an accelerating social pathology related to deindustrialization, deurbanization, and loss of democracy may be most severe for ethnic minorities in the US, the larger, embedding, cultural dysfunction has already spread upward along the social hierarchy, and is quickly entraining the majority population as well.

This is an explanation whose policy implications stand in stark contrast to current individual-oriented exhortations about 'taking responsibility for one's behavior' or 'eating less and getting more exercise' (e.g. Hill et al., 2003). The US 'liberal' approach is to mirror the explanations of the failed drug war: People overeat because there's a McDonald's on every street corner, companies market bigger portions, the food they sell is fatty, and so on. In contrast, we find that the fundamental cause of the obesity epidemic over the last twenty years in the US is not television, the automobile, or junk food. THese were already widespread beginning in the late 1950's, and into the 1980's without any corresponding obesity epidemic.

It is our thesis that the fundamental cause of the US obesity epidemic lies in a massive threat to the population caused by continuing deterioration of basic US social, economic, and related structures. This has occurred in the particular context of a ratcheting of dominance relations resulting from the concentration of effective power within a shrinking elite. This synergistic phenomenon generates a degree of community, family, and individual insecurity which is literally writing a life-threatening image of itself onto the bodies of all sectors of the American population. There is already a large and growing literature on other aspects of the sharpening inequalities within the US system (e.g. Wilkinson, 1996 and related material), and our conclusions fit within that body of work.

The basic and highly plieotropic nature of the biological relation between structured psychosocial stress and cognitive physiological systems ensures that magic bullet drug and other interventions will be largely circumvented: in the presence of a continuing socioeconomic and political ratchet, medical modalities are likely to provide little more than the equivalent of a choice of dying by hanging or by firing squad.

Effective intervention against obesity in the US would require creation of a broad, multi-level, ecological control program which must include redress of the power relations between groups, rebuilding of urban (and, increasingly, suburban) minority communities, and effective reindustrialization. This implies the necessity of a resurgence of the labor union, religious, civil rights, and community-based political activities which have been the traditional response to cultural patterns of injustice in the US. Responses to the diffusion of pathological norms from the US to other societies will, of course, be conditioned by their particular cultures and histories, and cannot be expected to be nonviolent.

The ecologist E.C. Pielou (1977) asserts that mathematical models of complicated ecosystem phenomena are most useful, not in answering questions, but rather in raising them for subsequent empirical study. In that spirit, it is our thesis that market-driven diffusion of pathological cultural norms from the powerful US empire and its allies, in particular structures and practices generating increasing levels of employment and other resource insecurity, has begun to distort traditional forms of economic and social relations in East Asian societies. This is generating a profound sense of threat expressing itself in developmental processes of chronic HPA axis dysfunction, producing widespread upward ratcheting of bodymass

set point: increasing global obesity.

References

Adams F., 2003, the informational turn in philosophy, *Minds and Machines*, 13:471-501.

Allan, J., 1998,Explanatory models of overweight among African American, Euro-American, and Mexican American women,*Western Journal of Nursing Research*, 20:45-66.

Atlan H. and I. Cohen, 1998, Immune information, self-organization and meaning, *International Immunology*, 10:711-717.

Avital E. and E. Jablonka, 2000, *Animal Traditions: behavioral inheritance in evolution*, Cambridge University Press, UK.

Banerji M., D. Kaufman, I. Shoreman, E. Smith, L. Rosenbaum, J. Coplan, and J. Kral, 2005, Insulin resistance metabolic syndrome in juvenile monkeys after early-life stress, **Obesity Reviews**, 6(Suppl. 1):127.

Barker D., 2002, Fetal programming of coronary heart disease, *Trends in Endocrinology and Metabolism*, 13:364-372.

Binney J., N. Dowrick, A. Fisher, and M. Newman, 1986, *The theory of critical phenomena*, Clarendon Press, Oxford, UK.

Bjorntorp P., 2001, Do stress reactions cause abdominal obesity and comorbidities?, *Obesity Reviews*, 2:73-86.

Brunner E., M. Marmot, K. Nanchahal, M. Shipley, S. Stansfeld, M. Juneja and K. Alberti, 1997, Social inequality in coronary risk: central obesity and the metabolic syndrome. Evidence from the Whitehall II study, *Diabetologia*, 40:1341-1349.

CDC, 2003, 1991-2001 Prevalence of Obesity Among US Adults, by Characteristics: Behavioral Risk Factor Surveillance System (1991-2001);Self-reported data.

http://www.cdc.gov/nccdphp/dnpa/obesity/trend/prev$_c$*har.htm*

Chrousos G., 2000, The role of stress and hypothalamic-pituitary-adrenal axis in the pathogenesis of the metabolic syndrome: neuro-endocrine and target tissue-related causes, *International Journal of Obesity and Related Metabolic Disorders*, Suppl. 2:S50-S55.

Coplan J., M. Andrews, L. Rosenblum, M. Owens, S. Friedman, J. Gorman, and C. Nemeroff, 1996, Persistent elevations of cerebrospinal fluid concentrations of corticotropin-releasing factor in adult nonhuman primates exposed to early-life stressors: implications for the patholopsychology of mood and anxiety disorders, *Proceedings of the National Academy of Sciences*, 103:1619-1623.

Coplan J., R. Trost, M. Owens, T. Cooper, J. Gorman, C. Nemeroff, and L. Rosenblum, 1998, Cerebrospinal fluid concentrations of somatostatin and biogeneic amines in grown primates reared by mothers exposed to manipulated foraging conditions, *Archives of General Psychiatry*, 55:473-477.

Coplan J., E. Smith, M. Altemus, B. Scharf, M. Owens, C. Nemeroff, J. Gorman, and L. Rosenblum, 2001, Variable foraging demand rearing: sustained elevations in cisternal cerebrospinal fluid corticotropin-releasing concentrations in adult primates, *Biological Psychiatry*, 50:200-204.

Coplan J., M. Altemus, S. Mathew, E. Smith, B. Sharf, P. Coplan, J. Kral, J. Gorman, M. Owens, C. Nemeroff, and L. Rosenblum, 2005, Synchronized maternal-infant elevations in primate CSF-CRF concentrations in response to variable foraging demand, *CNS Spectrum*,10:530-536.

Cover T. and J. Thomas, 1991, *Elements of Information Theory*, John Wiley Sons, New York.

Caswell H., 2001, *Matrix Population Models*, Aldine.

Dimitrov A. and J. Miller, 2001, Neural coding and decoding: communication channels and quantization, *Computation and Neural Systems*, 12:441-472.

Durham W., 1991, *Coevolution: Genes, Culture, and Human Diversity*, Stanford University Press, Palo Alto, CA.

Duryea P., 1978, Press release dated Friday 27 January, Office of the New York State Assembly Republican Leader, Albany, NY. Copy available from R. Wallace.

Eldredge N., 1985, *Time Frames: The Rethinking of Darwinian Evolution and the Theory of Punctuated Equilibria*, Simon and Schuster, New York.

Feynman R., 1996, *Feynman Lectures on Computation*, Addison-Wesley, Reading, MA.

Fullilove M., 2004, *Root Shock: Upheaval, Resettlement and Recovery in Urban America*, Random House, in press.

Ginsberg-Fellner F., L. Jagendorf, H. Carmel, and T. Harris, 1981, *American Journal of Nutrition*, Overweight and obesity in preschool children in New York City,34:2236-2241.

Gohil B., L. Rosenblum, J. Coplan, and J. Kral, 2001, Hypotalamic-pituitary-adrenal axis function and the metabolic syndrome X of obesity, *CNS Spectrum*, 6:581-586, 589.

Gould S., 2002, *The Structure of Evolutionary Theory*, Harvard University Press, Cambridge, MA.

Heine S. 2001, Self as a cultural product: an examination of East Asian and North American selves, *Journal of Personality*, 69:881-906.

Hill J., H. Wyatt, G. Reed, and J. Peters, 2003, Obesity and the environment: where do we go from here? *Science*, 266:853-858.

Hirsch J., 2002, Obesity: matter over mind?, *Cerebrum*, xx:7-18.

Holling C., 1973, Resilience and stability of ecological systems, *Annual Review of Ecological Systems and Systematics*, 4:1-25.

Isshiki, Y., K. Morimoto, M. Nakajima, S. Maruyama, and T. Takeshita, 2002, Increasing obesity among male workers in Japan: 1992-1997, *Environmental Health and Preventive Medicine*, 6:256-259.

Ives A., 1995, Measuring resilience in stochastic systems, *Ecological Monographs*, 65:217-233.

Masuda T., and R. Nisbett, 2006, Culture and change blindness, *Cognitive Science*, 30:381-399.

Markus H. and S. Kitayama, 1991, Culture and the self-implications for cognition, emotion, and motivation, *Psychological Review*, 98:224-253.

Matsushita Y., N. Yoshiike, F. Kaneda, K. Yoshita, and H. Takimoto, 2003, Trends in childhood obesity in Japan over

the last 25 years from the national nutrition survey, *Obesity Research*, 12:205-214.

Mokdad A., M. Serdula, W. Dietz, B. Bowman, J. Marks, J. Koplan, 1999, The spread of the obesity epidemic in the United States, 1991-1998 *JAMA*, 282:1519-1522.

Nisbett R., K. Peng, C. Incheol, and A. Norenzayan, 2001, Culture and systems of thought: holistic vs. analytic cognition, *Psychological Review*, 109:291-310.

Pappas G., 1989, *The Magic City*, Cornell University Press, Ithaca, NY.

Pielou, E., 1977, *Mathematical Ecology*, John Wiley and Sons, New York.

Rojdestvensky I. and M. Cottam, 2000, Mapping of statistical physics to information theory with applications to biological systems, *Journal of Theoretical Biology*, 202:43-54.

Rosenblum L., C. Forger, S. Noland, R. Trost, and J. Coplan, 2001, Response of adolescent Bonnet macaques to an acure fear stimulus as a function of early rearing conditions, *Developmental Psychobiology*, 39:40-45.

Spiegel K., R. Leproult, and E. Van Cauter, 1999, Impact of sleep debt on metabolic and endocrine function, *The Lancet*, 354:1435-1439.

Strauss R. and H. Pollack, 2001, Epidemic increase in childhood overweight, 1986-1998, *Journal of the American Medical Association*, 286:2845-2848.

Ullmann J., 1988, *The Anatomy of Industrial Decline*, Greenwood-Quorum, Westport, CT.

Wallace D., and R. Wallace, 1998, *A Plague on Your Houses*, Verso Publications, New York

Wallace D. and R. Wallace, 2000, Life and death in Upper Manhattan and the Bronx: toward an evolutionary perspective on catastrophic social change, *Environment and Planning A*, 32:1245-1266.

Wallace R. and R.G. Wallace, 1998, Information theory, scaling laws and the thermodynamics of evolution, *Journal of Theoretical Biology*, 192:545-559.

Wallace R., and R.G. Wallace, 1999, Organisms, organizations, and interactions: an information theory approach to biocultural evolution, *BioSystems*, 51:101-119.

Wallace R., D. Wallace, J. Ullmann, and H. Andrews, 1999, Deindustrialization, inner-city decay, and the diffusion of AIDS in the USA, *Environment and Planning A*, 31:113-139.

Wallace R., and D. Wallace, 2005, Structured psychosocial stress and the US obesity epidemic, *Journal of Biological Systems*, 13:363-384.

Wallace R., 2000, Language and coherent neural amplification in hierarchical systems: renormalization and the dual information source of a generalized stochastic resonance, *International Journal of Bifurcation and Chaos*, 10:493-502.

Wallace R., 2002a, Immune cognition and vaccine strategy: pathogenic challenge and ecological resilience, *Open Systems and Information Dynamics*, 9:51-83.

Wallace R., 2002b, Adaptation, punctuation and rate distortion: non-cognitive 'learning plateaus' in evolutionary process, *Acta Biotheoretica*, 50:101-116.

Wallace R., D. Wallace, and R.G. Wallace, 2003, Toward cultural oncology: the evolutionary information dynamics of cancer, *Open Systems and Information Dynamics*, 10:159-181.

Wallace R. and D. Wallace, 2003, Resilience reconsidered: case histories from disease ecology.
http://www.arxiv.org/abs/q-bio.NC/0310009

Wallace R., 2004, Comorbidity in psychiatric and chronic physical disease: autocognitive developmental disorders of structured psychosocial stress, *Acta Biotheoretica*, 52:71-93.

Wallace R., 2005a, *Consciousness: A mathematical treatment of the Global Neuronal Workspace Model*, Springer, New York.

Wallace R., 2005b, A global workspace perspective on mental disorders, *Theoretical Biology and Medical Modelling*, 2, 49.

Wallace R., 2006, Pitfalls in biological computing: canonical and idiosyncratic dysfunction of conscious machines, *Mind and Matter*, 4:91-113.

Wallace R., 2007, Culture and inattentional blindness: a global workspace perspective, *Journal of Theoretical Biology*, doi:10.1016/j/jtbi.2006.10.006.

Wellman N. and B. Friedberg, 2002, Causes and consequences of adult obesity: health, social and economic impacts in the United States, *Asia Pacific Journal of Clinical Nutrition*, Suppl.8:S705-S709.

Wilkinson R., 1996, *Unhealthy Societies: the afflictions of inequality*, Routledge, London and New York.

Wilson, K., 1971, Renormalization group and critical phenomena. I Renormalization group and the Kadanoff scaling picture, *Physical Review B*, 4:3174-3183.

Yoshiike N., F. Kaneda, and H. Takimoto, 2002, Epidemiology of obesity and public health strategies for its control in Japan, *Asia Pacific Journal of Clinical Nutrition*, 11(Suppl): S727-S731.

Wu, Y., 2007, Overweight and obesity in China, *British Medical Journal*, 333:362-363.