Preliminary Draft

'Fat people and bombs': HPA axis cognition, structured stress, and the US obesity epidemic

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Abstract

We examine the accelerating 'obesity epidemic' in the US from the perspective of recently developed theory relating a cognitive hypothalamic-pituitary-adrenal axis to an embedding 'language' of structured psychosocial stress. Using a Rate Distortion argument, the obesity epidemic is found to represent the literal writing of an image of a ratcheting pathological social hierarchy onto the bodies of American adults and children. This process, while stratified by the usual divisions of class and ethnicity, is nonetheless relentlessly engulfing even affluent majority populations. Our perspective places the common explanation that 'obesity occurs when people eat too much and get too little exercise' in the same category as the remark by US President Calvin

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Coolidge on the eve of the Great Depression that 'unemployment occurs when large numbers of people are out of work'. Both statements ignore profound structural determinants of great population suffering which must be addressed by collective actions of equally great reform.

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Introduction

Obesity is epidemic in the United States, has been so for more than two decades, and continues to increase. Current rates of overweight and obesity in the US 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).

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.

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 hypothalamic-pituitary-adrenal (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. psychosocial to HPA.

Here we will adapt recent developments regarding the punctuated information dynamics of evolutionary 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, 2002a, b).

'Stress', we aver, is not often random in human societies, but is rather a highly structured 'language', having both a grammar and a syntax, so that certain stressors are 'meaningful' in a particular 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 'languages' which we will find critical to an understanding of how psychosocial stress affects the HPA axis, and, ultimately, writes a distorted image of itself on the human body as visceral fat deposition.

This analysis presents a slightly different picture of the obesity epidemic, but one having profound implications for intervention policy.

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, memory and brain cognition 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. This is almost certainly an evolutionary adaptation of considerable significance.

Following the approach of Wallace (2000, 2002a), we make a very general model of that process.

Pattern recognition-and-response, as we characterize it, proceeds by convoluting (i.e. 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 model how the pattern recognition system is 'trained', and hence we adopt a weak model, regardless of learning paradigm, which can itself 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.

The model is as follows.

A pattern of sensory input is convoluted (compared) with internal 'ongoing' activity to create a path of convoluted signal $x = (a_0, a_1, ..., a_n, ...)$. This path is fed into a highly nonlinear 'decision oscillator' which generates an output $h(x)$ that is an element of one of two (presumably) disjoint sets B_0 and B_1. We take

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

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

Thus we permit a graded response, supposing that if

$$h(x) \in B_0$$

the pattern is not recognized, and 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 $h(a_0, ..., a_m) \in B_1$.

For each positive integer n let $N(n)$ be the number of paths of length n which begin with some particular a_0 having $h(a_0) \in B_0$ and lead to the condition $h(x) \in B_1$. We shall call such paths 'meaningful' and assume $N(n)$ to be considerably less than the number of all possible paths of length n – pattern recognition-and-response is comparatively rare. We further assume 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*.

We may thus define an 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.

Different 'languages' will, of course, be defined by different divisions of the total universe of possible responses into different pairs of sets B_0 and B_1, or 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 require 'higher order' cognitive decisions by other modules.

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

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

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

(2)

in the context of a 'transfer function' $f(\mathbf{y}^j \cdot \mathbf{w}^j)$ such that the probability of the neuron firing and having a discrete output $z^j = 1$ is $P(z^j = 1) = f(\mathbf{y}^j \cdot \mathbf{w}^j)$.

Thus the probability that the neuron does not fire at time j is just $1 - P$. In the usual terminology the m values y_i^j constitute the 'sensory activity' and the m weights w_i^j the 'ongoing activity' at time j, with $a_j = \mathbf{y}^j \cdot \mathbf{w}^j$ and the path $x \equiv a_0, a_1, ..., a_n, ...$ A little more work leads to a standard neural network model in which the network is trained by appropriately varying $\mathbf{w}$ through least squares or other error minimization feedback. This can be shown to replicate rate distortion arguments, as we can use the error definition to define a distortion function which measures the difference between the training pattern y and the network output $\hat{y}$ as a function, for example, of the inverse number of training cycles, K. As we will discuss in another context, 'learning plateau' behavior emerges naturally as a phase transition in the parameter K in the mutual information $I(Y, \hat{Y})$.

Thus we will eventually 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 (2002b), 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 memoryless 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 memoryless ergodic' (APME). 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.

Interacting information sources

We 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, one of the fundamental limit theorems of 20th Century mathematics, 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

$$I(X,Y) = H(X) - H(X|Y) = H(X) + H(Y) - H(X,Y)$$

(3)

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

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

(4)

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 immune system's reset cognition and the system of external signals and stressors, and set $K = 1/P$, following the development of those earlier studies. Then we have

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

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

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

(5)

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 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; Wallace et al., 2003).

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

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

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 analyses, 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)

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

(6)

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 $I(Y_1, Y_2|Y_3)$. Note that our results are almost exactly parallel to the Eldredge/Gould model of evolutionary punctuated equilibrium (Eldredge, 1985; Gould, 2002).

The simplest HPA axis model

Stress, as we envision it, 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. We view the stress experienced by an individual as APME 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, which we will characterize as 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

$$X_{t+1} \approx < \mathbf{R} > X_t + b_0, \tag{7}$$

where $< \mathbf{R} >$ is the matrix of regression coefficients and b_0 a vector of constant terms.

We then suppose that, in the presence of a perturbation by structured stress

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

$$\equiv < \mathbf{R} > X_t + \epsilon_{t+1},$$

(8)

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

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

$$\mathbf{Q} X_{t+1} = (\mathbf{Q} < \mathbf{R} > \mathbf{Q}^{-1}) \mathbf{Q} X_t + \mathbf{Q} \epsilon_{t+1},$$

(9)

or equivalently as

$$Y_{t+1} = < \mathbf{J} > Y_t + W_{t+1},$$

(10)

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

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 (APM) ergodic, we can either time or ensemble average both sides of equation (10), so that the one-period offset is absorbed in the averaging, giving an 'equilibrium' relation

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

or

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

(11)

where $\mathbf{I}$ is the $n \times n$ identity matrix.

Now we reverse the argument: Suppose that Y_k is chosen to be some fixed eigenvector of $< \mathbf{R} >$. 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

$$< Y_k > = \frac{1}{1 - < \lambda_k >} < W_k >$$

(12)

where $< \lambda_k >$ 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 'tuning' the eigenmodes of $< \mathbf{R} >$, the HPA axis can be trained to galvanized response in the presence of particular forms of perturbation.

This is because, if $< \mathbf{R} >$ 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. This is why we have written $< \mathbf{R} >$ instead of simply $\mathbf{R}$: The regression matrix is a tunable set of variables.

Suppose we require that $< \lambda >$ 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

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

(13)

This places particular constraints on the behavior of the 'learned average' $< \mathbf{R} >$, 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.

Discussion and conclusions

Current theory clearly identifies 'stress' as critical to the etiology of visceral obesity, the metabolic syndrome, and their pathological sequelae, mediated by the HPA axis and several other physiological subsystems which we have not addressed here.

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 '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, for humans, a cultural artifact, one of many such which interact intimately with human physiology. Indeed, much current theory in evolutionary anthropology focuses on the fundamental (but not unique) role culture plays in human biology (e.g. Durham, 1991; Avital and Joblonka, 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 pathogenic social hierarchy 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'. 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, and so on. In contrast, we find that the fundamental cause of the obesity epidemic over the last twenty years is not television, the automobile or junk food. All were prominent from the late 1950's into the 1980's without an obesity epidemic. The fundamental cause of the US obesity epidemic is a twenty-year ratcheting of dominance relations within the US socioeconomic and political system which is literally writing a life-threatening image of itself onto the bodies of American adults and children. 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 fundamental and plieotropic nature of the biological relation between structured psychosocial stress and cognitive physiological systems ensures that 'magic bullet' interventions will be largely circumvented: in the presence of a continuing 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 will involve creation of a broad, multi-level, ecological control program. It is evident that any such program must include address of the power relations between groups. This implies the necessity of a resurgence of the labor union, religious, and community-based political activities which have been traditionally directed against cultural patterns of injustice in the past, activities which, ultimately, liberate all.

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