Version 3

A Global Workspace perspective on mental disorders

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November 16, 2005

Abstract

Recent developments in Global Workspace theory suggest that human consciousness can suffer interpenetrating dysfunctions of mutual and reciprocal interaction with embedding environments which will have early onset and often insidious staged developmental progression, possibly according to a cancer model.

A simple rate distortion argument implies that, if an external information source is pathogenic, then sufficient exposure to it is sure to write a sufficiently accurate image of it on mind and body in a punctuated manner so as to initiate or promote similarly progressively punctuated developmental disorder.

There can, thus, be no simple, reductionist brain chemical 'bug in the program' whose 'fix' can fully correct the problem. On the contrary, the growth of an individual over the life course, and the inevitable contact with a toxic physical, social, or cultural environment, can be expected to initiate developmental problems which will become more intrusive over time, most obviously following some damage accumulation model, but likely according to far more subtle, highly punctuated, schemes analogous to tumorigenesis.

The key intervention, at the population level, is clearly to limit such exposures, a question of proper environmental sanitation, in a large sense, a matter of social justice which has long been understood to be determined almost entirely by the interactions of cultural trajectory, group power relations, and economic structure, with public policy. Intervention at the individual level appears limited to triggering or extending periods of remission, as is the case with most cancers.

Key words: cancer, cognition, consciousness, culture, economic structure, information theory, mental disorder, power relations, public policy

Introduction

Mental disorders in humans are not well understood. Indeed, such classifications as the *Diagnostic and Statistical Manual of Mental Disorders - fourth edition*, (DSM-IV, 1994), the standard descriptive nosology in the US, have been characterized as 'prescientific' by Gilbert (2001) and others. Arguments from genetic determinism fail, in part because of an apparently draconian population bottleneck which, early in our species' history, resulted in an overall genetic diversity less than that observed within and between contemporary chimpanzee subgroups. Arguments from psychosocial stress fare better, but are affected by the apparently complex and contingent developmental paths determining the onset of schizophrenia – one of the most prevalent serious mental disorders – dementias, psychoses, and so forth, some of which may be triggered in utero by exposure to infection, low birthweight, or other stressors. Gilbert suggests an evolutionary perspective, in which evolved mechanisms like the 'flight-or-fight' response are inappropriately excited or suppressed, resulting in such conditions as anxiety or post traumatic stress disorders. Our own work suggests that many sleep disorders may also be broadly developmental (Wallace, 2005b).

Serious mental disorders in humans are often comorbid among themselves – depression and anxiety, compulsive behaviors, psychotic ideation, etc. – and with serious chronic physical conditions such as coronary heart disease, atherosclerosis, diabetes, hypertension, dyslipidemia, and so on. These too are increasingly recognized as developmental in nature (see Wallace, 2004, 2005a for references), and are frequently compounded by behavioral problems like violence or substance use and abuse. Indeed, smoking, alcohol and drug addiction, compulsive eating, and the like, are often done as self-medication for the impacts of psychosocial and other stressors, constituting socially-induced 'risk behaviors' which synergistically accelerate a broad spectrum of mental and physical problems.

A central failure of current theory surrounding mental disorders has been the exclusion, for powerful ideological reasons, of virtually all scientific discussion of consciousness in humans, a circumstance more completely discussed by Baars (1988). Mental disorders are, quintessentially, disorders of consciousness, and the collapse of the scientific exploration of consciousness in the early part of the 20th Century clearly accounts for much of the current unsatisfactory state of the epidemiology of mental disorders.

This paper will review some recent advances in the theory of consciousness in humans, and apply the results toward a

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better understanding of mental disorders, using an information theory formalism which draws a parallel between punctuated evolutionary and cognitive/learning forms of information transmission (Wallace, 2002).

The comparison of punctuated evolutionary adaptation with cognitive learning plateaus is counterintuitive: evolution is not a cognitive process. Cognition involves an active selection of one out of a complex repertory of possible responses to a sensory input, based on comparison with a learned representation of the outer world (e.g. Cohen, 2000; Atlan and Cohen, 1998). Although genes, or in the case of human biology, a composite of genes-and-culture (e.g. Richerson and Boyd, 2004), do indeed constitute a kind of 'memory' of past interaction with the world, response to selection pressure is not through direct comparison with that 'memory', but rather through the reproductive success of a random variation constrained by the path of evolutionary history. This is not cognition, and there can be no 'intelligent purpose' to adaptive or evolutionary process. Nonetheless, selection pressures represent systematic patterns of interaction with an embedding and highly structured ecosystem in which each species is itself manifest. We will, below, use this perspective to infer a rough analog between developmental onset and progression of a broad class of mental disorders and the onset and progression of a certain class of cancers.

Recent resumption of scientific research on consciousness in humans follows from Baars' (1988) pioneering restatement of the problem in terms of a global workspace theory (Baars, 1988; Baars and Franklin, 2003), to which the reader should refer for more details.

The central ideas are as follows (Baars and Franklin, 2003):

(1) The brain can be viewed as a collection of distributed specialized networks (processors).

(2) Consciousness is associated with a global workspace in the brain – a fleeting memory capacity whose focal contents are widely distributed (broadcast) to many unconscious specialized networks.

(3) Conversely, a global workspace can also serve to integrate many competing and cooperating input networks.

(4) Some unconscious networks, called contexts, shape conscious contents, for example unconscious parietal maps modulate visual feature cells that underlie the perception of color in the ventral stream.

(5) Such contexts work together jointly to constrain conscious events.

(6) Motives and emotions can be viewed as goal contexts.

(7) Executive functions work as hierarchies of goal contexts.

Although this basic approach has been systematically elaborated upon for nearly twenty years by a number of quite eminent researchers, consciousness studies has only recently, in the context of a deluge of data from brain imaging experiments, come to the point of actually digesting the perspective and moving on.

Baars' model has received increasing experimental verification (Dehaene and Naccache, 2001; Massimini et al, 2005). Since it particularly attempts to properly represent the matter of embedding and interpenetrating contexts, it provides a basis for understanding mental disorders in humans, and for drawing a parallel with the initiation and progression of cancer as a disorder of information.

My own recent work provides a rigorous mathematical formulation of the GW blackboard model, in terms of an iterated, second-order, contextually-embedded, hierarchical General Cognitive Model (GCM) crudely analogous to hierarchical regression. It is, however, based on the Shannon-McMillan rather than on the Central Limit Theorem, and is strongly supplemented by methodologies from topological manifold theory and differential geometry (Wallace, 2005a, b, c). Recent results (Wallace, 2005c) suggest that, in fact, it should be possible to make a rigorous theory of 'all possible' GW blackboard models, much in the same sense that the Church lambda calculus describes 'conventional' computers and the Nix/Vose Markov chain treatment describes many possible genetic algorithms (Nix and Vose, 1992; Vose, 1999).

We begin with a simplified analysis focusing on modular networks of interacting cognitive substructures, and particularly study the importance of their embedding in progressively larger systems. More complicated examples, involving renormalization treatment of phase transitions affecting information sources, iterated to second order, can be found in Wallace (2005a).

The simplest modular network Global Workspace model

Cognition as 'language' Cognition is not consciousness. Indeed, most mental, and many physiological, functions, while cognitive in a particular formal sense, hardly ever become entrained into the Global Workspace of consciousness. For example, one seldom is able to consciously regulate immune function, blood pressure, or the details of binocular tracking and bipedal motion, except to decide 'what shall I look at', 'where shall I walk'. Nonetheless, many cognitive processes, conscious or unconscious, appear intimately related to 'language', broadly speaking. The construction is surprisingly straightforward (Wallace, 2000, 2005a).

Atlan and Cohen (1998) and Cohen (2000) argue, in the context of immune cognition, that the essence of cognitive function involves comparison of a perceived signal with an internal, learned picture of the world, and then, upon that comparison, choice of one response from a much larger repertoire of possible responses.

Cognitive pattern recognition-and-response, from this view, proceeds by functionally combining an incoming external sensory signal with an internal ongoing activity – incorporating the learned picture of the world – and triggering an appropriate action based on a decision that the pattern of sensory activity requires a response.

More formally, a pattern of sensory input is mixed in an unspecified but systematic manner with a pattern of internal ongoing activity to create a path of combined signals $x = (a_0, a_1, ..., a_n, ...)$. Each a_k thus represents some algorithmic composition of internal and external signals.

This path is fed into a highly nonlinear, but otherwise similarly unspecified, 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. Let

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

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

Assume 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.

The principal objects of interest are 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 $h(a_0, ..., a_m) \in B_1$. Wallace (2005a) examines the possibility of more complicated schemes as well.

For each positive integer n, let $N(n)$ be the number of high probability 'grammatical' and 'syntactical' 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', assuming, not unreasonably, that $N(n)$ will be considerably less than the number of all possible paths of length n leading from a_0 to the condition $h(x) \in B_1$.

While combining algorithm, the form of the nonlinear oscillator, and the details of grammar and syntax, are all unspecified in this model, the critical assumption which permits inference on necessary conditions is that the finite limit

$$H \equiv \lim_{n \to \infty} \frac{\log[N(n)]}{n} \tag{1}$$

both exists and is independent of the path x.

We call such a pattern recognition-and-response cognitive process *ergodic*. Not all cognitive processes are likely to be ergodic, implying that H, if it indeed exists at all, is path dependent, although extension to 'nearly' ergodic processes is possible (Wallace, 2005a).

Invoking the spirit of the Shannon-McMillan Theorem, it is possible to define an adiabatically, piecewise 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 satisfy the classic relations

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

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

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

This information source is defined as *dual* to the underlying ergodic cognitive process (Wallace, 2005a).

The Shannon uncertainties $H(...)$ are cross-sectional law-of-large-numbers sums of the form $-\sum_k P_k \log[P_k]$, where the P_k constitute a probability distribution. See Khinchine (1957), Ash (1990), or Cover and Thomas (1991) for the standard details.

The giant component A formal equivalence class algebra (and hence a groupoid, sensu Weinstein, 1996) can be constructed by choosing different origin points a_0 and defining equivalence by the existence of a high probability meaningful path connecting two points. Disjoint partition by equivalence class, analogous to orbit equivalence classes for dynamical systems, defines the vertices of the proposed network of cognitive dual languages. Each vertex then represents a different information source dual to a cognitive process.

We now suppose that linkages can fleetingly occur between the ordinarily disjoint cognitive modules defined by this algebra. In the spirit of Wallace (2005a), this is represented by establishment of a non-zero mutual information measure between them: cross-talk.

Wallace (2005a) describes this structure in terms of fixed magnitude disjunctive strong ties which give the equivalence class partitioning of modules, and nondisjunctive weak ties which link modules across the partition, and parametizes the overall structure by the average strength of the weak ties, to use Granovetter's (1973) term. By contrast the approach here, initially, is to simply look at the average number of fixed-strength nondisjunctive links in a random topology. These are obviously the two analytically tractable limits of a much more complicated regime which we believe ultimately includes 'all possible' global workspace models.

Since we know nothing about how the cross-talk connections can occur, we will – for purposes of illustration only – assume they are random and construct a random graph in the classic Erdos/Renyi manner. Suppose there are M disjoint cognitive modules – M elements of the equivalence class algebra of languages dual to some cognitive process – which we now take to be the vertices of a possible graph.

As Corless et al. (1996) discuss, when a graph with M vertices has $m = (1/2)aM$ edges chosen at random, for $a > 1$ it almost surely has a giant connected component having approximately gM vertices, with

$$g(a) = 1 + W(-a \exp(-a))/a,$$

(2)

where W is the Lambert-W function defined implicitly by the relation

$$W(x)\exp(W(x)) = x.$$

(3)

Figure 1 shows $g(a)$, displaying what is clearly a sharp phase transition at $a = 1$.

Such a phase transition initiates a new, collective, shifting, cognitive phenomenon: the Global Workspace, a tunable blackboard defined by a set of cross-talk mutual information measures between interacting unconscious cognitive submodules. The source uncertainty, H, of the language dual to the collective cognitive process, which defines the richness of the cognitive language of the workspace, will grow as some function of g, as more and more unconscious processes are incorporated into it. Wallace (2005a) examines what, in effect, are the functional forms $H \propto \exp(\alpha g), \alpha \ln[1/(1-g)]$, and $(1/(1-g))^{\delta}$, letting $R = 1/1-g$ define a 'characteristic length' in the renormalization scheme. While these all have explicit solutions for the renormalization calculation (mostly in terms of the Lambert-W function), other, less tractable, expressions are certainly plausible, for example $H \propto g^{\gamma}, \gamma > 0$, γ real.

Given a particular $H(g)$, the quite different approach of Wallace (2005a) involves adjusting universality class parameters of the phase transition, a matter requiring much mathematical development.

By contrast, in this new class of models, the degree of clustering of the graph of cognitive modules might, itself, be tunable, producing a variable threshold for consciousness: a topological shift, which should be observable from brain-imaging studies. Second order iteration would lead to an analog of the hierarchical cognitive model of Wallace (2005a).

Wallace (2005a) focuses on changing the average strength of weak ties between unconscious submodules rather than the average number of fixed-strength weak ties as is done here, and tunes the universality class exponents of the phase transition, which may also imply subtle shifts in underlying topology.

Following Albert and Barabasi (2002, Section V), we note that real networks differ from random graphs in that their degree distribution, the probability of k linkages between vertices, often follows a power law $P(k) \approx k^{-\gamma}$ rather than the Poisson distribution of random networks,

$P(k) = a^k \exp(-a)/k!, k \geq 0$. Since power law networks do not have any characteristic scale, they consequently termed scale-free.

It is possible to extend the Erdos/Renyi threshold results to such 'semirandom' graphs. For example, Luczak (1992) has shown that almost all random graphs with a fixed degree smaller than 2 have a unique giant cluster. Molloy and Reed (1995, 1998) proved that, for a random graph with degree distribution $P(k)$, an infinite cluster emerges almost surely when

$$Q \equiv \sum_{k \geq 1} k(k-2)P(k) > 0.$$

(4)

Following Volz, (2004), cluster tuning of random networks leads to a counterintuitive result. Define the clustering coefficient C as the proportion of triads in a network out of the total number of potential triads, i.e.

$$C = \frac{3N_{\Delta}}{N_3},$$

(5)

where N_{Δ} is the number of triads in the network and N_3 is the number of connected triples of nodes, noting that in every triad there are three connected nodes. Taking the approach of Molloy and Reed (1995), Volz shows quite directly that, for a random network with parameter a, at cluster value C, there is a critical value given by

$$a_C = \frac{1}{1 - C - C^2}.$$

(6)

If $C = 0$, i.e. no clustering, then the giant component forms when $a = 1$. Increasing C *raises* the average number of edges which must be present for a giant component to form. For $C \geq \sqrt{5}/2 - 1/2$, which is precisely the Golden Section, where the denominator in this expression vanishes, no giant component can form, regardless of a. Not all network topologies, then, can actually support a giant component, and hence, in this model, consciousness. This is of some importance, having obvious and deep implications ranging from the evolutionary history of consciousness to the nature of sleep.

A more complete exploration of the giant component can be found, e.g. in Newman et al. (2001), especially the discussion leading to their figure 4. In general, 'tuning' of the GC will generate a family of curves similar to figure 1, but with those having threshold to the right of that in the plot 'topping out'

at limits progressively less than 1: higher thresholds seem usually to imply smaller giant components. In sum, the giant component is itself highly tunable, replicating, in this model, the fundamental stream of consciousness.

Note that we do not, in this paper, address the essential matter of how the system of interacting cognitive modules behaves away from critical points, particularly in the presence of 'external gradients'. Answering this question requires the imposition of generalized Onsager relations, which introduce complications of topological 'rate distortion manifolds', metric structures, and the like (e.g. Wallace, 2005a, b).

Mutual and reciprocal interaction: evading the mereological fallacy Just as a higher order information source, associated with the GC of a random or semirandom graph, can be constructed out of the interlinking of unconscious cognitive modules by mutual information, so too external information sources, for example in humans the cognitive immune and other physiological systems, and embedding sociocultural structures, can be represented as slower-acting information sources whose influence on the GC can be felt in a collective mutual information measure. The measure will, through the Joint Asymptotic Equipartition Theorem which generalizes the Shannon-McMillan Theorem, be the splitting criterion for high and low probability joint paths across the entire system.

The tool for this is network information theory (Cover and Thomas, 1991, p. 387). Given three interacting information sources, Y_1, Y_2, Z, the splitting criterion, taking Z as the 'external context', is given by

$$I(Y_1, Y_2|Z) = H(Z) + H(Y_1|Z) - H(Y_1, Y_2, Z), \tag{7}$$

where $H(..|..)$ and $H(.., .., ..)$ represent conditional and joint uncertainties (Khinchine, 1957; Ash, 1990; Cover and Thomas, 1991).

This generalizes to

$$I(Y_1, ...Y_n|Z) = H(Z) + \sum_{j=1}^{n} H(Y_j|Z) - H(Y_1, ..., Y_n, Z). \tag{8}$$

If we assume the Global Workspace/GC/blackboard to involve a very rapidly shifting, and indeed highly tunable, dual information source X, embedding contextual cognitive modules like the immune system will have a set of significantly slower-responding sources $Y_j, j = 1..m$, and external social, cultural and other 'environmental' processes will be characterized by even more slowly-acting sources $Z_k, k = 1..n$. Mathematical induction on equation (8) gives a complicated expression for a mutual information splitting criterion which we write as

$$I(X|Y_1, .., Y_m|Z_1, .., Z_n). \tag{9}$$

This encompasses a fully interpenetrating 'biopsychosociocultural' structure for individual consciousness, one in which Baars' contexts act as important, but flexible, boundary conditions, defining the underlying topology available to the far more rapidly shifting global workspace (Wallace, 2005a, b).

This result does not commit the mereological fallacy which Bennett and Hacker (2003) impute to excessively neurocentric perspectives on consciousness in humans, that is, the mistake of imputing to a part of a system the characteristics which require functional entirety.

Punctuation phenomena for information systems

As quite a number of researchers have noted, in one way or another, – see Wallace, (2005a) for discussion – equation (1),

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

is homologous to the thermodynamic limit in the definition of the free energy density of a physical system. This has the form

$$F(K) = \lim_{V\to\infty} \frac{\log[Z(K)]}{V}, \tag{10}$$

where F is the free energy density, K the inverse temperature, V the system volume, and $Z(K)$ is the partition function defined by the system hamiltonian.

Wallace (2005a) shows at some length how this homology permits the natural transfer of renormalization methods from statistical mechanics to information theory. In the spirit of the Large Deviations Program of applied probability theory, this produces phase transitions and analogs to evolutionary punctuation in systems characterized by piecewise, adiabatically stationary, ergodic information sources. These 'biological' phase changes appear to be ubiquitous in natural systems and can be expected to dominate machine behaviors as well, particularly those which seek to emulate biological paradigms. Wallace (2002) uses these arguments to explore the differences

and similarities between evolutionary punctuation in genetic and learning plateaus in neural systems. Punctuated phenomena will emerge as important in the discussions below of subtle information system malfunctions, be those systems biological, social, or mechanical.

The dysfunctions of consciousness and intelligence: a cancer model

Somewhat surprisingly, equation (9), informed by the homology with equation (10), permits general discussion of the failure modes of global workspace systems of all kinds, in particular of their second order iteration which appears to be the analog to consciousness in humans and other higher animals.

The foundation for this lies in the Rate Distortion Theorem. Under the conditions of that theorem, equation (9) is the splitting criterion defining the maximum rate at which an external information source can write an image of itself having a given maximum of distortion, according to some defined measure (Cover and Thomas, 1991; Dembo and Zeitouni, 1998). Inverting the argument, equation (9) suggests that an external information source can, if given enough time, write an image of itself upon consciousness. If that external source is pathogenic, then, given sufficient exposure, some measure of consciousness dysfunction becomes inevitable.

A more general discussion of comorbid mind/body disorders in humans emerges quite naturally (Wallace, 2004). The picture, in humans, then, is of a multifactorial and broadly interpenetrating mind/body/sociocultural dysfunction, often having early onset and insidious, irregular, developmental progression. These disorders are, broadly speaking, distorted images of pathogenic external environments which are literally written upon the developing embryo, on the growing child, and on the maturing adult (Wallace, 2005a, Ch. 6). Equation (9) suggests that, in similar form, these images will be inevitably written upon consciousness as well.

Further consideration implies critical parallels with the initiation and progression of cancer in multicellular organisms, a quintessential disorder of information transmission (Wallace et al., 2003).

The analogy requires some development.

Nunney (1999) suggests that in larger animals, whose lifespans are proportional to about the 4/10 power of their cell count, prevention of cancer in rapidly proliferating tissues becomes more difficult in proportion to their size. Cancer control requires the development of additional mechanisms and systems with increasing cell count to address tumorigenesis as body size increases – a synergistic effect of cell number and organism longevity.

As Nunney puts it,

> "This pattern may represent a real barrier to the evolution of large, long-lived animals and predicts that those that do evolve... have recruited additional controls [over those of smaller animals] to prevent cancer."

In particular different tissues may have evolved markedly different tumor control strategies. All of these, however, are likely to be energetically expensive, permeated with different complex signaling strategies, and subject to a multiplicity of reactions to signals.

Work by Thaler (1999) and Tellilion et al. (2001) suggests that the mutagenic effects associated with a cell sensing its environment and history could be as exquisitely regulated as transcription. Invocation of the Rate Distortion or Joint Asymptotic Equipartition Theorems in address of the mutator necessarily means that mutational variation comes to significantly reflect the grammar, syntax, and higher order structures of embedding environmental processes. This involves far more than a simple 'colored noise' – stochastic excursions about a deterministic 'spine' – and most certainly implies the need for exquisite regulation. Thus there are deep information theory arguments in favor of Thaler's speculation.

Thaler further argues that the immune system provides an example of a biological system which ignores conceptual boundaries between development and evolution.

Thaler specifically examines the meaning of the mutator for the biology of cancer, which, like the immune system it defies, is seen as involving both development and evolution.

Thus Thaler, in essence, looks at the effect of structured external stress on tumorigenesis and describes the 'local evolution' of cancer within a tissue in terms of a 'punctuated interpenetration' between a tumorigenic mutator mechanism and an embedding cognitive process of mutation control, including but transcending immune function.

The mutation control process constitutes the Darwinian selection pressure determining the fate of the (path dependent) output of a mutator mechanism. Externally-imposed and appropriately structured environmental signals then jointly increases mutation rate while decreasing mutation control effectiveness through an additional level of punctuated interpenetration. This is envisioned as a single, interlinked biological process.

Various authors have argued for 'non-reductionist' approaches to tumorigenesis (e.g. Baverstock (2000) and Waliszewski et al. (1998)), including psychosocial stressors as inherent to the process (Forlenza and Baum, 2000). What is clear is that, once a mutation has occurred, multiple systems must fail for tumorigenesis to proceed. It is well known that processes of DNA repair (e.g. Snow, 1997), programmed cell death – apoptosis – (e.g. Evans and Littlewood, 1998), and immune surveillance (e.g. Herberman, 1995) all act to redress cell mutation. The immune system is increasingly viewed as cognitive, and is known to be equipped with an array of possible remediations (Atlan and Chohen, 1998; Cohen, 2000). It is, then, possible to infer a larger, jointly-acting 'mutation control' process incorporating these and other cellular, systemic, and, in higher animals, social mechanisms. This clearly must involve comparison of developing cells with some internal model of what constitutes a 'normal' pattern, followed by a choice of response: none, repair, programmed cell death, or full-blown immune attack. The comparison with an internal picture of the world, with a subsequent choice from a response repertoire, is, as Atlan and Cohen (1998) point out, the essence of cognition.

One is led to propose, in the sense of equation (9), that a mutual information may be defined characterizing the interaction of a structured system of external selection pressures with the 'language' of cellular cognition effecting mutation control. Under the Joint Asymptotic Equipartition or Rate Distortion Theorems, that mutual information constitutes a splitting criterion for pairwise linked paths which may itself be punctuated and subject to sudden phase transitions.

Pathologically structured externally environmental signals can become jointly and synergistically linked both with cell mutation and with the cognitive process which attempts to redress cell mutation, enhancing the former, degrading the latter, and significantly raising the probability of successful tumorigenesis.

Raised rates of cellular mutation which quite literally reflect environmental pressure through selection's distorted mirror do not fit a cognitive paradigm: The adaptive mutator may propose, but selection disposes. However, the effect of structured environmental stress on both the mutator and on mutation control, which itself constitutes the selection pressure facing a clone of mutated cells, connects the mechanisms. Subsequent multiple evolutionary 'learning plateaus' (Wallace, 2002) representing the punctuated interpenetration between mutation control and clones of mutated cells constitute the stages of disease. Such stages arise in the context of an embedding system of environmental signals which, to use a Rate Distortion argument, literally writes an image of itself on all aspects of the disease.

These speculations are consistent with, but suggest extension of, a growing body of research. Kiecolt-Glaser et al. (2002), for example, discuss how chronic inflammation related to chronic stress has been linked with a spectrum of conditions associated with aging, including cardiovascular disease, osteoporosis, arthritis, type II diabetes, certain cancers, and other conditions. Dalgleish (1999, 2002) and others (O'Byrne and Dalgleish, 2001; Ridley, 1996) have argued at length that chronic immune activation and inflammation are closely related to the etiology of cancer and other diseases. As Balkwill and Mantovani (2001) put the matter, "If genetic damage is the 'match that lights the fire' of cancer, some types of inflammation may provide 'fuel that feeds the flames' ".

Dalgleish (1999) has suggested application of non-linear mathematics to examine the role of immune response in cancer etiology, viewing different phenotypic modes of the immune system – the Th1/Th2 dichotomy – as 'attractors' for chaotic processes related to tumorigenesis, and suggests therapeutic intervention to shift from Th2 to Th1. Such a shift in phenotype might well be viewed as a phase transition.

This analysis implies a complicated and subtle biology for cancer in higher animals, one in which external environmental 'messages' become convoluted with both pathogenic clone mutation and with an opposing, and possibly organ-specific, variety of tumor control strategies. In the face of such a biology, anti-inflammants (Coussens and Werb, 2002) and other 'magic bullet' interventions appear inadequate, a circumstance having implications for control of the aging of conscious systems which we infer from these examples.

Although chronic inflammation, related certainly to structured environmental stress, is likely to be a contributor to the enhancement of pathological mutation and the degradation of corrective response, it is unlikely to be the only such trigger. The constant cross-talk between central nervous, hormonal, immune, and tumor control systems in higher animals guarantees that the 'message' of the external environment will write itself upon the full realm of individual physiology in a highly plieotropic, punctuated, manner, with multifactorial impact on both cell clone mutation and tumor control.

Discussion and conclusions

These examples, particularly the cancer model, suggest that consciousness in higher animals, the quintessence of information processing, is necessarily accompanied by elaborate regulatory and corrective processes, both internal and external. Only a few are well known: Sleep enables the consolidation and fixation in memory and semiautomatic mechanism of what has been consciously learned, and proper social interaction enhances mental fitness in humans. Other long-evolved, but currently poorly understood, mechanisms probably act as correctives to keep Gilbert's evolutionary structures from going off the rails, e.g. attempting to limit flight-or-fight HPA responses to 'real' threats, and so on.

Animal consciousness has thus had the benefit of several hundred million years of evolution to develop the corrective and compensatory structures for its stability and efficiency over the life course. These are currently not well characterized.

The explicit inference, then, is that human consciousness can suffer interpenetrating dysfunctions of mutual and reciprocal interaction with embedding environments which will have early onset and often insidious staged developmental progression, possibly according to a cancer model. There will be no simple, reductionist brain chemical 'bug in the program' whose 'fix' can fully correct the problem. On the contrary, the growth of an individual over the life course, and contact with toxic features of the outside world, can be expected to initiate developmental disorders which will become more intrusive over time, most obviously following some damage accumulation model, but likely according to far more subtle, indeed punctuated, schemes.

The obvious rate distortion argument suggests that, if an external information source is pathogenic, then sufficient exposure to it is sure to write a sufficiently accurate image of it on mind and body in a punctuated manner so as to initiate or promote similarly progressively punctuated developmental dysfunction.

The key intervention, at the population level, is clearly to limit such exposures, a matter of proper environmental sanitation, in a broad sense, largely a question of social justice which has long been understood to be primarily determined by the interactions of cultural trajectory, group power relations, and economic structure with public policy (e.g. Fullilove, 2004; Wallace and Wallace, 1998; Gandy and Zumla, 2003; Farmer, 2003).

At the individual level, intervention against mental disorders is far more problematic, just as it is in the treatment of cancer, and for analogous reasons. In effect, one does not cure developmental disorders once they have begun. The best that can be achieved is to trigger remission, either by encouraging or strengthening natural control and recovery mechanisms, or through direct chemical intervention. Proper treatment can sometimes induce long-lasting remission, subject to the determinants of medical failure, which are often precisely the matters of cultural trajectory, group power relations, economic structure, and public policy which are at the base of many population-level disease patterns. For an extended discussion see, for example, Wallace and Wallace (2004), who find that structured psychosocial stress can write a literal image of itself onto the success or failure of individual-level therapeutic intervention, drug-related or not, just as it can on the development and functioning of human intelligence and consciousness.

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Figure Caption

Figure 1. Relative size of the largest connected component of a random graph, as a function of $2\times$ the average number of fixed-strength connections between vertices. W is the Lambert-W function, or the ProductLog in Mathematica, which solves the relation $W(x)\exp[W(x)] = x$. Note the sharp threshold at $a = 1$, and the subsequent topping-out.'Tuning' the giant component by changing topology generally leads to a family of similar curves, those having progressively higher threshold with correspondingly lower asymptotic limits (e.g. Newman et al., 2001, fig. 4).

RELATIVE SIZE OF LARGEST CONNECTED COMPONENT