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The sleep cycle: a mathematical analysis from a global workspace perspective

Rodrick Wallace, Ph.D.
The New York State Psychiatric Institute*

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Abstract

Dretske's invocation of necessary conditions from communication theory in the characterization of mental process serves as a basis for deriving Hobson's AIM treatment of the sleep/wake cycle from Baars' Global Workspace model. One implication of the analysis is that certain sleep disorders may be recognizably similar to many other chronic, developmental dysfunctions, including autoimmune and coronary heart disease, obesity, hypertension, and anxiety disorders, in that these afflictions often have roots *in utero* or adverse early childhood experiences or exposures to systematic patterns of structured stress. Identification and alteration of such factors might have considerable impact on population-level patterns of sleep disorders. This suggests the possibility of a comprehensive public health approach to sleep disorders rather than current exorbitantly expensive case-by-case medical intervention that does little more than prescribe hypnotic drugs which, in themselves, often carry a substantial health burden.

Key words AIM, consciousness, global workspace, information theory, phase transition, NREM, REM, sleep, structured stress.

Introduction.

Sleep and consciousness present complementary enigmas across a broad span of intellectual discourse. Philosophers, psychologists, psychiatrists, neuroscientists and others have long argued and speculated about their nature, content, purpose, and the relations between them. The debate has not been smooth, however. For most of the 20th Century, behaviorist ideology in psychology precluded the scientific study of consciousness, and at least one essential characteristic of sleep – the Rapid Eye Movement (REM) stage – was only discovered in the 1950's.

Currently two apparently rival candidates for large-scale 'standard models' have emerged describing the phenomena, Hobson's activation-input-modulation (AIM) treatment of the sleep-wake cycle (Hobson, 2001) and Baars' global workspace (GW) analysis of consciousness (Baars, 1988). The purpose of this paper is to extend a recent formal mathematical treatment of the GW model (Wallace, 2005) toward Hobson's perspective. The basis for this attempt is Dretske's recognition that the asymptotic limit theorems of information theory provide a set of necessary conditions for all mental process, just as they do for any phenomena involving the transmission and transformation of information (Dretske, 1981, 1988, 1993, 1994). The essential trick is to implement an obvious homology between information source uncertainty and the free energy density of a physical system. This permits importation of renormalization methods for information systems which undergo the punctuated 'phase changes' characteristic of accession to consciousness. It also permits development of an analog to the phenomenology of Onsager relations in a physical system, allowing description of behavior away from such critical points, in the context of a set of topological rate distortion manifolds which generalize the idea of an adaptable retina (Wallace, 2005, Ch. 4). Taking the model to second order in tuning the universality class associated with conscious punctuation gives the Baars result in a manner almost exactly similar to hierarchical regression. Here we use this formalism to produce, in what we claim is a highly natural manner, something much like Hobson's treatment of the sleep/wake cycle.

The reader should be broadly familiar with the different stages of sleep, their role in fixation of memory (e.g. Rauchs et al., 2005), and Hobson's work (Hobson, 2001), as well as with the general ideas of Baars' global workspace model (Baars, 1988). Regarding the latter, essential features include the shifting serial broadcast/recruitment of information among unconscious modules, and punctuated behavior (e.g. Sergeant and Dehaene, 2004). Note that REM sleep is now viewed as a state of consciousness which, compared to alert waking, is deficient in both neuromuscular function (i.e. sleep paralysis) and analytic ability, while particularly rich in emotional cognition. Finally, both waking up and going to sleep involve complicated physiological processes to effect a transition between states. Since the late 1940's researchers have understood the importance of the reticular activating mechanism in the change from sleep to waking (Evans, 2003; Moruzzi and Magoun, 1949). More recently, an arousal inhibitory mecha-

*Address correspondence to R. Wallace, PISCS Inc., 549 W. 123 St., Suite 16F, New York, NY, 10027. Telephone (212) 865-4766, email rdwall@ix.netcom.com. Affiliations are for identification only.

nism, a thalamo-cortical process, has been recognized which transfers the body from waking to sleep (Evans, 2003). The necessity of mechanisms to effect these transitions will prove central to the subsequent argument.

The mathematical development will be limited to first order and to the minimum needed to go from the GW to the AIM model, although the core of a more elaborate treatment is outlined in a mathematical appendix.

Extending the global workspace model.

Figure 1, from Hobson (2001) via Mahowald (2003), is a schematic of the activation-input-modulation model of sleep. It is a three dimensional classification of sleep/wake states based on (1) degree of central nervous system activation, the A axis, (2) classification of input into endogenous (internal) vs. exogenous (external), the I axis, and (3) modulation, M, based on aminergic vs. cholinergic-dominated neurochemistry. The normal trajectory, the dotted line, is the orderly progression believed necessary to efficiently fix memory (Rauchs et al., 2005).

To anticipate slightly, we shall argue that the CNS activation axis has its analog in the information dynamics version of global workspace theory through the probability of interaction between distinct neural modules, which generally process internal or external signals unconsciously, but can be recruited into the global workspace by long-range neural structures. Coma and (deep, slow-wave) NREM sleep are states in which such global recruitment does not, in fact, occur. The information theory analysis differs from Hobson's model in that this recruitment is highly punctuated – in effect a phase transition – once the probability of interaction between distant modules exceeds a threshold. Hence the sudden accession of some external or internal state to consciousness.

Modulation, from the information theory perspective, can likely be related to the 'richness', in a technical sense to be explored more fully below, of conscious cognitive process: REM sleep consciousness, since it is deficient in somatic function and analytic ability when compared to waking consciousness, is consequently far less rich, in this technical sense, than waking consciousness, although dreams have great and convincing, indeed delusional, emotional valence.

The trick will be to extend the information theory treatment of the GW model to include an independent axis analogous to Hobson's endogenous/exogenous Input axis.

In reality, of course, these AIM axes can be rotated, so that the independent parameters of the information theory treatment could then represent appropriate topological transformations or superpositions of Hobson's A, I, and M quantities. Hobson's degree of CNS activation seems the most likely to have an exact parallel in the information theory treatment. In essence, then, the information theory perspective must be extended to a three dimensional form topologically similar, in a general sense, to the AIM model. This requires some development.

Wallace (2000, 2005, Ch. 3) argues that cognitive process can always be associated with a 'dual' information source emitting a stream of internally correlated signals having a grammar and syntax which make it a kind of language subject to the asymptotic limit theorems of information theory. The system may have to be 'coarse grained', i.e. reduced from continuous to discrete, to allow the use of information theory arguments, in a precise analog to symbolic dynamics. Note that cognition does not imply consciousness, which must be addressed through a second order iteration exactly similar to a hierarchical linear regression. That second order model, however, can also undergo phase transitions much like those we describe here.

The dual information source of a cognitive process has a source uncertainty H which is assumed to be parametized by the 'strength of weak ties' between subcomponents of a network structure upon which the dual language is 'spoken'. Again, see Wallace (2005) for a full discussion.

Strong ties are those relations between individual elements of a network of interacting modules which disjointly partition the structure into subcomponents. Weak ties are interactions which do not disjointly partition the network. These are, respectively, analogous to local and mean field couplings in a physical system.

Higher probability of weak ties, i.e. nondisjoint coupling, is associated with higher overall channel capacity, and hence higher information source uncertainty: greater possible language richness. A disjointly partitioned network will not transmit information across its segregated subgroups.

Figure 2 shows H as a function of $X = 1/P$ where P is the probability of weak ties between network/module subcomponents. In typical psychophysics style, $H(X)$ is assumed to take a reverse S-shaped form. The phase transition at X_0 arises from the obvious homology between information source uncertainty and the free energy density of a physical system (Wallace, 2005, Ch. 2, Section 7; Rojdestvensky and Cottam, 2000; Wallace and Wallace, 1998). To the left of X_0 the components can transmit information across the whole structure, to the right, the system has crystallized into its individual submodules, and ceases large-scale broadcast. This is a model of the accession to consciousness, where dispersed functional structures are recruited into a shifting global workspace.

The second stage of the argument invokes a phenomenology similar to the Onsager relations of physical theory, which determine the behavior of physical systems under nonequilibrium thermodynamic conditions. If the source uncertainty H is parametized by a vector of quantities $\mathbf{K} \equiv (K_1, ..., K_m)$, then we are interested in the *disorder*,

$$S \equiv H(\mathbf{K}) - \sum_{j=1}^{m} K_j \partial H / \partial K_j. \quad (1)$$

The traditional phenomenological 'generalized Onsager relations', defining system dynamics, are then

$$dK_j/dt = \sum_i L_{j,i} \partial S/\partial K_i, \tag{2}$$

where the $L_{i,j}$ are constants depending on the nature of the underlying structures. They are to be viewed empirically, in the same way as the slope and intercept parameters of a regression model, and may not at all have the simple canonical forms expected of an ordinary thermodynamic system.

These equations are taken as determining the time-dynamics of the unconstrained system under the 'forces' $\partial S/\partial K_i$. The central problem, of course, lies in characterizing the constraints. A more formal perspective is outlined in the appendix.

For cognitive, as opposed to physical processes, Wallace (2005) argues that the general tendency will be for the system to be driven away from peaks in S: cognitive process does not like residing on a cliff, from this perspective (Wallace, 2005), but prefers to be either at the top or bottom of an S-shaped, or reverse S-shaped curve.

In figure 2, then, there should be a strong tendency for a system already at high H to remain there, with S acting as a barrier to transition to disjoint status. Conversely, a system with $X > X_0$, a disjoint structure, would face a barrier to becoming coherent. This implies that active physiological mechanisms should be required to go from consciousness to unconsciousness, or vice versa: in the context of sleep, the arousal inhibitory mechanism and the reticular activating mechanism, respectively.

The most direct way to produce an analog to the AIM model from this structure is to impose a second reverse S-shaped curve on figure 2, on an axis *perpendicular to the* $H - X$ *plane*. This would represent the decline in richness of cognitive function as one goes from waking consciousness to REM consciousness. Figure 3a and 3b show the full model, based on a particular 'double reverse S' form of H in 3a and the resulting S in 3b. H, S, and X are as above, while Y is an appropriate parameter representing a shift in consciousness from waking to REM sleep.

Note the barriers between the states of figure 3 marked W, R, and N, presumably waking consciousness, REM sleep, and dissociated, unconscious NREM sleep. This suggests the necessity of active mechanisms, not only for going from sleep to awake and back, but for going from waking to REM sleep: Hobson's AIM model is particularly rich in its description of intermediate pathological states like trance, waking hallucinations, the effect of psychedelic drugs, and the like, and is probably applicable to hypnosis. The analysis suggests searching for some physiological phenomenon similar to, but separate from, the reticular activating and arousal inhibitory mechanisms. This must implement any direct transition from waking consciousness to REM sleep.

A detailed mathematical analysis based on figure 3 and generalizations of equations (1) and (2) is presented in the appendix. Like all models of what are basically complicated ecosystem phenomena, using the words of of the mathematical ecologist E.C. Pielou (1976, p. 106), this one too is most useful in "...[R]aising questions for empirical study rather than in answering them."

Discussion and conclusions.

One series of essential questions raised by the modeling exercise is as follows: Clearly equation (2) (and, indeed, the more elaborate development of the appendix), by itself, cannot describe the normal sleep/wake cycle, which isn't at all automatic, and requires distinct physiological mechanisms – reticular activating, arousal inhibiting and possibly others – to effect transition between the quasi-stable states W, N, and R, and to ensure the orderly progression so important to the fixation of memory. Wallace (2004; 2005, Ch. 6) has invoked higher order 'Zero Mode Identification' (ZMI) cognitive modules analogous to an adaptable retina in the operation of a broad spectrum of cognitive physiological and psychological processes ranging from the immune system and Hypothalamic-Pituitary-Adrenal (HPA) axis to emotional and conventional cognitive function. Chronic 'autocognitive' disease is then viewed as the identification of an excited mode, supposed to be only temporarily activated, as the normal, zero-state of the system. Systematic misidentification is supposed to account for autoimmune dysfunction, certain anxiety disorders, and the like (Wallace, 2004, 2005).

It is tempting, then, to postulate a generalized ZMI structure for the sleep/wake cycle, but with *three* rather than one zero-modes. Call it, for want of a better name, the Sleep Mode Identification (SMI) module, a 'retina' with three focal zones, if you will. Normal operation of the SMI is the progression $W \rightarrow N \rightarrow R \rightarrow W$ among quasi-stable states. $W \rightarrow R$ would be characteristic of hypnosis and related conditions, while recurrent 'stalling' at an intermediate – usually unstable – focal point would define chronic sleep disorders, in much the same way that chronic inflammation characterizes coronary heart disease (CHD) and chronic excitation of the blood pressure regulatory mechanism defines hypertension.

It is widely believed that a broad group of chronic 'autocognitive' disorders like hypertension, obesity, CHD, other autoimmune diseases, anxiety disorder, and the like, are developmental in nature, with roots in utero or early childhood. Wallace (2005) provides a long list of references. Chronic mind/body dysfunction, from this perspective, instantiates an image of embedding structured psychosocial or other stressors on child development (Wallace, 2004, 2005). Consideration suggests that some sleep disorders are also likely to be developmental in nature, with similar origins, and possibly progressive onset. These will then constitute distorted, internalized images of embedding patterns of structured external stressors. As with many conditions, sleep disorders are likely to be comorbid with other mind/body dysfunctions.

Recent studies of insomnia seem consistent with this analysis. Bixler et al. (2002) found, for a large study sample in

Central Pennsylvania, that the principal determinants of insomnia in a logistic regression model were depression, female status, and non-Caucasian ethnicity. Indeed, non-Caucasians were found to suffer insomnia at nearly twice the rate of Caucasians, 12.9 vs. 6.6 percent. Female and minority status in the US, of course, subject a person to substantial gender and racial discrimination over the entire life course. Similarly, Ohayon et al. (2002) have found working on a rotating daytime shift to cause significant sleep disturbances, resulting in higher rates of on-the-job sleepiness, work-related accidents, and sick leaves. Assignment to shift work is often a quintessential consequence of employee disempowerment. These examples indeed suggest that external stressors can write an image of themselves on sleep pattern, as well as on other physiological and psychological processes, by a variety of mechanisms.

With regard to comorbidity, Vitiello et al. (2002) found that sleep disorders in older adults are not a consequence of age itself, but rather cosegregate with other medical and psychiatric disorders and health-related burdens.

In sum, it does indeed seem possible to relate Hobson's AIM model to Baars' Global Workspace model in a 'natural' manner, based on Dretske's necessary conditions analysis as extended by the obvious homology between information source uncertainty and the free energy density of a physical system. Independent of the details of the mathematical modeling exercise, this perspective suggests that some chronic sleep disorders may be similar to many other chronic mental and physical disorders, not only by virtue of representing failure of supervisory 'mode identification' cognitive modules, but by being broadly developmental in nature. If true, some sleep disorders might significantly respond, at the population level, to highly efficient and relatively inexpensive public health interventions which do not simply shift disease management burden to those afflicted, but rather prevent suffering altogether.

Mathematical Appendix

Equations 1 and 2 can be derived in a simple parameter-free 'covariant' manner which relies on the underlying topology of the 'information source space' implicit to the development. We suppose that different physiological cognitive phenomena have, in the sense of Wallace (2000, 2005, Ch. 3), dual information sources, and are interested in the local properties of the system near a particular reference state. In this we suppose the system to be far from phase transition, for example sleepy, but not yet in NREM sleep, or in REM sleep, but not yet awakened by the reticular activating mechanism. We impose a topology on the system, so that, near a particular 'language' A, dual to an underlying cognitive process, there is (in some sense) an open set U of closely similar languages $\hat{A}$, such that $A, \hat{A} \subset U$. Note that it may be necessary to coarse-grain the physiological responses to define these information sources. The problem is to proceed in such a way as to preserve the underlying essential topology, while eliminating 'high frequency noise'. The formal tools for this can be found, e.g., in Chapter 8 of Burago et al. (2001).

Since the information sources dual to the cognitive processes are 'similar', for all pairs of languages $A, \hat{A}$ in U, it is possible to:

(1) Create an embedding alphabet which includes all symbols allowed to both of them.

(2) Define an information-theoretic distortion measure in that extended, joint alphabet between any high probability (i.e. grammatical and syntactical) paths in A and $\hat{A}$, which we write as $d(Ax, \hat{A}x)$ (Cover and Thomas, 1991). Note that these languages do not interact, in this approximation.

(3) Define a metric on U, for example,

$$\mathcal{M}(A, \hat{A}) = |\lim \frac{\int_{A,\hat{A}} d(Ax, \hat{A}x)}{\int_{A,A} d(Ax, A\hat{x})} - 1|, \tag{3}$$

using an appropriate integration limit argument over the high probability paths. Note that the integration in the denominator is over different paths within A itself, while in the numerator it is between different paths in A and $\hat{A}$.

Consideration suggests $\mathcal{M}$ is a formal metric, having $\mathcal{M}(A, B) \geq 0, \mathcal{M}(A, A) = 0, \mathcal{M}(A, B) = \mathcal{M}(B, A), \mathcal{M}(A, C) \leq \mathcal{M}(A, B) + \mathcal{M}(B, C)$.

Other approaches to constructing a metric on U may be possible.

Since H and $\mathcal{M}$ are both scalars, a 'covariant' derivative can be defined directly as

$$dH/d\mathcal{M} = \lim_{\hat{A} \to A} \frac{H(A) - H(\hat{A})}{\mathcal{M}(A, \hat{A})}, \tag{4}$$

where $H(A)$ is the source uncertainty of 'language' A.

Suppose the system to be set in some reference configuration A_0.

To obtain the unperturbed dynamics of that state, we impose a Legendre transform using this derivative, defining another scalar

$$S \equiv H - \mathcal{M} dH/d\mathcal{M}. \tag{5}$$

The simplest possible Onsager relation – again an empirical equation like a regression model – in this case becomes

$$d\mathcal{M}/dt = LdS/d\mathcal{M},$$

(6)

where t is the time and $dS/d\mathcal{M}$ represents an analog to the 'thermodynamic force' in a chemical system. This is seen as acting on the reference state A_0. For

$$dS/d\mathcal{M}|_{A_0} = 0, d^2S/d\mathcal{M}^2|_{A_0} > 0$$

(7)

the system is quasistable, and externally imposed physiological forcing mechanisms will be needed to effect a transition to a different state.

Explicit parametization of $\mathcal{M}$, for example by X and Y in figure 3, introduces standard – and quite considerable – notational complications (e.g. Wald, 1984; Burago et al., 2001; Auslander, 1967). Letting the parameters be a vector $\mathbf{K}$ having components $K_j, j = 1..m$, we can write $\mathcal{M}$ in terms of a 'metric tensor' $g_{i,j}(\mathbf{K})$ as

$$\mathcal{M}(A_0, A) = \int_A^{\hat{A}} [\sum_{i,j}^{m} g_{i,j}(\mathbf{K}) \frac{dK_i}{dt} \frac{dK_j}{dt}]^{1/2} dt$$

(8)

where the integral is taken over some parametized curve from the reference state A_0 to some other state A. Then equation (6) becomes

$$[\sum_{i,j} g_{i,j}(\mathbf{K}) \frac{dK_i}{dt} \frac{dK_j}{dt}]^{1/2} = L \frac{dS}{d\mathcal{M}}.$$

(9)

This states that the 'velocity' $d\mathbf{K}/dt$ has a magnitude determined by the local gradient in S at A_0, since the summation term on the left is the square root of an inner product of a vector with itself.

The first condition of equation (7) gives

$$\sum_{i,j} g_{i,j} \frac{dK_i}{dt} \frac{dK_j}{dt} = 0.$$

(10)

Thus the initial velocity is again zero, in the coordinates $\mathbf{K}$.

To go much beyond this obvious tautology we must, ultimately, generate a parametized version of equation (6) and its dynamics, but expressing the metric tensor, and hence redefining the geometry, in terms of derivatives of S by the K_i. The result requires some development.

Write now, for parameters K_i – analogous to X and Y in figure 3 – the Onsager relation

$$dK_i/dt = L\partial S/\partial K_i,$$

(11)

where the K_i have been appropriately scaled.

Again place the system in a reference configuration A_0, having a vector of parameters $\mathbf{K}_0$, so that

$$d\mathbf{K}/dt|_{\mathbf{K}_0} = L\nabla S|_{\mathbf{K}_0} \equiv 0.$$

(12)

Deviations from this state, $\delta K \equiv K - K_0$, to first order, obey the relation

$$d\delta K_i/dt \approx L \sum_{j=1}^{m} (\frac{\partial^2 S}{\partial K_i \partial K_j}|_{K_0}) \delta K_j.$$

(13)

In matrix form, writing $U_{i,j} = U_{j,i}$ for the partials in S, this becomes

$$d\delta\mathbf{K}/dt = L\mathbf{U}\delta\mathbf{K}.$$

(14)

Assume appropriate regularity conditions on S and $\mathbf{U}$, and expand the deviations vector $\delta\mathbf{K}$ in terms of the m eigenvectors $\mathbf{e}_i$ of the symmetric matrix $\mathbf{U}$, having $\mathbf{U}\mathbf{e}_i = \lambda_i \mathbf{e}_i$, so that $\delta\mathbf{K} = \sum_{i=1}^{m} \delta a_i \mathbf{e}_i$.

Equation (14) then has the solution

$$\delta\mathbf{K}(t) = \sum_{i=1}^{m} \delta a_i \exp(L\lambda_i t)\mathbf{e}_i.$$

(15)

If all $\lambda_i \leq 0$, then the system is bounded quasistable, and a physiological forcing mechanism will be required to change status.

Next let $d\delta K_i/dt \equiv \delta V_i$. In first order the magnitude of the vector $\delta\mathbf{V}$ is

$$|\delta\mathbf{V}|^2 = \frac{L^2}{2} \sum_{i,j} [\sum_k U_{i,k} U_{k,j}] \delta K_i \delta K_j$$

(16)

Redefining

$$g_{i,j} \equiv \frac{L^2}{2} \sum_k U_{i,k} U_{k,j}$$

(17)

gives, after some notational shift, the symmetric Riemannian metric

$$dV^2 = \sum_{i,j} g_{i,j}(K_i, K_j) dK_i dK_j,$$

(18)

so that the metric, and hence the geometry, is now defined in terms of derivatives of S by the K_j.

The 'distance' between points a and b along some dynamic path in this geometry is, again,

$$s(A,B) = \int_A^B [\sum_{i,j} g_{i,j} \frac{dK_i}{dt} \frac{dK_j}{dt}]^{1/2} dt.$$

(19)

Application of the calculus of variations to minimize this expression produces a geodesic equation for the slowest dynamical path, and hence the most physiologically stable configuration. This has the traditional component-by-component form

$$d^2K_i/dt^2 + \sum_{j,m} \Gamma^i_{j,m} \frac{dK_j}{dt} \frac{dK_m}{dt} = 0,$$

(20)

where the $\Gamma^i_{j,k}$ are the famous Christoffel symbols involving sums and products of $g_{i,j}$ and $\partial g_{i,j}/\partial K_m$ (e.g. Auslander, 1967; Burago et al., 2001; Wald, 1984, etc.).

The analog to equation (7) in this new geometry, defining a quasi-stable state, is that there exists a positive number $\mathcal{K} \ll R$, where R is the maximal possible number characteristic of the entire system, such that, for all geodesics $\mathbf{K}(t)$ which solve equation (20),

$$|\mathbf{K}(t)| \leq \mathcal{K}.$$

(21)

at all times t.

Under such circumstances geodesics sufficiently near the reference state A_0 are all bound, and external physiological forcing must be imposed to cause a transition to a different condition. This result is analogous to the 'Black Hole' solution in General Relativity: recall that, within a critical radius near a sufficiently massive point source – the 'event horizon' – all geodesics, representing possible paths of light, are gravitationally bound without, however, the possible grace of a physiological *deus ex machina*.

References

Auslander L., 1967, *Differential Geometry*, Harper and Row, New York.

Baars B, 1988, *A Cognitive Theory of Consciousness*, Cambridge University Press.

Burago D, Y. Burago, and S. Ivanov, 2001, *A Course in Metric Geometry*, American Mathematical Society, Providence RI.

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

Dretske F., 1981, *Knowledge and the Flow of Information*, MIT Press, Cambridge, MA.

Dretske F., 1988, *Explaining Behavior*, MIT Press, Cambridge, MA.

Dretske F., 1993, Mental events as structuring causes of behavior. In *Mental Causation*, (ed. A. Mele and J. Heil), pp. 121-136. Oxford University Press.

Dretske F., 1994, The explanatory role of information, *Philosophical Transactions of the Royal Society A*, 349:59-70.

Evans B., 2003, Sleep, consciousness and the spontaneous and evoked electrical activity of the brain. Is there a cortical integrating mechanism? *Neurophysiologie Clinique*, 33:1-10.

Hobson J.A., 2001, *The Dream Machine: Chemically Altered States of Consciousness*, MIT Press, Cambridge, MA.

Mahowald M., 2003, Understanding consciousness, *Perspectives in Biology and Medicine*, 42:297-304.

Moruzzi G. and H. Magoun, 1949, Brain stem reticular formation and activation of the EEG, *Electroencephology and Clinical Neurophysiology*, 1:455-473.

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

Rauchs G., B. Desgranges, J. Foret, and F. Eustache, 2005, The relationships between memory systems and sleep stages, *Journal of Sleep Research*, 14:123-140.

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

Sergeant C. and S. Dehaene, 2004, Is consciousness a gradual phenomenon? Evidence for an all-or-none bifurcation during the attentional blink. In press, *Psych. Sci.*

Wald R., 1984, *General Relativity*, University of Chicago Press, Chicago.

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

Wallace R., 2004, Comorbidity and anti comorbidity: autocognitive developmental disorders of structured psychosocial stress, *Acta Biotheoretica*, 50:101-116.

Wallace R., 2005, *Consciousness: A Mathematical Treatment of the Global Neuronal Workspace Model*, Springer-Verlag, New York.

Figure captions.

Figure 1. Hobson's AIM model (Hobson, 2001) via Mahowald (2003). Three dimensions characterize the model: (A) Level of CNS activation (low vs. high), (I) Sensory input, (internal vs. external) and (M), neurotransmitter modulation, (aminergic vs. cholinergic). Normally only small, well defined positions of this space are traversed, wakefulness, NREM sleep, and REM sleep. Under pathological circumstances, however, any portion may be occupied, resulting in altered states of consciousness and behavior, for example phases of narcolepsy are explained as admixtures of wakefulness and REM sleep.

DOA = disorders of arousal (sleepwalking, sleep terrors, confusional arousals). RBD = REM sleep behavior disorder.

Figure 2. Two dimensional model of consciousness according to Global Workspace theory. $X = 1/P$ is the inverse probability of nondisjunctive 'weak' ties between cooperating, distant, neural modules. $H(X)$ is the source uncertainty dual to the cognitive process associated with consciousness, and $S = H - XdH/dX$ is the disorder construct. Note that X_0 is the critical point at which the workspace falls apart into component parts: consciousness ceases. According to theory, S constitutes a barrier which must be overcome by active physiological mechanisms. H is likely to be analogous to Hobson's M.

Figure 3. (a) Extension of H in figure 2 by addition of a second reverse S-shaped curve perpendicular to the H-X axis. This new curve represents the decline in 'richness' of consciousness between waking and REM sleep. (b) $S = H - XdH/dX - YdH/dY$ from H in (a). Note that three states, marked W, N and R lie in regions of relative stability, requiring, in this formulation, active physiological mechanisms for transitions between them. We infer the existence of a cognitive 'Sleep Mode Identification' module – SMI – analogous to a tunable retina whose 'focus' determines sleep progression. The normal course is $W \rightarrow N \rightarrow R$. Sleep pathologies – SMI 'focus' on intermediate states – represent, in this model, rate-distorted images of embedding structured stressors written on the SMI by developmental process.

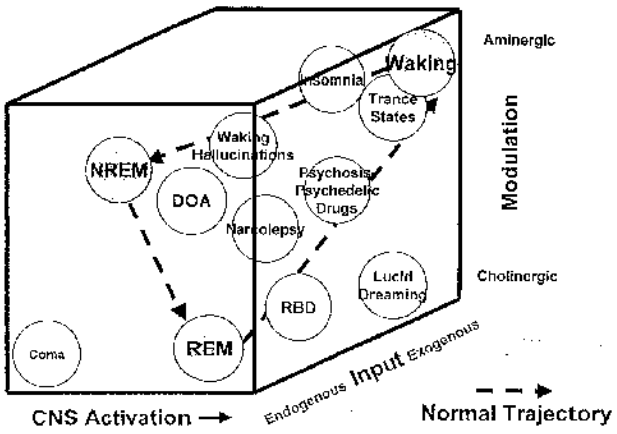

Fig. 1

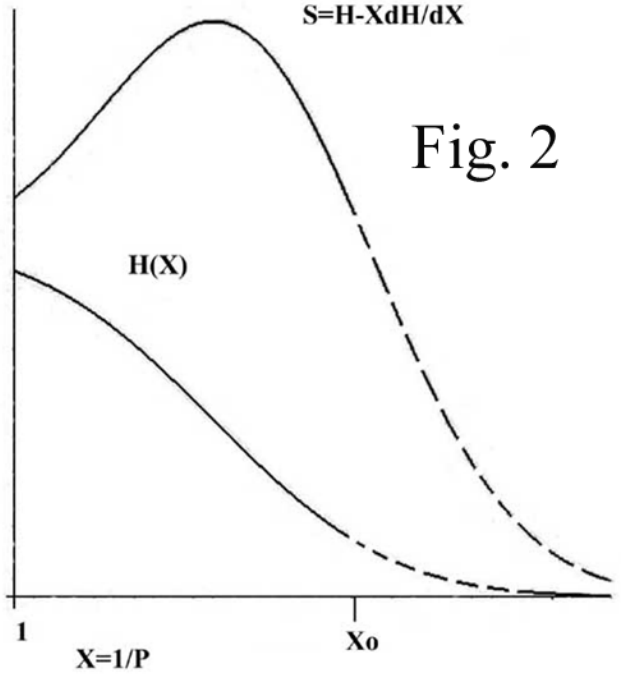

Fig. 2

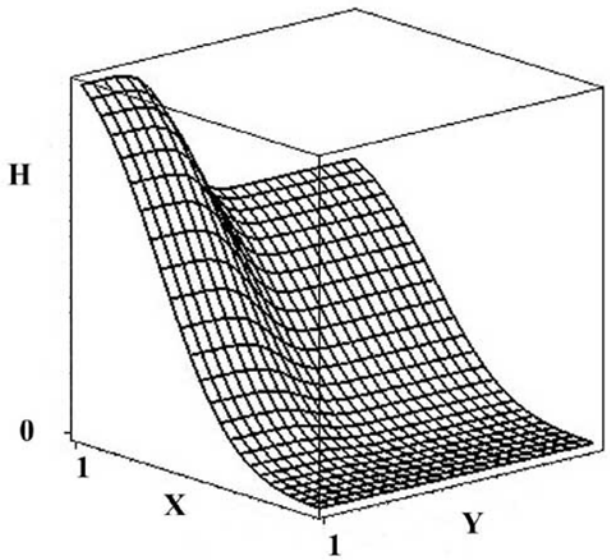

Fig. 3a

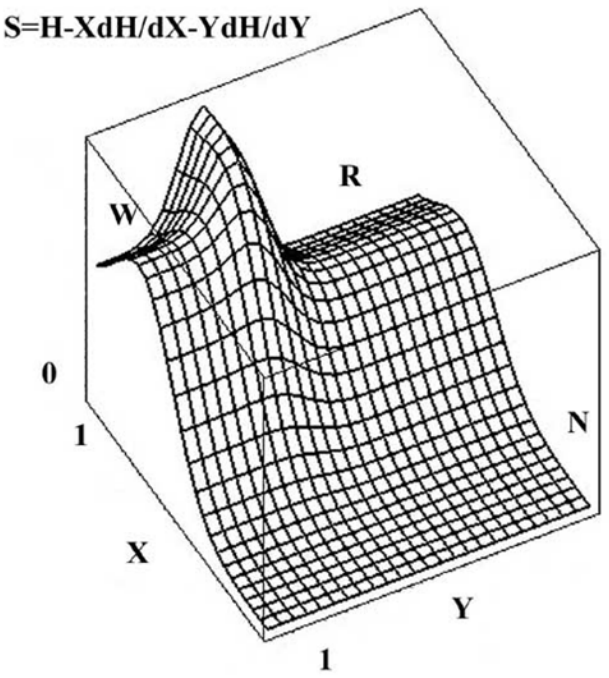

Fig. 3b