THE METABOLIC ROOTS OF CONSCIOUSNESS

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Abstract. Clinical research dealing with metabolic dysfunctions indicates that the evolution of disorders commonly referred to as *psychogenic, mental, behavioral* and *stress-related* is governed predominantly by intermediary metabolic activity. These dysfunctions, characterized by weak or poorly coupled interactions within Kreb's cycle and the Embden-Meyerhoff pathways, can be nutritionally countereffected thereby limiting and often eliminating the extent to which they are cognitively, affectively and behaviorally articulated. This research suggests that genetic factors and nutritional input are primary determinants of psychometabolic pathology. The implications of this research are profound, extend far beyond the domains of clinical psychology and medicine, and may go on to impact disciplines as diverse as psycholinguistics, sociobiology, criminology, cultural anthropology and zoology to mention only a few.

The status of clinical research regarding the role of intermediary metabolism in shaping cognitive performance is reviewed in this article. A generic mathematical formalism of metabolic activity is developed, and a metric mapping metabolic activity into cognitive activity is proposed and discussed. KEYWORDS: metabolism, zeitgebers, psychopathology, cognitive function, redundancy, consciousness, nutrition.

I. Introduction.

Two key observations shed light on a critical biological mechanism which mediates cognitive activity. Nutritionally induced changes in intermediary metabolic function gauged by shifts in venous plasma pH correlate with the evolution of so-called *psychogenic* or *psychiatric* disorders. These changes are also accompanied by alterations in chemorhythmic activity characterized by circadian, ultradian and infradian cycles. These observations are discussed explaining how metabolism alters cognitive states. Specifically, it is shown how psychopathology may be precipitated and terminated via nutritional input. The metabolic and chemorhythmic mechanisms mediating these cognitive changes are discussed in this context. A mathematical formalism is also proposed in an attempt to establish a rational methodology which predicts how the human organism must be metabolically manipulated to allow it to predictably undergo alterations in cognitive and experiential states. This formalism is used to canonically describe metabolic activity in terms of first principles of enzyme rate kinetics and to posit a metric for psychometabolic organization. It is shown that this metric is formally identical to the information theoretic noise norm, *redundancy*. Finally, it is also mathematically shown that richly coupled metabolic systems governed by Michaelis-Menten rate kinetics can be made to evolve in more organized fashion than their more poorly coupled counterparts, a conclusion which is supported by clinical observations.

II. Metabolic Classification.

Without exception, every individual within any randomly selected population may at any given time be classified as belonging to any one of three intermediary metabolic categories. Initially described in terms of glucose oxidation rate (ref. 1, 2, 3, 4, 5), these metabolic categories or types are perhaps better characterized in terms of acid/alkaline shifts in venous plasma pH to one part in one thousand (ref. 10). Each of these three types (referred to hereafter as acid, alkaline and mixed mode metabolizers) are genetically programmed to metabolically respond in a uniquely different fashion to an identical set of nutritional regimens administered over prolonged periods of time (ref. 10). This observation may in fact be used to define these types as follows. A standardized nutritional regimen rich in nucleoprotein and modest in carbohydrate and fat will elicit the following metabolic responses from each type. An acid metabolizer's average diurnal venous plasma pH will drop below 7.43. An alkaline metabolizer's average diurnal venous plasma pH will rise above 7.47. A mixed mode metabolizer's average diurnal venous plasma pH will settle in the immediate vicinity of 7.45. The terms acid and alkaline are therefore used to refer to *relative* shifts in venous plasma pH about a baseline range. Shifts above and below this baseline range are defined as alkaline and acid respectively. These terms obviously do *not* refer to acid/alkaline shifts about pH neutrality (7.00) of pure water since venous plasma in vivo is *always* alkaline with respect to pure water.

These deviations are significant in that when pH values fall below or rise above 7.43 and 7.47 respectively, they almost invariably accompany the onset of psychophysiological dysfunction. Conversely, frequency of occurrence of psychophysiological dysfunction accompanied by average diurnal pH variation in the neighborhood of 7.43-7.47 is extremely low. In essence, these results strongly suggest that in a population which does not suffer from neurological impairment, the greater the deviation of

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venous plasma pH from mid range values (7.43-7.47), the greater the extent of psychophysiological dysfunction and vice versa. In the extreme, individuals possessing average diurnal venous plasma pH values either less than 7.39 or exceeding 7.50 invariably suffer severe and incapacitating cognitive dysfunction.

Given the fact that sustained administration of the same nutritional regimen will elicit markedly different psychochemical reactions from different metabolic types, it is clear then why each type will require a different set of regimens to allow its member individuals to minimize psychophysiological dysfunction and ultimately optimize psychometabolic performance.

Relative optimal psychometabolic performance may be achieved as follows:

1. Acid metabolizers will require low carbohydrate, moderate fat and high nucleoprotein regimens with the following supplemental micronutrients (and no others): A, E (mixed tocopherols), B12, C, Inositol, Choline, Niacinamide, Pantothenic Acid, Ca, P, Zn, I.

2. Alkaline metabolizers will require high carbohydrate, low fat and near zero nucleoprotein regimens with the following supplemental micronutrients (and no others): A, D, E (d-α tocopherol), B1, B2, B3, B6, C, Paba, Mg, Mn, K, Cu, Cr, Fe.

3. Mixed mode metabolizers possess biochemical profiles which consist of a superposition of the acid and alkaline profiles, biased to respond favorably to nutrient intake similar to that appropriate for acid metabolizers. Consequently, mixed mode types will require an admixture of the two aforelisted regimens and supplements in a 3:1 caloric and dosage ratio biased in favor of the regimen appropriate for acid metabolizers.

It is noted in passing that the regimens and companion micronutrient supplements required to optimize psychometabolic performance within the acid and alkaline groups are mutually exclusive.

These regimens could incidentally be used to redefine each of the three aforelisted metabolic types consistent with the definition given initially. Specifically, in response to the regimen and micronutrient combination appropriate for acid metabolizers, average diurnal pH values for acid types will be allowed to settle within the 7.43 to 7.47 range, while average diurnal pH for alkaline and mixed mode types will be forced to exceed 7.50 or will be driven within the 7.48-7.50 range respectively. This result says in effect that the regimen/micronutrient combination appropriate for acid types countereffects venous plasma acidosis in that it is alkaline inducing. This regimen/micronutrient combination drives pH upward thus inducing severe alkalosis in alkaline types and mild alkalosis in mixed mode types. Similarly, in response to the regimen/micronutrient combination appropriate for alkaline metabolizers, average diurnal pH values for alkaline types will be allowed to settle within the 7.43 to 7.47 range, while average diurnal pH for acid and mixed mode types will be forced to drop below 7.39 or will be driven within the 7.40-7.43 range respectively. This result says in effect that the regimen/micronutrient combination appropriate for alkaline types countereffects venous plasma alkalosis in that it is acid inducing. This regimen/micronutrient combination drives pH downward thus inducing severe acidosis in acid types and moderate acidosis in mixed mode types.

These clinical results point to the fact that the administration of an *antagonistic* regimen with its companion set of micronutrients will invariably result in significant *erosion* of cognitive and behavioral performance. Specifically, inappropriate administration of regimen/micronutrient combinations will drive pH upward in the case of alkaline metabolizers, downward in the case of acid metabolizers and destabilize pH trajectories in the case of mixed mode metabolizers. In all cases, metabolic disequilibration of this nature will trigger acute psychophysiologic dysfunction which is clinically indistinguishable from states of psychopathy historically believed to be sociogenic.

The inability to distinguish between these disorders, even through the use of psychometric inventories suggests that perhaps most if not all chronic psychophysiological dysfunction is the result of metabolically mismatched nutritional input and not, as is widely believed in both popular and academic circles, the result of social or informational input.

The metabolic types discussed thus far characterize static metabolizers in that their psychometabolic response to nutritional input is invariant over time. Static acid, alkaline and mixed mode metabolizers will always respond favorably to the regimen and companion micronutrients compatible with their respective metabolic types. Conversely they will always respond adversely to the regimen and companion micronutrients incompatible with their respective metabolic types. There are however also numerous individuals possessing time varying metabolic profiles which can be expressed as time dependent functions of these three types. A few of the more prevalent profiles are characterized by diurnal, monthly, seasonal and random cycling. In general, cycling is characterized by transitions from one of the three aforelisted metabolic states to another. Cycling generally does not occur gradually, but occurs rather in stepwise discontinuous fashion. Cyclic metabolic types will of course require different nutritional regimens and companion micronutrient supplements at different times in order to countereffect emerging metabolic imbalances as they arise. Diurnal and random cycling are the least common of the four, the latter exhibiting shifts in pH which are extremely difficult to countereffect. Seasonal cycling, also relatively uncommon is characteristic of Seasonal Affective Disorder (SAD). Monthly cycling is not uncommon among premenopausal females and is likely the reason why postpubescent women comprise more than 75% of all patients seeking assistance through primary health care. It is not surprising to see why these patients are in frustration inappropriately viewed by the medical community as prime candidates for psychiatric treatment.

Metabolic inhomogeneity within human populations is very likely the result of genetic inhomogeneity. This inhomogeneity as well as variations in eating habits suggest that population density distributes itself in Gaussian fashion when plotted against deviations in venous plasma pH and (thus) psychophysiological dysfunction. This distribution's spread cannot be accounted for solely in terms genetic inhomogeneity, but results in large part form diversity in eating habits. The area subtended by this

Gaussian distribution's central hump characterizes that portion of the population experiencing relatively low level distress. The two extremes of the distribution characterize that portion of the population whose symptoms are severe enough to warrant long term institutionalization. The remaining area beneath this distribution characterizes that portion of the population which is distressed enough to require short term episodic institutionalization, and which repeatedly seeks out assistance from a medical community which erroneously and routinely classifies its distress as *psychosomatic, psychogenic* or (more recently) as *stress related.* This error should be underscored since it is estimated that more than 75% of all individuals in the U.S. receiving assistance through the primary health care system are told that their distress is at least in part psychogenic. Labeling in this fashion inescapably implies that these disorders are somehow *learned* through informational cues conveyed in familial and social settings. Conversely, this type of labeling implies that these disorders may somehow be *unlearned* or extinguished through verbal interaction taking on any one of the many forms of psychotherapy. *Given the evidence cited thus far, it is not at all surprising to find that with few and limited exceptions (most notably behavior modification therapy), psychotherapy is wholly ineffective in dealing with these and other classes of disorders* (ref. 9, 16).

III. A General Mathematical Formalism.

It has been demonstrated that venous plasma pH is an excellent synoptic indicator of the intensity or absence of psychophysiological distress. Recall, pH is tied to activity of Kreb's cycle and the Embden-Meyerhoff pathways (ref. 5). This metabolic activity can in turn be readily manipulated through nutritional input, thus altering intensity of psychophysiological distress. As already discussed, mismatching this input with metabolic type is primarily responsible for many if not most non-neurologically induced cognitive and behavioral disorders. Future psychometabolic research will however probably uncover other metabolic activity whose impact upon psychophysiological function will also be profound. The fact that venous plasma pH may not be tied directly to this activity suggests that another more general indicator must be found if the psychometabolic connection is to be understood in terms of first principles of biochemistry and thermodynamics. Clinical observations suggest that certain key and pervasive phenomena may in fact universally characterize underlying psychometabolic activity (ref. 6, 7, 8). It is proposed that three phenomena described below be viewed as conditions which should be satisfied if a candidate mathematical formalism is to adequately describe the psychometabolic connection.

C.1:Time dependent concentrations of biochemical species in human physiology vary rhythmically, describing well defined oscillations or circadian rhythms with small amplitudes about their respective baseline values. Biochemical species as differentiated from enzymes and co-enzymes are defined as those chemical species whose co-interactions are facilitated by the presence of enzymes.

C.2:The oscillatory characteristics of these species' concentrations are related directly to the enzymes facilitating interactions among the species. Changes in enzyme concentrations alter frequency and phase of the species' chemo-oscillations.

C.3:Enzyme blocking results in a reduction in chemorhythmic activity, damped chemo-oscillations and increased cognitive dysfunction.

It is proposed that Michaelis-Menten enzyme-species rate kinetics characterize the endogenous mechanism mediating psychometabolic function. This mechanism may be represented mathematically by a set of n coupled spatio-temporal differential equations describing the co-interactions of n biochemical species as part of an n x n reaction-diffusion process.

The n x n reaction diffusion system, adequate in describing small amplitude systems of the variety encountered in this context is given as follows:

$$d\hat{X}/dt = \bar{K}\hat{X} + \nabla^2\bar{D}\hat{X} \tag{1}$$

$\hat{X}$ is a column vector representing the difference between the species' concentrations and their steady state concentrations. $\bar{K}$ represents the n x n enzyme reaction matrix. $\bar{D}$ Represents the n x n diffusion matrix characterizing how each of the biochemical species will migrate spatially as a function of time.

Equation (1) may be solved as follows for $\bar{K}$ and $\bar{D}$ both hermitian. Generalization to the nonhermitian case will be carried out later. Define $\hat{X} = \hat{\chi} - \hat{\chi}_{ss}$ where $\hat{\chi}$ represents species concentration and $\hat{\chi}_{ss}$ represents steady state species concentration. Separating $\hat{X}$spatially and temporally obtain:

$$\hat{\chi} = \hat{\chi}_{ss} + \sum_{i=1}^{n} < \hat{\chi}(0) - \hat{\chi}_{ss} | \hat{e}_i > e^{-\lambda_i t}\hat{e}_i \tag{2}$$

where λ_i and $\hat{e}_i$ are the eigenvalues and the normalized eigenvectors of $\bar{K}$ respectively.

$< \hat{\chi}_{ss} | \hat{e}_i >$ and $\hat{\chi}_{ss}$ are found by solving equation 2 for $\frac{d\hat{\chi}(0)}{dt}$ by exploiting the orthogonality of $\hat{e}_i$ as follows:

$$< \hat{\chi}_{ss} | \hat{e}_i > = < \frac{1}{\lambda_i} \cdot \frac{d\hat{\chi}(0)}{dt} | \hat{e}_i > + < \hat{\chi}(0) | e_i > \tag{3a}$$

$$\hat{\chi}_{ss} = \sum_{i=1}^{n} < \frac{1}{\lambda_i} \cdot \frac{d\hat{\chi}(0)}{dt} | \hat{e}_i > \hat{e}_i + < \hat{\chi}(0) | \hat{e}_i > \hat{e}_i \tag{3b}$$

Consequently,

$$\hat{\chi}(t) = \sum_{i=1}^{n} < \frac{1 - e^{-\lambda_i t}}{\lambda_i} \cdot \frac{d\hat{\chi}(0)}{dt} + \hat{\chi}(0) | \hat{e}_i > \hat{e}_i \tag{4}$$

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Spatio-temporal separation of the hermitian system's solutions is justified since the hermitian system may always be diagonalized via a similarity transform. Hermitian systems are therefore always off diagonally *enzyme blocked*, have only real eigenvalues and thus lack oscillatory time dependence.

In contrast, consider the nonhermitian system. While the nonhermitian system will be examined later in this article within another context as a perturbation of the hermitian system, in general it may be stated that the emergence of complex eigenvalues may be brought about by non-zero off diagonal $\tilde{K}$ matrix entries which cannot be reduced to zero (or blocked) by any similarity transform. The imaginary components of these complex eigenvalues give $\hat{\chi}$ temporal periodicity which would otherwise be absent in the hermitian case. In the extreme, the nonhermitian system whose eigenvalues are pure imaginary consists of chemical substrates or species whose concentrations oscillate temporally in undamped fashion. Consequently, in the context of Michaelis-Menten reaction rate kinetics, it is seen that hermitian systems by definition display widespread enzyme blocking, are exponentially damped, and thus do not display oscillatory activity. Nonhermitian systems on the other hand can be constructed which display less enzyme blocking than their hermitian counterparts and whose species' concentrations describe well defined temporal oscillation which are circadian, infradian or ultradian depending upon their respective frequencies.

Chemorhythms are therefore seen as natural and direct consequences of the generalized n x n Michaelis-Menten law of reaction rate kinetics where the preponderance of temporal oscillations is by and large governed by the richness of off diagonal enzyme couplings.

While some enzyme-species systems may in fact be nonlinear, linear rate kinetics provide an adequate representation of systems whose activity is characterized by small amplitude oscillations. The reaction-diffusion equations therefore provide a canonical representation of an endogenous mechanism which gives rise to chemo-rhythmic activity. This representation reduces to an eigenvalue format where oscillatory activity imparted by the eigenvector solutions describes well defined small amplitude temporal oscillations of species' concentrations. These chemo-oscillations can in turn be classified as circadian, ultradian or infradian depending upon the period of oscillation. In this case, the off diagonal enzyme coupling agents facilitating reactions among the n biochemical species are commonly referred to as the *zeitgebers* which impart periodicity to the system. Consequently, the first and second conditions listed above (C.1, C.2) are satisfied.

A metric quantifying metabolic dysfunction will now be proposed in addressing the third aforelisted condition (C.3). Rigorous proof of this metric's validity is not given at this time. Nevertheless, the fact that this metric may be analytically expressed as a function of $\tilde{K}, \tilde{D}, \hat{\chi}$ and any set of boundary conditions provides a framework for validation through computer simulation and clinical observation which are currently underway.

Historically, entropy (S) has been proposed (ref. 11, 12, 13) as a metric to quantify systemic organization. Disorganization in turn has been equated to systemic dysfunction which has been tentatively described as a monotonic increasing function of entropy - the greater the entropy the greater the dysfunction. As a result of Nernst's theorem however, this definition implies that metabolic systems can be made to achieve minimum entropy at $0°K$. Consequently, equating entropy to systemic dysfunction allows one to fallaciously conclude that metabolic systems in the quantum mechanical ground state are entirely free of dysfunction. It is therefore unreasonable to propose entropy as a meaningful measure of systemic dysfunction in this particular context. In attempting to steer clear of the implications of Nernst's theorem, it is more meaningful to think in terms of an entropic norm. In this case, redundancy (R), defined as $1-\frac{S}{S_{max}}$ is proposed as a candidate metric. It is noted that redundancy does not uniquely describe systemic organization. Any one of an infinite number of entropic noise norms are adequately suited. For the sake of simplicity, unnormalized redundancy defined as $\Delta S = S_{max} - S$ will be used herein to illustrate how organization varies as a function of those parameters which canonically describe metabolic systems through the reaction rate kinetics differential equations. S_{max} is defined as the maximum entropy which the system may achieve at any given time when the system is denied any external input (an imposition of the Neumann boundary conditions), and when each of its constituent biochemical species are enzymatically uncoupled from all others ($\tilde{K} = \tilde{0}$). External and internal uncoupling in this fashion allows the system to achieve stable thermodynamic equilibrium. Consequently, $\lim_{T \to 0} R = \lim_{S \to S_{max}} 1-\frac{S}{S_{max}} = 0$. Defined in this fashion, redundancy maps $\tilde{K}, \tilde{D}, \hat{X}$ and the system's boundary conditions into the real numbers from 0 to 1 where these respective values correspond to maximum and minimum dysfunction. While no interpretation of redundancy 1 is offered here, it is clear that redundancy 0 in effect represents the dead system.

The use of redundancy in synoptically describing systemic organization is not new and was first proposed by information theorists (ref. 14). Unnormalized redundancy or negative entropy of a set of events, each of which is assigned a probability of occurrence p_i where $\sum_i p_i = 1$, has historically been defined as $-\sum_i p_i \log p_i$. This metric therefore provides a measure of uncertainty in that it is unbounded when all events are equiprobable, and approaches 0 as the probability of occurrence of one event approaches unity. In the case of a metabolic system, redundancy may be expressed as a particulate noise norm which is formally identical to that of its information theoretic counterpart. Specifically, the following expression may be derived for any statistical mechanical ensemble:

$$R = 1 - \frac{\sum_{ij} p_{ij} \log p_{ij}}{\sum_i p_i \log p_i + \log L} \qquad (5)$$

p_{ij} is now defined as the number of particles of species i per unit cell j, or concentration $\chi_i(\vec{r})$ at any given location within the system. p_i is the total number of particles of species i in the entire system, given as $\sum_{j=1}^{L} p_{ij}$, or as $\int \chi_i(\vec{r}',t)d^3r'$, where L is the number of cells into which the system has been divided. Alternatively, the variables p_{ij} may be defined as quantum mechanical wave functions $\Psi_i(\vec{r},t)$ for any species. The quantum mechanical variants of the rate kinetics equations discussed above are given as follows;

$$-i\hbar\frac{d\hat{\Psi}}{dt} = \tilde{V}\hat{\Psi} + \nabla^2\hat{\Psi}' \tag{6}$$

where $\Psi_i' = -\hbar^2\Psi_i/2m_i$, and $\tilde{V}$ is the reaction matrix whose entries V_{ij} are interaction potentials created by each enzyme facilitating interactions between species i and j.

In this case, equiprobable distributions constitute spatial homogeneity in the absence of localized potential fields which would otherwise be provided by catalytic agents or enzymes. In other words, equiprobable distributions define systems which have been internally and externally uncoupled and thus occupy the zero redundancy state. Relative weighting of the Ψ_i functions, achieved through the introduction of nonzero k_{ij} values in the reaction-diffusion equations serves to increase R. Conversely, for any given set of boundary conditions, redundancy is decreased as the entries in $\tilde{K}$ approach zero. Decreasing these factors in essence suppresses chemo-oscillations by having $\hat{\chi} \rightarrow \hat{\chi}_{ss}$.

The relationship between redundancy, $\tilde{K}$ and chemo-oscillatory activity may be viewed from another perspective. Given $\tilde{D}$, $\hat{\chi}(0)$ and $d\hat{\chi}0/dt$, the system's redundancy may be maximized if $\tilde{K}$ ensures the persistence of oscillations. These oscillations will delay the occurrence of $\hat{\chi} = \hat{\chi}_{ss}$, thus postponing the occurrence of S_{max}. In this regard, maximizing redundancy may be achieved by judicious selection of $\tilde{K}$. Specifically, it is desirable to increase the magnitude of the imaginary component of $\hat{\chi}$'s hamiltonian (the argument of $\hat{\chi}$'s evolution operator) at the expense of its real component, since the imaginary component determines frequency of $\hat{\chi}$'s oscillations while the real component determines the rate at which $\hat{\chi}_i$ will exponentially approach $\hat{\chi}_{ss}$. Hermitian $\tilde{K}$ matrices therefore become undesirable in that their eigenvalues will always be real and will accelerate decay of redundancy. Conversely, the complementary set of matrices will preserve or increase redundancy. A degree of *hermiticity* may be defined by exploiting the fact that hermitian matrices have eigenvector solutions which are orthogonal. Hence an inner product norm H, may be defined as follows:

$$H = \left| \sum_{\substack{ij \\ i \neq j}}^{n} < \hat{e}_i \,|\, \hat{e}_j > /n \right| \leq 1 \tag{7}$$

$\hat{e}_i$ is a unit eigenvector and n is the dimensionality of $\tilde{K}$. Since a hermitian system ($H = 0$) will possess eigenvectors which are mutually orthogonal, it will also not produce oscillations, and should therefore act to suppress redundancy. Increased oscillatory activity should on the other hand be produced by systems where $H > 0$. Hence, the relative orientation of $\tilde{K}$'s eigenvectors may also provide a measure of redundancy. The higher the value of H, the higher the redundancy for systems which are otherwise identical. Uncoupling the eigenvectors in this fashion may also result in enzyme uncoupling or enzyme blocking in the metabolic system since hermitian matrices can be off diagonally blocked via similarity transforms. It is therefore proposed that widespread enzyme blocking which has been observed to increase pathology, will also decrease organization since blocking decreases redundancy by suppressing H. This relationship between redundancy, hermiticity and enzyme blocking is given some additional support below.

It will now be demonstrated that for every n x n hermitian matrix there exists an n x n nonhermitian matrix whose unnormalized redundancy ΔS exceeds that of its hermitian counterpart.

Define entropy (S) as

$$S = -\sum_{ij}(N_{ij}/N)\log(N_{ij}/N) \tag{8a}$$

where N_{ij} is the number of particles of species i in subcell j, and N is the total number of particles in the system.

Consequently,

$$S_{max} = \lim_{N_{ij} \rightarrow N_i/V} S \tag{8b}$$

where V is the system's volume.

Defining $\Delta S = S_{max} - S$, obtain

$$N\Delta S = N\log V + \sum_{ij} N_{ij}\log N_{ij} - \sum_{i} N_i \log N_i \tag{9a}$$

For the continuous case, $N_{ij} = \chi_i$ and $N_i = \int \chi_i dr^3$. Thus,

$$N\Delta S = \int <\hat{\chi}|\log\hat{\chi}> dr^3 - <\int \hat{\chi}dr^3> |\log\int \hat{\chi}dr^3> + N\log V \tag{9b}$$

In the case of small amplitude oscillations the following is obtained via first order Taylor series expansion without significant loss of generality:

$$N\Delta S = N\log V + \sum_{ij} N_{ij} + N_{ij}\log N_{ij}(0) - N_{ij}(0) - N_i - N_i \log N_i(0) + N_i(0) \tag{10}$$

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Recognizing that $\sum_{ij} N_{ij} = \sum_i N_i = N$ for any time, then equation 10 may be rewritten as follows:

$$N\Delta S = \sum_{ij} N_{ij} \log N_{ij}(0) - \sum_i N_i \log N_i(0) + N \log V \tag{11a}$$

or

$$N\Delta S = \int < \hat{\chi} | \log \hat{\chi}(0) > dr^3 - < \int \hat{\chi} dr^3 | \log \int \hat{\chi}(0) dr^3 > + N \log V \tag{11b}$$

Substituting equation 4 into 11b, obtain the following for the hermitian system:

$$N\Delta S = N\Delta S(0) + \sum_{i=1}^{n} \frac{1 - e^{-\lambda_i t}}{\lambda_i} \cdot \left[\int < \frac{d\hat{\chi}(0)}{dt} | \hat{e}_i >< \hat{e}_i | \log \hat{\chi}(0) > dr^3 - < \int \frac{d\hat{\chi}(0)}{dt} dr^3 | \hat{e}_i >< \hat{e}_i | \log \int \hat{\chi}(0) dr^3 > \right] \tag{12}$$

Equation 12 illustrates how the unnormalized entropic noise norm $N\Delta S$ evolves as a function of $N\Delta S(0)$ and the system's initial conditions $\hat{\chi}(0)$ and $d\hat{\chi}(0)/dt$. Equation 12 may be rewritten as follows:

$$N\Delta S = N\Delta S(0) + \sum_{i=1}^{n} \frac{1 - e^{-\lambda_i}}{\lambda_i} \cdot N\Delta dS_i(0)/dt \tag{13}$$

where $N\Delta dS_i(0)/dt$ is the i^{th} component of the initial time rate of change of the entropic noise norm.

Now consider the nonhermitian system with identical initial conditions. This system is formed by perturbing the hermitian system by introducing off diagonal K_{ij} elements which transform $\lambda_i \rightarrow \lambda_i + \delta_i$ for $\lambda_i \gg \delta_i$, and $\hat{e}_i \rightarrow \hat{e}_i + \hat{\varepsilon}_i$ for $|\hat{e}_i| \gg |\hat{\varepsilon}_i|$, where δ_i is real.

Defining the unnormalized entropic norm for the nonhermitian system as $[N\Delta S]'$ obtain,

$$[N\Delta S]' = N\Delta S(0) + \sum_{i=1}^{n} F(t) \left[\int < \frac{d\hat{\chi}(0)}{dt} | \hat{e}'_i >< \hat{e}'_i | \log \hat{\chi}(0) > dr^3 - < \int \frac{d\hat{\chi}(0)}{dt} dr^3 | \hat{e}'_i >< \hat{e}'_i | \log \hat{\chi}(0) dr^3 > \right] \tag{14a}$$

where $F(t) = \left[1 - e^{-(\lambda_i + \delta_i)t}\right] \left[(\lambda_i + \delta_i) | \hat{e}_i + \hat{\varepsilon}_i |^2\right]^{-1}$ and $\hat{e}'_i = \hat{e}_i + \hat{\varepsilon}_i$.

Equation 14a may be rewritten as follows to first order

$$[N\Delta S]' = N\Delta S(0) + \sum_{i=1}^{n} \left[\frac{1 - e^{-\lambda_i t}}{\lambda_i} - \frac{\delta_i}{\lambda_i^2}\left(1 - e^{-\lambda_i t}\right) + \frac{\delta_i t e^{-\lambda_i t}}{\lambda_i} \right] N\Delta dS_i(0)/dt \tag{14b}$$

It is noted that the term $N\Delta dS_i(0)/dt$ is identical to its corresponding term in the Hermitian case as a direct result of first order Taylor series expansion and the assumption that $< \hat{e} | \hat{\varepsilon} > / |\hat{e}| \, |\hat{\varepsilon}| \ll 1$.
Hence,

$$[N\Delta S]' - [N\Delta S] = \sum_{i=1}^{n} \left[-\frac{\delta_i}{\lambda_i^2}\left(1 - e^{-\lambda_i t}\right) + \frac{\delta_i}{\lambda_i} t e^{-\lambda_i t} \right] [N\Delta dS_i(0)/dt] \tag{15a}$$

or

$$[N\Delta S]' - [N\Delta S] = \sum_{i=1}^{n} \frac{\delta_i}{\lambda_i^2}\left(e^{-\lambda_i t} - 1 + \lambda_i t e^{-\lambda_i t}\right) [N\Delta dS_i(0)/dt)] \tag{15b}$$

Since $\left[e^{-\lambda_i t} - 1 + \lambda_i t e^{-\lambda_i t}\right] \lambda_i^{-1} \geq 0$ and $\lambda_i > 0$, then for any $[N\Delta dS_i(0)/dt]$, a value of δ_i may be selected (either greater or less than zero) so that $[N\Delta S]' - [N\Delta S] > 0$. Hence for any given n x n hermitian system, there exists an n x n nonhermitian system with identical initial conditions where the latter evolves in more organized fashion than the former *at every point in time.*

Equation 15 b also implies that there exists a nonhermitian system which evolves at lower levels of organization. Equation 6 however implies that off diagonal entries serve to decrease the system's number of physical degrees of freedom and thus increase systemic organization (ΔS). Consequently, it is hypothesized that while the condition $[N\Delta S]' - [N\Delta S] < 0$ is permitted mathematically, it is not physically permissible.

IV. Conclusions.

Cognitive function is deeply rooted in metabolic activity, and metabolic activity in turn is routinely and continuously influenced by nutritional input. Specifically, metabolically compatible nutrition plays a major role in shaping cognitive, affective, experiential and behavioral activity. At the present time venous plasma pH appears to be an excellent synoptic indicator of intermediary metabolic function. Deviations in pH beyond upper and lower limits will with few exceptions precipitate the onset of disorders which are erroneously believed to be psychogenic and which are therefore attributed to sociogenic factors. Seen in this context, mainstream research in the field of nutrition, medicine and psychology has unwittingly focused upon nutritional input which could be called *pH neutral.* As this designation suggests, the impact of this type of nutritional input upon intermediary metabolic activity is nominal. It is not surprising therefore to see that this input will usually have little if any effect upon behavior, and will (if inadvertently mismatched) backfire by precipitating acute psychophysiological dysfunction. Absent an understanding of metabolic inhomgeneity, researchers will continue to be at a loss to understand these diverse and apparently paradoxical responses. In an attempt to explain this diversity, clinical psychologists will likely continue to attribute it to social factors.

Since patterns of circadian rhythms seem to correlate with psychometabolic activity, a general model has been proposed whereby chemo-oscillations are formally accounted for in terms of first principles of reaction rate kinetics. This model is potentially capable of resolving one aspect of the artificial mind/body dichotomy which has and will continue to produce gross misunderstandings in both academic and popular circles. The proposed theory of psychometabolic function is utilized in an attempt to relate metabolic activity to cognitive activity. A more extensive metabolic mapping of the human organism will be required as part of an effort to understand how the organism must be metabolically reorganized (either nutritionally or otherwise) to cause it to *predictably* transition between levels of cognitive function, affect and behavior.

Perhaps an overriding conclusion relates to the fact that many if not most disorders which have historically been viewed as sociogenic are far less connected to social dynamics than had been anticipated. Evidently, the human organism contains biological mechanisms which strongly influence its behavior in a fashion which is far less dependent upon social factors than has been historically suspected. This statement has considerable support in genetics research dealing with identical twins reared apart at birth versus fraternal twins reared together (ref. 15). This research suggests that identical twins reared apart have far more behaviorally and attitudinally in common than fraternal twins reared together. Indeed, from a metabolic perspective it may be argued that behavioral differences between identical twins reared apart may to a significant extent be the result of different nutritional regimens.

In conclusion, some behaviors are learned whereas others are not. While one might learn to play the piano, tap dance or solve a differential equation, it is highly likely that behavioral disorders such as schizophrenia, chronic depression, free floating anxiety among a host of others which are routinely labeled as *psychogenic*, *psychosomatic* or *stress-related*, have less to do with social/familial dynamics than with endogenous neurometabolic activity. The ramifications of this observation inescapably force one to suspect the extent to which behavior and affect in general are neurochemically driven versus being informationally driven. It is indeed plausible to conclude that the modality within which the human organism will interact with its informational environment is, to a very large extent, driven by the organism's *chemoneural wiring*. Simply stated, *well organized individuals possessing high levels of metabolic redundancy will on average interact in a far more adaptive fashion within the same informational environment than their more poorly organized counterparts.*

The human organism is a superposition of quantum mechanical eigenstates, and is to a far lesser extent a set of stimulus-response reflex arcs. In effect, humans are probably far more closely related to *Schrodinger's Cat* than to *Pavlov's Dog*. In this context, a solution to the nature versus nurture problem may finally be in sight.

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