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Why Metabolic Systems Are Rarely Chaotic

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Abstract

One of the mysteries surrounding the phenomenon of chaos is that it can rarely be found in biological systems. This has led to many discussions of the possible presence and interpretation of chaos in biological signals. It has caused empirical biologists to be very sceptical of models that have chaotic properties or even employ chaos for problem solving tasks. In this paper, it is demonstrated that there exists a possible mechanism that is part of the catalytical reaction mechanisms which may be responsible for controlling enzymatic reactions such that they do not become chaotic. It is proposed that where these mechanisms are not present or not effective, chaos may still occur in biological systems.

Key words: Control of Chaos, Rate Control, Biological Complexity, Chemical Chaos

1. Introduction

In biology, it can be argued that virtually all chemical reaction steps are under some form of enzymatic control. This can be analysed to determine the rate of production of the different metabolites and then modelled using traditional Michaelis-Menten type kinetics [1]. More recently, the total flux of a chemical pathway can be analysed using control analysis which allows the relative efficiencies of each intermediate step to be quantified [2]. Even though these pathways are of tremendous complexity, they appear to be stable [3]. Chaos occurs in chemical (and physical) processes regularly and it seems that biochemical processes can either prevent it from occurring or have evolved to avoid chaotic domains.

Because there appears to be little indication for chaotic behaviour to be a particularly bad (or possibly good) survival trait, it is assumed that the biochemical catalytical process itself avoids chaotic domains by virtue of its specific organisation. Enzymatic processes control the rate of production of their substrates and it is suggested in this paper that this is a built-in safeguard that limits the system to stable dynamics.

It is assumed that the Michaelis-Menton model is sufficiently accurate to describe the rate of reaction which depends non-linearly on the substrate concentrations. The Michaelis-Menton model exhibits rate saturation at higher concentrations of the substrates and it is this concept that will be used to describe a rate control method for chaotic control.

$$v = \frac{SV_m}{S + K_m}$$ (1)

The Michaelis-Menton equation (1) describes a rate curve $v$ where $S$ is the substrate concentration and $K_m$ the Michaelis constant with $V_m$ the limiting rate. For this equation, it is assumed that enzyme-substrate reactions, as part of the catalytical process, are so rapid that the process is at virtual equilibrium. This effectively means that the breakdown of the enzyme-substrate complex into the product is
the rate limiting step of the catalysis [2]. Although this is only a conceptual model of enzyme activity because it assumes that the rates are close to equilibrium, it seems to be adequate for most reaction rate analysis problems. The steady state assumption implied by this can only be true if indeed most of these problems are near steady-state or in a state which is indistinguishable from a steady state. This may seem to be an arbitrary distinction but its relevance can be more readily understood if it is considered that a system in steady-state and a controlled chaotic system are dynamically similar. A chaotic system that is under control of some effective control method, shows the same dynamical properties as a stable system because the control method forces the system to revert to more classical dynamics. Only careful analysis, and interference with the control method, will show the difference. This property of chaotic control is one of the reasons why chaotic control has been studied in great depth and has been used to control different systems [4].

The aim of controlling chaotic dynamic systems is, generally, to stabilise specific points or orbits within the phase space. The chaotic nature of a system can thus be reduced to stable states. Different methods have been developed that are either variations on the OGY system of control [5,6] or delay control [7,8,9]. The OGY control methods require knowledge of the unstable periodic orbits (UPOs) contained in the attractor. Therefore, an analytical understanding of the chaotic system is necessary to control the system. The delay control method uses the control function \( F(y) = K(y(t) - y(t - \tau)) \) which does not require any knowledge of the UPOs but it needs appropriate choices for the control constant \( K \) and the delay \( \tau \). If the choices for \( K \) and \( \tau \) are not correctly chosen then the system will not stabilise into an orbit. Note that some chaotic systems can not be stabilised using the single delay control method such as the Lorenz system [10] due to the fact that these contain negative Floquet exponents but these systems can be controlled by an extended delayed feedback control [11,9].

To study the possibility that metabolic processes contain a mechanism to prevent chaotic states from developing, two different biochemical kinetic models will be shown to be readily controlled using the novel rate control method described in this paper. Additionally, the principles of the rate control method of chaotic control are outlined using two classic chaotic models.

2. Rate Control

In this section, a novel method of chaotic control is presented that does not depend on a priori knowledge of the presence of unstable periodic orbits in a chaotic system. Additionally, it requires only the current state of each of the variables by limiting the relative rate of expansion of each of those variables. An extensive analysis of this method (including an analysis of the method in terms of the Lyapunov exponents) is in preparation [12]. This will also demonstrate how the rate control method can overcome the limitations of the single delay control method for specific chaotic systems, such as the Lorenz system.

Consider the form of \( n \) linear rate equations that govern a particular dynamic system:

\[
\begin{align*}
\dot{x}_1 &= F_1(x_1) + G_1(x_2) \\
\dot{x}_2 &= F_2(x_2) + G_2(x_2) \\
&\quad \vdots \\
\dot{x}_n &= F_n(x_n) + G_n(x_n)
\end{align*}
\]

In this case, the system depends on one of the functions \( F \) or \( G \) to grow or decrease at some rate. Additionally, either function may be non-linear and may depend on more than one variable. If one considers the nature of a chaotic flow equation, in the range where stretching and folding occurs, i.e. where the local Lyapunov exponents tend to have at least one positive value, the global behaviour is dominated by some of the \( n \) equations. These tend to be the non-linear parts of the system that allow the system to expand at an exponential rate. The local rate of expansion is proportional to the local behaviour of each of the variables. If a chaotic system is near an unstable periodic orbit, it can be kept near or on this orbit by small proportional adjustments to one or more of the variables. This is the principle of the OGY method of control. In rate control, the exponential expansion of the system away from the orbit is used to limit the rate of expansion, thereby preventing the chaotic system from leaving the orbit. Note that the rate control method does not necessarily target specific orbits.

To achieve a rate control of a chaotic system, the rate of expansion can be estimated from the growth terms in each equation of the system. By determining the current proportion of the variable of the space it occupies, a measure for each variable can be found, e.g. for the Rössler system,
The rate function may be the Rössler system \[13\] was modified to include the rate control function \[6\]. The rate function for the divergence of \(x\) and \(z\) is a constant and \(\mu\) and \(\xi\) are variable but by default \(f = 1, \xi = -1\) and \(\mu = 150\). With any appropriately chosen value for \(\mu\), the parameter \(\xi\) may be varied resulting in different unstable periodic orbits to become stabilised. In figure 1 the evolution of the Rössler \(z\) variable is shown when \(\xi = -1.5\) and with the rate control enabled at timestep 50. The system rapidly becomes stabilised into a stable one period. The rate control function \(\sigma\) is shown in figure 2 and is constant at 1 until the \(z\) variable increases rapidly, it then decreases in proportion to the change in \(z\) and thereby “slows” down the \(z\) variable. This results in the system becoming stable periodic. By changing the rate parameter \(\xi\), it is possible (whilst all other parameters and initial conditions are the same) to stabilise different unstable periodic orbits. In figure 3 are shown three orbits that can be stabilised when the values for \(\xi\) are \(-1.5, -1.0\) and \(-0.4\). From right to left can be seen the stable period 1 orbit for \(\xi = -1.5\), two orbits of the stable period 4 orbit for \(\xi = -0.4\), the period 1 orbit when \(\xi = -1.0\) and the other two orbits of the period 4 for \(\xi = -0.4\). It can be recognised from these two figures that the shape of the Rössler attractor is stretched in the \(x\) and \(y\) plane when \(\xi < 1.0\). Otherwise, it has a shape similar to the chaotic attractor, including stretching and folding when \(x > 0\) and \(y > 0\).

### 3. Application of rate control to chaotic systems

#### 3.1. Rössler system

To demonstrate the rate control on a flow system, the Rössler system \[13\] was modified to include the rate control function \[6\]. The rate function may be applied to all the three Rössler variables but it is sufficient to apply the rate control to the third \(z\) variable alone. The resulting modified rate controlled Rössler system is then as follows:

\[
\begin{align*}
\frac{dx}{dt} &= -(y + z) \\
\frac{dy}{dt} &= x + \frac{y}{\alpha} \\
\frac{dz}{dt} &= \beta \left(\frac{z}{\alpha} + (z)x\right) - (\gamma z)
\end{align*}
\]

The growth term for equation (4) is \((x, z)\) (and an additional constant term). The proportion of each of these two variables to the growth rate is given by

\[
q_x = \frac{x}{x + \mu_x} \quad \text{and} \quad q_z = \frac{z}{z + \mu_z}
\]

where \(\mu_x\) and \(\mu_z\) are constant. For this non-linear equation, the divergence of the two variable \(x\) and \(z\) determines the increase in rate of growth of the \(z\) variable itself. Therefore, by limiting the \((x, z)\) term in proportion to the divergence rate, the system should be stabilised. To express this in more general terms, the following equation describes a generic rate control function for the divergence of \(x\) and \(z\):

\[
\sigma(x, z) = f e^{\xi q_x q_z} = f e^{\left\{\frac{\xi(x, z)}{(x, z) + (z)x + (z)z + \mu}\right\}}
\]

where \(\sigma(x, z)\) is a constant and \(\xi\) and \(f\) are variable scalars that can be used to stabilise different orbits. This rate control function can be used in many different chaotic systems of which two examples are presented below before the rate control is applied to biochemical models. The rate control function is capable of both limiting the rate of expansion of a variable as well as promoting the rate of expansion which can also lead to the stabilisation of an unstable periodic orbit depending on the local dynamics.

#### 3.2. Ikeda map

To demonstrate that the rate control method applies to chaotic maps as well as flows, the rate control is introduced in the Ikeda map. The Ikeda map is a good example of a complex chaotic map that contains several points of nonlinearity \[14\]. By plotting the real and imaginary parts of the complex equation

\[
z(n + 1) = a - b z_n e^{i\phi - \frac{i\phi}{1 + |z_n|^2}}
\]

3
with $x = Re(z)$ and $y = Im(z)$, the Ikeda map can be described as

$$
\phi = \kappa - \frac{\eta}{(1 + x_n^2 + y_n^2)} \quad (12)
$$

$$
x_{n+1} = a + b(x_n \cos \phi - y_n \sin \phi) \quad (13)
$$

$$
y_{n+1} = b(x_n \sin \phi + y_n \cos \phi) \quad (14)
$$

with $a = 1, b = 0.9, \kappa = 0.4$ and $\eta = 6.0$. The rate control equation (6) is applied to both $x_n$ and $y_n$ by limiting the function $\phi$ (12). The function $\phi$ therefore becomes

$$
\phi' = \kappa - \frac{\eta}{(1 + \sigma(x_n, y_n)x_n^2 + \sigma(x_n, y_n)y_n^2)} \quad (15)
$$

where the rate control parameters are chosen to be $\mu = 5$ and $\xi$ is variable.

Some resulting unstable periodic orbits are shown in figure 4 where three different orbits, two period three orbits and a period six orbit, are indicated on top of the uncontrolled Ikeda map. Orbits are stabilised depending on the proximity of the system to the orbit when control is enabled. The control is initially disabled and becomes enabled after 5000 iterative time steps. The resulting evolution of the $x$ variable of the Ikeda map for $\xi = 2$ is shown in figure 5 where the map will very quickly stabilise into the three orbit when control is enabled.

The rate control function $\sigma$ that is applied to the map is shown in figure 6, where the control function is equal to one when rate control is disabled and becomes periodic when control is enabled and stabilised. Note that $\sigma$ is close to one for two of the unstable points, which implies that the local dynamics is small (i.e. one of the local Lyapunov exponents is small positive), not much rate control is needed to prevent the system from leaving the orbit. The third unstable point requires a relative large limitation of the expansion rate for $\sigma = 0.4$.

4. Control of a chemical oscillator

The relevance of rate control of chaos to the biochemical processes can be illustrated by introducing rate control to the growth terms of a known multi-variable chemical model. No assumptions on the properties of the control parameters are initially made but different values for the control parameters $\xi$ and $f$, see equation (6), have been tested. The model described below is a three variable model that
Figure 4. Ikeda attractor with three different unstable periodic orbits, $\xi = 2$ (3 orbit, circles), $\xi = 2.25$ (3 orbit, squares) and $\xi = 1.75$ (six orbit, diamonds).

Figure 5. Evolution of controlled $x$ variable of the Ikeda map, control is turned on at 5000.

Figure 6. Rate control variable $\sigma$ of the Ikeda map, with control enabled at time step 5000.

is derived from a two-variable autocatalator system [15]. It has six reaction steps with rates given by law-of-mass-action kinetics:

- $P \rightarrow A$ \hspace{1cm} Rate= $k_0 p_0$
- $P+C \rightarrow A+C$ \hspace{1cm} Rate= $k_c p_0 \gamma$
- $A \rightarrow B$ \hspace{1cm} Rate= $k_u \alpha$
- $A+2B \rightarrow 3B$ \hspace{1cm} Rate= $k_1 \alpha \beta^2$
- $B \rightarrow C$ \hspace{1cm} Rate= $k_2 \beta$
- $C \rightarrow D$ \hspace{1cm} Rate= $k_3 \gamma$

This system is considered to be open with a constant precursor $p_0$. In a dimensionless form the last three relevant rate equations can be written as:

$$\frac{da}{dt} = u(k+c) - a - a b^2$$

$$\frac{db}{dt} = a + a b^2 - b$$

$$\frac{dc}{dt} = b - c$$

where

$$a = \sqrt{\frac{k_1 k_u}{k_2^2} \alpha}, \hspace{1cm} b = \sqrt{\frac{k_1}{k_u} \beta}, \hspace{1cm} c = \sqrt{\frac{k_1 k_2^2}{k_u k_2^2} \gamma}$$

are dimensionless concentrations and $t = k_u \tau$ is dimensionless time. The parameters $d = 0.02$, $k = 65$ and $s = 0.005$ are dimensionless and derived from the original rate equations [15]. The parameter $u$ shows asymptotic behaviour when varied through a supercritical Hopf bifurcation at $u = 0.016$, followed by a period doubling cascade that begins at $u = 0.143$. For $0.1534 < u < 0.1551$, the system is chaotic [15]. For the subsequent simulations, the value of $u = 0.154$, well inside the chaotic range.

To introduce the rate control method, the proportional quantities $q_a, q_b, q_c$ are determined in equations (20), (21) and (22). For variable $a$, the rate of growth is given by the term $u(k+c)$, for variable $b$ the rate of growth is given by the term $(a + a b^2)$ and for variable $c$ the rate of growth is given by the term $b$. Apart from equation (17), the rate of growth is given by a single variable. It can therefore be expected that the rate of growth of equation (17) may diverged faster than the other two. The complete rate control of these equations can now be formulated, including the rate equations $\sigma_n$, as follows.
\[ q_a = \frac{a}{a + \mu_a} \]  
\[ q_b = \frac{b}{b + \mu_b} \]  
\[ q_c = \frac{c}{c + \mu_c} \]  
\[ \sigma_a(q_c) = f_a e^{\xi_a q_c} \]  
\[ \sigma_b(q_a, q_b) = f_b e^{\xi_b q_a q_b} \]  
\[ \sigma_c(q_b) = f_c e^{\xi_c q_b} \]  
\[ \frac{d a}{d t} = \sigma_a(u(k + c)) - a - a b^2 \]  
\[ s \frac{d b}{d t} = \sigma_b(a + a b^2) - b \]  
\[ d \frac{d c}{d t} = \sigma_c b - c \]  

Note that the rate control functions \( \sigma_n \) will be equal to 1 when control is disabled. When control is active, it will vary around the value 1 depending on the rate of expansion. For this model, \( \mu_a = 1 \), \( \mu_b = 150 \), \( \mu_c = 30 \) and the rate control parameters \( \xi_n \) and \( f_n \) may be varied. In figure 7 is shown the chaotic attractor of the model when control is disabled (i.e. \( \xi_n = 0 \)). In figure 8 is shown the effect of control when \( \xi_n = -1 \) for the variable \( b \) of the chaotic chemical model. At (non-discrete) time 9 is the control enabled and the system rapidly stabilises into a period one orbit. The corresponding control function \( \sigma_b \) is shown in figure 9. Notice that the rate control varies between 0.92 and 1, i.e. the rate limiting function only reduces the rate of expansion with a factor of < 0.08. This is nevertheless sufficient to stabilise the chaotic system. The values of the rate parameters \( \xi_a, \xi_b \) and \( \xi_c \) can be varied and may result in the stabilisation of different unstable period orbits.

5. Control of an autocatalytic system

To demonstrate the dynamic effect of the rate control mechanism on an different autocatalytic system, the control method is introduced into a model of extracellular matrix degradation balance [16]. This model, illustrated in figure 10, describes the cyclical generation and degradation of extracellular matrix proteins including the autocatalytic degradation of the enzymes that drive this cycle. Protein-cleaving enzymes, proteases \( p \), degrade the unsoluble extracellular matrix \( m \) into soluble fragments \( f \). These fragments \( f \) are converted back into unsoluble matrix by an enzyme transglutaminase \( g \) (and other enzymes not included in this model). The proteases \( p \) and transglutaminase \( g \) are therefore antagonistic.
in building and degenerating the matrix. The matrix \( m \) is created *de novo* at a constant rate \( r_{im} \); the presence of the fragments \( f \) will stimulate the production of both enzymes \( p \) and \( g \) by surrounding cells. These cells detect the presence of the intercellular fragments which will stimulate the production of \( p \) and \( g \). All proteins are subject to degradation by the proteases \( p \), including \( p \) itself. The model can be described by the dimensionless equations (29), (30), (31) and (32).

\[
\frac{dm}{dt} = k_p \frac{fg}{K_p + f} - \frac{mp}{1 + m} + r_{im} \quad (29)
\]
\[
\frac{df}{dt} = -k_p \frac{fg}{K_p + f} + \frac{mp}{1 + m} - \frac{fp}{1 + f} \quad (30)
\]
\[
\frac{dp}{dt} = \alpha \frac{f^n}{K_p^n + f^n} - k_a p^2 \quad (31)
\]
\[
\frac{dg}{dt} = \beta \frac{f^l}{K_g^l + f^l} - k_{deg} \frac{gp}{K_{deg} + g} \quad (32)
\]

Here the parameters \( n \) and \( l \) in equations (31) and (32) are the Hill-numbers for the two enzymes where \( n = l = 4 \). \( \alpha \) and \( \beta \) are the proteases and transglutaminase synthesis rate respectively and are assumed to be much smaller than the turnover rates for the matrix \( m \) and fragments \( f \). The model parameters are derived from experimental data and for the following experiments the parameters were set as \( \alpha = 0.026, \beta = 0.00075, K_G = 0.1, K_{deg} = 1.1, k_p = k_{deg} = 0.05 \) and \( k_a = \frac{k_{deg}}{K_{deg}} = 0.0455 \). For different values of \( r_{im} \) the model exhibits a wide range of dynamic behaviour, including periodic cycles, bistability and chaos. For the rate control experiments, the values of \( r_{im} \) used were \( r_{im} = 0.0098 \) for chaotic dynamics, \( r_{im} = 0.00990 \) for bistability and \( r_{im} = 0.00995 \) for a period-6 cycle [16].

The rate control equation (6) can be introduced separately in each or all of the equations of the autocatalytic model. If it is introduced into equation (29) or (30), the rate control will successfully stabilise the system into a periodic orbit. More interesting consequences of the rate control mechanism can be demonstrated by introducing the rate control equation in either the synthesis of the proteases \( p \) (31) or the synthesis of the transglutaminase \( g \) (32) or both. The modifications necessary to include rate control in these equations are shown in equations (33) to (37).

Because both the synthesis of \( p \) and \( g \) depend on the presence of the protein fragments \( f \), the control is proportional to the rate of expansion of the phase space in the direction of \( f \) only. This is sufficient to control the system into a stable 4-orbit which is shown superimposed in figure 11 and in figure 12.
where is plotted $f$ versus time. Parameter values for the control equations are $f_p = 0.5, f_g = 0.5, \xi_p = 0.01, \xi_g = -0.01, \mu_f = 1$. In figure 13 is shown the values of the control equations $\sigma_p$ (34) and $\sigma_g$ (35) that stabilise the autocatalytic model into the 4-orbit. Very small changes to the rate of expansion of the enzyme synthesis rates is sufficient to prevent the model from becoming chaotic. This demonstrates that the periodic stabilisation of the extracellular matrix degradation balance can be controlled effectively and efficiently by the surrounding cells by monitoring the presence of the protein fragments and adjusting the rate of synthesis of the two enzymes. The rate control method ensures that the surrounding cells need not supervise all the processes outside the cell but can control these by monitoring only one aspect of the degradation balance. Even if the model parameters change due to a change in the balance, the rate control method can prevent the recurrence of chaotic dynamics.

For example, if other surrounding cells increase the rate of de novo generation of insoluble extracellular matrix proteins, which could lead to a bifurcation from chaos to bistability (or periodicity), the same control method can maintain the periodic dynamics at the cost of a change in period. This also requires a very small increase in the control parameters to maintain effective control $\xi_p = 0.02, \xi_g = -0.02$. By increasing the rate of synthesis of the extracellular matrix $r_{im}$ from 0.0098 to 0.00995, the model changes dynamics from chaos to a period 6-orbit. This is shown in figure 14 where the extracellular matrix production rate is increased at the dashed line, switching the dynamics of the model from a stabilised unstable periodic orbit to a different periodic orbit. If the model is uncontrolled, the system would cycle in a 6-period as is shown in figure 15. In this figure, the uncontrolled behaviour...
Figure 14. Controlled periodic orbits of the autocatalytic model, where the model switches from controlled chaotic to periodic dynamics at the dashed line.

Figure 15. Phase space of the autocatalytic model showing the uncontrolled 6-orbit (thin line) and the controlled orbit (thick line).

of the model is shown with $r_{lm} = 0.00995$ (thin line) and the controlled dynamics with the same parameters is shown superimposed (thick line). This demonstrates clearly that the control modifies the dynamic behaviour of the model to remain periodic which does not dependent on the uncontrolled system being either chaotic or periodic. This implies that extracellular processes can be readily controlled by the surrounding cells without the need for complex feedback or other control mechanisms.

6. Conclusion

The rate control of chaotic systems is a novel and effective method to stabilise unstable periodic orbits contained in chaotic systems. It does not require a priori knowledge of the UPOs contained in the system, however, it requires access to some of the system variables and the rate of change of some, or all, of those variables. Finding and stabilising different UPOs can easily be achieved experimentally. The mechanism is only active (in the sense that the control function $\sigma$ is significantly different from one) when the variables in the rate control equation (6) are changing rapidly. This means that when the chaotic system is not near a folding or stretching manifold the control is inactive. The proportional change of the variables and therefore the local shape of the attractor are the only limited elements of rate control. It may be possible to include a scalar to determine the relative contribution of the rate limiting function to the chaotic system. This may be used to direct a chaotic system towards any unstable orbit which can then be stabilised using other methods, e.g., external periodic input or delay control.

The described method of rate limiting control can be argued to be similar to other rate controlling mechanisms such as enzymatic control of biochemical processes. In such systems the flux of the product is determined by the relative control of each of the enzymatic controlled steps [2]. Such a mechanism would prevent the occurrence of chaotic dynamics by preventing the system to become chaotic. A bifurcation doubling cascade, for example, would lead to a stable (controlled unstable periodic orbit) period until the parameter has been changed back into the stable domain.

The control of the autocatalytic model shows that the rate control mechanism is very effective at controlling the dynamics of an extracellular reaction balance without the need to control each variable. It monitors the rate of expansion of the protein fragments which then influences the rate of synthesis of the enzymes that drive the system. Only small modifications are required to achieve a high level of control and will remain in stable periodic dynamics even if external parameters are modified (albeit with different periods).

Modelling the rate control of chaos on a chemical chaotic oscillator does not yet prove that the mechanism for rate control of chaos and the enzymatic rate control function perform a similar task of prevent-
ing the occurrence of chaos. The two mechanisms of the reaction rate control are not directly related but both show similar properties. More extensive modelling of rate control of different chemical oscillators and better simulation of the enzymatic reaction steps may provide some ideas.

7. Future work

Attempts to control some different chaotic systems have been successful (the Duffing system, Henon map and Hindmarsh-Rose system) but it appears that some variables in a system are more effective at controlling the chaotic dynamics than others. This is more obvious in simple system, such as Duffing’s model and the Henon map, but in more complex dynamic systems this is not readily evident. A systematic analysis of the relative contributions of rate control of different system variables may be required to determine if this effect is due to the control method or to properties of the chaotic system itself. The strength of the control parameters provides a useful indication of the efficacy of the method for a given set of equations.

If the rate control of different system variables indeed result in variable control of the chaotic system this may give some clue to identifying which variables are more effective rate limiting than others as can be seen in the rate control of the autocatalytic model. This can give an indication of the relevance of a particular enzyme or metabolite for the overall process. It may also be the case that the total control of the chaotic system is distributed over all rate variables in different proportions. This mechanism could have evolved readily in biological systems and would appear to be a simple way to eliminate undesirable chaos. In those pathways or mechanisms where rate control is not applicable, chaotic states may still be found. For example, even though there are rate control mechanisms limiting the ionic flow through membrane channels, these may not be able to control the transmembrane potential as previously has been assumed. It has already been shown that in pathological cases, neural activity can show chaotic activity [17]. It has been suggested, by different authors [18,19,20,21,22] that chaos is a possible means of information processing. Understanding the rate control methods of normal pathways may help in determining if this hypothesis is viable.

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