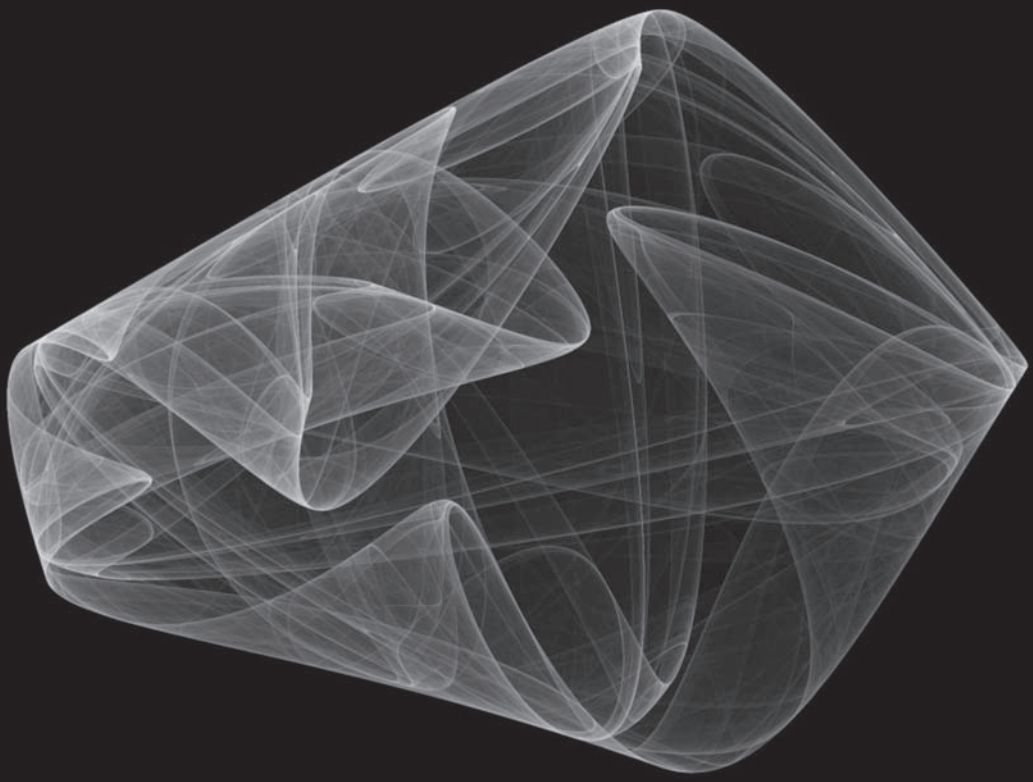
Models and Applications of Chaos Theory in Modern Sciences

Elhadj Zeraoulia



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Preface

This unique book is a compilation of selected papers from the past decade presenting a perspective underlying the thematics and strategies related to the chaos theory and its applications in modern sciences, in particular, physical sciences and also human behavior, both individual and social (including the latest investigations in chaos theory and its interrelated problems in diverse theoretical and practical disciplines, incorporating the main engineering applications.) This collection of selected papers provide a comprehensive view on some models and applications of chaos theory in medicine, biology, ecology, economy, electronics, mechanical and human sciences…etc. The papers, written by many of the leading experts in the field, cover both the experimental and theoretical aspects of the subject. This volume presents a variety of fascinating topics of current interest and problems arising in the study of both discrete and continuous time chaotic dynamical systems modeling the several phenomena in nature and society. Exciting techniques stemming from the area of nonlinear dynamical systems theory are currently being developed to meet these challenges.

Models and Applications of Chaos Theory in Modern Sciences is devoted to setting an agenda for future research in this exciting and challenging field.

I would like to thank everyone who help me in writing this book.

Readership: Advanced undergraduates and graduate students in natural and human sciences and engineering such as physics, chemistry, biology or bioinformatics…etc; academics and practitioners in nonlinear physics and in various other areas of potential application; researchers, instructors, mathematicians, nonlinear scientists and electronic engineers interested in chaos, nonlinear dynamics and dynamical systems and all interested in nonlinear sciences.

Dr. Elhadj Zeraoulia
April, 2011

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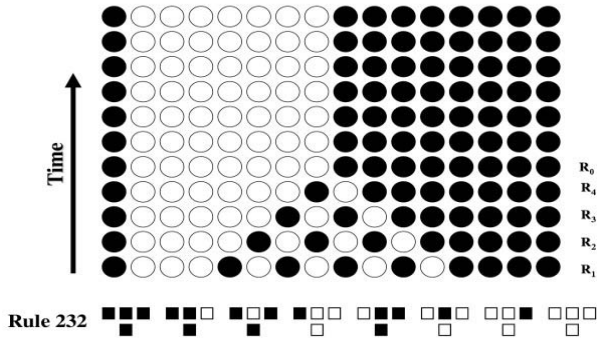
Models and Applications of Chaos Theory in Medicine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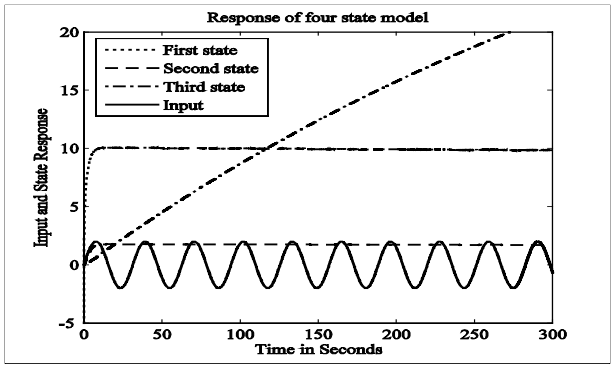
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Chapter 1

A Chaotic View of Behavior Change: A Quantum Leap for Health Promotion†

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ABSTRACT

Background: The study of health behavior change, including nutrition and physical activity behaviors, has been rooted in a cognitive-rational paradigm. Change is conceptualized as a linear, deterministic process where individuals weigh pros and cons, and at the point at which the benefits outweigh the cost change occurs. Consistent with this paradigm, the associated statistical models have almost exclusively assumed a linear relationship between psychosocial predictors and behavior. Such a perspective however, fails to account for non-linear, quantum influences on human thought and action. Consider why after years of false starts and failed attempts, a person succeeds at increasing their physical activity, eating healthier or losing weight. Or, why after years of success a person relapses. This paper discusses a competing view of health behavior change that was presented at the 2006 annual ISBNPA meeting in Boston.

Discussion: Rather than viewing behavior change from a linear perspective it can be viewed as a quantum event that can be understood through the lens of Chaos Theory and Complex Dynamic Systems. Key principles of Chaos Theory and Complex Dynamic Systems relevant to understanding health behavior change include: 1) Chaotic systems can be mathematically modeled but are nearly impossible to predict; 2) Chaotic systems are sensitive to initial conditions; 3) Complex Systems involve multiple component parts that interact in a nonlinear fashion; and 4) The results of Complex Systems are often greater than the sum of their parts. Accordingly, small changes in knowledge, attitude, efficacy, etc. may dramatically alter motivation and behavioral outcomes. And the interaction of such variables can yield almost infinite potential patterns of

†Reused with permission from: Ken Resnicow, Roger Vaughan, A chaotic view of behavior change: a quantum leap for health promotion, *International Journal of Behavioral Nutrition and Physical Activity* 2006, 3: 25. http://www.ijbnpa.org/content/3/1/25.

motivation and behavior change. In the linear paradigm unaccounted for variance is generally relegated to the catch all "error" term, when in fact such "error" may represent the chaotic component of the process. The linear and chaotic paradigms are however, not mutually exclusive, as behavior change may include both chaotic and cognitive processes. Studies of addiction suggest that many decisions to change are quantum rather than planned events; motivation arrives as opposed to being planned. Moreover, changes made through quantum processes appear more enduring than those that involve more rational, planned processes. How such processes may apply to nutrition and physical activity behavior and related interventions merits examination.

Background

"What we call **chaos** is just patterns we haven't recognized. What we call random is just patterns we can't decipher. What we can't understand we call nonsense. What we can't read we call gibberish" Chuck Palahniuk.

The study of health behavior change, including nutrition and physical activity behaviors, has historically been rooted in a cognitive-rational paradigm. Extant models, such as Social Cognitive Theory, the Health Belief Model, the Theory of Planned Behavior, the Transtheoretical Model and others, have generally viewed change as an interaction of cognitive factors such as knowledge, attitude, belief, efficacy and intention [1,2]. Change is conceptualized as a linear, deterministic process where individuals weigh the pros and cons, and at the point at which the benefits outweigh the cost, "decisional balance" tips them toward change. An implicit assumption within this perspective is that change is a gradual process under conscious control. Consistent with this framework, the associated statistical models have almost exclusively assumed a linear relationship between psychosocial predictors and behavior (change); i.e., greater increases in knowledge, attitudes and intentions will lead to greater change in behavior.

However, the theoretical and statistical assumptions underlying this linear paradigm may be seriously flawed. In particular, such a perspective fails to account for nonlinear, quantum influences on human thought and action. The limitations of a rational-linear conceptualization of behavior change may in part (in addition to measurement error) explain the modest proportion of behavioral variance accounted for by such models; which typically has been in the range of around 10%–20% and rarely higher than 50% [3–11]. The fact that the majority of studies have employed cross-sectional designs and relied on self-report to measure behavior further suggests that the true variance accounted for by linear models may be even lower [12]. Below we provide an alternative model of health behavior change based on non-linear dynamics.

Discussion

An alternative view is that decisions to initiate (and possibly maintain) behavior change are quantum rather than linear events [13]. Such quantum leaps result from a surge of motivation or inspiration that is greater than the sum of its cognitive parts. It is not so much a planned decision, but something that arrives beyond cognition. The more

dramatic form of quantum change is described by Miller [14]. "Buried in the statement 'I just decided', however can be another kind of experience that has been confused with ordinary decision making. It is the insightful type of quantum change. When people talk about such experiences in shorthand, they may say 'it just happened' or 'I just decided'. Inquire a little more closely, however, and it becomes apparent that the process is somewhat more complex." (page 37)

Miller delineates two types of quantum change, sudden insights and mystical epiphanies. Both kinds leave an indelible impact and often lead to lasting and pervasive change. Both usually involve a significant alteration in how the person perceives him/her self, others and the world. Although the cases described in Miller's book tend to involve an overwhelming transformation, less dramatic, less mystical "mini-epiphanies" may contribute to many behavior change decisions. From this perspective, behavior change can be understood through the lens of Chaos Theory and Complex Dynamic Systems. Four key principles from these theories relevant to understanding health behavior change are:

1. Chaotic systems can be mathematically modeled, usually in non-linear terms, but are nearly impossible to predict;
2. Chaotic systems are sensitive to initial conditions;
3. Complex Systems involve multiple component parts that interact in a nonlinear fashion; and
4. The results of Complex Systems are often greater than the sum of their parts.

Examples of chaotic systems include the weather, war, love, population growth, many epidemics and stock market prices. Chaos Theory has been used to explain psychologic health as well as specific health behaviors such as smoking and physical activity [15–17].

One of the first published works on Chaos Theory came from a meteorologist named Edward Lorenz. In the 1960's he was developing computer models of weather prediction. One day after running a predictive equation he decided to run the model a second time. But to save time he started the calculation in the middle of the sequence, plugging in manually some key numbers. But the predicted output diverged sharply from the original. He eventually discerned that in the original computation the number used was .506127 but in the simulation he had only entered the first three digits, .506 [18]. This phenomenon, eventually labeled "sensitivity to initial conditions", posits that a minor change at the beginning (or at various points) of a sequence of events can dramatically alter the long-term outcome of the system. This is commonly referred to as the butterfly effect.

The flapping of a single butterfly's wing today produces a tiny change in the state of the atmosphere. Over a period of time, what the atmosphere actually does diverges from what it would have done. So, in a month's time, a tornado that would have devastated the Indonesian coast doesn't happen. Or maybe one that wasn't going to happen, does. (Ian Stewart, Does God Play Dice? The Mathematics of Chaos, pg. 141) [19].

The weather is considered a classic chaotic system, as described in the text below. Yet, simple substitution of health behavior terminology for meteorological terminology reveals striking similarity. The weather (**BEHAVIOR CHANGE**) is an example of a chaotic

system. In order to make long- term weather forecasts (**PREDICTIONS OF BEHAVIOR CHANGE**) it would be necessary to take an infinite number of measurements, which would be impossible to do. Also, because the atmosphere (**HUMAN BEHAVIOR**) is chaotic, tiny uncertainties would eventually overwhelm any calculations and defeat the accuracy of the forecast. Even if it were possible to fill the entire atmosphere of the earth with an enormous array of measuring instruments, e.g., thermometers, wind gauges, and barometers (**PSYCHOSOCIAL, BIOLOGIC, AND ENVIRONMENTAL MEASURES**) uncertainty in the initial conditions would arise from the minute variations in measured values between each set of instruments in the array. Because the atmosphere (**HUMAN BEHAVIOR**) is chaotic, these uncertainties, no matter how small, would eventually overwhelm any calculations and defeat the accuracy of the forecast (**PREDICTION**).

Another metaphor for sensitivity to initial conditions involves rolling two identical balls down a tall rocky mountain. Starting the balls even an inch or less apart at the top of the mountain could result in the two balls ending hundreds of feet apart at the bottom; having traversed vastly different courses. The different pathways created by slight differences in the impact point on a billiard ball is another example.

One additional concept from Chaos Theory, fractal patterns, may also be relevant to understanding human behavior. Fractals, which have been identified in natural science in the mapping of the microvascular system and snow flake geometry, are recurring patterns within larger systems that are self-similar, that is, a shape appears similar at all scales of magnification. In terms of human behavior, there may be common patterns of behavior change within and across individuals that follow certain complex, non linear patterns. Thus, although behavior change may unfold in an almost infinite combination of knowledge, attitude, efficacy, and intention, there may be recurrent patterns of change that may be used to identify audience segments which could be targeted by common interventions.

Linear	**Quantum**
Cognitive-Rational	Intuitive
Motivation is arrived at	Motivation arrives
Planned	Epiphany
Cortical	Limbic
Left Brain	Right Brain
Maintenance of Change	Initiation of Change
Engineers/Physicists	Artists

Figure 1 Continuum of Motivational Processes.

Application to Health Behavior

Health behavior may mirror other Complex Systems found in nature in that they involve multiple component parts that interact in a nonlinear fashion. Factors such as knowledge, attitude, belief, and efficacy no doubt exert some influence on health behavior change.

However, the interaction of these factors represent a complex system bound by chaotic regulation. For example, which particular bits of knowledge, attitude, belief, etc. and the amount of each required to "tip" the system for a particular individual is virtually impossible to predict, and the outcome is sensitive to initial conditions. Initial conditions within individuals, e.g., relevant prior experience with a particular disease (e.g., family history) or a genetic predisposition may alter the interaction in profound ways. And, the slightest change in the system, i.e., the addition of one more piece of information or persuasion could dramatically alter the outcome. Such complex relationships are well represented by the swirling patterns created by mixing multiple colors of dye with a stick. Given the non-linear nature of complex systems they are usually represented mathematically by quadratic or other non-linear models. In the linear framework unaccounted for variance is generally relegated to the catch all "error" term, when in fact such "error" may represent the chaotic component of the outcome. Stated otherwise, "error" may be the result of imposing a linear model on a non-linear phenomenon. Additionally, in complex dynamic systems the interaction of factors can yield almost infinite potential patterns. In linear terms, this may be analogous to higher order interaction terms that could involve 5, 10, or 15-way interactions. Although linear methods can be used to model such interactions, they are limited statistically and conceptually. First, the ability to detect such interactions would be underpowered, so unless the magnitudes of these interactions are pre-specified so that the study could be adequately powered, these analyses would generally lead one to assume, perhaps falsely, that no interaction exists. Second, untangling a 3-way or higher order interaction generally extends beyond our ability to map and interpret such a finding; a relatively simple two-way interaction states that the effect of one variable on the outcome is not constant, but depends upon the level or status of yet a second variable (e.g., the intervention effect on cholesterol reduction is not constant, but is greater for males that for females). The extension to a 3-way interaction says that that observed gender by treatment interaction is itself not always better for males than for females, but depends upon the status of a third variable (perhaps the intervention does better for tall males, but no better than it works for short females, etc.). And this is a reduced example where each variable in the interaction only has two levels. In complex systems the levels of interactions are copious. Finally, from a chaotic perspective the confluence of interactions both within and between individuals is highly variable and the system is sensitive to initial conditions making prediction of such complex interactions virtually impossible. From a chaotic perspective, rather than searching for main effects or simple 2-way interaction effects, behavior change is assumed to involve multiple levels of interaction that vary across individuals.

Linear models of behavior change are then both conceptually inappropriate and statistically futile. In traditional statistical terms this would equate to analyzing and reporting separate main effects for multiple independent variables when there are known interactions (non linear in nature) of these variables. The solution does not do justice to the complexity of the phenomena.

A potential important element of this model that should also be considered is the occurrence of random external and intrapsychic events. Chaotic systems are not synonymous with randomness, nonetheless, random events can significantly impact complex systems.

Consider why after years of false starts and failed attempts, a person succeeds at increasing their physical activity, eating healthier or losing weight. Or, why after years of success a person relapses. One explanation is that success or failure is determined by random events. The event may be external, such as hearing about someone they knew who lost weight, quit smoking, or perhaps passed away. This is similar to the "Cues" concept in the Health Belief Model [20,21].

The random event may also be intrapsychic. Without conscious thought, the person may experience a surge of motivation that they need to and/or are able to change or a craving may arise unexpectedly that triggers a relapse. Such feelings may be stimulated by associations created by classical conditioning about which the individual may not be conscious. Regardless, motivation and impulse arrives as opposed to being planned. Consistent with this perspective, West et al. recently reported an analysis of how smokers decided to quit.

Approximately half of the ex- and current smokers in their sample reported that their most recent quit attempt was unplanned and those who did quit this way were more likely to stay quit than those who made a specific plan to quit [22]. Another study of smokers found that more than half of quit attempts were spontaneous rather than planned [23]. West et al. explain their findings using "catastrophe theory" [22], which posits that dramatic outcomes can result from continuous pressure of a force on a system. An example often used to illustrate this concept is the result of gradually bending a plastic ruler until it snaps or the point at which water becomes vapor. So too, motivation may break or boil when enough pressure is applied to the system.

Chaotic patterns can stimulate behavior change in two distinct ways. In the first, single external random events such as a conversation, a public service announcement, newspaper article, word about the death of a friend or relative, etc. may serve as a tipping point for motivational change. Conversely, absent an external event, resident chunks of knowledge or attitude may randomly coalesce to form a perfect motivational storm. Miller also delineates two types of quantum change, with one being more a dramatic, mystical experience and the second being more a sudden insight or sense of finding one's truth. Common to both pathways is that they occur outside of conscious reasoning; that they happen to the person [13]. As Miller notes, the individual experiences a "fast forward to self actualization". Interestingly in a study of problem drinkers, those whose decision to quit drinking arose from a transformational experience (having experienced a negative/traumatic event such as hitting rock bottom or having a spiritual awakening) were twice as likely to be non-problem drinkers at followup whereas those who reported weighing the pros and cons of drinking were actually more likely to have drinking problems at followup [24]. The cognitive approach to behavior change in this study was associated with worse outcomes. Thus not only do there appear to be linear and quantum pathways to change, the two processes may impact behavioral outcomes differently. Another perspective that may be useful to include in this alternative paradigm is the concept of "Tipping Points". Tipping points are dramatic changes in social behavior that arise quickly and usually unexpectedly [25]. Whether it be a jingle or slogan; a political idea or mass purchase of a

"fad" product, such tipping points are virtually impossible to predict, yet retrospectively coherent explanations for the phenomena are routinely offered. Similarly, each night after the stock market closes, pundants explain why certain events of the day or week "caused" the price fluctuations. Yet, a priori, few pundants could have predicted the impact of said events. If they possessed such prognostication ability they would be extremely wealthy. The stock market provides an excellent metaphor for chaos, as on an almost daily level, tipping points occur that lead to what has been called the random walk theory of wall street [26]. Additionally, just as our interventions often work, the stock market tends to rise. The former may be due to an inherent will to live and the latter inherent optimism of consumers. However, in both cases, there may be underlying human dynamics that predispose systems to moving in a particular direction.

Threshold effects or tipping points are commonly used in epidemiology. For example cutpoints for obesity, hyperlipidemia, and blood pressure are in part based on non-linear thresholds at which disease risk begins to rise at a faster rate [27]. In behavioral terms, the tipping point refers to the threshold at which individuals or groups of individuals adopt a particular idea or practice. Relating this to the obesity epidemic for example, there may be a societal tipping point at which a large percent of the population decides to alter their diet and activity patterns. A recent tipping point occurred in 2004–2005 when as much as 15% of the US population had tried the Atkins diet or some other low carbohydrate regimen [28], despite little scientific evidence demonstrating effectiveness [29–31]. Such non linear shifts have also occurred in the prevalence of smoking and illicit drug use [32,33]. However, they are difficult to predict let alone cause. It is important to note that the chaotic perspective of behavior change offered here focuses mostly on the individual intrapsychic dimension. Environmental factors such cost, availability, legal restrictions etc. also interact with intrapsychic determinants. In some cases, environmental determinants can overwhelm system constraints. For example, raising cigarette taxes by several dollars per pack, has a suppressing impact on individual smoking behaviors, whereas lack of availability of fruits and vegetables can constrain dietary choices.

Resistance to Chaos

Accepting randomness as a primal determinant of human behavior may be contrary to the deterministic view characteristic of western thought. Randomness may conflict with an innate tendency for humans to infer causality and a need for predictability. For example, when a punter wins the lottery, a completely random event, many individuals will assume that the winner used some replicable strategy that led to them to "earn" their prize or that some higher order "kharma" deemed the winner worthy. Accepting randomness requires that we relinquish the faith that reward and punishment; fortune and misfortune are doled out in an orderly, just fashion. Perhaps not surprisingly, Chaos theory and non-linear dynamics have met considerable resistance within the scientific community [18]. For public health professionals it requires a new conceptualization of health behavior as well as how and why we influence change.

In the complex system approach, the role of health communications may be analogous to the spinning of ping pong balls in a lottery machine. Say that each ping pong ball represents a chunk of knowledge, attitude, efficacy, or intention. On each ball lies a few strips of Velcro; the soft side. Inside the human psyche lies strips of the opposite, hard side of Velcro, which serve as potential motivational "receptors". Some of the motivational ping pong balls may have resided in the system for years while others may have been more recently implanted through a health education program, clinical counseling encounter, or health communication campaign. Rather than attempting to predict which piece or pieces of motivation may "tip" the individual, from the chaotic perspective, the role of the health professional is to ensure the balls are kept spinning at various intervals and velocities to maximize the chances that they adhere to their receptors. When sufficient balls have adhered a tipping point may occur. Which balls or combination of balls may trip the motivational switch as well as when and why they may stick, are chaotic events that defy accurate prediction. From a non-linear perspective, the goal of health professionals may be to encourage wing flapping.

The linear and chaotic paradigms are not necessarily mutually exclusive. Behavior change includes both chaotic and rational processes. As shown in the figure below, the Cognitive-Planned and Chaotic-Quantum aspects of motivation can be placed along a continuum. The continuum may be seen as a framework to both classify motivational styles (across individuals) or behavioral decisions (within individuals).

Some individuals may by their nature be prone to employ rationale decision making processes typically associated with left hemispheric function. On the other hand some may be more predisposed to quantum processes where change is more dramatic and less planned. Most individuals are likely influenced by both linear and quantum processes, perhaps depending on mood or other initial conditions. Another way to conceptualize the interaction of linear and quantum processes is that cognitive-rational factors may provide the fertile soil on which chaotic events may sprout. Thus, health promotion may be viewed as priming individuals so that when chaotic environmental or intrapsychic events occur, they have a greater likelihood of taking root. Whether individuals possess a predisposition to either style is an important issue with considerable implications for health communications. If valid, one implication is that program planners may need to tailor intervention content and delivery to match individual cognitive/motivational styles. Whereas quantum processes may be more operative at initiation of change, it is possible that cognitive-rational processes may be more relevant to maintenance of behavior change.

Summary and Implications for Practice and Future Research

The random component of health behavior change, though difficult to predict or control, can nonetheless be incorporated into practice and research. For example, using the "perfect storm" analogy, it may be important to provide individuals with periodic interventions so that the motivational ping pong balls are spun under varying "atmospheric" (i.e., psychologic and/or life circumstances) conditions. Periodic exposure is consistent with the approach used in many chronic disease management programs. Such program, from

this new perspective can be viewed as providing repeated opportunities to produce the motivational storm. This approach is also consistent with counseling models such as motivational interviewing, which provide clients with considerable opportunity to explore life with and without their risk behavior; that is to spin the balls [34,35].

Another implication is that individually tailored interventions may be particularly promising as a means to maximize the likelihood of a perfect motivational storm [36–40]. Individually tailored communications increase both receiver attention and message salience, which together increase the chances that the "balls" are spun and that they have a optimal chance of sticking. There are also statistical implications. The potential variance in behavior accounted for by traditional cognitive factors should perhaps be assumed to have an upper limit far below 100%. Given prior studies, a reasonable upper limit may be in the 50% range. And rather than assuming unaccounted for variance simply reflects "error", non-linear models could be used to explore alternative mathematical relationships. And although the relationship of predictor variables may be complex and non-linear, there may be identifiable patterns, i.e., fractals in the parlance of chaos theory, that manifest across individuals that would allow for sophisticated audience segmentation and potentially powerfully tailored interventions.

We are not proposing that linear statistical models and linear-based health promotion interventions are of no value and need be discarded entirely. There is a vast scientific base indicating that our interventions can successfully change behavior. What we are proposing, however is that we need to rethink why our interventions work and for whom. Group interventions, we propose, work because they have spun the "balls" of motivation (or deactivated barrier balls) in a large group of individuals, and for a subset of these individuals the balls fit their motivational receptors and other psychologic and biologic settings. It is important to note that current theories and communication methodologies can greatly inform which "balls" we select to highlight in our interventions. Motivation is not random. Tailoring motivational messages to the audience remains a critical step in chieving positive outcomes, and our current theories can help select the most effective set of balls.

Additionally, although patterns of change likely follow unique, i.e., chaotic, patterns across individuals, it may nonetheless may be useful to know that, in aggregate, balls that have similar characteristic profiles tend to "pool" in a defined geographic area once rolled down the metaphorical intervention mountain, helping us to perhaps understand which ping pong balls to keep circulating and for whom. That is, there may be common pathways to change based on individual parameters that can be used to develop sophisticated audience segmenta ution analyses and more effective interventions that account for the chaotic element of change. A "mixture model" of both chaotic and linear progression may be one that helps us best understand change.

The proposition that a significant proportion of human behavior operates from a chaotic perspective, at first blush, may appear to defy empirical verification. However, with the advent of technologies such as Functional Magnetic Resonance Imaging (fMRI) and momentary psychologic assessment, it may be possible to examine where, neurologically different types of motivation arise, and even predict when and why quantum transformations occur. Theoretical and statistical research examining behavior change from a quantum

perspective is encouraged. In particular, the degree to which transformational motivation observed in the addiction field operates in the nutrition and physical activity domains, and whether changes spurred by inspiration are more enduring than changes arrived at from the more cognitive, conscious pathway merits examination.

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Chapter 2

Crisis and Chaos in Behavioral Nutrition and Physical Activity†

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ABSTRACT

Resnicow & Vaughn challenged the field of behavioral nutrition and physical activity to conduct research in new ways. They challenged the predictiveness of our models, sensitivity to initial conditions, factors predisposing to change and measurement procedures. While the predictiveness of our models will reflect the sophistication of our thinking and research, and the sensitivity to initial conditions is subsumed under the sophistication of our models, research on conditions predisposing to change (e.g., epiphanies), more longitudinal designs, refined measurement procedures and testing of critical issues can only enhance the quality of our research. Improved research quality should lead to enhanced efficacy and effectiveness of our interventions, and thereby our making meaningful contributions to mitigating the chaos in our field and the crisis from the rising epidemic of obesity.

Background

Our field of behavioral nutrition and physical activity should be operating in crisis mode. The prevalence of obesity and overweight (an essentially nutrition and physical activity problem in its etiology and control) continues to increase at alarming rates in all age, demographic and gender groups in the US [1], Europe [2], and many other parts of the world [3,4]. There is concern that this will reverse the recent advances in chronic disease control [5]. In the face of this encroaching epidemic, obesity treatment programs have tended to have weak effects mostly for short periods of time [6]; and review after review have shown that obesity prevention programs also tend not to work [7–9]. Furthermore,

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using the mediating variable model (see Fig.1) as a structured framework, it is not clear we know what changes in diet or physical activity behaviour have led to the current problems and thereby provide the best behavioral targets for change [link A in Fig.1] [10,11]; nor what mediating variables are most strongly related to these behaviors and thereby provide the best mechanisms for change [link B in Fig.1] [12]; nor how best to manipulate the mediating variables to obtain behavior change and lower obesity [link C in Fig.1] [13]. This is a frightful state of affairs. We should all be doing innovative theoretically guided, but high risk, research to quickly build a stronger knowledge base from which more effective interventions could be crafted. Yet, most of us appear to be acting in our usual way of doing things: "same old, same old,"

In this context, Resnicow & Vaughn [14] challenged our "same old" way of thinking about our field. They correctly specified the assumption of linearity in our predictive models, and proposed Chaos and Dynamic Systems Theories as iterative nonlinear models. They did not throw out all our theories per se, but challenged how we interrelated the variables, how we related them to behaviors, and offered some new variables predisposing to change. While Glass & McAtee [15] recently pointed out deficiencies in the social dimensions of our research, Resnicow & Vaughn targeted our thinking about behavior change. Some of the issues they raised are non-issues, but others deserve that we morph our basic methods to test the new ideas.

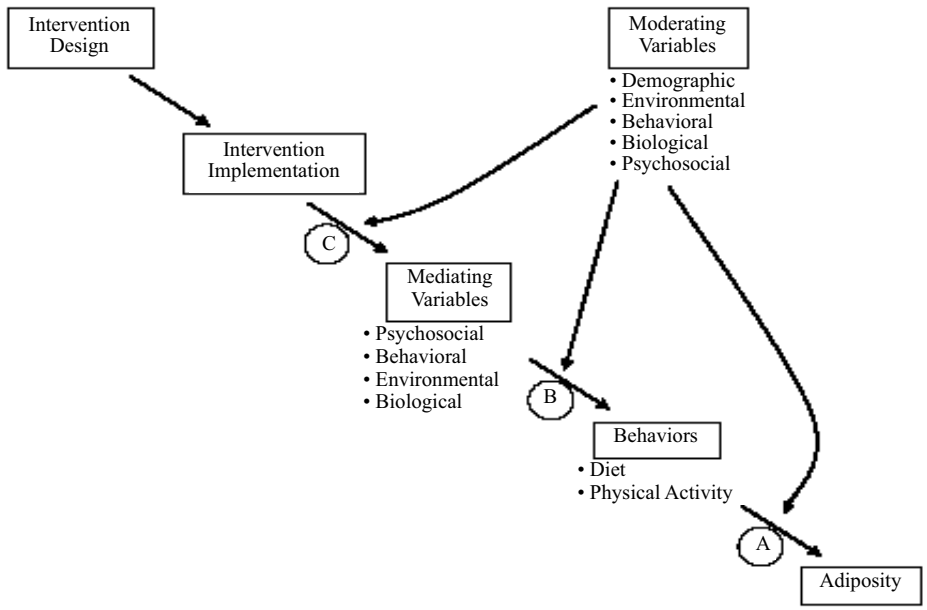

Figure 1 Mediating variable model for obesity.

Discussion

Nonissues

Since we use statistical methods, all our models are by definition probabilistic, rather than deterministic (unless we could account for 100% of the variance, which will not happen in our lifetimes).

A key issue in their article was the predictiveness of our current models. They correctly identified the very limited predictiveness of the current models. The key issue, however, is the level of predictiveness that could possibly be achieved in predicting behavior. For example, our biological research colleagues are not satisfied unless their models account for 90% or more of the variance in their phenomena of interest. We are well below that [16]. Resnicow & Vaughn have not taken into account the emerging research on environmental influences, e.g., home availability [17], neighbourhood characteristics [18,19]; biological influences, e.g., genes [20], sensitivity to tastes [21], the hormone rages of adolescent development [22,23]; emotional influences [23,24]; nor the likely interrelationships and interactions among these variables and our more usual psychosocial and behavioral predictors [25,26]. The higher the predictiveness of our models, the more we can engage in our logical approach to designing interventions based on these models. The larger number of and more diverse variables incorporated into these models, the more complex our interventions will need to be to address components of the model. And the interventions will need to both segment the population for differing types of interventions to different gender, age, ethnic, socioeconomic, and/or neighbourhood groups, and tailor the intervention to individual characteristics within these groups [27]. At this time, we need to build and test the more comprehensive models. This is a daunting, but exciting, challenge.

Resnicow & Vaughn proposed the principal of sensitivity to initial conditions, as if this were a new idea. All of our models of longitudinal relationships (as equations) have built into them sensitivity to initial conditions, i.e., the initial values of the variables. How diverse the outcomes depend on the nature of the relationships. As our models become more comprehensive and complex, fairly similar initial conditions could lead to quite divergent outcomes. In part this is a function of the sophistication of our knowledge base. We need to build more sophisticated predictive models.

The idea of a tipping point or when it might occur, is not well defined [28]. In some ways it reifies a change, as if there is something intrinsic to or magical about the change process. If a tipping point is nothing more than a critical point on a variable beyond which change occurs, it is not clear the concept adds much, but identifying those points would be helpful.

Issues Deserving Intensive Research

Investigators could take away from the Resnicow & Vaughn message that change is random and cannot be predicted, and thereby cannot be understood by our usual research methods on behavior or its change. This would be very unfortunate. Resnicow & Vaughn will have made a major contribution, only if it leads to innovative research and new insights.

Even in the vast complexities of molecular science, investigators are hammering away at delineating linear and nonlinear patterns to better understand the biology. Chances are we can do the same in behavior research.

Resnicow & Vaughn proposed that change does not occur in a linear "persuasion slowly overcoming resistance" manner, but rather in what they characterized as "quantum leaps," i.e., an epiphany or "aha!" event occurs from which the person decides to change. This is an interesting idea and should be testable. Innovative methods will be needed to identify people soon after the aha! experience to learn more about it. Perhaps interviewing new recruits to Weight Watchers™ or to fitness centers would accomplish this? A related issue would be what could we do to encourage aha! experiences? Are they a response to an overload of information (probably not, since we have done a lot of this already)? To repeated thinking about the issues (we could rogram prompts to thinking)? To setting off some emotional experience related to the behaviour (we might be able to tailor messages to issues people found emotionally charged)? Resnicow & Vaughn invoked the concept of "cues" from the Health Belief Model. There has been some research on cues [29–31], but this has not as yet led to substantial insights. Relating cues to aha! experiences could be an important avenue for research. Developing valid and reliable retrospective methods to identify and recall aha! experiences would be necessary to make much progress.

Resnicow & Vaughn correctly pointed out the cross sectional nature of most of our research. Dynamic Systems modelling proposes that dynamic research be done, and this would be focused on change over time which requires longitudinal designs [32]. The importance of longitudinal designs was emphasized when Nigg [33] found that physical activity predicted ensuing self efficacy, but not the other way around. If self efficacy is really caused by physical activity, but doesn't cause physical activity, it doesn't make sense to try to increase self efficacy in interventions.

While it is challenging to recruit and maintain longitudinal cohorts, such cohorts are required to address issues of direction of causality and thereby which variables should be targets for change in intervention programs. While ten year cohorts may not be necessary, perhaps 3 mo or 6 mo cohorts would provide tests for the changes we need. Longitudinal dynamic systems research has been initiated in other fields [34,35], which should provide a guide for our further development.

Whether behavior change can only be understood in retrospect instead of prospectively is an empirical issue. In part this is a function of how much variance our models will ultimately predict. Perhaps a few retrospective analyses will be necessary, perhaps using qualitative methods, to map out the processes occurring? But predictive science should be where we are headed, since predictive relationships clearly demonstrate what we know.

Resnicow & Vaughn correctly identified our current approaches to measurement as providing severe limitations to how we could understand our phenomena of interest. There have been limits on the extent to which existing measurement methods (e.g., classical test theory) have been used and reported [36], and limits on the predictiveness of existing measurement models [37]. One innovation in measurement theory that has recently drawn attention is Item Response Theory (IRT) [38]. IRT fits latent variables to items (and

respondents) which identifies portions of the underlying variable being poorly measured [39], and assesses reliability across the range of the underlying variable [39,40]. Having items measuring specific locations on the underlying variable permits an assessment of whether the measures work differently after participation in an experiment [41]; differ by ethnic, gender or other groupings; and permit more efficient multidimensional modelling of the variable [42]. Use of IRT offers great promise for better understanding and minimizing the problems due to measurement of our constructs, and deserves much wider use.

An issue Resnicow addressed in his oral presentation in Boston (but not in his paper) was the falsifiability of a theory, and whether our current cognitive models are really theories. He correctly stated that in our current approach to research, no theories have been discarded (which would be considered a sign of progress and development in a field). Our best current research fits multivariate models to sets of variables [43], and determines which variables were significantly related to other variables in the model. This is useful for assessing the predictiveness of particular variables in certain situations, but does not necessarily address the usefulness of the larger theory. To move our field forward we need more attention to theoretical issues in our research, tests of clearer more specific predictions from theory applied to particular issues [44,45], and delineation of "critical issues" where two theories would make different predictions or model fitting research would need to test the fit of competing models [24], where the alternative models were predicated on different theories. More highly controlled experimental research on critical issues will also be necessary. Accumulation of findings across "critical" studies would enable the field to find more comprehensive and more predictive theoretical frameworks, and capitalize upon them in more likely to be effective interventions. There has been distaste for theory in our field [46], and some have proposed continuing conducting intervention research until randomly hitting on intervention procedures that work [47].

Alternatively, I believe highly predictive theory should guide the design of effective interventions. In a complicated set of many possible variables and relationships, a random search may never result in finding effective change techniques, and even if it did, we wouldn't have the conceptual framework to understand why it happened in order to exploit it.

Conclusion

Resnicow & Vaughn challenged the field of behavioural nutrition and physical activity to conduct research in new ways. While the predictiveness of our models will reflect the sophistication of our thinking and research, and the sensitivity to initial conditions is subsumed under the sophistication of our models, research on conditions predisposing to change (e.g., epiphanies), more longitudinal designs, refined measurement procedures and testing of critical issues can only enhance the quality of our research. Improved research quality should lead to enhanced efficacy and effectiveness of our interventions, and thereby our making meaningful contributions to mitigating the chaos in our field and the crisis from the rising epidemic of obesity.

Abbreviations

US = United States
IRT = Item Response Theory

Competing interests

The author(s) declare that they have no competing interests.

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Chapter 3

Memory in Astrocytes: A Hypothesis †

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ABSTRACT

Background: Recent work has indicated an increasingly complex role for astrocytes in the central nervous system. Astrocytes are now known to exchange information with neurons at synaptic junctions and to alter the information processing capabilities of the neurons. As an extension of this trend a hypothesis was proposed that astrocytes function to store information. To explore this idea the ion channels in biological membranes were compared to models known as cellular automata. These comparisons were made to test the hypothesis that ion channels in the membranes of astrocytes form a dynamic information storage device.

Results: Two dimensional cellular automata were found to behave similarly to ion channels in a membrane when they function at the boundary between order and chaos. The length of time information is stored in this class of cellular automata is exponentially related to the number of units. Therefore the length of time biological ion channels store information was plotted versus the estimated number of ion channels in the tissue. This analysis indicates that there is an exponential relationship between memory and the number of ion channels. Extrapolation of this relationship to the estimated number of ion channels in the astrocytes of a human brain indicates that memory can be stored in this system for an entire life span. Interestingly, this information is not affixed to any physical structure, but is stored as an organization of the activity of the ion channels. Further analysis of two dimensional cellular automata also demonstrates that these systems have both associative and temporal memory capabilities.

Conclusion: It is concluded that astrocytes may serve as a dynamic information sink for neurons. The memory in the astrocytes is stored by organizing the activity of ion channels and is not associated with a physical location such as a synapse. In order for this form of memory to be of significant duration it is necessary that the ion channels in the astrocyte syncytium be electrically in contact with each other. This function may be served by astrocyte gap junctions and suggests that agents that selectively block these gap junctions should disrupt memory.

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Background

Until recently astrocytes were considered to play no more than a supportive role for neurons in the central nervous system. This view has now been supplanted by a more active participation of astrocytes in information processing, where the astrocytes not only receive and respond to neuronal input, but also transmit signals to neurons [1–9]. These findings indicate that astrocytes contribute to the processing of information. In support of this concept it was recently demonstrated that spinal cord astrocytes are necessary to support hyperalgesia produced by peripheral injury [10–12]. Blocking gap junctions in the astrocytes suppressed hyperalgesia, which suggested that the astrocytes were processing the nociceptive information and regulating the function of spinal cord neurons [10]. These results are similar to work reported by Hertz et al. and Ng et al. who demonstrated that astrocytes are critical for the establishment of learned behaviors [13,14]. Furthermore, recent studies indicate that several general anesthetics suppress the function of astrocyte gap junctions at concentrations that are relevant for loss of consciousness [15,16]. These data suggest that the anesthetic properties of these agents may be mediated at least in part by their actions on astrocytes and may indicate some role for astrocytes in consciousness.

In a recent review Robertson outlined an astrocentric hypothesis of memory [17] as an alternative to the current neurocentric or synaptic based theories. In this hypothesis Robertson concludes that because astrocytes form large syncytium via gap junctions and that they are connected to neurons through synapses these cells can store and "bind" diverse information.

In this intriguing review Robertson hypothesizes that information is stored as a result of gap junctional plaques converting to a crystalline configuration that is a closed, high resistance, state of the gap junctions. As a result of these altered gap junctions ion flow between astrocytes is restricted resulting in a functional memory.

In examining the idea that astrocytes might play a major role in information processing it seemed prudent to examine other potential memory mechanisms that could support information processing in astrocytes. In experiments examining electrical potentials and calcium fluxes in astrocytes it was demonstrated that these cells can, on an individual basis, support potentials for several seconds [1,2,6,7]. These data suggest that ion channel activity in a group of gap junction linked astrocytes could retain information for substantial periods of time. Thus, the ion channels mediating the astrocyte potentials could function to store and process information in the central nervous system.

This paper examines the possible role of ion channels in storing information in astrocytes.

Results and Discussion

Similarity of Ion Channels to Cellular Automata

Ion channels communicate with each other via changes in voltage, changes in calcium concentrations or through other second messenger systems. In voltage gated ion channels, for example, the rules governing the relationship between channels specify that if neighboring channels alter the local membrane potential to some threshold the channel under observation will change state, i.e., open or close. Each ion channel functions as an independent unit that

monitors information transmitted from its nearest neighbors. As a result of the information processing occurring at the single ion channel level ensembles of ion channels are capable of performing relatively complex functions, such as the generation of action potentials. This form of information processing by ion channels is remarkably similar to models known as cellular automata [18,19]. In cellular automata simple units that are capable of existing in a finite number of states are linked together using rules for the transfer of information between the units. The states occupied by the units and the rules of information transfer determine what state each unit will occupy in the next time period. These models have been extensively studied and demonstrate the emergence of complex behavior [20,21]. Some cellular automata have even demonstrated universal computation [22]. To illustrate how a cellular automata stores and processes information a one dimensional cellular automaton in which the units are binary (they are either in state 0 or state 1) is presented in Fig.1. The rule used was the mean of three units rounded to the nearest integer determines the state of the middle unit in the next iteration. This model was studied at length by Wolfram and this rule is Wolfram's rule number 232 [20,21].

In Fig.1 the initiating event (Representation 1(R_1)) was produced by randomly setting the states of the units in the automata. The time series was then calculated. In the figure it is evident that from R_1 to R_4 the automaton changes representations, but after R_4 the cellular automaton reaches a steady state and the representations no longer change. This stabile representation is the attractor R_0. The transition period from R_1 to R_0 is the memory of

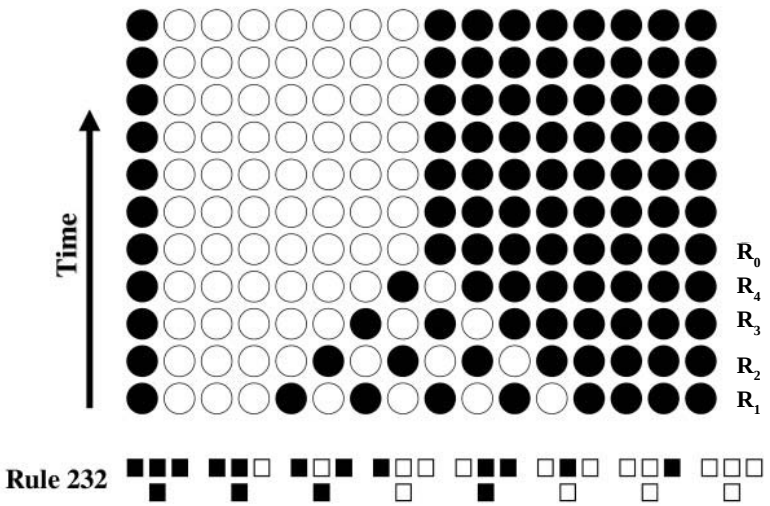

Figure 1 Memory in cellular automata. A sixteen unit one dimensional cellular automaton was constructed using binary units and Wolfram's rule number 232. This rule is illustrated at the bottom of the figure where the three squares on top are the current states of three adjacent units and the single square below is the resultant state of the middle unit during the next iteration. Open squares indicate state 0 and filled squares indicate state 1. The initial representation (R_1) was generated by randomly setting the state of each unit to either 0 (open) or 1 (filled). The time series was then calculated. Note that the memory of this system extends from R_1 to R_4 where the representations change with each iteration. Starting at R_0 the units no longer change state indicating that all information about R_1 is lost.

the automaton. At each iteration prior to R_0 the automaton retains information that can be used to determine something about the initial configuration. However, when the automaton reaches R_0 all information about the initial configuration has been lost. In astrocytes the ion channels in the membrane are distinct units with a finite number of states and they communicate with each other through a simple set of rules, i.e., a change in voltage or in Ca2+ concentration. Therefore, the astrocytes' membrane ion channels are acting as a two dimensional cellular automaton. As with the automaton presented in Fig.1 the initiating event can be inferred based on the configuration of the entire ensemble of ion channels up until the ion channel configuration returns to the attractor representation (R_0). At this point all information about the initiating event is lost. This concept suggests that ion channels working in collection can store information for at least brief periods of time. The remaining question is the maximum duration of memory in this type of system.

Memory in Cellular Automata

In a series of interesting experiments Langton examined the properties of cellular automata that optimize information storage and processing [23]. In these experiments he varied the rules by which the cellular automata operated and measured the resulting chaotic nature of the system. Langton found that automata whose rules made them operate at the junction between ordered and chaotic behavior were able to store information for the longest period of time. Memory dropped off markedly on either side of this phase transition. To illustrate how the chaotic nature of the cellular automata might influence memory a two dimensional cellular automaton with four different rule sets and a Moore neighborhood (8 neighbors) was set up (Fig. 2.A). The units in the automaton could occupy four different states, i.e., one open, one closed and two inactive. The cellular automaton was seeded with two units in the open state to invoke the initial representation R_1.

The left hand column illustrates a rule set that produces ordered behavior. Note that a signal cannot propagate in this cellular automaton. The second column demonstrates another form of ordered behavior where the behavior immediately becomes repetitive. This cellular automaton, like the one to the left of it, cannot process information due to the inability of the automaton to transition to novel representations. The third column is a rule set that produces behavior at the border between order and chaos. The net result is the smooth propagation of an "action potential" throughout the cellular automaton with the system eventually returning to the attractor representation R_0. The final column illustrates a chaotic system that evolves rapidly into a random pattern of channel openings. The nearly random behavior prevents proper processing of information since there is no relationship between successive representations. Figure 2.B illustrates the "potentials" produced by these different rule sets by plotting the number of open channels versus time. These models demonstrate that only the rule set with behavior at the transition between order and chaos produces a potential that is similar to an action potential observed in biological systems. Note that the rules that produce ordered behavior either returned to the attractor representation R_0 very rapidly or never returned to R_0, suggesting that the systems are incapable of supporting information storage. The chaotic rule set also never returns to the

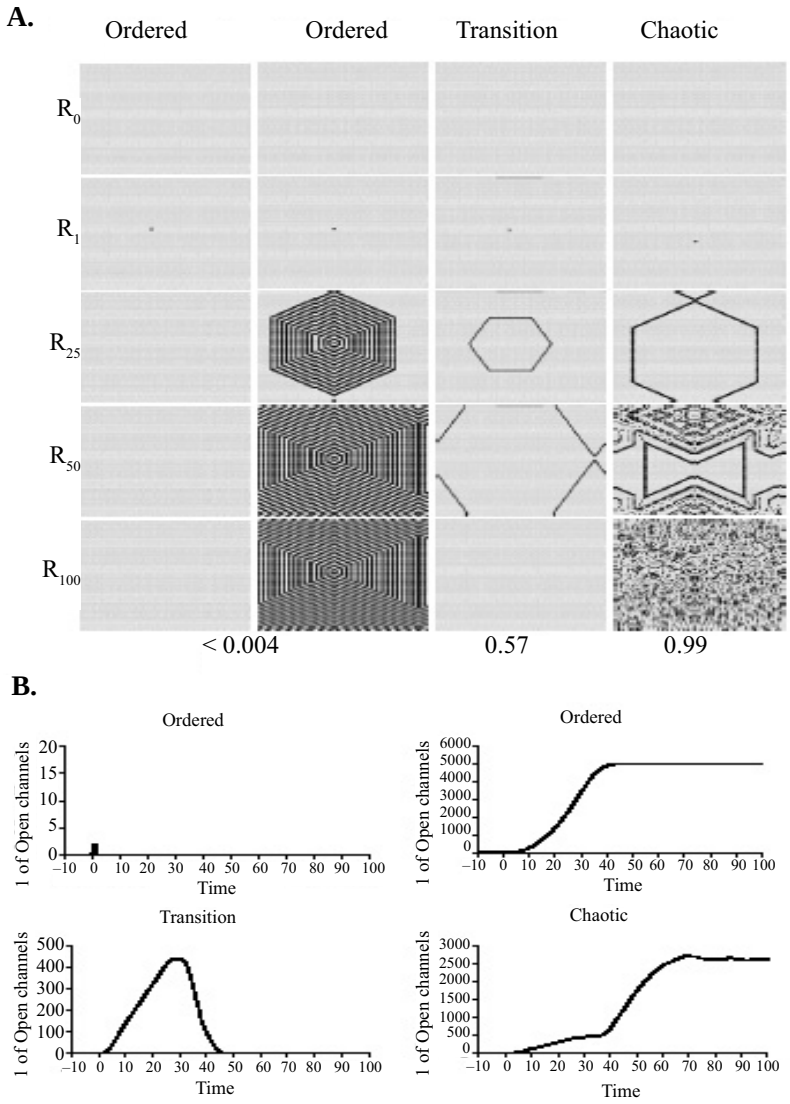

Figure 2 Two dimensional cellular automata operating between order and chaos behave like excitable membranes in biological cells. **A.** A two dimensional cellular automaton was constructed with the program CaSim using units with four states, i.e., one open, one closed and two inactive states. Four different rule sets were used to generate the four time series in the figure. The cellular automaton was seeded at R_1 by setting two units to the open state and the times series calculated. The configuration of the cellular automata at iterations 0, 1, 25, 50 and 100 are presented in the figure for the four rule sets. The entropy of the rule sets was determined by calculating the probability of each state (P_s) from 10 runs of 1000 iterations. For these calculations 10 percent of the units were set to the open state at R_1. Entropy was calculated using the equation: $\text{entropy} = -\sum P_s \ln(P_s)$. The entropy of each rule set was then expressed as a ratio of the calculated entropy to the maximum entropy (bottom of the figure). The maximum entropy is when all four states have a probability of 0.25. **B.** The "potentials" generated by the rule sets in A were graphed by plotting the number of open channels versus time. These plots indicate that only the transition rule set produces channel openings that are similar to action potentials in biological membranes.

attractor, which also indicates that the system cannot retain information for significant periods of time. Only the rule set that produced behavior between order and chaos could retain information about the initial event R_1 for a period of time and then return to the attractor representation. Based on the similarity of the potentials generated by the transition rule set these models suggest that the ion channels in the membranes of biological cells function as cellular automata with rules that set the behavior at the boundary between order and chaos. This region of the order to chaos spectrum balances information storage with transmission, which, in turn, supports information modification [23].

In addition to examining the length of memory in cellular automata relative to the chaotic nature of the automata, Langton [23] evaluated how the number of units in an automaton influenced memory. In these experiments Langton used rules that produced automata that operated in the order/chaos phase transition and then varied the number of units in the automata. He found that there was a log-linear relationship between the time that the cellular automata stored information and the number of units in the automata. This indicated that the addition of units to the automata exponentially increased the amount of time the automata stored information. This relationship is an extremely powerful property of cellular automata that has evolutionary significance for biological systems that process information with ion channels. The exponential relationship between memory and the number of units in an automaton indicates that a biological system simply has to add more units (ion channels) to its calculating device in order to dramatically increase its memory. With an increase in memory duration the complexity of the calculations that can be performed also increases [23].

The Human Cellular Automaton

The findings of Langton indicate that as a cellular automaton is increased in size the duration of memory increases. In the astrocentric hypothesis large numbers of astrocytes are connected through gap junctions [10,17,24–27], which suggests that astrocytes form extensive ion channel cellular automata. To examine the potential memory duration for a human brain sized cellular automaton data was collected from the literature for maximum ion channel open and closed times, duration of potentials evoked in single cells by very brief stimuli and the duration of potentials in brain slices and mollusk ganglia. The recordings in the slices and ganglia used for this analysis represented a large number of cells in the tissue rather than a single cell in the slice or a population response to a single synaptic event. Since data are limited for astrocytes, potentials from all forms of excitable cells were collected. In Fig. 3 the log maximum length of time reported for single ion channels to transition through an open and closed cycle and the log of the duration of evoked whole cell potentials were plotted versus the number of ion channels. For whole cells the number of ion channels was estimated to be 10^6. A regression line was fitted to these two sets of data. The duration of potentials from the slices and ganglia were then plotted on this line and the number of ion channels needed to produce these potentials was estimated by extrapolation. These potentials appeared to be generated by 10^7 to 10^8 ion channels. This finding suggests that Langton's relationship of the number of units to length of time that information is stored in cellular automata holds true for ion channel cellular automata.

Note that for convenience there was no attempt to limit the data collected to any one type of ion channel, cell type, or species. The assumption used here is that all biological systems evolved a similar mechanism to process information with ion channels and, as such, their ion channels have similar properties.

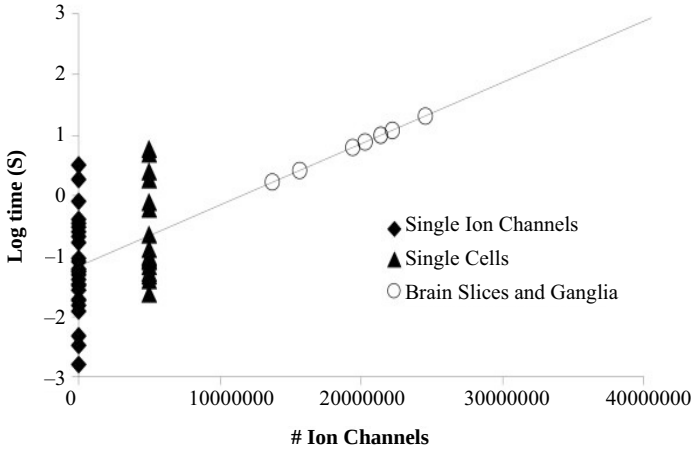

Figure 3 Memory as a function of the number of ion channels. Data was collected from the literature for the open/closed times for single ion channels, the length of potentials evoked in single cells and the length of potentials in groups of cells in brain slices or mollusk ganglia. The logs of the single ion channel and single cell data were graphed versus the number of ion channels. Cells were estimated to have 10^6 ion channels. The slope of the line defined by these two points was determined and the length of the potentials in the brain slices and mollusk ganglia were plotted onto the graph.

To generate an estimate of the total number of ion channels in a human astrocyte cellular automaton the number of astrocytes was approximated to be 10^{13} [28]. With 10^6ion channels/cell this suggests 10^{19} ion channels in a human cellular automaton. Using the estimate of 10^{19} ion channels in the human cellular automaton the predicted duration of memory was extrapolated from the slope of the line in Fig. 3. The relationship between memory and the number of ion channels was estimated to be $t = e^{2.3\times10-7N}$. Where t is time and N is the number of ion channels in the system. This calculation yielded a predicted maximum memory for a human sized astrocyte cellular automaton of $10^{10^{12}}$ years. Therefore, for all practical purposes, the predicted maximum duration of memory in human cellular automata is infinite. What is most notable about this memory is that it occurs without fixing the information to any physical structure such as a synapse or cell as predicted in Hebb's postulate [29]. The information is stored as a succession of representations, or ion channel configurations, with each individual representation lasting only a short period of time. The configuration of the ion channels is organized by the incoming information and then as this organization dissipates over time the information is lost. In thermodynamic terms the entropy of the system is decreased by the storage of information and, as the calculation presented above indicates, it takes a substantial amount of time for the entropy to return to baseline levels. Admittedly, the estimates for the number

of ion channels and the number of astrocytes that make up a single syncytium are crude; however, even if the estimates are off by several orders of magnitude the overall conclusion that the potential duration of memory in a human ion channel cellular automaton is infinite, from a biological frame of reference, remains valid.

Another interesting comparison to be made between the astrocentric hypothesis and the neurocentric hypothesis is that there are $k^{10^{19}}$ distinct representations or unique configurations of the ion channels.

Using 10^{12} neurons each possessing 10^3 synapses we can estimate that there are 10^{15} synapses in a human brain [28] and a potential for $k^{10^{15}}$ distinct representations or unique configurations of the synapses. The term k is the number of states that an individual ion channels or synapse can take. These calculations demonstrate that the potential information processing capacity of the astrocytes using ion channels is many orders of magnitude larger than the capacity of neurons using synapses.

Associative Memory in Cellular Automata

An important component of memory is the ability to associate two or more events. In an ion channel cellular automata this is accomplished by the fact that the series of representations produced by a single event is significantly different from that produced by two events. Fig. 4 demonstrates the ability of a cellular automaton to associate information from two events. In the first column a single event produces a series of representations as the automaton progresses. In the second column two events occur simultaneously. The two events produce a series of representations that are distinct from the single event presented in the left column. This indicates that the two events have been associated to produce a unique memory.

Another interesting facet of ion channel cellular automata is that because they are dynamic systems they can readily store temporal differences between events. In the right hand column the two events are separated by ten units of time resulting in a series of representations that differs from either the single event in the left hand column or the two simultaneous events in the middle column. These observations suggest that the proposed astrocyte memory system can associate memories and that temporal information can be stored.

Research Supporting Astrocyte Cellular Automata as Memory Systems

In studies published over forty years ago Hyden demonstrated that glia were critical for memory [30–32]. More recent work using the one-trial aversive learning paradigm in chicks has confirmed Hyden's findings [13,14,33]. In these studies inhibitors of astrocyte function were found to block both short term and intermediate term memory, but, when administered later, had no effect on the long term retention of the learned behavior. During the short and intermediate periods it was demonstrated that ion fluxes in astrocytes are critical [13,33,34] for memory suggesting that the astrocyte ion channels may store information in the chicks for a brief period of time, approximately 60 minutes, while the appropriate rewiring of the neuronal circuitry takes place. It is important to note that this behavioral model involves

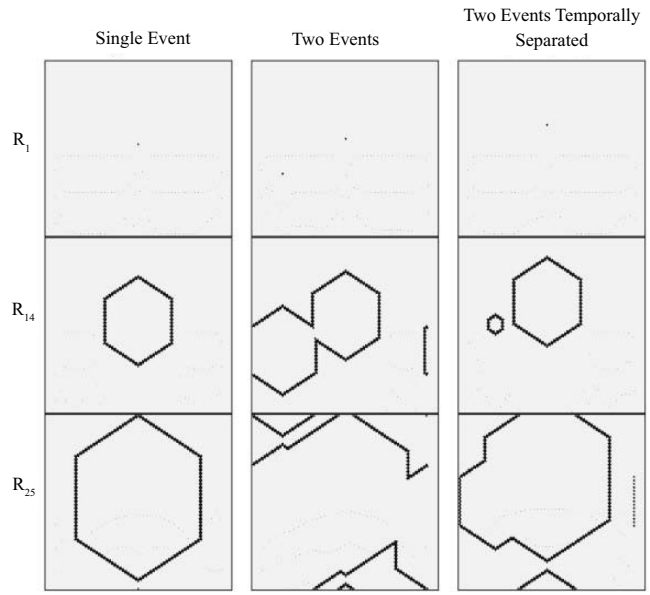

Figure 4 Associative memory in cellular automata. A cellular automaton operating at the transition between order and chaos was setup as described in Fig. 2 using the program CaSim. Three different stimuli were used. Iterations 1, 14 and 25 are presented in the figure. In the left column the cellular automaton was seeded by setting one unit to the open state at R_1 (Single Event). In the center column two units were seeded at R_1 (Two Events). In the right hand column the cellular automaton was seeded by setting one unit to the open state at R_1 and a separate unit to the open state at R_{10} (Two Events Temporally Separated). Note that each time series generates a different pattern of channel openings (representations) indicating that the two events in the second and third columns have produced unique memories by associating the events. Also note that the difference in representations produced by the automaton in the second and third columns indicates that the cellular automaton stores temporal information about the events. Therefore it is concluded that a two dimensional ion channel cellular automata is capable of associative memory.

both memory and learning, while the cellular automata hypothesis presented here is related purely to memory. Memory is the ability of an organism to store information about events in a retrievable format, whereas learning involves a change in behavior or potential behavior. Thus, a consolidated learned behavior, as occurs in the one-trial aversive learning paradigm, is likely to be the result of neuronal rewiring.

Furthermore, it does not require the organism to retain any specific memory of the event that precipitated the change in behavior beyond the length of time necessary to produce the rewiring. In this light, the chick in the aversive learning paradigm may actually recall the aversive stimulus for the short and intermediate term memory periods, which require astrocytes, but may not retain any recollection of the event once the aversive behavior has been established. It is enough for the chick to avoid certain objects without remembering why it needs to avoid them. The distinction between memory and learning is important because the two processes are likely mediated by different mechanisms. In the current hypothesis the ion channel cellular automata would be responsible for the specific memory of the event while changes in synaptic strength of the neurons would be responsible for

learning and maintaining the new behavior. Astrocyte memory could support learning, but learning does not necessarily support the memory of events.

In addition to proposing that glia were involved in memory, Hyden predicted that mental diseases may involve glia [35] as reported in [34]. In the ion channel cellular automata hypothesis it is critical that the ion channels operate at the junction between order and chaos. Departure from this behavior is predicted to produce pathology. Deviation to the ordered side of the spectrum might produce depressive types of behaviors in the organism and memory deficits while deviation to the chaotic side might produce psychotic or manic types of behaviors that are also associated with memory deficits.

Several studies have demonstrated that long term treatment with antidepressant drugs at clinically relevant doses alters protein expression and function in astrocytes [36–41] and long term treatment with lithium ion results in suppression of mRNA for sodium-dependent inositol transporter in astrocytes [34]. The length of treatment required for the change in astrocyte proteins is consistent with the onset of the therapeutic effect of these agents. These studies suggest that these psychoactive agents may adjust the activity of astrocyte ion channel cellular automata toward the order/chaos border, thus improving the function of the memory system. Therefore, a number of studies, spanning over forty years, indicate that astrocytes are important for memory and possibly for the therapeutic effect of psychoactive drugs, which is consistent with the astrocyte ion channel cellular automata hypothesis.

Conclusion

In this study the hypothesis that astrocytes could store information in the central nervous system was considered. Based on the similarity of membrane ion channels to mathematical models known as cellular automata it seems reasonable to conclude that ion channels in astrocytes could store information for significant periods of time. This storage system does not rely on physically fixing information to any structure such as a synapse; rather information is stored by organizing the activity of the ion channels. If this concept is correct it suggests that neurons may use astrocytes as a dynamic information sink. In theory, this information would remain readily available to the neurons for extended periods of time.

Furthermore, this hypothesis indicates that to store information for significant periods of time the ion channels in the astrocyte syncytium must be in electrical contact with each other. This function could be served by the astrocytes' gap junctions. Thus, we can predict that agents that selectively block astrocyte gap junctions should disrupt memory. Clearly, further work is needed to verify this theoretical framework for memory in nervous systems.

Methods

One Dimensional Cellular Automaton

A 16 unit one dimensional cellular automaton was set up with each unit having 2 states. The rule used for this automaton was Wolfram's rule number 232 [20,21]. In this rule each

unit is updated by averaging the states of the unit with its two nearest neighbors and then rounding to the nearest integer. The time series for this cellular automaton was calculated by hand.

Two Dimensional Cellular Automata

To examine the effects of different rule sets on 2 dimensional cellular automata the program CaSim [42] was used. A matrix of *100×100* units with a Moore neighborhood (eight neighbors) was set up with various rules. Each unit had 4 states. The entropy of the different rule sets was calculated using the equation entropy $= -\sum P_s \ln(P_s)$, where P_s is the probability of a unit occupying a particular state. The probabilities of the different states were determined from 10 runs of 1000 iterations for each cellular automaton. For these calculations the cellular automaton was seeded for each run by randomly setting ten percent of the units to the open state. The maximum entropy was calculated using the probability of 0.25 for each of the four states. The ratio of the calculated entropy of the rule set to the maximum possible entropy was used as an indicator of the chaotic nature of the system. Thus an entropy ratio of 0 is a completely ordered rule set and a ratio of 1 is a completely chaotic rule set.

For the examples presented in the figures the cellular automata where seeded with either 1 or 2 units set to the open state.

Duration of Memory Versus the Number of Ion Channels

To calculate the relationship between the number of ion channels in a system and the duration of information storage by the ion channels data was collected from published sources. The maximum open and closed times for various ion channels were obtained [43–54] and the open to closed cycle was used as the duration of memory in single ion channels. Similarly, potentials recorded in single cells were obtained [55–62] and used as an indication of the activity of multiple ion channels in concert. The log of the values for the duration of the responses in the ion channels and cells were plotted versus the number of ion channels. The number of ion channels in the cells was estimated to be 10^6. A line was then fitted to the two points and the log of the duration of potentials in slices and ganglia [63–71] were plotted on the line.

Acknowledgements

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Chapter 4

Nonlinear Dynamics and Chaos in a Fractional-Order HIV Model †

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ABSTRACT

We introduce fractional order into an HIV model. We consider the effect of viral diversity on the human immune system with frequency dependent rate of proliferation of cytotoxic T-lymphocytes (CTLs) and rate of elimination of infected cells by CTLs, based on a fractional-order differential equation model. For the one-virus model, our analysis shows that the interior equilibrium which is unstable in the classical integer-order model can become asymptotically stable in our fractionalorder model and numerical simulations confirm this. We also present simulation results of the chaotic behaviors produced from the fractional-order HIV model with viral diversity by using an Adams-type predictor-corrector method.

Introduction

An important part of the human immune response against viral infections is cytotoxic T Lymphocytes (CTLs) [1].They recognize and kill cells which are infected by virus. There are many immune models describing the virus dynamics with CTL immune response. Nowak and Bangham [2,3] proposed an ODE model which explores the relation among CTL immune responses, virus load, and virus diversity. In [2], a rate of specific CTL(Zj) proliferation in response to the corresponding specific infected cells (Ij) depends on the mass action law cI_jZ_j. This model has been important in the field of mathematical modelling of HIV infection. In their model, there is no interaction among different types of CTL(Zj). Iwami et al. [4] assumed that the correlation is incorporated as a function of the frequency

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that the specific CTLs(Z_j)-encounter in the specific infected cells (I_j). In a similar manner, they considered the rate of elimination of specific infected cells (I_j) by the specific CTLs(Z_j) to be proportional to this frequency. However, these models do not take into account the fractional order derivatives that have been extensively applied in many fields (e.g., [5–17]) and the reference cited therein). Recently many mathematicians and applied researchers have tried to model real processes using the fractional order differential equations (FODE) [16]. In biology, it has been deduced that the membranes of cells of biological organism have fractional order electrical conductance [13] and then, they are classified into group of noninteger order models. Also, it has been shown that modelling the behavior of brainstem vestibule-oculumotor neurons by FODE has more advantages than classical integer order modelling [8].

Particular emphasis is that a major difference between fractional order models and integer order models is that fractional order models possess memory [5,12], while the main features of immune response involve memory [18]. Hence, we attempt to model HIV infection with immune response using a fractional order system. Our presentation is based on the immune model of HIV infection which is developed by Iwami et al. [4]. For the one-virus model, we carry out a detailed analysis on stability of equilibrium. Our analysis shows that the interior equilibrium which is unstable in the classical integer order model can become asymptotically stable in our fractional order model. We also find that chaos does exist in the fractional order HIV model with viral diversity.

Model Derivation

We first give the definition of fractional order integration and fractional order differentiation [14,16]. For the concept of fractional derivative we will adopt Caputo's definition which is a modification of the Riemann-Liouville definition and has the advantage of dealing properly with initial value problems.

Definition 2.1. The fractional integral of order $\alpha > 0$ of a function $f : R^+ \to R$ is given by

$$I^{\alpha} f(x) = \frac{1}{\Gamma(x)} \int (x-t)^{\alpha-1} f(t)dt \tag{2.1}$$

provided the right side is pointwise defined on R^+.

Definition 2.2. The Caputo fractional derivative of order $\alpha \in (n-1, n)$ of a continuous function $f : R^+ \to R$ is given by

$$D^{\alpha} f(x) = I^{n-\alpha} D^n f(x), \quad D = \frac{d}{dt}. \tag{2.2}$$

Now we introduce fractional order into the ODE model by Iwami et al. [4]. The new system is described by the following set of FODE:

$$D^{q_1} T = \lambda - dT - \sum_{l=1}^{n} B_l^{'} T V_l,$$

$$
\begin{aligned}
&D^{q_{2j}} I_j = \beta_j^i T I_j - a I_j - b Z_j (I_j / T + \sum_l^n I_l^{'}),\\
&D^{q_{4j}} V_j = k a I_j - u V_j, (j = 1,2,...,n),\\
&D^{q_{3j}} Z_j = c Z_j \frac{I_j}{T + \sum_l^n I_l^{'}} - \delta Z_j, \quad (j = 1,2,...,n),
\end{aligned} \tag{2.3}
$$

where *T(t)* represents the concentration of uninfected cells at time *t*, $I_j(t)$ represents the concentration of infected cells with a virus particle of type *j*, $V_j(t)$ the concentration of free virus particle of type *j*, and $Z_j(t)$ denotes the magnitude of the specific CTL response against variant *j*. Here, $0.95 \le q_{1j}, q_{2j}, q_{3j}, q_{4j} \le 1$ *(j=1,2, ...,n)* are restricted such that fractional derivative can be approximately described the rate of change in number.

Following [4], uninfected cells are assumed to be generated at a constant rate λ. Uninfected cells, infected cells, free viruses, and CTLs decline at rates *d, a, u*, and δ, respectively. The total number of virus particles produced from one cell is *k*. The rate of CTL proliferation in response to antigen is given by $cZ_i I_i /(T + \sum_{l=1}^{n} I_l)$ and the specific infected cells are killed by specific CTLs at rate $bZ_i I_i /(T + \sum_{l=1}^{n} I_l)$ while infected cells are produced from uninfected cells and free virus at rate $\beta_l^{'} T V_l$, that is, a rate of specific CTL(*Zj*) proliferation in response to the corresponding specific infected cells (I_j) depends on the frequency, instead of the mass action law.

To simplify the model, it is reasonable to assume that the decay rate of free virus, *u*, is much larger than that of the infected cells, *a*, and this system describes the qualitative dynamics of the asymptomatic phase of HIV infection. Thus, we may introduce as a good approximation that the virus is in steady state (i.e., $Dq_{4j} V_j = 0$) and hence $V_j = k a I_j / u$ (see [4, 19]). This leads to the following simplified system of FODE:

$$
\begin{aligned}
&D^{q_1} T = \lambda - dT - \sum_{l=1}^{n} \beta_l T I_l,\\
&D^{q_{2j}} I_j = \beta_j^i T I_j - a I_j - b Z_j I_j /(T + \sum_l^n I_l^{'}),\\
&D^{q_{3j}} Z_j = c Z_j I_j / \left(T + \sum_l^n I_l^{'} \right) - \delta Z_j, \quad (j = 1,2,...,n),
\end{aligned} \tag{2.4}
$$

Where $\beta_j = \dfrac{k a \beta_j^{'}}{u}$.

One-Virus Model

In this section, we discuss in detail an important special case of model (2.4) and perform an equilibrium and stability analysis for this special case.

We consider the one-virus model (n = 1) and assume that $q_1=q_{21}=q_{31}=\alpha$ ($0.95 \le \alpha \le 1$). This one-virus model is described by the following system of FODE:

$$\begin{aligned} D^{\alpha} T &= \lambda - dT - \beta_1 T I_1, \\ D^{\alpha} I_1 &= \beta_1 T I_1 - a I_1 - \frac{b Z_1 I_1}{T + I_1}, \\ D^{\alpha} Z_1 &= \frac{c Z_1 I_1}{T + I_1} - \delta Z_1. \end{aligned} \tag{3.1}$$

To evaluate the equilibria, let

$$\begin{aligned} D^{\alpha} T &= 0 \\ D^{\alpha} I_1 &= 0 \\ D^{\alpha} Z_1 &= 0. \end{aligned} \tag{3.2}$$

Then system (3.1) has three equilibria: the uninfected equilibrium $E_H = (\lambda / d, 0, 0)$ the boundary equilibrium $E_I = (T^*; I^*, 0)$ where $T^* = \frac{\alpha}{\beta_1}, I_1^* = \frac{\lambda}{\alpha} - \frac{d}{\beta_1}$ and the interior equilibrium $E_c = (\hat{T}, \hat{I}_1, \hat{Z}_1)$, where

$$\hat{T} = \frac{-d + \sqrt{d^2 + 4\lambda\hat{\beta}}}{2\hat{\beta}}, \hat{I} = \frac{\delta}{c - \delta}\hat{T}, \tag{3.3}$$

$$\hat{Z} = \frac{c\hat{T}}{b(c - \delta)}(\beta_1 T - \alpha), \hat{\beta} = \frac{\delta \beta_1}{c - \delta}. \tag{3.4}$$

Following the analysis in [4], we introduce a basic reproduction number which is defined by

$$R_0 = \frac{\lambda \beta_1}{\alpha d}. \tag{3.5}$$

Denote $R_+^3 = \{(T, I_1, Z_1) \in R^3 / T \ge 0, I_1 \ge 0, Z_1 \ge 0\}$ and we always assume that $c > \delta$. Note that $D\alpha Z_1 < 0$ always holds true if $c \le \delta$. By generalized mean value theorem [15], we get $Z_1(t)$ is decreasing if $c \le \delta$.

Next we will discuss the existence and stability of the equilibria of the model (3.1).

Theorem 3.1. (a) *The uninfected equilibrium EH is locally asymptotically stable (LAS) if* $R_0 \in (0,1)$ *and unstable if* $R_0 > 1$.
(b) *If* $R_0 > 1$, *then the boundary equilibrium* E_I *exists. This equilibrium is LAS if* $1 < R_0 < (\alpha\delta / d(c - \delta)) + 1$ *and unstable if* $R_0 > (\alpha\delta / d(c - \delta)) + 1$.
(c) *If* $R_0 > \alpha\delta / d(c - \delta)) + 1$, *then* E_c *exists in* $\mathrm{Int}R_+^3$, *where* $\mathrm{Int}R_+^3$ *is the interior of* R_+^3.
Proof: (a) The Jacobian matrix $J(E_H)$ for system (3.1) evaluated at E_H is given by

$$J(E_H)=\begin{pmatrix} -d & -\dfrac{\beta_1\lambda}{d} & 0 \\ 0 & \dfrac{\beta_1\lambda}{d}-\alpha & 0 \\ 0 & 0 & -\delta \end{pmatrix} \tag{3.6}$$

E_H is locally asymptotically stable if all of the eigenvalues p of the Jacobian matrix $J(E_H)$ satisfy the following condition (6,17):

$$\left|\arg(p)\right| > \frac{\alpha\pi}{2} \tag{3.7}$$

The eigenvalues of $J(E_H)$ are $-d$, $\beta_1\lambda/(d-a)$, $-\delta$. It is clear that E_H is LAS if $R_0<1$ and is unstable if $R_0>1$.

(b) If $R_0>1$, then the existence of E_1 is obvious. The Jacobian matrix $J(E_1)$ for system (3.1) evaluated at E_1 is given by

$$J(E_H)=\begin{pmatrix} -d-\beta_1 I_1^* & -\beta_1 T^* & 0 \\ \beta_1 I_1^* & 0 & -\dfrac{bI_1^*}{T^*+I_1^*} \\ 0 & 0 & \dfrac{cI_1^*}{T^*+I_1^*}-\delta \end{pmatrix} \tag{3.8}$$

For $J(E_1)$ given by (3.8), the characteristic equation becomes

$$[p^2-(d+\beta_1 I_1^*)p+\beta_1^2 I_1^*](p-\frac{cI_1^*}{T^*+I_1^*}+\delta)=0 \tag{3.9}$$

and hence all the eigenvalues

$$\begin{cases} p_{1,2}=\dfrac{-(d+\beta_1 I_1^*)p\pm\sqrt{-(d+\beta_1 I_1^*)^2-4\beta_1^2 I_1^* T^*}}{2}, \\ p_3=\dfrac{cI_1^*}{T^*+I_1^*}-d. \end{cases} \tag{3.10}$$

If $R_0>1$, then $T^*>0$, $I_1^*>0$ and $p_{1,2}$ have negative real parts. Furthermore, if $1<R_0<(\alpha\delta/d(c-\delta))+1$, then $p_3<0$ and E_I is LAS.

If $R_0>(\alpha\delta/d(c-\delta))+1$, then $p_3>0$ and E_I is unstable.

(c) If $R_0>(\alpha\delta/d(c-\delta))+1$, then we obtain $\hat{Z}_1>0$. Thus, E_c exists in Int $\mathrm{Int}R_+^3$. Therefore, the proof is complete.

To discuss the local stability of the interior equilibrium E_c, we consider the linearized system of (3.1) at E_c. The Jacobian matrix at E_c is given by

$$J(E_H)=\begin{pmatrix} -d-\beta_1 I_1^* & -\beta_1\hat{T} & 0 \\ \beta_1 I_1^* + -\dfrac{b\hat{Z}_1\hat{I}_1}{(\hat{T}+\hat{I})^2} & \beta_1\hat{T}-\alpha-\dfrac{b\hat{Z}_1\hat{I}_1}{(\hat{T}+\hat{I})^2} & -\dfrac{b\hat{I}_1}{\hat{T}+\hat{I}} \\ -\dfrac{c\hat{Z}_1\hat{I}_1}{(\hat{T}+\hat{I})^2} & \dfrac{c\hat{Z}_1\hat{T}}{(\hat{T}+\hat{I})^2} & \dfrac{b\hat{I}_1}{\hat{T}+\hat{I}}-\delta \end{pmatrix} \tag{3.11}$$

For convenience, we denote $\hat{T}=T,\hat{I}_1=I,\hat{Z}_1=Z$ and $\beta_1=\beta$. In view of the above assumptions and using

$$\beta T-\alpha-\frac{bZ}{T+I}=0,\lambda-dT-\beta TI=0,\frac{cI}{T+I}=\delta,\frac{T}{T+I}=\frac{c-\delta}{c}, \tag{3.12}$$

$J(E_c)$ can now be written as follows:

$$J(E_c)=\begin{pmatrix} -d-\beta I & -\beta T & 0 \\ \beta I+\dfrac{\delta}{c}(\beta T-\alpha) & \dfrac{\delta}{c}(\beta T-\alpha) & -\dfrac{b\delta}{c} \\ -\dfrac{\delta}{b}(\beta T-\alpha) & \dfrac{c-\delta}{b}(\beta T-\alpha) & 0 \end{pmatrix}. \tag{3.13}$$

Then the characteristic equation of the linearized system of (3.1) is

$$\Phi(p)=p^3+\alpha_1 p^2+\alpha_2 p+\alpha_3=0, \tag{3.14}$$

Where

$$\begin{aligned} \alpha_1&=d+\frac{\delta\alpha}{c}+\frac{\delta^2\beta T}{c(c-\delta)}, \\ \alpha_2&=\beta T(\beta I+\frac{\delta(\beta T-\alpha)}{c})+\frac{\delta(c-\delta)(\beta T-\alpha)}{c}-\frac{\delta}{c}(d+\beta I)(\beta T-\alpha), \\ \alpha_3&=\frac{\delta(c-\delta)(\beta T-\alpha)}{c}-(d+\beta I)+\frac{\beta\delta^2}{c}(\beta T-\alpha). \end{aligned} \tag{3.15}$$

Proposition 3.2. *The interior equilibrium E_c is LAS if all of the eigenvalues p of $J(E_c)$ satisfy* $|\arg(p)>\dfrac{\alpha\pi}{2}$.

Denote

$$D(\Phi)=\begin{vmatrix} 1 & \alpha_1 & \alpha_2 & \alpha_3 & 0 \\ 0 & 1 & \alpha_1 & \alpha_2 & \alpha_3 \\ 3 & 2\alpha_1 & \alpha_2 & 0 & 0 \\ 0 & 3_1 & 2\alpha_1 & \alpha_2 & 0 \\ 0 & 0 & 3 & 2\alpha_1 & \alpha_1 \end{vmatrix}$$

$$=18\alpha_1\alpha_2\alpha_3+(\alpha_1\alpha_2)^2-4\alpha_3\alpha_1^3-4\alpha_2^3-27\alpha_3^2. \tag{3.16}$$

Using the results of [5,20], we have the following proposition:

Proposition 3.3. *One assumes that* E_c *exists in* $IntR_+^3$.

i) *If the discriminant of* $\Phi(p)$, $D(\Phi)$ *is positive and Routh-Hurwitz conditions are satisfied, that is,* $D(\Phi)>0,\ \alpha_1>0,\ \alpha_1\alpha_2>\alpha_3$, *then the interior equilibrium* E_c *is LAS.*

ii) *If* $D(\Phi)<0,\alpha_1>0,\alpha_3>0,\alpha_1\alpha_2=\alpha_3,\alpha\in[0,1]$, *then the interior equilibrium* E_c *is LAS.*

iii) *If* $D(\Phi)<0,\alpha_1<0,\alpha_3<0,\alpha>\frac{2}{3}$, *then the interior equilibrium* E_c *is unstable.*

In our first example we set $\lambda=10, d=0.02$ which are chosen according to [21] and set $\alpha=\delta=0.04, c=b, \beta_1=4\times10^{-4}$ which come from [4]. With these parameter values: $R_0>5/(\alpha\delta/d(c-\delta))+1,\ \alpha_1\alpha_2<\alpha_3$,
$D(\Phi)=-9.4073\times10^{-7}<0$. By Pro.3.2., we obtain the interior equilibrium E_c=(362.0335,19.0544,49.9289) is LAS when $\alpha<0.9916$. Numerical simulations show that trajectories of system (3.1) approach to the interior equilibrium (see Figs. 1(a) and 1(b)) However, when $\alpha=1$ that is the case of classical integer order, E_c is unstable by the Routh-Hurwitz criterion (see Figs. 2(a) and 2(b)).

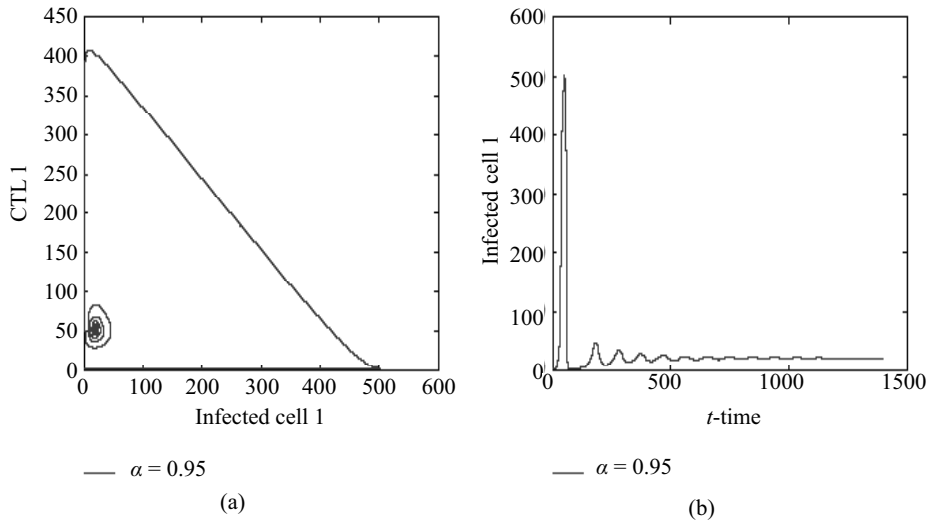

Figure 1 Numerical solutions of system (3.1). The plots show that trajectories of system (3.1) approach to the interior equilibrium for α=0.95.

Two-Virus Model

In this section, we consider viral diversity.We examine the two-virus model using numerical simulations. By examining the behavior of this simpler model we hope to get an idea as to how the more general models in system (2.4) may behave. The two-virus model is given by the following system of FODE:

$$
\begin{aligned}
D^{q_1} T &= \lambda - dT - \beta_1 T I_1 - \beta_2 T I_2, \\
D^{q_{21}} I_1 &= \beta_1 T I_1 - a I_1 - b Z_1 \frac{I_1}{T + I_1 + I_2}, \\
D^{q_{22}} I_2 &= \beta_2 T I_2 - a I_1 - b Z_2 \frac{I_2}{T + I_1 + I_2}, \\
D^{q_{31}} Z_1 &= c Z_1 \frac{I_1}{T + I_1 + I_2} - \delta Z_1, \\
D^{q_{32}} Z_2 &= c Z_2 \frac{I_2}{T + I_1 + I_2} - \delta Z_2,
\end{aligned}
\tag{4.1}
$$

with initial value condition

$$
T(0) = T_0, I_i(0) = I_{i0}; Z_i(0) = Z_{i0}, i = 1.2, \tag{4.2}
$$

where $0.95 \le q_1, q_{2j}, q_{3j}\ (j = 1, 2) \le 1$.

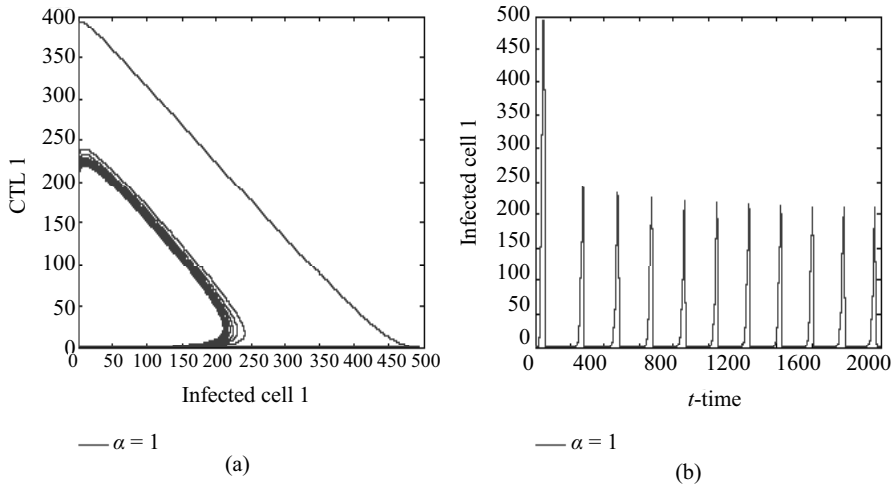

Figure 2 Numerical solutions of system (3.1). The plots show that the interior equilibrium is unstable for $\alpha = 1$.

To find numerical solution to (4.1) and (4.2) in the interval $[0, T]$, we reduce the systems (4.1) and (4.2) to a set of fractional integral equations, by using an equivalence (see [16, Theorem 3.24])

$$
D^{\alpha} X = f(x) \Leftrightarrow X(t) = X(0) + I^{\alpha} f(x). \tag{4.3}
$$

Then we apply the generalized Adams-type predictor-corrector method or, more precisely, Predict, Evaluate, Correct, Evaluate (PECE) methods (see [22, 23]).

For notational convenience, we denote $\alpha = (q_1, q_{21}, q_{22}, q_{31}, q_{32})$.

We carry out numerical simulations for system, (4.1) and (4.2) with parameters $\lambda = 10$, $b = c = 0.08, \alpha = 0.031, \delta = 0.03, d = 0.02, \beta_1 = 4 \times 10^{-4}$ and $\beta_2 = 2.08 \times 10^{-4}$ for the step

size 0.07. Numerical solutions of systems (4.1) and (4.2) support that the system exhibits a chaotic behavior and systems (4.1) and (4.2) have a strange attractor in $IntR_+^5$ for $\alpha = [0.95, 0.95, 1, 1, 1]$ (see Figs. 3(a)–3(c)). It is clear that chaos does exist in our fractional order model with viral diversity as in the case of integer order model. The effect of viral diversity and the frequency dependence results in collapse of the immune system and make the behavior of the system dynamics complex [4]. However, as the value of some component or more components of the order α further decreases, for example, $\alpha = [0.95, 0.95, 0.95, 1, 1]$, the chaotic motion disappears and the systems (4.1) and (4.2) stabilize to a fixed point (see Figs. 4(a)–4(c)).

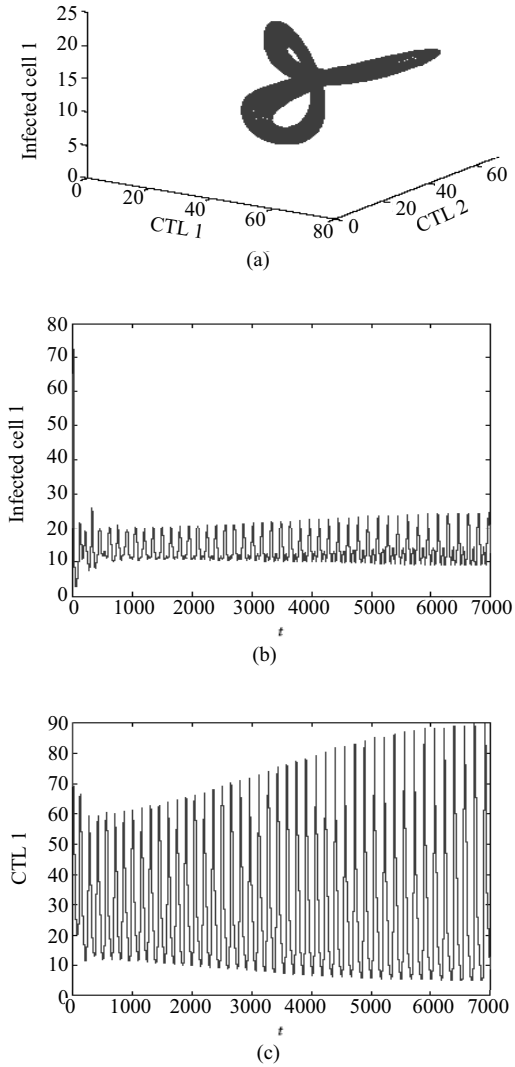

Figure 3 Numerical solutions of system (4.1) for $\alpha = [0.95, 0.95, 1, 1, 1]$. (a) A strange attractor in the Z_1-Z_2-I_1 phase. (b) Infected cell 1. (c) CTL 2.

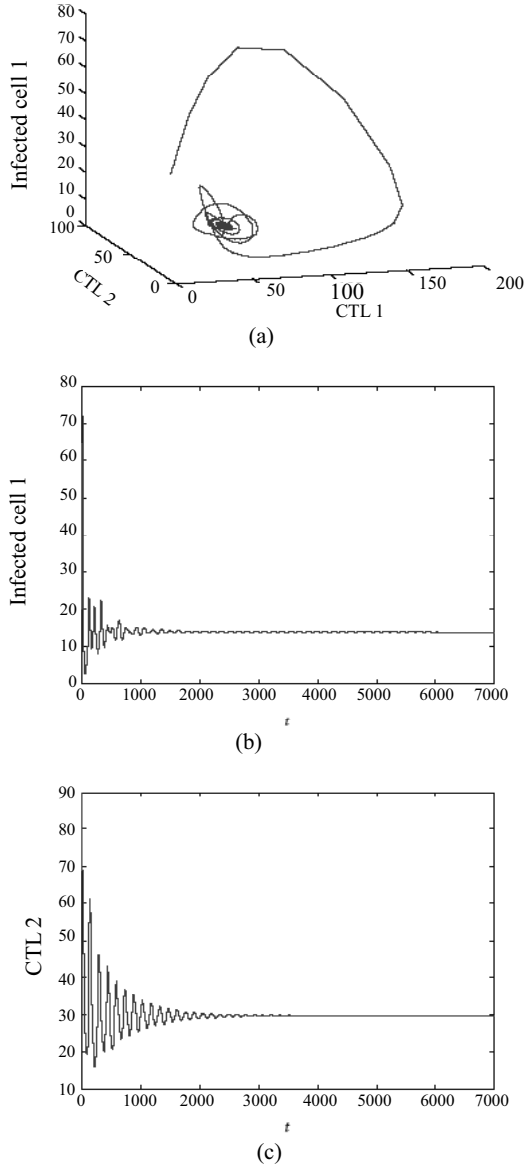

Figure 4 Numerical solutions of system (4.1) for $\alpha = [0.95, 0.95, 0.95, 1, 1]$. (a) Z_1-Z_2-I_1 phase. (b) Infected cell 1. (c) CTL 2.

Conclusions

In this paper, we have proposed a fractional order HIV model, as a generalization of an integer order model, developed by Iwami et al. [4]. The premise of the proposed model is the fact that fractional order models possess memory while the main features of immune response involve memory. It is an attempt to incorporate fractional order into the mathematical model of HIV-immune system dynamics and it is still an interesting exercise to determine, mathematically, how the order of a fractional differential system affects the dynamics of system.

In the case of one-virus model, the fractional order system has an interior equilibrium under some restriction. By using stability analysis on fractional order system, we obtain sufficient condition on the parameters for the stability of the interior equilibrium. Our analysis shows that the interior equilibrium which is unstable in the classical integer order model can become asymptotically stable in our fractional order model. Note that the interior equilibrium is globally asymptotically stable (GAS) (see [24]) if the terms associated with immune reactions are given by cZ_1I_1 and bZ_1I_1 instead of $\frac{cZ_1I_1}{T+I_1}$ and $\frac{bZ_1I_1}{T+I_1}$ in (3.1). That is, the interior equilibrium of the one-virus model can become unstable because of the frequency dependence (see [4]). However, in our fractional order model with the frequency dependence, the interior equilibrium can also become asymptotically stable if the order $\alpha < 0.9916$.

We then consider viral diversity. If the terms associated with immune reaction depend on the mass action law instead of frequency, an interior equilibrium in [24] is GAS. Similar to the integer order model in [4], we find that strange chaotic attractors can be obtained under fractional order model with frequency dependence. That is, the effect of viral diversity and the frequency dependence results in collapse of the immune system and make the behavior of the system dynamics complex. However the chaotic motion may disappear and the fractional order system stabilizes to a fixed point if the value of the order α decreases. The specific biological meaning is deserved to further study.

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Chapter 5

Research on the Relation of EEG Signal Chaos Characteristics with High-Level Intelligence Activity of Human Brain†

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ABSTRACT

Using phase space reconstruct technique from one-dimensional and multi-dimensional time series and the quantitative criterion rule of system chaos, and combining the neural network; analyses, computations and sort are conducted on electroencephalogram (EEG) signals of five kinds of human consciousness activities (relaxation, mental arithmetic of multiplication, mental composition of a letter, visualizing a 3-dimensional object being revolved about an axis, and visualizing numbers being written or erased on a blackboard). Through comparative studies on the determinacy, the phase graph, the power spectra, the approximate entropy, the correlation dimension and the Lyapunov exponent of EEG signals of 5 kinds of consciousness activities, the following conclusions are shown: (1) The statistic results of the deterministic computation indicate that chaos characteristic may lie in human consciousness activities, and central tendency measure (CTM) is consistent with phase graph, so it can be used as a division way of EEG attractor. (2) The analyses of power spectra show that ideology of single subject is almost identical but the frequency channels of different consciousness activities have slight difference. (3) The approximate entropy between different subjects exist discrepancy. Under the same conditions, the larger the approximate entropy of subject is, the better the subject's innovation is. (4) The results of the correlation dimension and the Lyapunov exponent indicate that activities of human brain exist in attractors with fractional dimensions. (5) Nonlinear quantitative criterion rule, which unites the neural network, can classify different kinds of consciousness activities well. In this paper, the results of classification indicate that the consciousness activity of arithmetic has better differentiation degree than that of abstract.

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Introduction

EEG signal is a spontaneous bioelectricity activity that is produced by the central nervous system. It includes abundant information about the state and change of the neural system; therefore it is widely used in clinic and neural-electricity physiological research. In recent years, with the development of the nonlinear dynamics, more and more evidences indicate that the brain is a nonlinear dynamic system, and EEG signal can be regarded as its output [1,2]. In 1985, Babloyantz et al. first put forward that II and IV stage EEG signals of human sleep cycle are chaotic [3]. Hereafter, a large number of study results were reported that the EEG was derived from chaotic systems [4–8]. Therefore, people try to analyze EEG signals by way of nonlinear dynamics to get new knowledge of the brain. Lindenberg, Lehnertz and Ferri et al. researched several kinds of physiological and pathologic conditions; and computed the relevant data under various conditions. They point out finally, the nonlinear characteristic of the physiological EEG signals greatly differs from that of the pathology; when clear-headed, the brain has higher chaotic degree, processes information more quickly and can make more responses [9–12]. Chaos is unordered, but in some situations, it has organizing structures and high order and is the source of system information [13]. Therefore, in this paper, we study the relation of chaos characteristic of EEG signals with high-level intelligence activity of human brain through comparative studies of the nonlinear dynamic characteristic of the dynamic physiological EEG information of brain under different consciousness conditions.

Theory and Method

Chaotic system is described by strange attractors in the phase space [13]. In order to construct the phase space, we adopt the phase space reconstruct technique which was put forward by Packard et al. [14] and made reliable mathematical base by Takens [15]. Its principle is: Reconstruct *m*-dimensional phase space from EEG time series $\{x_n \mid n = 1, 2, \cdots, N\}$, then we get a group of phase space vectors

$$\boldsymbol{X}_i = \{x_i, x_{i+\tau}, \cdots, x_{i+(m-1)\tau}\},\ i = 1, 2, \cdots, M,\ \boldsymbol{X}_i \in R^m;$$

where τ is the time-delay; $m \geq 2\delta + 1$, δ is the number of the system independent variables. M is less than N and they have the same order of magnitude. To reconstruct phase space, it is critical to analyze the phase graph, compute correlation dimension and Lyapunov exponent.

CTM Algorithm and the Determinism Computation of EEG Signals

Whether the brain is a deterministic system, determines the applicability of the nonlinear dynamic method of studying EEG signal [16]. Generally, the deterministic computation of the EEG signal requires much data; and supposes the spread of adjacent lines of EEG series in the phase space are similar. However, unstable data often generates false results. CTM algorithm is a method to express the second-order difference plot (SODP) characteristic of trajectory tangent vector quantificationally. It can be used in the deterministic computation of nonlinear time series effectively. This algorithm is real-time, stable and anti-noisy [17].

The tangent vector of trajectory in the reconstructing phase space is

$$\boldsymbol{Y}(t) = \boldsymbol{x}(t+1) - \boldsymbol{x}(t).$$

The angle between the tangent vectors can be expressed by its cosine value

$$A(t) = \frac{\boldsymbol{Y}(t+1) \cdot \boldsymbol{Y}(t)}{\left\|\boldsymbol{Y}(t+1)\right\| \left\|\boldsymbol{Y}(t)\right\|}.$$

Compared with the angle itself, the cosine value can resist noises better. The SODP of signal expresses the change rate of the tangent vectors angle $A(n+2) - A(n+1)$ to $A(n+1) - A(n)$, its CTM value is

$$\mathrm{CTM} = \frac{1}{N-2} \sum_{n=1}^{N-2} \sqrt{\left[A_{n+2} - A_{n+1}\right]^2 + \left[A_{n+1} - A_n\right]^2}.$$

The value of CTM reflects the smooth degree of the attractors' trajectory: the smaller the CTM value is, the less the changes of tangent vector angle, the smoother the trajectory is; and vice versa. The determinacy of the signal S can be measured by the ratio of the CTM value of the EEG series data and the surrogate data. The bigger S is, the stronger the randomicity of EEG signal is. The researches show: the deterministic signal S < 0.3; the random signal S > 0.7; as to part deterministic signal 0.3 < S < 0.7.

Approximate Entropy

In 1991, Pincus put forward a rule to measure the complexity and the statistic quantification of time series, i.e., approximate entropy [18]. The approximate entropy can weigh the probability of creating new pattern of time series. The bigger the probability is, the more complex the time series gets. Because only less data is needed to compute the stable estimated value of the approximate entropy, the approximate entropy is suitable for the classification of nonsteady consciousness EEG signal. For example, the sampling frequency for most EEG machines are between 100–1000Hz, but computing the approximate entropy needs 100–1000 data points, so the EEG data length used for classification can be taken as 0.5–1s. Although there are false mark disturbance and power frequency disturbance while gathering EEG signals, the EEG data needed is very short. So the approximate entropy has strong anti-chirp and antijamming ability. At present, there is still dispute on whether EEG is derived from chaotic systems or disorderly linear random systems [19]. The approximate entropy is suitable for deterministic and random signal, which further shows that the approximate entropy has better practicability.

The concrete algorithm for approximate entropy is described as follows: Suppose the initial data as $x(1), x(2), \cdots, x(N)$.

1. Form a group of *m*-dimensional vector according to the serial number order: $\boldsymbol{X}(i) = [x(i), x(i+1), \cdots, x(i+m-1)]$ $(i = 1, 2, \cdots, N-m+1)$.
2. Define the distance between $\boldsymbol{X}(i)$ and $\boldsymbol{X}(j)$ as $d[\boldsymbol{X}(i), \boldsymbol{X}(j)] = \max_{k=0\sim m-1} [\left|x(i+k) - x(j+k)\right|]$, and compute the distance $d[\boldsymbol{X}(i), \boldsymbol{X}(j)]$ between $\boldsymbol{X}(i)$ and other vectors $\boldsymbol{X}(j)$ $(j = 1, 2, \cdots, N-m+1; j \neq i)$ for every *i* value.

3. Given the threshold value *r*, count the number of $d[\boldsymbol{X}(i), \boldsymbol{X}(j)]$ which is smaller than *r* for every *i* value, and compute the ratio of this number to the total distance $N-m$: $C_i^m(r) = \frac{1}{N-m}\{\text{number of } [d[\boldsymbol{X}(i), \boldsymbol{X}(j)] < r]\}$ $(i = 1, 2, \ldots, N-m+1)$.
4. The average value of *i* is computed according to logarithm of $C_i^m(r)$: $\phi^m(r) = \frac{1}{N-m+1}\sum_{i=1}^{N-m+1} \ln C_i^m(r)$.
5. Add the dimension by 1 again to $m+1$, repeat steps (1) to (4), and compute $C_i^m(r)$ and $\phi^m(r)$.
6. The theoretical value of the approximate entropy is $ApEn(m,r) = \lim_{N\to\infty}[\phi^m(r) - \phi^{m+1}(r)]$.

Generally speaking, the boundary value mentioned above exists by probability 1. *N* can't be ∞ in practice. When *N* is a finite value, the result is the estimated value of *ApEn* when the series length is *N*, which is defined as $ApEn(m,r,N) = \phi^m(r) - \phi^{m+1}(r)$. Obviously, the value of *ApEn* is related with the value of *m* and *r*. According to Pincus's work, $m = 2$ and $r = 0.1 : 0.25SD_x$ are suggested (SD_x is the standard deviation (SD) of initial data $x(i)$ (i = 1, 2,..., *N*).

Multi-lead Correlation Dimension

In the study of nonlinear dynamics of EEG signals, the Takens's time delay reconstruction phase space method used EEG data of single channel record to reconstruct multi-dimensional EEG attractor, which reflects the time correlation of the system. In order to show the characteristic of the system from time and space, Eckmann and Ruelle proposed the multichannel reconstructing (multivariable embedding) method that can show the correlation of space and time simultaneously. When applied in time series with short-time noise, it can avoid problems such as the choice of delayed parameters and system errors with higher embedding dimension. Rombouts et al. thought the multichannel reconstructing method can provide more reliable results [20]. Take EEG signals as an example, recording variable of each lead is taken as a component of the reconstructing vector while reconstructing, the reconstructing dimension is decided by the electrode number of EEG signals.

Based on the multi-lead data, the principal step of computing the correlation dimension with GP algorithm [21] is: The *m*-dimensional embedding-space $\{\boldsymbol{X}\}$ is got from *m*-lead observing time series.

1. Suppose $\boldsymbol{X}(n) = \{x_1(n), x_2(n), \ldots, x_m(n)\}$ $(n \le N, m \le M)$, here *m* is the number of the required variables.
2. For a given distance *r*, compute the correlation integral $C(r) = \frac{2}{N(N-1)}\sum_{j=1}^{N-1}\sum_{i=j+w}^{N} H(r - \|\boldsymbol{X}_i - \boldsymbol{X}_j\|)$, here $\boldsymbol{X}$ is the vector in embedded space, *N* is the number of the vector, *w* is Theiler window, *H* is Heaviside function.

3. For an enough small *r*, the correlation integral approaches to the following formula: $LnC_m(r) = LnC + d_m Ln(r)$.
4. Evaluate the slope of the fitting straight line in the linearity range of $LnC_m(r) \cong Ln(r)$, namely the estimated value of the correlation dimension D_2.

Generally speaking, the correlation dimension of EEG represents the invariable measure for the self-similarity and the criterion irrelevance of the EEG signal, and shows the complex degree of the EEG signal.

Small Data Sets Method of Computing Lyapunov Exponent

The ordinary method of studying whether the actual observable series has chaotic characteristic or not, is to compute the biggest Lyapunov exponent λ_1 of the observable series. When $\lambda_1 > 0$, the observable system is believed to be chaotic. Since Wolf proposed and computed the Lyapunov exponent according to the observable series in 1985, there are some sophisticated methods in this respect, such as Jacobian method, *p* norm method and the small data sets method proposed by Rosenstein et al. [22]. The small data sets method is more robust than other methods to embedded dimension of the phase space, the reconstruction time delay, observable noises and so on.

Mark the constructed phase space as $\mathbf{X}=[\mathbf{X}_1,\mathbf{X}_2,\ldots,\mathbf{X}_N]$, phase point is $\mathbf{X}_j = \left[x_{j-(m-1)J}, x_{j-(m-2)J}, ..., x_j\right] (j = 1,2,..., N)$, here N is the total number of the phase points, m is the embedding dimension of the phase space, J is the reconstructing time delay. Generally, $J = k\Delta t$, k is a positive integer, Δt is sampling interval. For $\forall\, \mathbf{X}_j \in \mathbf{X}$, define $d_j(0) = \inf_{\mathbf{X}k\in} \left\|\mathbf{X}_j - \mathbf{X}_k\right\| = \left\|\mathbf{X}_j - \mathbf{X}_{\hat{j}}\right\|$, and $\left|j - \hat{j}\right| > p$, p is the average cycle of the time track. If $\exists \boldsymbol{X}_{j+i} \in \mathbf{X}$ and $\mathbf{X}_{\hat{j}+i} \in \mathbf{X}$, define $d_j(i) = \left\|\mathbf{X}_{j+i} - \mathbf{X}_{\hat{j}+i}\right\|$, then the advanced distance $d_j(i)$ has the following approximate relation

$$d_j(i) \approx d_j(0) e^{\lambda_1 i \Delta t} \tag{1}$$

here Δt is the sampling interval or the step length of the observable series; i is the sliding step ordinal of the phase point along the time track. Take natural logarithm to both sides of the formula (1), we can get $\ln d_j(i) \approx \ln d_j(0) + \lambda_1 i \Delta t$. When $d_j(i) = \left\|\boldsymbol{X}_{j+i} - \boldsymbol{X}_{\hat{j}+i}\right\|$ ($\|\cdot\|$ denotes the vector 2 norm), we get the empirical formula which Rosenstein et al. used to compute λ_1 [22]. In view of the influence of local computation, the last empirical formula is

$$\frac{1}{\Delta t} < \ln d_j(i) > \approx \frac{1}{\Delta t} < \ln d_j(0) > + \lambda_1 i .$$

Here $<\cdot>$ is to get average.

Power Spectra

Using Auto-Regressive (AR) parameter model method to compute the self power spectra estimated value of the EEG signal [23]: The AR model of the EEG time series x_n is provided by the following formula

$$x_n = -\sum_{k=1}^{p} a_k x_{n-k} + w_n \tag{2}$$

here p is the order of the AR model; $a_k (k = 1,2,\cdots p)$ is AR model parameter; w_n is the unpredictable part of x_n, namely residual error. If the model can well match the EEG time series, w_n should be white noise process. According to the AR model given by formula (2), we can get the estimated value of the AR spectra

$$P_x(\omega) = \frac{\sigma_\omega^2}{\left|A(e^{j\omega})\right|^2} = \frac{\sigma_\omega^2}{\left|1 + \sum_{k=1}^{p} a_k e^{-j\omega k}\right|^2} \tag{3}$$

here σ_ω^2 is the variance of AR model residual error. From the formulas (2) and (3), we know the key to get the AR spectra estimation is to estimate the AR parameters $a_k (k = 1,2,\cdots p)$ through the EEG time series. Usually, Yule-Walker equation and Levinson-Durbin algorithm are used to estimate AR parameters. In this paper, we use Burg algorithm. Burg algorithm is an autoregression power spectra estimated method, on the premise of Levinson-Durbin recursion restraint, making the sum of the front and back forecast error energy smallest. Burg algorithm avoids the computation of self-correlation function. It can distinguish the extremely close sine signal in low noise signals, and may use less data record to estimate, and the result is extremely close to real values. Moreover, the forecasting error filter obtaining from Burg algorithm is minimum phase. The choice of the model order p is a critical problem in the AR model spectra estimate. If p is too low, it will cause smooth spectra estimate; while if p is too high, it will cause spectral line excursion and spectral line abruption and generate general statistic instability. In this paper, we adopt Akaike information criterion (AIC) to estimate the value of the order

$$AIC(p) = N \ln \hat{\rho}_p + 2p,$$

here N is the number of the data points, $\hat{\rho}_p$ is the estimated value of the white noise variance (forecasting error power) of p order AR model.

SOM Neural Network

The neural network is a highly nonlinear system; and it also shares similar characteristics with brain, so it is used in various classifications extensively. SOM neural network is composed of entire connection neuron array and it is a non-teacher, self-organizing and self-learning network. Its idea is that neurons in different areas of the space have different functions. When the neural network accepts an external input mode, it will be divided into different response areas, and each area has different response characteristics to the input mode.

A typical characteristic of SOM network is that it can generate the characteristic topology classification of input signal on one-dimensional or two-dimensional processing unit array, so the SOM network can extract the pattern characteristics of the input signal.

Generally, SOM network only includes one-dimensional array and two-dimensional array, but it can also be generalized into multidimensional processing unit array. This research uses two-dimensional array. SOM network is made up of the following four parts.

1. Processing unit array. Using to accept the input event and forming "discriminant function" of these signals.
2. Comparison and choice of mechanism. Using to compare the "discriminant function". And choosing one processing unit which has the biggest output value.
3. Partial interconnection action. Using to drive the chosen processing unit and the processing unit closest to it simultaneously.
4. Adaptive process. Using to revise the parameter of driven unit in order to increase its output value to the specific input "discriminant function".

Experiment and Result

EEG Data Source

The data used in this paper is the consciousness activities EEG data of 7 subjects that offered by the EEG research center of Colorado State University [24]. There are five kinds of human consciousness activities, i.e., relaxation, mental arithmetic of multiplication, mental composition of a letter, visualizing a 3-dimensional object being revolved about an axis, and visualizing numbers being written or erased on a blackboard [25]. The experimental process of data acquisition is: Subjects sit in the sound-insulated and light-weak room with the electrode cap and complete some consciousness tasks according to the indications. The corresponding electrical signals of the brain will be recorded. The electrode is laid in C3, C4,P3,P4,O1,O2 and EOG (Electro-Oculogram) altogether 7 leads according to international $10 \sim 20$ system standard. The sampling frequency is 250Hz, the simulative filtering range is $0.1 \sim 100$Hz. Signals polluted seriously by winks are excluded. Experimental data of each consciousness task last 10s. Figure 1 is the EEG signal of subject 1 while relaxing. It is obvious that even under relaxing conditions; healthy people's EEG signals fluctuate in a complicated way, which contains abundant nonlinear dynamic information.

Phase Graph Analysis

Using the phase space reconstruct technique from one-dimensional time series to determine the time delay τ: In the experimental system, it should be through repeated trial method to confirm choice of τ. If τ is undersize, the track of the phase space will approach to a straight line; per contra τ is oversize, the data point will centralize in a small range of the phase space, and we can't get the attractors' local structures from the reconstructed phase graph [13]. Testing repeatedly, we find that selecting $\tau = 3$, data point $N = 2000$, it can well reconstruct the EEG attractors. We construct the EEG attractors of all five kinds of consciousness activities of 7 subjects and find that EEG attractors of various patterns have similar characteristics.

Figure 2 is a representative one. As can be seen from Fig. 2, the attractors' track often rotate in an extremely complex way, even smear a group black in the plane, but there is

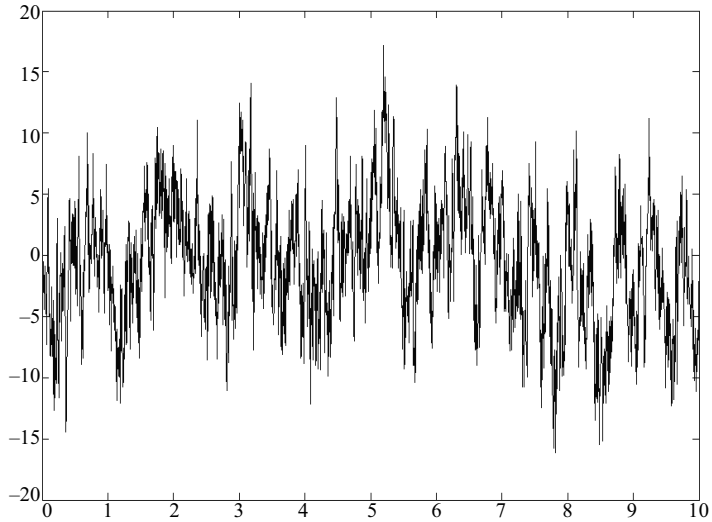

Figure 1 EEG signal waveform of subject 1 while relaxing.

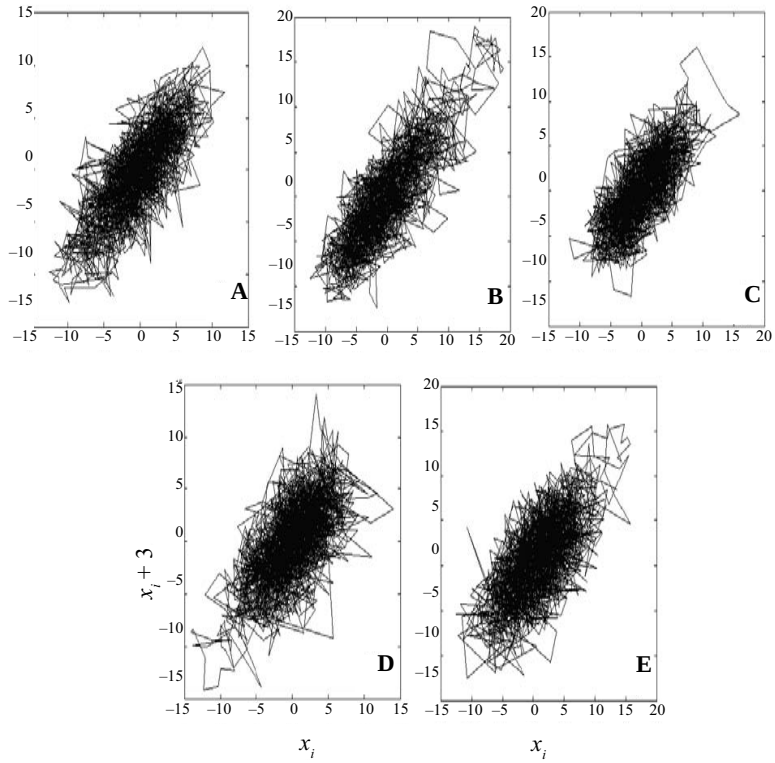

Figure 2 EEG attractors of five kinds of consciousness activities of subject 1. (a) Relaxation. (b) Mental arithmetic of multiplication. (c) Mental composition of a letter. (d) Visualizing a 3-dimensional object being revolved about an axis. (e) Visualizing numbers being written or erased on a blackboard.

still internal structure when the attractors is magnified. The attractors of relaxation, mental composition of a letter and visualizing a 3-dimensional object being revolved about an axis often distribute in a small ellipse region, while the point in the attractors of mental arithmetic of multiplication and visualizing numbers being written or erased on a blackboard centralize nearby the 45 degree line and there is a large distributing range along the 45 degree line. This is because while proceeding rational computation such as mathematics or imagination, the value of the adjacent sampling points of EEG signals are close, and the amplitude values of the whole EEG signals are great.

Power Spectra Analysis

Using the AR parameter model method, we select 250Hz sampling frequency to compute the power spectra of five kinds of tasks' EEG signal of 7 subjects. The parameters used in analysis are: the length of FFT M: 1024; the total number of the data N: 6000; order p: 320. By comparison of the power spectra of five kinds of tasks of 7 subjects, we find that the power spectra of five kinds of tasks for identical subject are similar and meet $1/f$ distribution. As can be seen from Fig. 3, although the attractors' difference is great (Fig. 2 (b) and 2(d)), their power spectra (Fig. 3(a) and 3(b)) show certain similarity. The peak in the high-frequency in Fig.3 is caused by the power frequency disturbance.

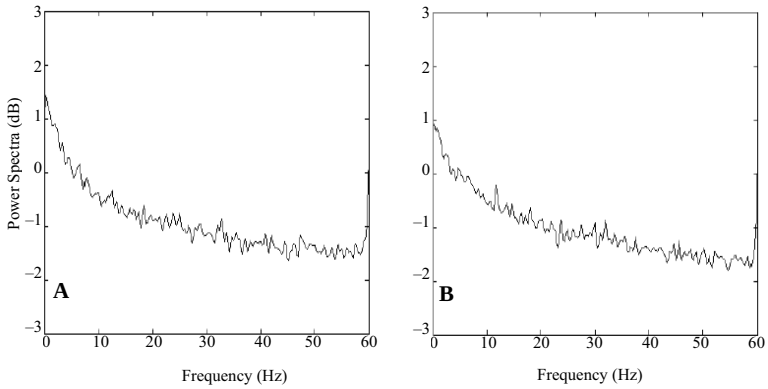

Figure 3 EEG power spectra of 2 kinds of tasks of subject 1. (a) Mental arithmetic of multiplication. (b) Visualizing a 3-dimensional object being revolved about an axis.

Practice prove: The EEG of human can be divided into four frequency sections: δ wave: the frequency is 1-4Hz, appears while sleeping, anaesthetizing deeply, oxygen deficit or the brain with organic disease; θ wave: the frequency is 4–8Hz, appears while feeling sleepy; α wave: the frequency is 8–13Hz, appears while closing eyes with clear-headed; β wave: the frequency is 14–30Hz, appears while opening eyes and looking at things or thinking. As can be seen from Fig. 3, although the spectral lines are similar, there are differences in the active frequency bands (8–30Hz) of different consciousness. So we add the energy of 8–13Hz and 14–30Hz separately in order to use it in SOM network to classify the consciousness.

CTM and the Deterministic Computation of the Signals

EOG signal is the main disturbance of each lead EEG signal, so we make a relevant analysis separately between the gathered EOG signal and another 6 leads in order to find several leads which are disturbed less. We choose $\tau = 3$ and $m = 16$ to compute the CTM. The method of surrogate data [26,27] is used to help detect nonlinear determinism. The surrogate data are linear stochastic time series that have the same power spectra as the EEG signal series. In this paper, we use "iteratively refined surrogate data", which have the same autocorrelation function, Fourier power spectrum, and probability distribution as the EEG time series. More detailed algorithms used in this study are present in the paper of Schreiber and Schmitz [27].

Figure 4 gives the statistic average histogram for each task of 100 times testing. It is obvious that the value of CTM accords with the phase graph 3 well. The statistic average results of the deterministic computations of the EEG signals are in the interval of $0.3 < S < 0.7$. It offers strong support that human brain which contains chaotic component is a highly nonlinear system. But while proceeding deterministic tests, we also find that its value's fluctuation is very big. As an empirical algorithm, when there is less data sample, its application also has certain limitation.

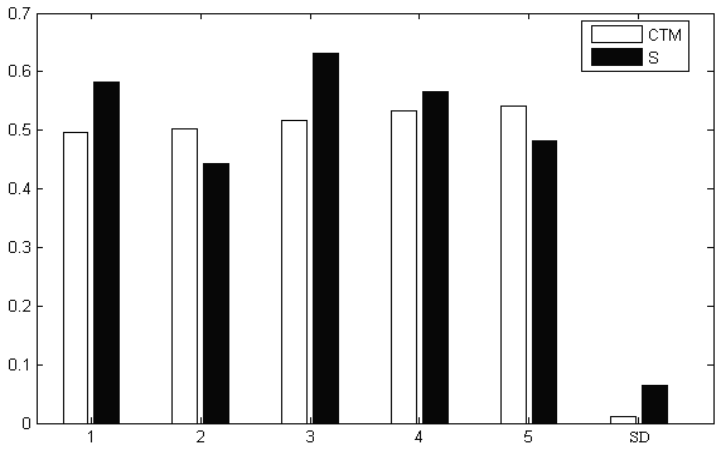

Figure 4 Statistic average histogram of CTM and S of 5 tasks of subject 1 (SD denotes standard deviation).

Approximate Entropy Computation

According to the characteristics of the processing data, we choose $r = 0.5SD_x$ and $r = SD_x$. The approximate entropy to 100 groups of data is computed separately. Because the data gathered from different electrodes may be asynchronous, we make interval eliminations to those unsuitable data. Figure 5 provides the statistic average histogram of the approximate entropy when $r = 0.5SD_x$ and $r = SD_x$.

Correlation Dimension Computation

According to the characteristics of the processing data, we precondition the EEG data first. Namely make a relevant analysis between the EOG and other leads, and sort them according to the order from weak to strong. Then carry through the phase space reconstruction. According to the discussion by Brandstater and Swinney [13]: The fluctuation of partial derivative in scale-free region should be less than 1%. Thus, the scale-free region can be determined. Then the least square method can be used to obtain the correlation dimension. After iterative trials, we found that the correlation dimension can be exactly determined with $m > 12$. Therefore, in these experiments, we choose $\tau = 3$ and $m = 16$ to compute the data of 4 subjects and each contains ten groups separately.

From Fig. 5, we can see, the consciousness activities (task 2 and 5), with more rational consciousness such as arithmetic, have relatively weaker ability to generate new pattern; while those consciousness activities (task 4), with more abstract consciousness such as visualizing graph rotating, have relatively stronger ability to generate new pattern, which means that the time series have more complexity. This also corresponds to the practice. Because mathematical computation is based on fixed rule, its ability to create new pattern ingredient is naturally lower.

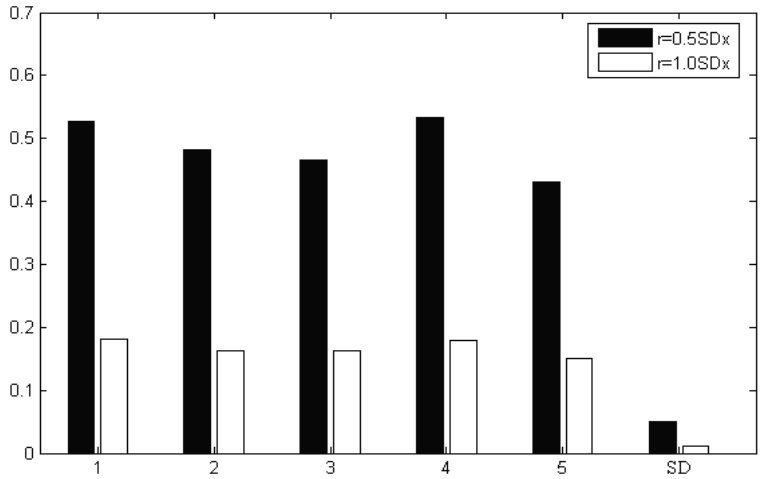

Figure 5 Statistic average histogram of the approximate entropy of subject 1 when $r = 0.5SDx$ and $r = 1.0SDx$.

Figure 6(a) is a representative curve *LnC*(*r*) *vs Ln*(*r*) of subject 1 while relaxing. Figure 6(b) provides the statistic results of the correlation dimension D_2 of 10 groups of data of five kinds of human consciousness activities (each vertical line represents the mean square error range of each task, the crossing point between the crosswise fold line and the vertical line is the mathematic expectation of the task). We can see from Fig. 6(b): For the same subject, do the same kind of tests in different time, its D_2 value may have great fluctuation, which means human brain has different excitable degree in different time slice. Figure 6(b) also shows that the error fluctuation of D_2 is minimum when implementing

mathematical computation (task 2). This is because mathematical computation can make the spirit centralized more easily than other consciousness activities. In addition, we also compute the data of D_2 for other 3 subjects and each contains 10 groups of data. These D_2 will also be used in the ideology classification of the SOM.

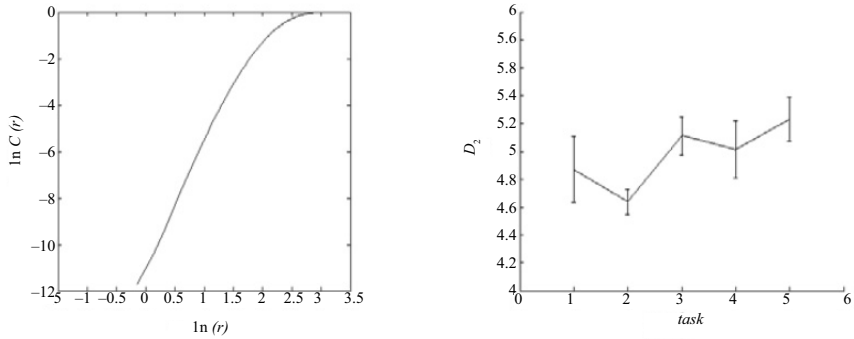

Figure 6 The result of the correlation dimension D_2 of subject 1. (a) *LnC*(*r*) *vs Ln*(*r*) curve while relaxing. (b) D_2 of 5 kinds of tasks and each contains 10 groups of EEG data.

Lyapunov Exponent Computation

Different consciousness activities stimulate different cerebrum regions, so the computation of single lead signal can't reflect the synthetic Lyapunov exponent of the brain consciousness activity well. The embedded dimension m is determined by iteratively trials. For the delay τ, the phase space of EEG signals is projected into the two-dimension plane. If τ is too small, the attractors will muster around the line $y = x$. If τ is too large, $m\tau$ will be much more than the average period. On this basis,τ is determined by iteratively trials. Furthermore, considering the fact that for different consciousness, different cerebrum region has different activity degree, implement sample splicing to the sampling data of each lead with $\hat{o} = 3$, $m = 16$ to reconstruct the phase space. Figure 7 is the biggest Lyapunov exponent λ_1 of 10 groups of EEG data with five kinds of human consciousness activities (each vertical line represents the result of mean square error range of each task, the crossing point between the histogram and the vertical line is the mathematic expectation of the task). It is obvious that the biggest Lyapunov exponents λ_1 of five human consciousness activities are all bigger than zero, which proves that human brain activity is chaotic.

SOM Network Consciousness Classification

The purpose of the investigation in this paper is to classify the intelligence consciousness activities. From the analysis above, we know that for the same subject, the methods described above may have better differentiation degree; but for different subjects, the above methods have difficulties to classify the consciousness activities, which also indicates that the brain is a highly complicated nonlinear system.

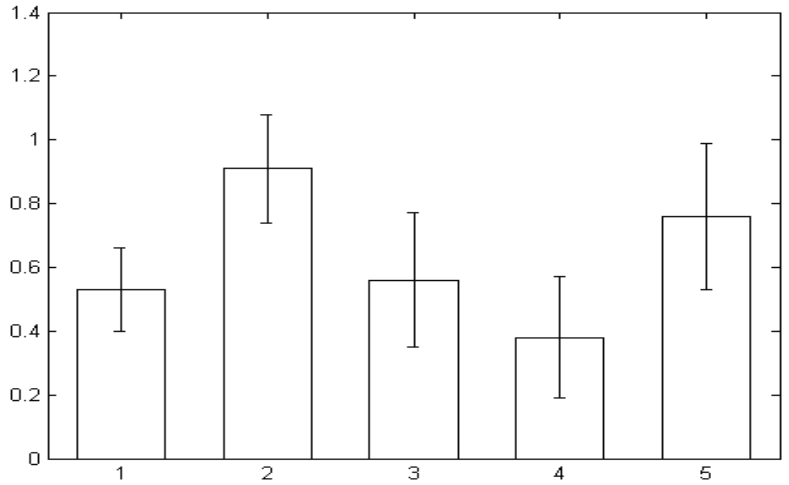

Figure 7 λ_1 of 5 kinds of tasks and 10 groups of EEG data of subject 1.

Therefore, we make the nonlinear criterions (mentioned above) into the prophase processing module, and input them to the input unit of the SOM network. That is, the SOM network has six inputs, including power spectra, CTM, S, approximate entropy, correlation dimension and Lyapunov exponents. According to the tests of the data, the competitive layer of the network is chosen as 8×6 structure. The predicted results of the network are shown in Fig. 8. In Fig. 8, the horizontal ordinate denotes the five outputs of the SOM network, and the vertical ordinate denotes the correct resolution. Figure 8(a) shows the correct resolution histogram of mixed tasks of single subject. In Fig. 8(a), the outputs of the SOM network are the mixed tasks which are combined in turn from the five kinds of human consciousness activities, i.e., relaxation, mental arithmetic of multiplication, mental composition of a letter, visualizing a 3-dimensional object being revolved about an axis, and visualizing numbers being written or erased on a blackboard. For example, "3" represents the combination of three tasks, i.e., relaxation, mental arithmetic of multiplication, and mental composition of a letter. Figure 8(b) shows the correct resolution histogram of four subjects. In Fig. 8(b), the outputs of the SOM network are the five individual tasks mentioned above. As can be seen from Fig. 8(b), the resolution of mathematical computation is relatively higher, while the resolutions of other tasks are about equivalent. The authors think that this is because the nonlinear quantitative parameters of the mathematical computation have great difference compared with other tasks. As can be seen from Fig. 8(a) and Fig. 8(b), the resolution of multi-individual drops obviously relative to single individual. This is because the nonlinear quantitative parameters of two subjects differ greatly, which makes the resolution of the network details drop. There will be better results if there are more individuals to train the network.

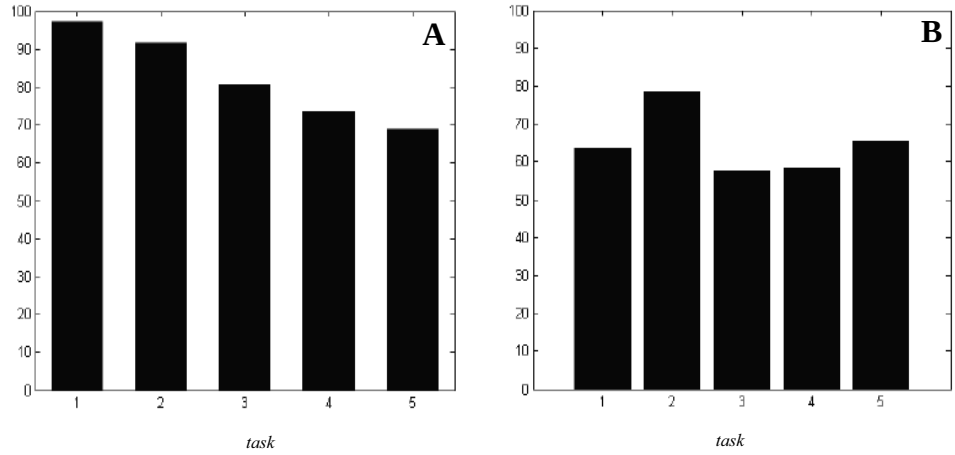

Figure 8 The predicted result of SOM network. (a) The correct resolution histogram of mixed tasks of single subject. (b) The correct resolution histogram of 4 subjects and 5 kinds of tasks'.

Discussion and Conclusion

1. In this paper, we use the determinacy, the phase graph, the power spectra, the approximate entropy, the correlation dimension and the Lyapunov exponent method etc. to study the EEG signal of 5 kinds of consciousness activities of 7 subjects. Although every method has merits and faults, the results show the nonlinear dynamic characteristics of the subject's brain from different perspective. Thereinto, from the deterministic computation we know that the EEG signal is between random signal and deterministic signal. This indicates that the brain may be a chaotic system. The analysis of the power spectra shows that various ideology of single subject is almost identical, but the activity frequency channels for different consciousness activities are different slightly. The analysis of the approximate entropy presents the degree of various consciousness activities on generating new pattern. The approximate entropy of different subjects exist discrepancy. The authors think that at the same state, the larger approximate entropy of the subject, the more innovational he has. The correlation dimension shows the change of chaos of different consciousness activities well, which can better indicate the activity degree of human consciousness, combining with the approximate entropy and the Lyapunov exponent. The above analyses indicate: Different consciousness activities have profound nonlinear dynamic differences. Some differences are difficult to perceive, and the nonlinear quantitative parameters of different individuals have great differences. So it is a critical problem to find a widely applicable criterion, which needs to be explored for a long time.