An Overview of Complex Adaptive Systems

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Abstract

Almost every biological, economic and social system is a complex adaptive system (CAS). Mathematical and computer models are relevant to CAS. Some approaches to modeling CAS are given. Applications in vaccination and the immune system are studied. Mathematical topics motivated by CAS are discussed.

Keywords: Complex adaptive systems; The immune system; Cellular automata; Game theory; Complex networks; Multi-objective optimization.

1 Basics of complex adaptive systems (CAS)

Definition (1): A complex adaptive system consists of inhomogeneous, interacting adaptive agents. Adaptive means capable of learning.

Definition (2): An emergent property of a CAS is a property of the system as a whole which does not exist at the individual elements (agents) level.
Typical examples are the brain, the immune system, the economy, social systems, ecology, insects swarm, etc..

Therefore to understand a complex system one has to study the system as a whole and not to decompose it into its constituents. This totalistic approach is against the standard reductionist one, which tries to decompose any system to its constituents and hopes that by understanding the elements one can understand the whole system.

2 Why should we study complex adaptive systems?

Most of living systems are CAS. Moreover they have intrinsic unpredictability which causes some ”seemingly wise” decisions to have harmful side effects. Therefore we should try to understand CAS to try to minimize such side effects. Here we give two examples of these side effects.

Mathematical models have played important roles in understanding the impact of vaccination programs. The complications of infectious diseases spread make the problem of predicting the impact of vaccinations a non-linear problem. Sometimes a counter-intuitive result appears e.g. the threshold phenomena [Edelstein-Keshet 1988]. Here another example will be mentioned.

Several vaccination programs are known e.g. mass vaccination where all population is vaccinated, target vaccination where only a certain group is vaccinated.

If one tries to understand the expected impact of a vaccination program one should take the following points into account:

1. Vaccination is not perfect hence a probability of vaccination failure should be assumed.

2. Sometimes vaccination takes time to be effective.

3. Immunity is waning i.e. may be lost with time.

4. Long range contacts can play a significant role e.g. SARS (severe acute respiratory syndrome) has been transmitted between countries via air travellers.
Rubella is a mild viral infectious disease. Typically it is most dangerous when infecting a pregnant female where it has severe effects on the fetus. Once one gets it he (she) gets a life long immunity. There are several vaccination strategies for rubella [Vynnycky et al 2003]. The US policy is to vaccinate all two years old children. The UK policy is to vaccinate only 14-years old girls. Another strategy which is adopted in some underdeveloped countries is not to vaccinate at all. It has been found [Jazbec et al 2003] that in most cases the UK strategy is equal or better than the US one despite being cheaper.

An interesting situation arose when some countries adopted a private sector vaccination to MMR (Measles, Mumps and Rubella) [Vynnycky et al 2003]. It was expected that the number of Congenital Rubella Syndrome (CRS) will decrease. However it did not and in some countries (e.g. Greece and Costs Rica) it increased. The reason can be understood as follows: This vaccination to part of the population decreases the probability of contracting the disease at young age. Hence the number of susceptible individuals at adulthood increases. Consequently the probability of contracting the disease at adulthood increases. This is an example of the counterintuitive effects of some vaccination programs.

Another example for bad side effects is Lake Victoria [Chu et al 2003] where a new species called Nile perch was introduced expecting that it is more economically profitable. Yet the following results have appeared:

(i) The local fishermen’s tools were not suitable for the new fish hence only large corporations benefited.

(ii) Due to its higher price the locals were unavailable to buy the new type.

(iii) The original fish used to eat the larva of mosquitoes but now mosquitoes’ numbers have increased significantly thus the quality of life of the locals have deteriorated!!

There are at least two sources for unpredictability in CAS. The first is the nonlinear interactions between its agents [West 1990]. The second is that CAS are open systems hence perturbation to one system may affect another related one e.g. perturbation to Lake Victoria affected the number of mosquitoes.
3 How to model a CAS?

The standard approaches are

1. Ordinary differential equations (ODE), difference equations and partial differential equations (PDE).
2. Cellular automata (CA) [Ilachinski 2001].
3. Evolutionary game theory [Hofbauer and Sigmund 1998].
4. Agent based models.
5. Networks [Watts and Strogatz 1998] etc..
6. Fractional calculus [Stanislavsky 2000].

Some of these approaches are included in [Boccaro 2004].

The ODE and PDE approaches have some difficulties as follows [Louzon et al 2003]:

(i) ODE and PDE assumes that local fluctuations have been smoothed out.

(ii) Typically they neglect correlations between movements of different species.

(iii) They assume instantaneous results of interactions.

Most biological systems show delay and do not satisfy the above assumptions. They concluded that a cellular automata (CA) [Ilachinski 2001] type system called microscopic simulation is more suitable to model complex biological systems. We agree that CA type systems are more suitable to model complex biological systems but such systems suffer from a main drawback namely the difficulty of obtaining analytical results. The known analytical results about CA type systems are very few compared to the known results about ODE and PDE. Some mathematical results about CA are given in the appendix.

Now we present a compromise i.e. a PDE which avoids the delay and the correlations drawbacks. It is called telegraph reaction diffusion equations
To overcome the non-delay weakness in Fick’s law it is replaced by

\[ J(x, t) + \tau \frac{\partial J(x, t)}{\partial t} = -D \frac{\partial c}{\partial x}, \]  

(1)

where the flux \( J(x, t) \) relaxes, with some given characteristic time constant \( \tau \) and \( c \) is the concentration of the diffusing substance. Combining Eq. (1) with the equation of continuity, one obtains the modified diffusion equation or the Telegraph equation:

\[ \frac{\partial c}{\partial t} + \tau \frac{\partial^2 c}{\partial x^2} = D \frac{\partial^2 c}{\partial x^2}. \]  

(2)

The corresponding Telegraph reaction diffusion (TRD) is given by

\[ \tau \frac{\partial^2 c}{\partial t^2} + \left(1 - \frac{df(c)}{dc}\right) \frac{\partial c}{\partial t} = D \frac{\partial^2 c}{\partial x^2} + f(c), \]  

(3)

where \( f(c) \) is a polynomial in \( c \).

Another motivation for TRD comes from media with memory where the flux \( J \) is related to the density \( c(x, t) \) through a relaxation function \( K(t) \) as follows

\[ J(x, t) = -\int_{0}^{t} K(t - \tau) \frac{\partial c(x, \tau)}{\partial x} d\tau. \]

It can be shown [Compte & Metzler 1997] that, with a suitable choice for the kernel \( K(t) \), the standard Telegraph equation is obtained.

A third motivation is that starting from discrete space time one does not obtain the standard diffusion equation but the telegraph equation [Chopard and Droz 1991].

Moreover it is known that TRD results from correlated random walk [Diekmann et al, 2000]. This supports the conclusion that Telegraph reaction diffusion equation is more suitable for modeling complex systems than the usual diffusion one.
4 The immune system as a complex system
[Segel and Cohen 2001, Ahmed and Hashish 2004]

The emergent properties of the immune system (IS) included:

* The ability to distinguish any substance (typically called antigen Ag) and determine whether it is damaging or not. If Ag is non-damaging (damaging) then, typically, IS tolerates it (responds to it).

* If it decides to respond to it then IS determines whether to eradicate it or to contain it.

* The ability to memorize most previously encountered Ag, which enables it to mount a more effective reaction in any future encounters. This is the basis of vaccination processes.

* IS is complex thus it has a network structure.

* The immune network is not homogeneous since there are effectors with many connections and others with low number of connections.

* The Ag, which enters our bodies, has extremely wide diversity. Thus mechanisms have to exist to produce immune effectors with constantly changing random specificity to be able to recognize these Ag. Consequently IS is an adaptive complex system.

* Having said that, one should notice that the wide diversity of IS contains the danger of autoimmunity (attacking the body). Thus mechanisms that limit autoimmunity should exist.

* In addition to the primary clonal deletion mechanism, two further brilliant mechanisms exist: The first is that the IS network is a threshold or ”window” one i.e. no activation exists if the Ag quantity is too low or too high (This is called low and high zone tolerance).

* Thus an auto reactive immune effector (i.e. an immune effector that attacks the body to which it belongs) will face so many self-antigens that it has to be suppressed due to the high zone tolerance mechanism.
* Another mechanism against autoimmunity is the second signal given by antigen presenting cells (APC). If the immune effector is self reactive then, in most cases, it does not receive the second signal thus it becomes anergic.

* Also long term memory can be explained by the phenomena of high and low zone tolerance where IS tolerates Ag if its quantity is too high or too low. So persisting Ag is possible and continuous activation of immune effectors may occur.

* There is another possible explanation for long term memory using the immune system (Extremal Dynamics).

* Thus design principles of IS can explain important phenomena of IS.

An interesting example is given by Matzinger [Matzinger 2002] where she argued that to prevent transplant rejection it may be more useful to design drugs that blocks signal II and not signal I (which the present drugs do). The reason is blocking signal II make the effectors (which originally were capable of recognizing the transplant) anergic while leaving the other immune effectors intact.

5 Conclusions

(i) CAS should be studied as a whole hence reductionist point of view may not be reliable in some cases.

(ii) CAS are open with nonlinear local interactions hence:

1. Long range prediction is highly unlikely [Strogatz 2000, Holmgren 1996].

2. When studying a CAS take into consideration the effects of its perturbation on related systems e.g. perturbation of lake Victoria has affected mosquitoes' numbers hence the locals quality of life. This is also relevant to the case of natural disasters where an earthquake at a city can cause a widespread power failure at other cities.

3. Expect side effects to any "WISE" decision.
4. Mathematical and computer models may be helpful in reducing such side effects.

(iii) Optimization in CAS should be multi-objective and not single objective [Collette and Siarry 2003].

(iv) CAS are very difficult to control. Interference at highly connected sites may be a useful approach [Dorogovtsev and Mendez 2004]. The interlinked nature of CAS elements complicates both the unpredictability and controllability problems. It also plays an important role in innovations spread.

(v) Memory effects should not be neglected in CAS. This lends more support for the proposed telegraph reaction diffusion Eq. (3). Also memory games have been studied [Smale 1980, Ahmed and Hegazi 2000]. Also delay and fractional calculus are relevant to CAS.

(vi) Mathematical topics motivated by CAS include ODE and PDE (non-autonomous, delayed, periodic coefficients, stability and persistence), multi-objective optimization (including biologically motivated methods e.g. Ant colony optimization, Extremal optimization, Genetic algorithm etc), difference equations, cellular automata, networks, fractional calculus, control (e.g. bounded delayed control of distributed systems), game theory, nonlinear dynamics and fuzzy mathematics.

Some of the mathematics motivated by CAS will be reviewed in the appendices.

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**Appendix (1): Some mathematical results for one-dimensional cellular automata**

**Definition (3):** A cellular automata consists of 4 components: A graph $G$, a set of states such that each site (vertex) of the graph has one of the possible states, a neighborhood set which assigns to each vertex a certain neighborhood and a transition function $f$ which defines the evolution of the state of each site as a function of the states of that site and those in its neighborhood.

We choose the set of possible states to be the ring $Z(p)$ i.e. the set of integers $0, 1, 2, ..., p - 1$, where addition is defined mod $p$. The total number of sites is denoted by $N$. In most of the cases, we choose $N, p$ to be relatively prime. The set of states of the sites at a given time is called a configuration. We now restrict us to a one-dimensional space. Let $x(j,t)$ be the state of site $j$ at time $t$.

**Definition (4):** A finite initial configuration is one such that there are two natural numbers $L, R$ such that $0 < L < R < N$, and $x(j,0) = 0$ if $j < L$ or $j > R$.

**Theorem (1) [Jen 1990]:** If $x(i,t), x(j,t), i < j$ are two periodic sequences i.e. $x(i,t) = x(i, t + p(i)), x(j,t) = x(j, t + p(j))$, then for every $k$ such that
$i < k < j$ then $x(k,t)$ is periodic.

**Corollary (1) [Jen 1990]:** If CA evolves according to the rule

$$x(i,t) = f(x(i-1,t), x(i,t), x(i+1,t)) \text{ mod } 2,$$

such that $000 \rightarrow 0, 100 \rightarrow 0, 001 \rightarrow 0$, then for any finite initial configuration the system is temporarily periodic i.e. the sequence $(x(i,t))$ is periodic for all $i$ such that $0 < i \leq N, 0 < T < t$.

**Proof.** The fact that $100 \rightarrow 0$ implies that $x(i,t) = 0$ for $i > R$, similarly $x(i,t) = 0$ for $i < L$ for all $t > 0$. Applying theorem (1) the result is proved.

In the case that $f$ in Eq. (4) is linear, one can use the methods of [Stevens et al 1993, Tadaki 1994] to get useful information about possible periodicity’s of the system. In this case the system can be written as

$$X(t+1) = UX(t),$$

where $U$ is called the evolution matrix. Then $X(t) = U^tX(0)$. In this case the asymptotic behavior of the system is governed by the characteristic polynomial of $U$ on the field $Z(p)$. Assuming periodic boundary conditions, the matrix $U$ is circulant matrix [Barnett 1990].

Let $P(N,\lambda)$ be the characteristic polynomial of the system (5) with $N$ sites, then typically it has the form

$$P(n,\lambda) = \lambda^n d(n,\lambda), \quad d(n,0) = 1.$$  \hspace{1cm} (6)

If $a > 0$, then the systems tends to a fixed configuration (which corresponds to a fixed point for discrete time continuous state dynamical systems). Reducing $d(n,\lambda)$ to its irreducible factors on the field of states then in most cases a cycle of length $p^k - 1$ exist for the system where $k$ is the degree of the irreducible factors.

As an example consider rule 90 [Martin et al 1984]

$$x(i,t+1) = x(i-1,t) + x(i+1,t) \text{ mod } 2.$$  \hspace{1cm} (7)

For $N = 5$, we have $P(5,\lambda) = \lambda(\lambda^2 + \lambda + 1)^2 \text{ mod } 2$, hence the system may evolve to a fixed configuration (e.g. $x(i,t) = 0$ for all $t > T > 0$, for all $0 < i \leq N$). It can also evolve to a cycle of period 3 ($= 2^2 - 1$).
Similarly for \( N = 9 \), \( P(9, \lambda) = \lambda(\lambda + 1)^2(\lambda^3 + l + 1)^2 \) on \( Z(2) \). Hence this system may evolve to a fixed configuration or to a periodic one with period 7. For \( N = 13 \), similar study implies that \( P(13, \lambda) = \lambda(\lambda^6 + \lambda^5 + \lambda^4 + \lambda + 1)^2 \) on \( Z(2) \). Hence fixed configurations and periodic ones with period 63 are expected. Such long periods may not be easy to find numerically. These results can be obtained using more elaborate methods [Martin et al 1984]; but the simplicity of the present approach is appealing.

Moreover it is directly applicable to nonlocal cases which have gained much attention after the pioneering work of Watts and Strogatz on small world network (SWN) [Watts and Strogatz 1998]. As an example consider the following system

\[ x(i, t + 1) = x(i - 1, t) + x(i + 1, t) + x(i + k, t) \mod 2, \tag{8} \]

where \( k \) is fixed. Some of the characteristic polynomials \( P(N, \lambda, k) \) are:

\[
\begin{align*}
P(11, 0, \lambda) &= \lambda^{11} + \lambda^{10} + \lambda^5 + \lambda^4 + \lambda + 1, \\
P(11, 3, \lambda) &= \lambda^{11} + \lambda^9 + \lambda^7 + \lambda^6 + \lambda^5 + \lambda^4 + \lambda + 1, \\
P(11, 1, \lambda) &= \lambda^{11} + \lambda^8 + \lambda^7 + \lambda^5 + \lambda^2 + 1,
\end{align*}
\]

Hence we have the following proposition:

**Proposition (1):**

a) The system (8) depends on \( k \).

b) The asymptotic behavior of (8) contains the following: For \( N = 11 \), \( k = 3 \), no fixed configuration but a periodic one with period 1023.

**Proof.**

a) For \( N = 11 \), \( k = 5 \), a homogeneous configuration is expected. This is not the case for \( N = 11 \), \( k = 0 \) or \( k = 3 \).

b) Use the procedure explained before.

Typically updating of CA is synchronous. It is important to notice that other types of updating e.g. a uniform random asynchronous one (where only one site is chosen randomly and updated at each time step) gives other patterns [Schonfisch and de Roos 1999]. The following lemma is useful

**Lemma (1):**

a) States which are stationary under synchronous updating
are also stationary under asynchronous one.

b) If there is a site \( j \) which is not updated for all time \( t > T > 0 \) then stationary configuration with respect to asynchronous updating may not be so under synchronous one.

**Proof.** a) If \( f(x(1), x(2), ..., x(N)) = (x(1), x(2), ..., x(N)) \) then \( f(j, x(j)) = x(j) \). This proves part a). Since site \( j \) is not updated for \( t > T > 0 \) then \( f(j, x(j)) \neq x(j) \) can still belong to a homogeneous configuration for the asynchronous updating but not the homogeneous one. This proves b).

Loosely speaking patterns present in asynchronous updating are mostly present in synchronous one. Motivated by these results we study sequential CA e.g. the sequential rule 90 is

\[
x(j, t + 1) = x(j - 1, t + 1) + x(j + 1, t) \mod 2.
\]

This can be written in the following equivalent form

\[
x(j, t + 1) = \sum_{k=2}^{j+2} x(k, t) \mod 2,
\]

where free periodic boundary conditions are assumed. The characteristic polynomials of the system (7) are:

\[
P(5, \lambda) = \lambda^5 + \lambda^3, \quad P(6, \lambda) = \lambda^6 + \lambda^5 + \lambda^3, \quad P(7, \lambda) = \lambda^7,
\]

\[
P(13, \lambda) = \lambda^7(\lambda^3 + \lambda^2 + 1)^2.
\]

Hence homogeneous configurations are expected for \( N = 5, 6, 7 \). For \( N = 13 \) a periodic configuration with period 7 is expected.

Studying the system (10) numerically showed that chaos (in the sense of sensitive dependence on initial conditions which is sometimes called damage spread) exists.

**Proposition (2):** Every initially finite configuration will evolve under the CA

\[
x(j, t + 1) = x(j - 1, t + 1) x(j + 1, t) \mod 2,
\]

into the zero configuration \( x(j, t) = 0 \) for all \( j, 0 < j \leq N \), for all time \( t > T > 0 \) where \( T < N \).
Proof. We have
\[ x(R, 1) = x(R + 1, 0) \times x(R - 1, 1). \]
But \( x(R + 1, 0) = 0 \) by definition of initially finite configuration thus \( x(R, 1) = 0 \). Repeating for \( x(R - 1, 2) \), one gets \( x(R - 1, 2) = 0 \) and continue.

Now the above results are applied to two known examples. The first is Domany-Kinzel (DK) model [Kinzel and Domany 1984], which is given by:

\[
\begin{align*}
\text{If } x(j - 1, t) + x(j + 1, t) &= 0 \text{ then } x(j, t + 1) = 0, \\
\text{If } x(j - 1, t) + x(j + 1, t) &= 1 \text{ then } x(j, t + 1) = 1, \\
\text{with probability } p_1, \\
\text{If } x(j - 1, t) + x(j + 1, t) &= 2 \text{ then } x(j, t + 1) = 1, \\
\text{with probability } p_2.
\end{align*}
\]

(13)

where \( x(j, t) \) are Boolean variables. For \( p_1 \to 1, p_2 \to 1 \), the system (13) corresponds to the CA

\[ x(j, t + 1) = x(j - 1, t) + x(j + 1, t) + x(j - 1, t) \times x(j + 1, t) \mod 2. \]  

(14)

Proposition (3): Any finite initial configuration with two consecutive ones will tend to the homogeneous configuration \( x(j, t) = 1 \) for all \( 0 \leq j \leq N, 0 < T < t, T \) is sufficiently large under the CA (12). Consequently the region \( p_1 \to 1, p_2 \to 1 \) in the DK CA does not show chaos (damage spread).

Proof. Assume that \( x(j, 0) = x(j + 1, 0) = 1 \). Then the system (14) implies

\[ x(j - 1, 1) = x(j, 1) = x(j + 1, 1) = x(j + 2, 1) = 1. \]

Continue one gets after \( t \) time steps \( x(k, t) = 1 \), where \( j - t \leq k \leq j + t + 1 \). This proves the first part. Now since the CA (14) will tend to \( x(j, t) = 1 \) for all \( 0 \leq j \leq N, 0 < T < t \), then any change in the initial conditions that preserves the condition \( x(j, 0) = x(j + 1, 0) = 1 \) for some \( j \) will not affect the asymptotic behavior of the CA (14). This completes the proof.
The case $p_1 \to 0$ in the DK model corresponds to the CA
\[ x(j, t + 1) = x(j - 1, t) x(j + 1, t) \mod 2. \]  
(15)

Following similar steps as those in proposition (3) one can prove the following:

**Proposition (4):** Any finite initial configuration with two consecutive zeros will tend to the homogeneous configuration $x(j, t) = 0$ for all $0 \leq j \leq N$, $0 < T < t$, $T$ is sufficiently large under the CA (15). Consequently the region $p_1 \to 1$, $p_2 \to 1$ in the DK CA does not show chaos (damage spread) or periodic configurations.

In the limit $p_2 \to 0$, $p_1 \to 1$, DK model corresponds to rule 90
\[ x(j, t + 1) = x(j - 1, t) + x(j + 1, t) \mod 2, \]
which is known to be chaotic.

All of the above results agree with numerical simulations. Bagnoli et al model [Bagnoli et al 2002] is given by

If $x(j - 1, t) + x(j, t) + x(j + 1, t) = 0$ then $x(j, t + 1) = 0$.

If $x(j - 1, t) + x(j, t) + x(j + 1, t) = 1$,
\[ \text{then } x(j, t + 1) = 1 \text{ with probability } p_1. \]

If $x(j - 1, t) + x(j, t) + x(j + 1, t) = 2$,
\[ \text{then } x(j, t + 1) = 1 \text{ with probability } p_2. \]

If $x(j - 1, t) + x(j, t) + x(j + 1, t) = 3$ then $x(j, t + 1) = 1$.

where $x(j, t)$ are Boolean variables. The limit $p_1 \to 1$, $p_2 \to 1$ corresponds to the CA
\[ x(j, t + 1) = x(j - 1, t) + x(j + 1, t) + x(j, t) x(j + 1, t) + x(j - 1, t) x(j + 1, t) x(j, t) + x(j - 1, t) x(j, t) x(j + 1, t) \mod 2. \]  
(16)

The limit $p_1 \to 0$, $p_2 \to 0$ corresponds to the CA
\[ x(j, t + 1) = x(j - 1, t) x(j, t) x(j + 1, t) \mod 2. \]  
(17)

**Proposition (5):** a) Any nonzero finite initial configuration will evolve
under the CA (17) into the homogeneous configuration \( x(j, t) = 1 \) for all \( 0 \leq j \leq N \), for \( t \) is sufficiently large. Hence the limit \( p_1 \rightarrow 1 \), \( p_2 \rightarrow 1 \) in Bagnoli et al model does not show chaos or periodic configurations.

b) Any finite initial configuration containing at least one zero site will evolve under the CA (18) into the homogeneous configuration \( x(j, t) = 0 \) for all \( 0 \leq j \leq N \), for \( t \) is sufficiently large. Hence the limit \( p_1 \rightarrow 0 \), \( p_2 \rightarrow 0 \) in Bagnoli et al model does not show chaos or periodic configurations.

**Proof.** similar to proposition (3).

The limit \( p_1 \rightarrow 1 \), \( p_2 \rightarrow 0 \) corresponds to the CA

\[
x(j, t+1) = x(j-1, t) + x(j+1, t) + x(j, t) + 2x(j-1, t)x(j, t)x(j+1, t) \mod 2
\]

which is similar to rule 150 \( x(j, t+1) = x(j-1, t) + x(j+1, t) + x(j, t) \), hence chaos is expected in Bagnoli et al model in this limit. All of the above results agree with numerical simulations.

It is interesting how CA unite polynomials on finite fields, circulant matrices, graph theory techniques and many other branches of mathematics into one branch which is important both mathematically and from the point of view of applications in complex systems.

**Appendix (2): Overview of networks in CAS**

Complex systems are often modeled as graphs where agents are the vertices and the interactions form the edges of the graph. Typically graphs are either regular lattices (e.g. square or cubic), random or scale free where the probability that a vertex has degree \( k \) is \( p(k) \approx k^{-\gamma} \). Most of the real networks are of the scale free type. Some proposed mechanisms for this fat tailed distribution [Dorogovtsev and Mendes 2004] are self organization (c.f. biological systems) and optimization involving many agents (c.f. economy).

Random graphs were first studied by the mathematicians Erdős and Rényi [Erdős and Rényi 1960]. Their model consists of \( N \) nodes, such that every pair of nodes is connected by a bond with probability \( p \). The recent increase in computing power and the appearance of interdisciplinary sciences has lead to a better understanding of the properties of complex networks.
Two main properties of complex networks are clustering and small world effect.

Small-world effect means the average shortest node to node (vertex to vertex) distance is very short compared with the whole size of the system (total number of vertices). For social networks, the social psychologist Milgram [Milgram 1967] concluded that the average length of the path of acquaintances connecting each pair of people in the United States is six. This concept is known as the six degrees of separation. Such an effect makes it easier for an effect (e.g. an epidemic) to spread throughout the network.

In a regular 1-dimensional lattice of size $N$, the average shortest path connecting any two vertices $l$ increases linearly with the system size. So regular lattices do not display small-world effect. On the other hand for a random graph, with coordination number $z$, one has $z$ first (nearest) neighbors, $z^2$ second neighbors and so on. This means that the total number of vertices $N = z^l$, this gives

$$l = \frac{\ln(N)}{\ln(z)}.$$  

The logarithmic increase with the size of the lattice allows the distance $l$ to be very short even for large $N$. Then random graphs display the small-world effect.

Clustering is a common property of complex networks. It means that every vertex has a group of connected nearest neighbours (NN) (collaborators, friends), some of them will often be a connected NN to another vertex. As a measure for the clustering property, a clustering coefficient $C$ is defined as the probability that connected pairs of NN of a vertex are also connected to each others. For a random graph, $C = z/N$ which goes to zero for large $N$. So random graphs do not display clustering property. On the other hand, a fully connected regular lattice itself forms a cluster, then its cluster coefficient is equal to 1.

Complex networks display a small-world effect like random graphs, and they have large clustering coefficient as regular lattices. For a review on many real-world examples, see [Dorogovtsev and Mendes 2004].

A small-world network (SWN) proposed initially by Watts and Strogatz [Watts and Strogatz 1998] is a superposition of a regular lattice (with high clustering coefficient) and a random graph (with the small world effect). SWN satisfy the main properties of social networks. Also, the structure of SWN combines between both local and nonlocal interactions which is ob-
served in many real systems. For example epidemic spreading show nonlocal interactions e.g SARS.

The concept of SWN has been applied successfully in modelling many CAS, e.g. some games [Ahmed and Elgazzar 2000 a], epidemics [Ahmed et. al. 2002], economic systems [Elgazzar 2002], and opinion dynamics [Elgazzar 2001].

An important property related to disease spread in a network is the second moment of the degree distribution i.e. $\langle k^2 \rangle$. If it is divergent then on average a vertex has an infinite number of second nearest neighbors thus if a single vertex is infected the disease will spread in the whole network. This explains the results that disease spread on scale free networks has zero threshold (contrary to the ODE and PDE models). However one should realize that real networks are finite hence a kind of threshold is expected.

Scale-free networks [Albert and Barabási 2002] are another class of complex networks. A scale-free network does not have a certain scale. Some nodes have a huge number of connections to other nodes, whereas most nodes have only a few, following a power law distribution.

Appendix (3): Basics of game theory

Game theory [Hofbauer and Sigmund 1998] is the study of the ways in which strategic interactions among rational players produce outcomes (profits) with respect to the preferences of the players. Each player in a game faces a choice among two or more possible strategies. A strategy is a predetermined program of play that tells the player what actions to take in response to every possible strategy other players may use. A basic property of game theory is that one’s payoff depends on the others’ decisions as well as his.

The mathematical framework of the game theory was initiated by von Neumann and Morgenstern in 1944. Also they had suggested the max-min solution for games which is calculated as follows: Consider two players A and B are playing against each other. Two strategies $S_1$, $S_2$ are allowed for both of them. This game is called two-player, two-strategy game. Assume that the constants $a, b, c$ and $d$ represent the payoffs (profits) such that, if the two players use the same strategy $S_1(S_2)$, their payoff is $a(d)$. When a player with strategy $S_1$ plays against another one with strategy $S_2$, the payoff of the $S_1$-player is $b$ and the payoff of the $S_2$-player is $c$ and so on. This is summarized in the payoff matrix as follows:
The max-min solution of von Neumann and Morgenstern is for the first player to choose \( \max\{\min(a, b), \min(c, d)\} \). The second player chooses \( \min\{\max(a, c), \max(b, d)\} \). If both quantities are equal then the game is stable. Otherwise use mixed strategies.

A weakness of this formalism has been pointed out by Maynard Smith in the hawk-dove (HD) game whose payoff matrix is

\[
\Pi = \begin{pmatrix}
H & D \\
H & \frac{1}{2}(v - c) & v \\
D & 0 & \frac{v}{2}
\end{pmatrix}
\]

The max-min solution implies (for \( v < c \)) that the solution is D yet as he pointed out this solution is unstable since if one of the players adopts H in a population of D he will have a very large payoff which will make other players switch to H and so on till number of H is large enough that they play each other frequently and get the low payoff \((v - c)/2\). Thus the stable solution is that the fraction of hawks should be nonzero. To quantify this concept one may use the replicator equation which intuitively means that the rate of change of the fraction of players adopting strategy \(i\) is proportional to the difference between their payoff and the average payoff of the population i.e.

\[
\frac{dx_i}{dt} = x_i \left[ (\Pi x)_i - x \Pi x \right], \quad i = 1, 2, ..., n, \quad \sum_{i=1}^{n} x_i = 1,
\]

where \(x_i\) is the fraction of players adopting strategy \(i\), and \(\Pi\) is the payoff matrix. Applying Eq. (20) to the HD game, one gets that the asymptotically stable equilibrium solution is \(x = v/c\), where \(x\) is the fraction of hawks in the population.

For asymmetric game the replicator dynamics equation is

\[
\frac{dx_i}{dt} = x_i \left[ (\Pi_1 y)_i - x \Pi_1 y \right], \quad \frac{dy_i}{dt} = y_i \left[ (\Pi_2 x)_i - y \Pi_2 x \right], \quad i = 1, 2, ..., n.
\]

A basic drawback of normal game theory is the assumption that all players interact globally. It is more realistic to study local games [Ahmed and Elgazzar 2000 b] e.g. games on a lattice where players interact only with their nearest neighbors. Also there are several modifications for game formulations.
Appendix (4): Unpredictability in CAS

There are at least two sources for unpredictability in CAS. The first is that CAS are open systems hence perturbing a CAS may affect another related one e.g. the insect population affected by the perturbation of Lake Victoria. Another reason is the nonlinear interactions [Strogatz 2000] between the elements of the CAS. The scientific and mathematical study of Chaos Theory contains many overlaps with the study of Complex Systems, but with differences related to method: Chaos Theory can be used to study Complex Systems, but is not restricted to the study of these systems. Chaos Theory "deals with deterministic systems whose trajectories diverge exponentially over time" (Bar Yam, NECSI website). It has been used to study Complex Systems, because these systems can be generally defined as a "deterministic system that is difficult to predict". On the other hand, complexity deals with systems composed of many interacting agents" The point being that Chaos Theory is one of many tools and methods that can be applied to the study of Complex Systems, but is not specifically devoted to the way these systems are designed, developed, studied, and modeled. That being stated, the famous example of the "Butterfly Effect" in a chaotic system is an example of an agent (a butterfly) evoking a non-linear response (the storm in New England) within a Complex System (Global Weather System).

A simple example of nonlinear interactions is the logistic difference equation

\[ x_{t+1} = rx_t(1 - x_t), \quad t = 0, 1, 2, ..., n, \quad r > 0. \]  

This equation has two equilibrium solutions \( x = 0, \quad x = 1 - 1/r \) \( (r > 1) \) which are asymptotically stable if \( r < 1 \) or \( 1 < r < 3 \) respectively. If \( 3 < r < 3.6 \) then cycles appears and if \( r > 3.6 \) chaos sets in. Intuitively chaos is sensitive dependence on initial conditions (for more mathematical definition see [Holmgren 1996]). Hence in chaotic systems one cannot make long range predictions c.f. weather. A useful measure of chaos are Lyapunov exponents

\[ \lambda = \frac{1}{n} \sum_{t=0}^{n-1} \ln |f'(x_t)|. \]  

Since CAS consists of several interacting agents one studies coupled systems e.g. coupled map lattices [Kaneko 1993] given by

\[ x_{i+1} = (1 - D)f(x_i) + \frac{D}{2} \left[ f(x_{i-1}) + f(x_{i+1}) \right], \quad i = 1, 2, ..., n. \]
The homogeneous equilibrium is given by \( x = f(x) \) and it is asymptotically stable if [Ahmed and Hegazi 2002]

\[
\left| \dot{f}(x) \left[ (1 - D) + D \cos \left( \frac{k \pi}{n} \right) \right] \right| < 1, \ k = 0, 1, ..., n - 1. \tag{24}
\]

The more realistic case is to assume that the map depends on the agents e.g.

\[
x_{i}^{t+1} = (1 - D)f_i(x_{i}^{t}) + \frac{D}{2} \left[ f_{i-1}(x_{i-1}^{t}) + f_{i+1}(x_{i+1}^{t}) \right], \ i = 1, 2, ..., n. \tag{25}
\]

But analytic studies for Eq. (25) are more difficult.

These systems shed some light on how to control (synchronize) some CAS [Ahmed et al 2003]. One may increase the coupling constant \( D \). Also if the network of the agents is more connected (e.g. SWN), then the system is easier to synchronize. Finally external control can be applied preferably at highly connected sites.

**Appendix (5): Elements of multi-objective optimization**

Almost every real life problem is multi-objective (MOB) [Collette and Siarry 2003]. Methods for MOB optimization are mostly intuitive.

**Definition (5):** A MOB problem is:

\[
\text{Minimize (min)} Z_i(x), \ i = 1, 2, ..., k, \ \text{subject to } g(x) \leq 0, \ h(x) \leq 0. \tag{26}
\]

**Definition (6):** A vector \( \vec{x}^* \) dominates \( \vec{x} \) if \( Z_i(\vec{x}^*) \leq Z_i(\vec{x}) \forall i = 1, 2, ..., k \) with strict inequality for at least one \( i \), given that all constraints are satisfied for both vectors.

A non-dominated solution \( \vec{x}^* \) is called Pareto optimal and the corresponding vector \( Z_i(\vec{x}^*) \), \( i = 1, 2, ..., k \) is called efficient. The set of such solutions is called a Pareto set.

Now we discuss some methods for solving MOB problems:
The first method is the lexicographic method. In this method objectives are ordered according to their importance. Then a single objective problem is solved while completing the problem gradually with constraints i.e.

\[
\begin{align*}
\min & \quad Z_1 \\
\text{subject to} & \quad g(x) \leq 0, \quad h(x) = 0,
\end{align*}
\]

then if ZMIN(1) is the solution, the second step is \( \min Z_2 \) subject to \( Z_1 = Z\text{MIN}(1) \), and the constraints in Eq. (26), and so on.

A famous application is in university admittance where students with highest grades are allowed in any college they choose. The second best group is allowed only the remaining places and so on. This method is useful but in some cases it is not applicable.

**Proposition (6):** An optimal solution for the lexicographic problem is Pareto optimal.

**Proof.** Let \( x^* \) be the solution to the Lexicographic problem \( P_l \). Thus

\[
\begin{align*}
x \neq x^*, \text{ then } Z_i(x) &= Z_i(x^*), \quad i = 1, 2, ..., l - 1 \text{ and } Z_l(x^*) < Z_l(x).
\end{align*}
\]

Thus \( x^* \) is not dominated.

The second method is the method of weights. Assume that it is required to minimize the objectives \( Z(j), j = 1, 2, ..., n \). (The problem of maximization is obtained via replacing \( Z(j) \) by \( -Z(j) \). Define

\[
Z = \sum_{i=1}^{k} Z_iw(i), \quad 0 \leq w(i) \leq 1, \quad \sum_{i=1}^{k} w(i) = 1.
\]

Then the problem becomes to minimize \( Z \) subject to the constraints. This method is easy to implement but it has several weaknesses. The first is that it is not applicable if the feasible set is not convex. The second difficulty of this method is that it is difficult to apply for large number of objectives. However it is quite effective for multiobjective problems with discrete parameters since in this case Pareto optimal set is discrete not a continuous curve.

The third method is the compromise method (sometimes called \( \varepsilon \)-constraint method \( P_\varepsilon(k) \)). In this case one minimizes only one objective while setting the other objectives as constraints e.g. minimize \( Z(k) \) subject to \( Z(j) \leq a(j), \quad j = 2, 3, ..., k - 1, k + 1, ..., n \), where \( a(j) \) are parameters to be
gradually decreased till no solution is found. The problem with this method is the choice of the thresholds \( a(j) \). If the solution is unique, then this method is guaranteed to give a Pareto optimal solution.

**Proposition (7):** If the solution is unique, then the \( \varepsilon \)-constraint method is guaranteed to give a Pareto optimal solution.

**Proof.** Let \( \mathbf{x}^* \) be the optimal solution for the \( \varepsilon \)-constraint method then

\[
\forall \mathbf{x} \neq \mathbf{x}^*, \text{ then } Z_k(\mathbf{x}^*) < Z_k(\mathbf{x}),
\]

hence \( \mathbf{x}^* \) is Pareto optimal. If \( \mathbf{x}^* \) is not unique, then it is weakly Pareto i.e. there is no \( \mathbf{x} \neq \mathbf{x}^* \) such that \( Z_i(\mathbf{x}^*) < Z_i(\mathbf{x}) \forall i = 1, 2, ..., n \).

A fourth method using fuzzy logic is to study each objective individually and find its maximum and minimum say \( \text{ZMAX}(j) \), \( \text{ZMIN}(j) \), respectively. Then determine a membership \( m(j) = (\text{ZMAX}(j) - Z(j))/\text{ZMAX}(j) - \text{ZMIN}(j) \). Thus \( 0 \leq m(j) \leq 1 \). Then apply \( \max\{\min\{m(j), j = 1, 2, n\}\} \). Again this method is guaranteed to give a Pareto optimal solution provided that the solution is unique otherwise it is weakly Pareto. This method is a bit difficult to apply for large number of objectives. A fifth method is Keeney-Raiffa method which uses the product of objective functions to build an equivalent single objective one.

**Appendix (6): Fractional calculus in CAS**

Recently [Stanislavsky 2000] it became apparent that fractional equations solve some of the above mentioned problems for the PDE approach. To see this consider the following evolution equation

\[
\frac{df(t)}{dt} = -\lambda^2 \int_0^t k(t - \dot{t}) f(\dot{t}) d\dot{t}.
\]  \hspace{1cm} (30)

If the system has no memory then \( k(t - \dot{t}) = \delta(t - \dot{t}) \) and one gets \( f(t) = f_0 \exp(-\lambda^2 t) \). If the system has an ideal memory, then

\[
k(t - \dot{t}) = \begin{cases} 
1, & t \geq \dot{t} \\
0, & t < \dot{t} 
\end{cases},
\]

hence \( f \approx f_0 \cos(\lambda t) \). Using Laplace transform
\[ L[f] = \int_{0}^{\infty} f(t) \exp(-st) dt, \]

one gets \( L[f] = 1 \) if there is no memory and \( L[f] = 1/s \) if there is ideal memory hence the case of non-ideal memory is expected to be given by \( L[f] = 1/s^\alpha, \) \( 0 < \alpha < 1. \) In this case Eq. (28) becomes

\[
\frac{df(t)}{dt} = \int_{0}^{t} (t - \hat{t})^{\alpha-1} f(\hat{t}) d\hat{t}, \tag{31}
\]

where \( \Gamma(\alpha) \) is the Gamma function. This system has the following solution

\[ f(t) = f_0 E_{\alpha+1}(-\lambda^2 t^{\alpha+1}), \]

where \( E_{\alpha}(z) \) is the Mittag Leffler function given by

\[ E_{\alpha}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k + 1)}. \]

It is direct to see that \( E_1(z) = \exp(z), \) \( E_2(z) = \cos(z) \)

Following a similar procedure to study a random process with memory, one obtains the following fractional evolution equation

\[
\frac{\partial^{\alpha+1} P(x, t)}{\partial t^{\alpha+1}} = \sum_n \frac{(-1)^n}{n!} \frac{\partial^n [K_n(x)P(x, t)]}{\partial x^n}, \quad 0 < \alpha < 1, \tag{32}
\]

where \( P(x, t) \) is a measure of the probability to find a particle at time \( t \) at position \( x. \)

We expect that Eq. (30) will be relevant to many complex adaptive systems and to systems where fractal structures are relevant since it is argued that there is a relevance between fractals and fractional differentiation [Rocco and West 1999].

For the case of fractional diffusion equation the results are

\[
\frac{\partial^{\alpha+1} P(x, t)}{\partial t^{\alpha+1}} = D \frac{\partial^2 P(x, t)}{\partial x^2}, \quad P(x, 0) = \delta(x), \quad \frac{\partial P(x, 0)}{\partial t} = 0 \quad \Rightarrow
\]

\[
P = \frac{1}{2\sqrt{Dt}^\beta} M \left( \frac{|x|}{\sqrt{Dt}^\beta}; \beta \right), \quad \beta = \frac{\alpha + 1}{2}, \tag{33}
\]

\[
M(z; \beta) = \sum_{n=0}^{\infty} \frac{(-1)^n z^n}{n! \Gamma(-\beta n + 1 - \beta)}.
\]

For the case of no memory \( \alpha = 0 \Rightarrow M(z, 1/2) = \exp(-z^2/4). \)