Theory of the evolutionary minority game

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Abstract

We present a theory which describes a recently introduced model of an evolving, adaptive system in which agents compete to be in the minority. The agents themselves are able to evolve their strategies over time in an attempt to improve their performance. The present theory explicitly demonstrates the self-interaction, or market impact that agents in such systems experience.

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I. INTRODUCTION

Agent-based models of complex adaptive systems (CAS) provide invaluable insight into the highly non-trivial global behaviour of a population of competing agents [1]. These models typically involve agents with similar capability competing for a limited resource. The agents are given the same global information, which is in turn generated by the action of the agents themselves, and they learn from past experience. The growing field of econophysics [2–4] represents an area in which such CAS may be applicable: every agent knows the past ups and downs in the index of a stock market and must decide how to trade based on this global information. An important step forward in agent-based models of CAS was made by Challet and Zhang [5,6] who proposed the so-called Minority Game (MG) in which an odd number $N$ of agents successively compete to be in the minority. Each agent is randomly assigned a limited number of strategies at the beginning of the game, hence introducing some quenched disorder. As the game progresses, non-trivial fluctuations arise in the collective agents’ decisions – these can be understood in terms of the dynamical formation of crowds consisting of agents using correlated strategies, and anticrowds consisting of agents using the anticorrelated strategies [7]. Subsequent work by Challet and co-workers has provided a remarkable formal connection to spin glass systems [8].

The basic minority game, however, does not incorporate evolution. Agents are stuck with their initial strategies and hence the system cannot avoid this in-built frustration. In the real world, one would expect that agents would be able to evolve more successful strategies, or at least stop playing disastrous strategies. This motivated us to recently propose a simpler minority model which allowed for an evolving population [9–11] - we call this the evolutionary minority game (EMG). D’Hulst and Rodgers [12] subsequently proposed an analytic theory, based on a slightly modified version of our model. However, the two models actually give different numerical results [11].

Here we provide a theory for our evolutionary minority game (EMG) [9] which correctly includes the self-interaction of the agents. Results are in good agreement with numerical
data. The plan of the paper is as follows. We introduce the EMG in Sec.II and give the
main features observed in numerical simulations of the model. In Sec.III, we present the
formalism and derive the winning probability for an agent. Results from the present theory
are compared with numerical data in Sec.IV. Section V provides a discussion of the results.

II. EVOLUTIONARY MINORITY GAME

Consider an odd number \( N \) of agents repeatedly choosing to be in room 0 (e.g. sell) or
room 1 (e.g. buy). After each agent has independently chosen a room, the winners are those
in the minority room. A single binary digit denoting the minority room forms the outcome
for each time-step. Each agent is given the information of the most recent \( m \) outcomes. Each
agent also has access to a common register or “memory” containing the outcomes from the
most recent occurrences of all \( 2^m \) possible bit strings of length \( m \). Consider, for example,
\( m = 3 \) and denote \((xyz)w\) as the \( m = 3 \) bit string \((xyz)\) and outcome \(w\). An example memory
would comprise (000)1, (001)0, (010)0, (011)1, (100)0, (101)1, (110)0, (111)1. Following a
run of three wins for room 0 in the recent past, the winning room was subsequently 1.
Faced with a given bit string of length \( m \), it seems reasonable for an agent to simply predict
the same outcome as that registered in the memory. The agent will hence choose room 1
following the next 000 sequence. If 0 turns out to be the winning room, the entry (000)1 in
the memory is then updated to be (000)0. Simply put, each agent looks into the most recent
history for the same pattern of \( m \) bit string and predicts the outcome using the history. In
effect, each agent holds one strategy and all agents hold the same strategy, with the strategy
being dynamical. The strategy is hence to follow the trend. However, if all \( N \) agents act in
the same way, they will all lose. A successful agent is one who can follow a trend as long as it
is valid and to correctly predict when it will end. To incorporate this factor into our model,
each agent is assigned a single number \( p \), which we refer to as the “gene”-value. Following
a given \( m \)-bit sequence, \( p \) is the probability that the agent will choose the same outcome as
that stored in the memory, i.e. he will follow the current predictor. An agent will reject the
prediction and choose the opposite action with probability $1 - p$. To incorporate evolution into our model, we assign $+1$ ($-1$) point to every agent in the minority (majority) room at each time step. If an agent’s score falls below a value $d$ ($d < 0$), his gene-value $p$ is modified. The new $p$ value is chosen randomly from a range of values centered on the old $p$ with a width equal to $R$. We impose reflective boundary condition to ensure that $0 \leq p \leq 1$. Our conclusions do not depend on the particular choice of boundary conditions. For $R = 0$, the agents will never change their gene values - this represents the limiting case of in-built quenched disorder determined by the initial distribution of $p$ values. For any non-zero $R$ value, the system is able to evolve through gene modification. For $R = 2$, the new gene value is uncorrelated with the old one upon modification.

Initially, each agent is randomly assigned a gene value in the range $0 \leq p \leq 1$. Choosing $R \neq 0$ allows the population to evolve. We focus on two quantities, $P(p)$ and $L(p)$, in the asymptotic limit. Here $P(p)$ is the frequency distribution of gene values, typically taken in the long time limit over a time window and normalized to unity; $L(p)$ is the lifespan defined as the average length of time a gene value $p$ survives between modifications. To introduce the basic features observed in numerical simulations, Fig. 1 shows $L(p)$ and $P(p)$ (inset) as a function of $p$ for a range of values of $m$. The other parameters are taken to be $N = 101$, $R = 0.2$ and $d = -4$. The most interesting feature is that $P(p)$ becomes peaked around $p = 0$ and $p = 1$, with a similar behaviour in $L(p)$. Both of these quantities are symmetric about $p = 1/2$. The results are insensitive to the initial distribution of $p$ values. Surprisingly the results indicate that agents who either always follow or never follow what happened last time, generally perform better than cautious agents using an intermediate value of $p$. Figure 1 also shows that there is no explicit dependence on $m$ for $P(p)$ and $L(p)$ \cite{11,13}. The independence on $m$ of the results was also discussed recently by Burgos and Ceva \cite{13} using a random walk argument. Reference \cite{12} proposes a theory which gives a $P(p)$ somewhat similar to that shown in Fig.1. However, the theory was developed based on a model in which each agent is initially assigned one strategy from the strategy pool, and uses this strategy throughout the game: the corresponding $P(p)$ is then $m$-dependent \cite{11}.\[4\]
in contrast to the EMG results shown in Fig.1. The dependences on the other parameters of the EMG such as \(N\), \(d\), and \(R\) are reported in Ref. [11].

III. FORMALISM

We consider a game with \(N\) agents \((N \gg 1)\). After a sufficiently long time, the distributions \(P(p)\) and \(L(p)\) reach the stationary forms as shown in Fig. 1. Consider a certain moment of the game in this steady-state regime. Let the predictor, which is simply the strategy stored in the memory for the given history bit-string, be 1; i.e. go to room “1”. As long as the winning room is defined as the minority room, i.e. with a cutoff at \((N - 1)/2\), the following arguments do not depend on the actual value of the predictor and hence also hold if the predictor says 0. We define \(F_N(n)\) as the probability of the attendance being \(n\) in the predicted room. It follows from the central limit theorem that \(F_N(n)\) will be an approximately gaussian distribution with a mean \(N\bar{p}\) and variance \(N \int_0^1 P(p)p(1-p)dp\). Here \(\bar{p}\) is the mean of the gene value \(p\) given by \(\bar{p} = \int_0^1 pP(p)dp\), which is known if the distribution \(P(p)\) is known. However, \(P(p)\) is the unknown which we are going to solve for. In the steady state, \(F_N(n)\) becomes identical to the probability of the attendance in any one of the two rooms since the two possible outcomes occur equally often on average. Figure 2 shows the normalized \(F_N(n)\) in the steady state extracted from the numerical simulations.

In the spirit of self-consistent mean-field theories, the basic idea of the present formulation is to consider the interaction between a particular agent and the rest of the population. We present the formulation in a general way so that it can be readily generalized to different variations of our model. We consider the action of a particular agent, say the \(k\)-th player, in the background of the \(N - 1\) other agents. Let \(G_{N-1}^k(n)\) be the probability of the attendance being \(n\) in the predicted room, given that there are only \((N - 1)\) agents participating in the game (i.e. excluding the \(k\)-th agent). Then \(F_N(n)\) can be written in terms of \(G_{N-1}^k\) as

\[
F_N(n) = p_k G_{N-1}^k(n - 1) + (1 - p_k) G_{N-1}^k(n),
\]

where \(n \neq 0, N\). Here \(p_k\) is the \(p\)-value of the \(k\)-th agent at that moment. The physical
meaning of Eq.(1) is transparent. An attendance of \( n \) in room “1” is achieved if the attendance by the \((N - 1)\) agent background is \( n - 1 \) and the \( k\)-th agent decides to go to room “1”: this leads to the first term in Eq.(1). Alternatively the attendance by the \((N - 1)\) agent background is \( n \) and the \( k\)-th agent decides not to go to room “1”: this leads to the second term in Eq.(1).

Let \( \tau(p_k) \) be the winning probability of the \( k\)-th agent. Given the probability \( G^k_{N-1}(n) \), we can write

\[
\tau(p_k) = p_k \sum_{n=0}^{(N-3)/2} G^k_{N-1}(n) + (1 - p_k) \sum_{n=(N+1)/2}^{N-1} G^k_{N-1}(n). \tag{2}
\]

Equation (2) says that the \( k\)-th agent wins if (i) the attendance is below \((N - 3)/2\) in room “1” before he makes his move and he decides to go to room “1”, thereby giving the first term or (ii) the attendance is above \((N + 1)/2\) in room “1” before he makes his move and he decides not to go to room “1”, thereby giving the second term. Since the \( k\)-th agent is only characterized by his gene value \( p_k \), \( \tau(p_k) \) can also be interpreted as the success rate of an agent using gene value \( p_k \). It follows from Eq.(1) that

\[
\sum_{n=1}^{(N-3)/2} F_N(n) = \sum_{n=1}^{(N-3)/2} p_k \left(G^k_{N-1}(n-1) - G^k_{N-1}(n)\right) + G^k_{N-1}(n) = \sum_{n=1}^{(N-3)/2} G^k_{N-1}(n) + p_k G^k_{N-1}(0) - p_k G^k_{N-1}\left(\frac{N-3}{2}\right).
\]

Since \( F_N(0) = (1 - p_k) G^k_{N-1}(0) \), which follows from the consideration that room “1” is empty only if the other \( N - 1 \) agents do not go to room “1” and the \( k\)-th agent does not go to room “1”, we have

\[
\sum_{n=0}^{(N-3)/2} G^k_{N-1}(n) = \sum_{n=0}^{(N-3)/2} F_N(n) + p_k G^k_{N-1}\left(\frac{N-3}{2}\right). \tag{3}
\]

Similarly, we have from Eq.(1)

\[
\sum_{n=(N+1)/2}^{N-1} F_N(n) = \sum_{n=(N+1)/2}^{N-1} p_k \left(G^k_{N-1}(n-1) - G^k_{N-1}(n)\right) + G^k_{N-1}(n) = \sum_{n=(N+1)/2}^{N-1} G^k_{N-1}(n) + p_k G^k_{N-1}\left(\frac{N-1}{2}\right) - p_k G^k_{N-1}(N-1).
\]
Since $F_N(N) = p_k G^k_{N-1}(N-1)$, which follows from the consideration that all the agents go to room “1” only if all the other $N-1$ agents go to room “1” and the $k$-th agent goes to room “1”, we have

$$G^k_{N-1}(n) = F_N(n) - p_k G^k_{N-1}(\frac{N-1}{2}).$$

Substituting Eqs.(3) and (4) into Eq.(2), we obtain

$$\tau(p_k) = p_k \sum_{n=0}^{(N-3)/2} F_N(n) + p_k^2 G^k_{N-1}(\frac{N-3}{2}) + (1 - p_k) \sum_{n=(N+1)/2}^{N} F_N(n) - (1 - p_k)p_k G^k_{N-1}(\frac{N-1}{2}).$$

Using Eq.(1) to express $G^k_{N-1}(\frac{N-3}{2})$ in terms of $G^k_{N-1}(\frac{N-1}{2})$ and $F_N(\frac{N-1}{2})$, we then obtain

$$\tau(p_k) = p_k \sum_{n=0}^{(N-3)/2} F_N(n) + (1 - p_k) \sum_{n=(N+1)/2}^{N} F_N(n) + p_k \left( F_N(\frac{N-1}{2}) - 2(1 - p) G^k_{N-1}(\frac{N-1}{2}) \right)$$

$$= p_k \sum_{n=0}^{(N-1)/2} F_N(n) + (1 - p_k) \sum_{n=(N+1)/2}^{N} F_N(n) - 2p_k(1 - p_k) G^k_{N-1}(\frac{N-1}{2}).$$

Equation (5) separates $\tau(p_k)$ into 3 terms, each of which has a physically transparent interpretation. Consider an “outsider”, i.e. someone whose action does not affect the outcome but instead is only betting on which side is the winning room according to the probability $p_k$. His winning probability is given by the first two terms in Eq.(5). The third term gives the difference in the winning probability between an “outsider” of the game and an agent who actually participates in the game. This term is negative, reflecting the fact that an agent has a smaller probability of winning when he is actually participating in the game. Consider the case in which the background population is split evenly between room “0” and room “1”: the $k$-th agent loses no matter what action he takes. Thus the third term represents this self-interaction term, or so-called market impact in financial market terminology. The $p_k(1 - p_k)$ factor means that the winning probability increases as the gene value $p_k$ deviates more from the value $1/2$, and it produces a symmetry about $p = 1/2$ in $L(p)$ and $P(p)$ as
shown in Fig.1. Note that Eq.(5) also applies to the case when the predictor says 0: hence it is independent of the dynamics of the predictor which in turn is determined by the time evolution of the outcomes. This further implies that the resulting $P(p)$ and $L(p)$ do not depend on the value of $m$ in the model. For the present EMG, there is a lack of an *a priori* preferred room: therefore the outcomes 0 and 1 will occur similar numbers of times on the average. In this case, the summations in the first and second terms of Eq.(5) in the steady state yield the value 1/2 and hence $\tau(p)$ becomes

$$
\tau(p_k) = \frac{1}{2} - 2p_k(1 - p_k)G^k_{N-1}\left(\frac{N-1}{2}\right). \tag{6}
$$

In order to express the right hand side of Eq.(5) entirely in terms of the function $F$, we use Eq.(1) to find $G^k_{N-1}\left(\frac{N-1}{2}\right)$. From Eq.(1), we have

$$
p_kG^k_{N-1}(n-2) + (1 - p_k)G^k_{N-1}(n-1) = F_N(n-1). \tag{7}
$$

Subtracting the equations obtained by multiplying Eq.(1) by $(1-p_k)$ and multiplying Eq.(7) by $p_k$, we can eliminate $G^k_{N-1}(n-1)$ to obtain

$$(1 - p_k)F_N(n) - p_k F_N(n-1) = (1 - p_k)^2G^k_{N-1}(n) - p_k^2G^k_{N-1}(n-2).$$

Repeatedly applying Eq.(1), we can eliminate $G^k_{N-1}(n-2), G^k_{N-1}(n-3), \cdots$ to obtain

$$
\sum_{j=0}^{n}(-1)^{n-j}F_N(j)(\frac{p_k}{1-p_k})^{n-j} = (1 - p_k)G^k_{N-1}(n). \tag{8}
$$

Similarly, if we apply Eq.(1) with increasing values of $n$ instead of decreasing values of $n$, we obtain

$$
\sum_{j=n+1}^{N}(-1)^{j-n-1}F_N(j)(\frac{1-p_k}{p_k})^{j-n-1} = p_kG^k_{N-1}(n). \tag{9}
$$

Although the results are exact, in practice it makes sense to use Eq.(8) for small $p_k$ and Eq.(9) for $p_k \sim 1$. Using Eq.(8) or Eq.(9) for $n = \frac{N-1}{2}$ and substituting the result into Eq.(5), we obtain $\tau(p_k)$ entirely in terms of $F_N(n)$, and the label $k$ becomes irrelevant. As mentioned, $\tau(p_k)$ can be regarded as the winning probability of an agent who is using a gene value $p$, and henceforth we denote it by $\tau(p)$ for simplicity.
IV. RESULTS

In order to obtain $P(p)$ from $\tau(p)$, we note that these two quantities are related. In Ref. [12], it was pointed out that the stationary distributions $P(p)$ and $L(p)$ are proportional to each other:

$$\frac{P(p)}{L(p)} = \text{constant}, \quad (10)$$

where the right hand side is a constant independent of $p$. Equation (10) follows from the balance between the fluxes of agents into and out of a region in $p$-space in the steady state. Since an agent using the gene value $p$ loses $(1 - 2\tau(p))$ points each turn [12], the lifespan $L(p)$ is given by

$$\tau(p) = \frac{|d|}{1 - 2\tau(p)}.$$

From Eq.(10), we have

$$P(p) \propto \frac{1}{1/2 - \tau(p)}, \quad (11)$$

with the proportionality constant determined by the normalization of $P(p)$ to $\int_0^1 P(p) dp = 1$.

Based on the present theory, it is straightforward to construct an iterative calculation scheme for $P(p)$. The steps are the following: (a) assume a form for $P(p)$, (b) obtain $F_N(n)$ by evaluating $\overline{p}$ and the standard deviation from the assumed $P(p)$, (c) use Eq.(5) together with Eqs.(8) and (9) to obtain $\tau(p)$, (d) calculate $P(p)$ from $\tau(p)$ using Eq.(11) and the normalization condition, (e) check for convergence of $P(p)$ and, if necessary, repeat the steps until convergence is obtained. Note that Eq. (5) is employed since it is valid for all forms of initial guess for $P(p)$, including those which are non-symmetrical about $p = 1/2$.

Results for $P(p)$ and $L(p)$ obtained by carrying out the calculation scheme are shown in Figs.3 and 4 together with results of numerical simulation for $N = 51$ and $N = 101$. Note that $P(p)$, when properly normalized, is not sensitive to $N$, while $L(p)$ depends on $N$. Results from our theory are in good agreement with numerical data. The results for $P(p)$ as obtained in Ref. [12] are also shown in Fig. 3 for comparison: note that the results
of Ref. [12] show a plateau over a significant range of $p$ in contrast to the present theory and the numerical simulations. The comparison indicates that the results from the present theory are in better agreement with the numerical results. To further test the validity of our theory, we compare results for $\tau(p)$ as a function of $p$ with numerical data for $N = 51, 101,$ and $201$ in Fig. 5. The numerical data are found by simply counting the number of times an agent with gene value $p$ wins. It should be noted that $\tau(p)$ provides a better test than $P(p)$ for the validity of any theory, since many forms of $\tau(p)$ can give rise to similar forms for $P(p)$. In contrast to the numerical results and those of the present theory shown in Fig. 5, the expression for $\tau(p)$ given in Ref. [12] gives a very small $\tau(p)$ for a significant range of $p$ around $p = 1/2$ corresponding to the plateau in $P(p)$. Figure 5 suggests that the correct $\tau(p)$ in the steady state, which follows from Eq.(5) (see also Eq.(6)), has the form

$$\tau(p) \sim 1/2 - A(N)p(1 - p)$$

where $A(N)$ is an $N$-dependent constant which decreases with $N$ as $1/\sqrt{N}$. Such a scaling with $N$ makes sense from random walk arguments.

V. DISCUSSION

We have presented a theory of the EMG based on the consideration of a particular agent in the environment formed by the rest of the population. The winning probability $\tau(p)$ is given in terms of the population distribution in one of the rooms. By relating the population distribution, the winning probability and the lifespan, an iteration scheme is set up for calculating the frequency distribution of gene values $P(p)$. Results for $P(p)$, $L(p)$ and $\tau(p)$ are in good agreement with numerical data.

The present formalism can be used to describe different versions of the EMG. For example, a generalization of the EMG was recently introduced where the winning ‘room’ (i.e. winning decision) was assigned according to whether the attendance was lower than a certain cutoff [14]. For this case, one can modify the limits in the summations in Eq.(2) and carry out the calculations accordingly. We emphasize that Eq.(5) is applicable even if the steady state $P(p)$ is not symmetric about $p = 1/2$. An interesting feature in this generalized
EMG model is that when the cutoff percentage deviates significantly from 1/2 and becomes smaller (or larger) than a critical value, the steady state $P(p)$ takes on a form which depends on the initial distribution of $p$. In particular, the population distribution $P(p)$ freezes - no further modification of gene values arises as time evolves for large (or small) enough value of the cutoff. This phenomenon is discussed in more detail in Ref. [14]. Another generalization is to modify the way in which the $p$-value is updated [15]. Future work will focus on application of the present theoretical approach to such generalizations of the simple minority game set-up.
REFERENCES

[1] J.H. Holland, *Emergence: From chaos to order*, (1998) (Addison-Wesley, Reading);
*Hidden Order: How adaptation builds complexity* (1995) (Addison-Wesley, Reading).

[2] W.B. Arthur, Amer. Econ. Rev. 84, 406 (1994); Science 284, 107 (1999).

[3] H.E. Stanley, Computing in Science & Engineering Jan/Feb, 76 (1999); Physica A 269, 156 (1999).

[4] See the proceedings of the International Workshop on Econophysics and Statistical Finance published in Physica A 269, 1-183 (1999).

[5] D. Challet and Y.C. Zhang, Physica A 246, 407 (1997); *ibid.* 256, 514 (1998); *ibid.* 269, 30 (1999).

[6] R. Savit, R. Manuca and R. Riolo, Phys. Rev. Lett. 82, 2203 (1999).

[7] N.F. Johnson, M. Hart and P.M. Hui, Physica A 269, 1 (1999).

[8] D. Challet and M. Marsili, Phys. Rev. E 60, R6271 (1999); D. Challet, M. Marsili, and R. Zecchina, Phys. Rev. Lett. 84, 1824 (2000); D. Challet and M Marsili, cond-mat/9908480.

[9] N.F. Johnson, P.M. Hui, R. Jonson and T.S. Lo, Phys. Rev. Lett. 82, 3360 (1999).

[10] N.F. Johnson, P.M. Hui and T.S. Lo, Phil. Trans. Royal Soc. London A 357, 2013 (1999).

[11] P.M. Hui, T.S. Lo, and N.F. Johnson, cond-mat/0003309.

[12] R. D’Hulst and G.J. Rodgers, Physica A 270, 514 (1999).

[13] E. Burgos and H. Ceva, cond-mat/0003179.

[14] N.F. Johnson, D.J.T. Leonard, P.M. Hui and T.S. Lo, cond-mat/9905039.

[15] H. Ceva, cond-mat/9909424.
FIGURES

FIG. 1. The lifespan $L(p)$, which is the average duration between modifications for a gene value $p$, as a function of gene value $p$ for $m = 1, 2, \ldots, 8$. The inset shows the distribution of gene values $P(p)$ as a function of $p$ for different values of $m$. Both $L(p)$ and $P(p)$ are insensitive to $m$. The other parameters are $N = 101$, $d = -4$ and $R = 0.2$.

FIG. 2. The probability of the attendance in one of the two rooms in the steady state, which is identical to $F_N(n)$, obtained by numerical simulations. The parameters are $N = 101$, $m = 3$, $d = -4$ and $R = 0.2$. It is approximately a gaussian distribution as expected from the central limit theorem.

FIG. 3. The frequency distribution of the gene values $p$ as a function of $p$ for $N = 101$ and $N = 51$ (inset). The other parameters are $d = -4$ and $R = 0.2$. The dotted lines are the data from numerical simulation. The solid lines give the results of the present theory. The dashed lines give the results of the theory proposed in Ref.[12].

FIG. 4. The lifespan $L(p)$ as a function of $p$ for $N = 101$ and $N = 51$ (inset). The dotted lines are the data from numerical simulation. The solid lines give the results of the present theory. Other parameters are the same as those in Fig.3.

FIG. 5. The winning probability $\tau(p)$ as a function of $p$ for different values of $N$. The solid lines give the results of the present theory while the dotted and dashed lines are results from numerical simulations. The three sets of lines from top to bottom at $p = 1/2$ correspond to $N = 201$, 101, and 51, respectively. Other parameters are the same as those in Fig.3.
\[ \tau(p) \]