Sex and recombination in the Hötzel aging model

A.O. Sousa

Institute for Theoretical Physics, Cologne University, D-50937 Köln, Germany

Abstract

Why sex evolved and it prevails in nature remains one of the great puzzles of evolution. Most biologists would explain that it promotes genetic variability, however this explanation suffers from several difficulties. What advantages might sex confer? The present communication aims at certain investigations related to this question, in this way we introduce sexual recombination on the Hötzel model (with males and females) and we compare these results with those from asexual reproduction without recombination.

Keywords: Population dynamics; Aging; Monte Carlo Simulations; Evolution; Recombination

1 Introduction

Sex, which involves the alternation of meiosis and gamete fusion, is a rather inefficient means of self-propagation as compared to asexual reproduction, where offspring stem only from a mitotically produced cells. One of the most common reasons used to explain the origin and maintenance of sex is its ability to reduce the mutation load if consecutive mutations lead to an increasing decline in relative fitness, although it is not clear a priori that the heritable variance in fitness is significantly increased by sex. Despite decades of developing theoretical models to explain why sex is such a widespread phenomenon and how sexual reproduction may confer advantages that outweigh its disadvantages, until now no such general clear advantage has been found.

Investigations of evolutionary problems by physicists have in fact boomed in the last few years. Since computer simulations of natural systems can provide much insight into their fundamental mechanisms, they can be used to test theoretical ideas that could be otherwise viewed as too vague to deserve the status of scientific knowledge [1, 2]. In this way, many computer models in population dynamics have been proposed to investigate the evolution of sex and its justification, as well as the comparison between sexual and asexual reproduction, for instance, the Redfield model [3], the Penna bit-string model [4], a genomic bit-string model without aging [5] and Stauffer model [6, 7, 8].

1-e-mail: sousa@thp.uni-koeln.de

After 01.04.2003, correspondence should be addressed to: ICA1, University of Stuttgart Pfaffentalring 27, D-70569 Stuttgart, Germany
Of particular interest here is the Heumann-Hötzel model [9], which originally simulated the evolution of asexual population, composed of haploid individuals, without recombination. Thus now we introduce the recombination in this model, in order to find out if the sexual reproduction (with males and females) can produce better results than the simple asexual reproduction [10]. In the next section, we describe the standard and the modified Heumann-Hötzel model, in section 3, we present our results and in section 4, our conclusions.

2 Heumann-Hötzel Model

Since it has been proposed by Michael Heumann and Michael Hötzel in 1995, the Heumann-Hötzel model [9], which was an unsuccessful attempt to introduce more ages in the Dasgupta model [11], has remained forgotten due to the fact that after many generations it reduces to the two-age model of Partridge-Barton [12]. The Dasgupta model consists in taking into account some modifications such as hereditary mutations and food restrictions in the Partridge-Barton model. In fact, the Heumann-Hötzel paper [9], treats basically the computer simulations using the generalized Dasgupta aging model proposed by Michael Hötzel in his work, under the supervision of Dietrich Stauffer, in order to obtain the license to teach in German secondary school [13]. Michael Heumann, who was another teacher’s candidate, worked only on the inclusion of the "Dauer" state in the Dasgupta model [14].

Recently, the Heumann-Hötzel model was reinvestigated and, according to the authors, with “simple and minor change in the original model” this incapacity to describe populations with many ages seems to be surmounted [15].

In the original version of the Heumann-Hötzel, the genome of each (haploid) individual is represented by one set of probabilities $p_0, p_1, p_2, \ldots, p_{\text{maxage}-1}$, where $p_a$ is the survival probability that an individual has to reach age $a + 1$ from age $a$. At every time step $t$, $N(t) \times \text{maxage}$ individuals are chosen randomly to have their survival probability $p_a$ altered by mutations to $p_a' = p_a \times \exp(\epsilon)$, where the age $a$ is also randomly chosen. $N(t)$ is the size of the population at time $t$ and maxage is the maximum age one individual can live, which is set up in the beginning of the simulation. The quantity $\epsilon$ is chosen randomly as any number between $\epsilon_1$ and $\epsilon_2$ and when it is negative (positive) it corresponds to a deleterious (beneficial) mutation.

The effect of food and space restrictions is taken account by an age-independent Verhulst factor, which gives to each individual a probability $[1 - N(t)/N_{\text{max}}]$ of staying alive; $N_{\text{max}}$ represents the maximum possible size of the population. This mean-field probability of death for the computer simulations has the benefit of limiting the size of population to be dealt with. The passage of time is represented by the reading of a new locus in the genome of each individual in the population, and the increase of its age by 1. After taking account the natural selection and the action of Verhulst dagger, at the completion of each
period of life, each individual gives birth to one baby (age=0) which inherits its set of probabilities \((p'_0, p'_1, p'_2, \ldots, p'_{\text{maxage}-1})\).

In the recent reinvestigation of this model[15], individuals with age \(a\) in the interval \(a_{\text{min}} \leq a \leq a_{\text{max}}\) will generate \(b\) offspring and the mutations are allowed only on a fraction \(F\) \((0 \leq F \leq 1)\) of the babies.

Figure 1: Total population for asexual reproduction (squares), sexual reproduction: case (a) (triangles) and case (b) (stars). The inset shows the corresponding survival probability.

In the sexual version, each (diploid) individual of the population, which consists of males and females, is genetically represented now by two sets of survival probabilities, \(P(a)^1\) and \(P(a)^2\), to be read in parallel. In this way, we have studied the following cases (see below):

\[
P(a)^1 = (p^1_0, p^1_1, p^1_2, \ldots, p^1_{\text{maxage}-1})
\]

\[
P(a)^2 = (p^2_0, p^2_1, p^2_2, \ldots, p^2_{\text{maxage}-1})
\]
• **Case (a)** - The effective survival probability in some age will be the arithmetic average of the values present in both sets at that age:

\[
P(a)_{\text{effective}} = \left( \frac{p_0^1 + p_0^2}{2}, \frac{p_1^1 + p_1^2}{2}, \ldots, \frac{p_{\text{maxage}-1}^1 + p_{\text{maxage}-1}^2}{2} \right)
\]

• **Case (b)** - The effective survival probability in some age will be the maximum value between the values present in both sets at that age:

\[
P(a)_{\text{effective}} = \left( \max[p_0^1, p_0^2], \max[p_1^1, p_1^2], \ldots, \max[p_{\text{maxage}-1}^1, p_{\text{maxage}-1}^2] \right)
\]

![Figure 2: Survival probability for asexual reproduction (circles), sexual reproduction with \(a_{\text{max}} = 17\) and \(a_{\text{min}} = 1\) (triangles), 3 (stars), 5 (squares).](image)

If the female succeeds in surviving until the minimum reproduction age \(a_{\text{min}}\), it chooses, at random, an able male to mate \((a_{\text{min}} \leq \text{age} \leq a_{\text{max}})\) and it generates, with probability \(p_b\), \(b\) offspring every iteration until the maximum age of reproduction \(a_{\text{max}}\). The offspring inherits its set of survival probabilities from its
parents in the following way: the two sets of survival probabilities of the male, for instance, are broken in the same random position, and the complementary pieces, belonging to different strings, are joined to form two male gametes. One of the gametes is then randomly chosen to be passed to the offspring. After that, $m_m$ random mutations are introduced into this gamete, and the final result corresponds to one string of the baby genome. The same process occurs with the female genome, generating the second string of the baby, with $m_f$ mutations. At the end the offspring genome contains a total of $M = m_m + m_f$ mutations. Finally, the sex of the baby is randomly chosen, each one with probability 50%. This procedure is repeated for each of the $b$ offspring.
3 Results

The simulation starts with \( N_0 \) individuals (half for each sex) and runs for 400,000 time steps, at the end of which (the last 10,000 steps, when the population was stabilized) averages are taken over the population. The parameters of the simulations are:

- Initial population \( N_0 = 10,000 \);
- Maximum population size \( N_{\text{max}} = 100 \times N_0 \);
- Probability to give birth \( p_b = 1.0 \);
- Birth rate \( b = 1.0 \);
- Mutation rate \( m_m = m_f = 1 \) per gamete;
- \( \epsilon_1 = 0.02 \) and \( \epsilon_2 = -0.04 \) (the same values used in the original Ḧotzel model).

Our figures with \( N_0 = 10,000 \) are confirmed by larger simulations with \( N_0 = 100,000 \), and also by larger simulations with \( 10^7 \) time steps.

From Figure 1 and its inset we can see that the diploid sexual population is not only larger than the haploid asexual one, but also presents a higher survival probability.

In Figure 2 (case (a)) and Figure 3 (case (b)), we present the survival probability as a function of age for different period of reproduction \((a_{\text{min}} \leq i \leq a_{\text{max}})\).

Aging starts with reproduction: the survival rate decays as soon as reproduction age is reached. There are no individuals alive older than the maximum reproduction age \( a_{\text{max}} \). Figure 4 corresponds to the case in which all the individuals of the population reproduces only once - the so-called catastrophic senescence effect [12, 16]. In this way, two rules of reproducing were adopted: 1) The reproduction age is the same for all individuals \((a_{\text{max}} = a_{\text{min}} = 10)\), 2) The reproduction age is randomly chosen between \( a_{\text{min}} = 5 \) and \( a_{\text{max}} = 10 \). We can noticed that this effect is more pronounced for the former [12, 16], as well that the responsible for that are both breeding once and breeding for all individuals at the same age. The explanation for these effects observed in Figures 2-4 is based on the Darwin theory: individuals must stay alive in order to reproduce and perpetuate the species. If they can no longer generate offspring but remain in the population, they are killed by the accumulation of deleterious mutations [1].

In fact, real mutations can be divided into the common recessive (almost 90% of the real mutations are recessive) and the rare dominant mutations. In this way, if among the many genes of a species, one of the father’s genes differs from the corresponding one of the mother, then it adversely affects the child only if the mutation is dominant. Recessive mutations affect the child only if both the father and mother have them. In order to take into account this aspect in the sexual version of Ḧotzel, at the beginning of the simulation we choose randomly \( d \) dominant positions and keep them fixed during the whole simulation. The effective survival probability in the dominant positions (ages) will be the smallest value of the two located in the same position in both strands,
Figure 4: Survival probability for sexual reproduction in case of reproduction only at the reproduction age $a_{\text{max}} = a_{\text{min}} = 10$ (circles), and in case of reproduction at some age between $a_{\text{min}} = 5$ and $a_{\text{max}} = 10$ (triangles).

and for recessives ones the effective survival probability will be the arithmetic average of them. In Figure 5, we can see that the inclusion of dominance does not alter the lifespan of the population, although it has been observed that population evolved without dominance is larger than the other without dominance due the deaths in the former being bigger than the latter, since in these dominant positions the effective survival probability is the minimum value between the values present in both sets at that age. In the particular simulation shown, for age = 2 it is noticed a decrease in the survival probability when the dominance is considered, since the ages 2, 13, 10, 15 were dominant positions.

Figure 6 shows the time evolution of the total population of each age for sexual reproduction when the mutations are exclusively harmful. From this figure we can notice that a stable population for ages $a \leq 3$ is obtained, in contrast to the original model in which there are no individuals alive older than
Figure 5: Survival probability for asexual reproduction (circles), sexual reproduction with $a_{\text{min}} = 1$, $a_{\text{max}} = 17$ and $d = 1$ (squares), $2$ (triangles). Case (a) (top) and case (b) (bottom).

age $a > 2$, even if beneficial mutation and also a deleterious mutation rate 5 times smaller have been assumed. The result obtained here (Fig. 1), introducing sex in the original model, was found with the asexual Hötzel model [15] only when mutations were allowed on a very small fraction $F = 10\%$ of the babies and also a minimum age of reproduction $a_{\text{min}} = 8$ was considered. In our simulations, $F = 100\%$, $a_{\text{min}} = 1$ and $a_{\text{max}} = 17$.

4 Conclusions

We have shown that main problem related to the Hötzel model, which was its incapacity to treat populations with many age intervals, has been overcome by introducing recombination (with males and females) in this model, without any other assumptions. As well as, with the inclusion of sex in the model, the
Figure 6: The total population for ages 0, 1, 2, 3 (top to bottom, respectively) as a function of time for sexual reproduction when the mutations are exclusively harmful ($\epsilon_1 = 0.0$ and $\epsilon_2 = -0.20$).

population meltdown observed in the asexual version, when only deleterious mutations are considered, has been avoided. Moreover, in agreement with some earlier models, we have also obtained that the sexual reproduction (with males and females) produces better results than the asexual one.

Acknowledgments
I would like to thank Suzana Moss and Dietrich Stauffer for discussions and a critical reading of the manuscript, and DAAD for financial support.

References
[1] S. Moss de Oliveira, P.M.C. de Oliveira and D. Stauffer (1999) Evolution, Money, War and Computers, Teubner, Leipzig.
[2] M. Eigen (1971) Naturwissenschaften 58, 465; D. Charlesworth, M.T. Morgan and B. Charlesworth (1992) Genet. Res. Camb. 59, 49; L.D. Mueller and M.R. Rose (1996) Proc. Natl. Acad. Sci. USA 93, 15249; B. Charlesworth (2001) J. Theor. Biology 210, 47.

[3] R.J. Redfield (1994) Nature 369, 145; S. Siller, Nature 411, 689 (2001); R.S. Howard and C.M. Lively, Nature (London) 367, 554 (1994); C. Zeyl, T. Vanderford and M. Carter, Science 299, 555 (2003).

[4] A.T. Bernardes (1997) J. Stat. Phys. 86, 431; D. Stauffer, J.S. Sá Martins, S. Moss de Oliveira (2000) Int. J. Mod. Phys. C 11, 7; J.S. Sá Martins and D. Stauffer (2001) Physica A 294, 191.

[5] B. Örcal, E. Tüzel, V. Sevim, N. Jan, A. Erzan (2000) Int. J. Mod. Phys. C 11, 9736; E. Tüzel, V. Sevim, A. Erzan (2001) Proc. Natl. Acad. Sci. USA 98, 13774.

[6] D. Stauffer (2002) in Biological Evolution and Statistical Physics, edited by M. Lässig and A. Valleriani, Springer, Berlin-Heidelberg, p.258.

[7] D. Stauffer and J.P. Radomski (2001) Exp. Gerontol. 37, 175.

[8] A.O. Sousa, S. Moss de Oliveira and D. Stauffer (2001) Int. J. Mod. Phys. C 12, 1477.

[9] M. Heumann and M. Hötzel (1995) J. Stat. Phys. 79, 483.

[10] A.O. Sousa (2001) PhD. Thesis, Univ. Fed. Fluminense, Niterói, Rio de Janeiro, Brazil.

[11] S. Vollmar and S. Dasgupta (1994) J. Phys. I France 6 817; S. Dasgupta (1994) J. Phys. I 10, 1563.

[12] L. Partridge and N.H. Barton (1993) Nature 362, 305.

[13] Michael Hötzel (1994) Staatsexamensarbeit, Institute for Theoretical Physics, Cologne University, Cologne, Germany.

[14] Michael Heumann (1994) Staatsexamensarbeit, Institute for Theoretical Physics, Cologne University, Cologne, Germany.

[15] R.N. Onody and N.G.F. Medeiros (2001) Phys. Rev. E 64, 41915.

[16] M.R. Rose (1981) The Evolutionary Biology of Aging, Oxford Univ. Press; B. Charlesworth (1994) Evolution in Age-Structured Populations, Cambridge Univ. Press.