# Bistability in a Gene Pool GA with Mutation

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## Abstract

It is possible for a GA to have two stable fixed points on a single-peak fitness landscape. These can correspond to meta-stable finite populations. This phenomenon is called *bistability*, and is only known to happen in the presence of recombination, selection, and mutation. This paper models the bistability phenomenon using an infinite population model of a GA based on gene pool recombination. Fixed points and their stability are explicitly calculated. This is possible since the infinite population model of the gene pool GA is much more tractable than the infinite population model for the standard simple GA. For the needle-in-the-haystack fitness function, the fixed point equations reduce to a single variable polynomial equation, and stability of fixed points can be determined from the derivative of the single variable equation. We also show empirically that bistability can occur on a single-peak landscape where there is selective pressure toward the optimum at every point of the search space.

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# 1 Introduction

Intuitively, one would expect that a GA could have only a single stable fixed point on a single-peak fitness function. If all strings have distinct fitnesses, this is the case for both a mutation/selection GA Rowe (1999) and is conjectured to be the case for a crossover/selection GA Vose and Wright (1995). However, in this paper we show that a mutation/crossover/selection GA may have two stable fixed points on a single-peak fitness landscape. We call this phenomenon *bistability*.

When there is bistability, and when the infinite population model of the GA is started from the center of the simplex (i. e., from a population where all strings are represented equally), the GA model will converge to a population distribution which has a very small probability for the optimum string. However, when the model is started near the optimum string, the GA model will converge to a population distribution with a high probability for the optimum string. Thus, bistability can prevent a GA which is started with a random initial population from finding the global optimum. This is confirmed in Suzuki and Iwasa (1999) where they show that crossover can reduce the “time to convergence” on the needle-in-the-haystack fitness when the crossover rate is sufficiently small to be outside of the bistability region. However, as the crossover rate is increased to put the GA into the bistable region, then the “time to convergence” appears to be tending to infinity. (See section 7 for more details.)

Bistability occurs only for some settings of the parameters. For our models, the minimum string length is 6. As the string length increases, the minimum mutation rate for bistability decreases rapidly, while the maximum mutation rate is approximately proportional to the inverse string length.

Bistability is not an isolated phenomenon. While we use gene pool recombination and a needle-in-the-haystack fitness for our models, neither are necessary for bistability. Bistability occurs with uniform and $n$-point crossover, and with fitness functions that show a more gradual increase in fitness as one gets close to the needle (section 8). We can show by means of a model that bistability under binary tournament selection is very similar to bistability under proportional selection. (These results will appear elsewhere.)

Bistability was first discovered and investigated by Boerlijst, Bonhoeffer, and Nowak (1996) in the context of viral quasispecies and the AIDS virus. Other papers on bistability include Ochoa and Harvey (1997), Wright, Rowe, and Neil (2002). The last citation has a more complete review of the literature in this area. The current paper is a more thorough and rigorous analysis of bistability.

This paper presents an infinite population model[1] of a GA that uses gene pool recombination. Gene pool recombination is an alternative recombination method for GAs. An individual created by gene pool recombination is chosen from a probability distribution determined directly from the whole population rather than from two parents. Geiringer’s theorem Geiringer (1944) shows that gene pool recombination in the infinite population model can be viewed as the limit of repeated applications of two-parent recombination (without selection). For the infinite population model, gene pool recombination takes the population to linkage equilibrium in one step. Gene pool recombination is more tractable for analysis than two-parent recombination.

Using gene pool recombination is equivalent to a linkage equilibrium assumption in a model. Linkage equilibrium has been widely used as a simplifying assumption in approximate models of GAs. These include Suzuki and Iwasa (1999) and Prügel-Bennett and Rogers (2001).

[1] As shown in Vose (1999), running the GA on a finite population is equivalent to sampling from the infinite population model, so the infinite population model contains all of the information needed to run the GA on a finite population.

This paper uses Walsh basis analysis to show that for some interesting classes of fitness functions, the fixed points of a GA which uses gene pool recombination, mutation, and proportional selection can be expressed as solutions of single-variable polynomial equations. Further, for these classes of fitness functions, the stability of fixed points can be expressed in terms of the derivative of a single variable polynomial.

Vose and Wright (1998a), Vose and Wright (1998b), Vose (1999) used Walsh basis techniques to model the simple GA. Furutani (2001) used a Walsh techniques to analyze the simple GA on the one max fitness. The authors of this paper Wright, Rowe, Poli, and Stephens (2002) analyzed the algorithm of this paper on linear fitness functions.

Gene pool recombination was proposed in Syswerda (1993). Mühlenbein and Mahnig (2001), Mühlenbein and Mahnig (2000) have investigated the UMDA algorithm which is a GA that uses gene pool recombination and some kind of selection (proportional, tournament, truncation, or Boltzmann) and no mutation. They have experimentally verified that UMDA can successfully optimize a wide variety of fitness functions but is misled by deceptive problems. Further, gene pool recombination is used in population-based incremental learning (PBIL) Baluja and Caruana (1995), a machine learning technique.

Our results suggest that the gene pool GA model is a reasonable approximation to the two-parent recombination GA for the classes of fitness functions investigated in this paper. Thus, the gene pool GA model can be viewed as an approximate model of the two-parent recombination GA.

## 2 Notation

The search space for this paper is the set of all binary strings of length $\ell$ which will be denoted $\Omega$. The binary representation of a string induces a correspondence from the elements of $\Omega$ to the set of integers from 0 to $2^\ell - 1$. Thus, the integer 0 corresponds to the all-zeros string, and the integer $2^\ell - 1$ corresponds to the all-ones string. A sum over $i \in \Omega$ is equivalent to a sum from $i = 0$ to $i = 2^\ell - 1$.

The bitwise mod-2 sum of two strings $j$ and $k$ is denoted by $j \oplus k$; $\Omega$ is a group under this operation. Note that $j \oplus k$ is also the XOR of $j$ and $k$. The identity element is the string of zeros, which will be denoted by 0. The string of all ones will be denoted by $\mathbf{1}$. The bitwise AND (or mod-2 product) of strings $j$ and $k$ is denoted by $j \otimes k$. The ones-complement of $k$ is denoted by $\overline{k}$. The number of ones in a binary string $k$ is denoted by $\#k$.

For each $u \in \Omega$, $\Omega_u = \{i \in \Omega : i \otimes u = i\}$; $\Omega_u$ is the set of binary strings which have a 1 only in positions where $u$ has a 1. It is also a schema denoted by a string over $\{0,*\}$ where there are asterisks in those positions where the corresponding bit of $u$ is 1, and where the fixed positions are all zeros. For example, if $\ell = 6$ and $u = 010110$, then $\Omega_u$ is the special schema 0∗0∗∗0, and is also the set $\{000000, 000010, 000100, 000110, 010000, 010010, 010100, 010110\}$. Note that $\Omega_u$ is a subgroup of $\Omega$.

Let $\mathcal{L} = \{j \in \Omega : \#j = 1\}$. Under the identification of $\Omega$ with the integers, $\mathcal{L} = \{2^k : k = 0, 1, \ldots, \ell - 1\}$. Let $\mathcal{L}_u = \{j \in \mathcal{L} : j \otimes u = j\} = \mathcal{L} \cap \Omega_u$. For example, if $\ell = 6$ and $u = 21$, $\mathcal{L}_u = \{2^0, 2^2, 2^4\} = \{1, 4, 16\} = \{000001, 000100, 001000\}$.

The sets $\mathcal{L}$ and $\mathcal{L}_u$ will be used as index sets. This might seem unnatural since these are subsets of $\Omega$ which do not correspond to consecutive integers. If this bothers the reader, a product over $i \in \mathcal{L}$, such as $\prod_{i \in \mathcal{L}} S_i$, could be rewritten as $\prod_{k=0}^{\ell-1} S_{2^k}$.

A population (a multiset of $\Omega$) is represented as a population vector $x$ indexed by $\Omega$; $x_v = x_{v_0 v_1 \ldots v_{\ell-1}}$ is the fraction of the population which is string $v = v_0 v_1 \ldots v_{\ell-1}$, where

$v_0, v_1, \ldots, v_{\ell-1}$ are the bits of $v$. For example, if $\ell = 2$ and the population as a multiset is $\{00, 01, 01, 11\}$, then the corresponding population vector is $\langle \frac{1}{4},\ \frac{1}{2},\ 0,\ \frac{1}{4} \rangle$, where $\langle \ldots \rangle$ denotes a column vector. A population vector is a population-size independent representation of a population, and thus is natural for infinite-population models.

If a population $x$ depends on time (or GA generation) $t$, then $x(t)$ is the population at time $t$.

All population vectors are contained in the simplex $\Lambda = \{x : \sum_{j \in \Omega} x_j = 1 \text{ and } x_j \geq 0 \text{ for all } j\}$. The simplex is a natural setting for the dynamical systems model since it allows population vectors to range continuously over a subset of $\mathbb{R}^{2^\ell}$ and thus allows derivatives and calculus to be used.

A schema is a coset of $\Omega_u$ for some $u \in \Omega$. In other words, $\Omega_u \oplus v = \{i \oplus v : i \in \Omega_u\}$ is the schema determined by $u$ and $v$, where $u$ is a mask for the variable positions and $v$ specifies the fixed positions. We will always assume that $u \otimes v = 0$. In traditional notation, a schema is denoted by a string from the alphabet $\{0, 1, *\}$, where $*$ is a "don't care" symbol. The traditional notation schema $1{*}0{*}{*}1$ is $\Omega_{010110} \oplus 100001$. The string $u$ has 1s in the positions of the asterisks, and the string $v$ has 0s in the positions of the asterisks. The string $v$ agrees with the traditional notation string in those positions where there are not asterisks.

The $\Omega_u \oplus v$ notation for schemata emphasizes the algebraic structure of schemata, as opposed to the traditional notation which emphasizes the syntactic structure.

Let $x_v^{(u)}$ denote the schema frequency of the schema $\Omega_{\overline{u}} \oplus v$ relative to the population $x$. In other words,

$$x_v^{(u)} = \sum_{i \in \Omega_{\overline{u}} \oplus v} x_i = \sum_{i \in \Omega_{\overline{u}}} x_{i \oplus v}$$

Thus, $u$ is a mask for the fixed positions of the schema, and we assume that $v \otimes \overline{u} = 0$. Since $v \in \Omega_u$, $x^{(u)}$ can be regarded as a vector indexed over $\Omega_u$. $x^{(u)}$ is the vector of schema frequencies of the family of competing schemata whose variable positions are determined by $\overline{u}$. In other words, $x^{(u)}$ is the vector of marginal frequencies corresponding to the schema family $\{\Omega_u \oplus v : v \in \Omega_u\}$.

For another example, if $\ell = 6$, $u = 010110$ and $v = 100001$, then the schema frequency of the schema $1{*}0{*}{*}1 = \Omega_{010110} \oplus 100001$ is denoted by $x_{100001}^{(101001)}$.

The Walsh matrix $W$ is an $2^\ell$ by $2^\ell$ matrix defined by $W_{i,j} = (-1)^{\#(i \otimes j)}$. The Walsh matrix is symmetric and $W^{-1} = 2^{-\ell} W$.

If $x$ is a population vector, then the Walsh transform of $x$ is $Wx$ and is denoted by $\widehat{x}$. Similarly, a fitness function defines a vector over $\Omega$ by letting $f_i$ be the fitness of $i \in \Omega$. Then $\widehat{f} = Wf$ is the Walsh transform of this fitness vector.

Let $e_0, e_1, \ldots, e_{N-1}$ be the standard basis vectors for $\mathbb{R}^N$. Then the vectors $\widehat{e}_0, \widehat{e}_1, \ldots, \widehat{e}_{N-1}$ form the Walsh basis for $\mathbb{R}^N$. If $x$ is a vector then $\widehat{x} = Wx$ is expressed in the Walsh basis. In other words, $x_j$ is the $j$th coordinate of $x$ in the standard basis and $\widehat{x}_j$ is the $j$th coordinate of $x$ in the Walsh basis. If $A$ is a $2^\ell$ by $2^\ell$ matrix, then $WAW$ is the Walsh transform of $A$ and is denoted by $\widehat{A}$.

Note that our definition of the Walsh transform is slightly different from that of Vose (1999) in that our Walsh transform of a vector is $2^{\ell/2}$ times the Walsh transform given in Vose's book and our Walsh transform of a matrix is $2^\ell$ times the Vose Walsh transform of that matrix.

The delta function is defined by $\delta_{i,j} = 1$ if $i = j$ and $\delta_{i,j} = 0$ if $i \neq j$.

## 3 Linkage equilibrium, the Walsh basis, and schema averages

This section defines linkage equilibrium and gives some basic results about populations at linkage equilibrium.

**Lemma 1** *For any population vector $x$ and any $k \in \mathcal{L}$,*

$$x_0^{(k)} = \frac{1}{2}\left(1 + \widehat{x}_k\right) \qquad \textit{and} \qquad x_k^{(k)} = \frac{1}{2}\left(1 - \widehat{x}_k\right)$$
$$\widehat{x}_k = 2x_0^{(k)} - 1 = 1 - 2x_k^{(k)}$$

**Proof:** The lemma can be proved by comparing the definition of $x_j^{(k)}$ with $\widehat{x}_k$. It is also a special case of Theorem 19.2 of Vose (1999). $\square$

**Definition 2** *A population $x$ is in* **linkage equilibrium** *if*

$$x_k = \prod_{u \in \mathcal{L}} x_{u \otimes k}^{(u)}$$

Thus, a population is in linkage equilibrium if the frequency of each string is the product of the marginal distributions corresponding to each allele of each locus. (These marginal distributions are the 1-schema averages referred to in the definition.) For example, the population $\frac{1}{32}\langle 9,\ 3,\ 15,\ 5\rangle$ (with Walsh basis representation $\frac{1}{8}\langle 8,\ 4,\ -2,\ -1\rangle$) is in linkage equilibrium.

**Corollary 3** *Let $x$ be in linkage equilibrium. For any $v \in \Omega$,*

$$x_v = 2^{-\ell} \prod_{i \in \mathcal{L}} \left(1 + (-1)^{\#(i \otimes v)} \widehat{x}_i\right)$$

**Proof:** This simply rewrites the definition of linkage equilibrium using lemma 1. $\square$

**Theorem 4** *If population $x$ is in linkage equilibrium, then for any $k \in \Omega$,*

$$\widehat{x}_k = \prod_{j \in \mathcal{L}_k} \widehat{x}_j$$

A similar result is proved as theorem 10.9 of Vose (1999) and theorem 3.5 of Vose and Wright (1998b).

**Lemma 5** *If $x \in \Lambda$, then $|\widehat{x}_k| \leq 1$ for all $k \in \Omega$.*

**Proof:** The simplex $\Lambda$ is the convex hull of the basis vectors $e_0, e_1, \ldots, e_{N-1}$ of the standard basis. The vectors $\widehat{e}_0, \widehat{e}_1, \ldots, \widehat{e}_{N-1}$ are the same geometric points expressed in the Walsh basis, so the simplex is still the convex hull of these points. But these correspond to the columns of the Walsh matrix $W$, and every entry of this matrix is $\pm 1$. Thus $\widehat{x}_k$ is a convex combination of 1 and $-1$. $\square$

## 4 The Gene Pool Model in the Walsh Basis

First we describe the finite population gene pool GA. The population size is assumed to be $r$ and the string length is $\ell$.

1 Choose a random population $x$.
2 Calculate the order-1 schema averages $x_0^{(k)}$ for each $k \in \mathcal{L}$.
3 Construct a new linkage equilibrium population with the same order-1 schema averages $x_0^{(k)}$:
  3.1 **for** $i$ **from** $0$ **to** $r-1$ **do**
  3.1.1 **for** $k \in \mathcal{L}$ **do**
  3.1.2 Choose bit $k$ of individual $i$ to be a 0 with probability $x_0^{(k)}$ and 1 otherwise.
4 Apply selection to this population.
5 Apply mutation according to bitwise mutation rate $\mu$.
6 Test for termination.
7 Go to step 2.

Step 3 corresponds to gene pool recombination.

The infinite population model will be described in terms of the order-1 Walsh coefficients. The model will be specified as a function

$$\mathcal{G} = \mathcal{U} \circ \mathcal{F} \circ \mathcal{M} : \Lambda \longrightarrow \Lambda$$

where $\mathcal{U}$ is mutation and $\mathcal{F}$ is selection as defined below. This paper emphasizes the proportional selection and the NEEDLE fitness function.

### 4.1 Gene pool recombination

Define the gene pool recombination function $\mathcal{M} : \Lambda \longrightarrow \Lambda$ by

$$\widehat{\mathcal{M}(x)}_k = \begin{cases} \widehat{x}_k & \text{if } k \in \mathcal{L} \cup \{0\} \\ \prod_{j \in \mathcal{L}_k} \widehat{x}_j & \text{otherwise} \end{cases}$$

By definition, $\widehat{\mathcal{M}(x)}$ is in linkage equilibrium, and $\mathcal{M}(x)$ only depends on $\widehat{x}_k$, $k \in \mathcal{L}$.

### 4.2 Proportional selection

Following Vose (1999), the effect of proportional selection can be described by a function $\mathcal{F} : \Lambda \longrightarrow \Lambda$. The probability that an individual $k \in \Omega$ is chosen to be in the new population is $\mathcal{F}(x)_k$.

Let $\sigma_k$ denote the $2^\ell \times 2^\ell$ matrix defined by $(\sigma_k)_{i,j} = \delta_{i \oplus k, j}$. Note that $\sigma_k$ is symmetric. It is easy to show that $(\sigma_k x)_i = x_{i \oplus k}$.

The following lemma tells how to compute proportional selection in the Walsh basis. It is proved as theorem 4.4 of Vose and Wright (1998a).

**Lemma 6** *For all $k \in \Omega$:*

$$\widehat{\mathcal{F}(x)}_k = \frac{\sum_{i \in \Omega} \widehat{f}_{i \oplus k} \widehat{x}_i}{\widehat{f}^T \widehat{x}} = \frac{\sum_{i \in \Omega} \widehat{f}_i \widehat{x}_{i \oplus k}}{\widehat{f}^T \widehat{x}} = \frac{\widehat{f}^T \sigma_k \widehat{x}}{\widehat{f}^T \widehat{x}}$$

### 4.3 Mutation

Following Wright (1999), the effect of mutation can be described by a function $\mathcal{U} : \Lambda \longrightarrow \Lambda$. The probability that an individual $k \in \Omega$ is chosen to be in the new population is $\mathcal{U}(x)_k$.

The following lemma shows how to compute mutation in the Walsh basis. The proof is not difficult, but is not given here for space reasons. A similar result is proved in Wright (1999).

**Lemma 7**

$$\widehat{\mathcal{U}(x)}_k = (1-2\mu)^{\#k}\widehat{x}_k \qquad \textit{for} \quad k \in \mathcal{L}$$

### 4.4 The gene pool GA for a general fitness vector expressed in the standard basis

We make the assumption that the fitness of every individual is positive. Since proportional selection is invariant under multiplication of the fitness by a positive constant, we can assume that the minimum fitness is 1. Thus, the fitness vector $f$ can be written in the form

$$f = \mathbf{1} + \sum_{k\in\Omega}(f_k - 1)e_k$$

The Walsh transform of $\mathbf{1}$ is $2^\ell e_0$, where $e_0$ represents the vector with a 1 in the first position and zeros in all other positions. Thus,

$$\widehat{f}_i = \begin{cases} 2^\ell + \sum_{j\in\Omega}(f_j - 1) & \text{if } i = 0 \\ \sum_{j\in\Omega}(-1)^{\#(j\otimes i)}(f_j - 1) & \text{if } i \neq 0 \end{cases}$$

**Theorem 8** *Let $x$ be a population. Then for any $k \in \Omega$,*

$$\widehat{\mathcal{G}(x)}_k = (1-2\mu)^{\#k}\frac{2^\ell \prod_{j\in\mathcal{L}_k}\widehat{x}_j + \sum_{i\in\Omega}(-1)^{\#(i\otimes k)}(f_i - 1)\prod_{j\in\mathcal{L}}\left(1 + (-1)^{\#(i\otimes j)}\widehat{x}_j\right)}{2^\ell + \sum_{i\in\Omega}(f_i - 1)\prod_{j\in\mathcal{L}}(1 + (-1)^{\#(i\otimes j)}\widehat{x}_j)} \tag{1}$$

*For $k \in \mathcal{L}$, this specializes to:*

$$\widehat{\mathcal{G}(x)}_k = (1-2\mu)\frac{2^\ell \widehat{x}_k + \sum_{i\in\Omega}(-1)^{\#(i\otimes k)}(f_i - 1)\prod_{j\in\mathcal{L}}\left(1 + (-1)^{\#(i\otimes j)}\widehat{x}_j\right)}{2^\ell + \sum_{i\in\Omega}(f_i - 1)\prod_{j\in\mathcal{L}}(1 + (-1)^{\#(i\otimes j)}\widehat{x}_j)} \tag{2}$$

Formula (2) is a recurrence that defines the infinite population gene pool GA model in terms of the variables $\widehat{x}_k$ for $k \in \mathcal{L}$. It allows us to compute $\mathcal{G}(x)$ efficiently when most of the fitnesses of individuals are the same.

**Proof:** Let $y = \mathcal{M}(x)$ (so that $y$ is in linkage equilibrium).

First we calculate $\widehat{\mathcal{F}(y)}$ using Lemma 6. For $k \in \Omega$:

$$\begin{aligned}
\widehat{\mathcal{F}(y)}_k &= \frac{\sum_{i\in\Omega}\widehat{y}_i\widehat{f}_{i\oplus k}}{\sum_{i\in\Omega}\widehat{y}_i\widehat{f}_i} \\
&= \frac{2^\ell\widehat{y}_k + \sum_{i\in\Omega}\widehat{y}_i\sum_{j\in\Omega}(-1)^{\#(j\otimes(i\oplus k))}(f_j - 1)}{2^\ell + \sum_{i\in\Omega}\widehat{y}_i\sum_{j\in\Omega}(-1)^{\#(j\otimes i)}(f_j - 1)} \\
&= \frac{2^\ell\widehat{y}_k + \sum_{j\in\Omega}(-1)^{\#(j\otimes k)}(f_j - 1)\sum_{i\in\Omega}(-1)^{\#(i\otimes j)}\widehat{y}_i}{2^\ell + \sum_{j\in\Omega}(f_j - 1)\sum_{i\in\Omega}(-1)^{\#(j\otimes i)}\widehat{y}_i} \\
&= \frac{2^\ell\widehat{y}_k + \sum_{j\in\Omega}(-1)^{\#(j\otimes k)}(f_j - 1)2^\ell y_j}{2^\ell + \sum_{j\in\Omega}(f_j - 1)2^\ell y_j} \qquad \text{by the Walsh transform formula} \qquad (3) \\
&= \frac{2^\ell\widehat{y}_k + \sum_{i\in\Omega}(-1)^{\#(i\otimes k)}(f_i - 1)\prod_{j\in\mathcal{L}}\left(1 + (-1)^{\#(i\otimes j)}\widehat{y}_j\right)}{2^\ell + \sum_{i\in\Omega}(f_i - 1)\prod_{j\in\mathcal{L}}(1 + (-1)^{\#(i\otimes j)}\widehat{y}_j)} \qquad \text{by Corollary 3}
\end{aligned}$$

Since $\widehat{y}_k = \prod_{j\in\mathcal{L}_k} \widehat{x}_j$, and since $\mathcal{G}(x) = \mathcal{U}(\mathcal{F}(\mathcal{M}(x))) = \mathcal{U}(\mathcal{F}(y))$, for $k \in \mathcal{L}$, equation (1) follows.

If $k \in \mathcal{L}$, then $\#k = 1$ and $\widehat{y}_k = \widehat{x}_k$, so equation (2) follows. □

## 5 The NEEDLE fitness function

The NEEDLE (needle-in-the-haystack) fitness function assigns a fitness of $1 + a$ (where $a > 0$) to the string of all zeros, and assigns a fitness of 1 to all other strings.

There has been extensive work on the NEEDLE fitness function and on mutation/selection models using this fitness function; see Eigen, McCaskill, and Schuster (1988) and Nowak and Schuster (1989).

Equation (1) can be specialized to the case of the NEEDLE fitness function:

$$\widehat{\mathcal{G}(x)}_k = (1-2\mu)\frac{2^\ell \widehat{x}_k + a\prod_{i\in\mathcal{L}}(1+\widehat{x}_i)}{2^\ell + a\prod_{i\in\mathcal{L}}(1+\widehat{x}_i)} \tag{4}$$

The recurrence leads immediately to the fixed point equations:

$$\widehat{x}_k = \frac{(1-2\mu)a\prod_{i\in\mathcal{L}}(1+\widehat{x}_i)}{2^{\ell+1}\mu + a\prod_{i\in\mathcal{L}}(1+\widehat{x}_i)} \tag{5}$$

The right side of this equation is the same for all $k$, so if $\mu > 0$ and if $x$ is a fixed point of $\mathcal{G}$, then $\widehat{x}$ is symmetric in the sense that all $\widehat{x}_k$ for $k \in \mathcal{L}$ are equal. Such a population is compatible with unitation classes: all strings in the same unitation class have the same frequency.

Thus, the fixed point equation (5) can be simplified by replacing all components of $\widehat{x}$ with a scalar variable $w$. The resulting equation is:

$$2^{\ell+1}\mu w + a(w-1+2\mu)(1+w)^\ell = 0 \tag{6}$$

Thus, we have reduced the fixed point equations to a single polynomial equation in a single variable. This will allow for the more detailed analysis that we will do in the next sections.

If the GA model is started with a symmetric population (such as the population where all strings have equal probability and where $\widehat{x}_i = 0$ for all $i \neq 0$, or the uniform population consisting of copies of the all-zeros string where $\widehat{x}_i = 1$ for all $i$), symmetry will be maintained. In this case, the recurrence equations can also be written in terms of the variable $w$.

$$\mathcal{G}(w) = (1-2\mu)\frac{2^\ell w + a(1+w)^\ell}{2^\ell + a(1+w)^\ell} \tag{7}$$

These symmetry results are not true for other fitness functions of unitation. (Mühlenbein and Mahnig 2001, theorem 2.3) have shown that when $\mu = 0$, vertices of the simplex correspond to stable fixed points if and only if the corresponding binary string is fitter than its Hamming neighbors. This makes it easy to construct non-symmetric stable fixed points using fitness functions of unitation. For example if $\ell = 2$ and $f = \langle 1,2,2,1\rangle$, then the uniform populations corresponding to the strings 01 and 10 are stable fixed points. For a sufficiently small mutation rate, there are stable fixed points near these points in the interior of the simplex.

Next we investigate the stability of the fixed points.

If $g : \mathbb{R}^N \to \mathbb{R}^N$ is any differentiable function, the differential of $g$ is the $N \times N$ matrix defined by

$$(dg_x)_{i,j} = \frac{\partial g_i(x)}{\partial x_j}$$

It is well known that if all of the eigenvalues of $dg_x$ have modulus less than 1, then $x$ is an asymptotically stable fixed point of $g$.

Thus, we are interested in computing $d\mathcal{G}_{\widehat{x}}$ for the fixed points $\widehat{x}$ of $\mathcal{G}$. We will show that the maximum modulus eigenvalue of $d\mathcal{G}_{\widehat{x}}$ is equal to the derivative of the single-variable $\mathcal{G}$ function defined in equation (7). Thus, we show that the stability of fixed points in the cube $[-1,1]^\ell$ is the same as the stability of fixed points in the one variable space of symmetric populations. Thus, fixed points can be found by solving a polynomial equation in a single variable, and their stability can be determined by taking a single variable derivative. This is not true in general—for example if the fitness vector is $f = \langle 1, 2, 2, 1 \rangle$, then there is a fixed point at the center of the cube (or simplex) which is stable in the space of symmetric populations but unstable in the space of all populations.

**Lemma 9** *Let* $P = \prod_{i \in \mathcal{L}} (1 + \widehat{x}_i)$, $P_j = \prod_{i \in \mathcal{L} \setminus \{j\}} (1 + \widehat{x}_i)$. *Then for* $j \neq k$,

$$\frac{\partial \widehat{\mathcal{G}(x)}_k}{\partial \widehat{x}_j} = (1 - 2\mu) \frac{2^\ell a (1 - \widehat{x}_k) P_j}{(2^\ell + aP)^2}$$

$$\frac{\partial \widehat{\mathcal{G}(x)}_k}{\partial \widehat{x}_k} = (1 - 2\mu) \frac{2^{2\ell} + 2^\ell a P + 2^\ell a (1 - \widehat{x}_k) P_k}{(2^\ell + aP)^2}$$

*Furthermore, both of these partial derivatives are nonnegative for* $0 \le \mu \le 1/2$ *and* $\widehat{x}$ *representing a point in the simplex.*

**Proof:** Note that $\frac{\partial P}{\partial \widehat{x}_j} = P_j$. Then the computation of the partials is straightforward but tedious. For $\widehat{x}$ in the simplex, $-1 \le \widehat{x}_i \le 1$ by lemma 5, and this implies that $P$ is nonnegative. $\square$

**Lemma 10** *Let* $P = (1 + w)^\ell$, $P' = \ell(1 + w)^{\ell - 1}$. *Then the derivative of the symmetric* $\mathcal{G}$ *defined in equation (7) is given by:*

$$\frac{d\mathcal{G}}{dw} = (1 - 2\mu) \frac{2^{2\ell} + 2^\ell a P + 2^\ell a (1 - w) P'}{(2^\ell + aP)^2}$$

**Lemma 11** *Let* $A$ *be an* $\ell \times \ell$ *matrix where all of the diagonal entries are equal to* d *and all of the off-diagonal entries are equal to* e. *Then the eigenvalues of* $A$ *are* $d + (\ell - 1)e$ *with multiplicity 1 and* $d - e$ *with multiplicity* $\ell - 1$.

**Proof:** The eigenvalues of a matrix are unchanged if a multiple of one column is added to another column. We will apply these operations to transform $A$ into a upper triangular matrix.

First, add the sum of columns 2 to $\ell$ to column 1. Now every entry of column 1 is $d + (\ell - 1)e$. Then subtract column $\ell$ from each of columns 2 to $\ell - 1$. This makes the matrix upper triangular except for the last column, and the diagonal entries in columns 2 to $\ell-1$ are $d-e$. Add $-e/(d+(\ell-1)e)$ times column 1 to column $\ell$. This makes all off-diagonal entries of column $\ell$ equal to zero and makes the diagonal entry equal to $d-e$. So the matrix is now upper triangular, the first diagonal entry is $d+(\ell-1)e$, and the remaining diagonal entries are $d-e$.

The eigenvalues of a triangular matrix are the diagonal entries. □

**Theorem 12** *At a symmetric point $\widehat{x}$ where $\widehat{x}_k = w$ for all $k \in \mathcal{L}$,*

$$\frac{d\mathcal{G}}{dw} = \frac{\partial \widehat{\mathcal{G}(x)}_k}{\partial \widehat{x}_k} + (\ell-1)\frac{\partial \widehat{\mathcal{G}(x)}_k}{\partial \widehat{x}_j} \tag{8}$$

*Thus, the largest modulus eigenvalue of $d\mathcal{G}_{\widehat{x}}$ is equal to $\frac{d\mathcal{G}}{dw}$.*

**Proof:** The formula follows by comparing the formulas from lemmas 9 and 10. Lemma 9 shows that the entries of $d\mathcal{G}_x$ are nonnegative, and Lemma 11 shows that its largest eigenvalue is the one with multiplicity 1 given by the right side of formula (8). □

To summarize, we have shown that we can find the fixed points of the model by solving a single-variable degree $\ell+1$ polynomial equation, and the stability of fixed points in the cube can be determined by evaluating the derivative of a single-variable degree $\ell+1$ polynomial. We know of no other case where the fixed points of a 3-operator GA on an interesting class of fitness functions can be so easily found and analyzed for stability for large string lengths.

# 6 The fixed points for the NEEDLE fitness

In this section we analyze the fixed points for the NEEDLE fitness. One way to understand the fixed points is to plot the symmetric fixed point equation (6) as a function of $a$. Thus, we rewrite equation (6) by solving for $a$:

$$a = \frac{2^{\ell+1}\mu w}{(1-w-2\mu)(1+w)^\ell} \tag{9}$$

Figure 1 shows $a$ plotted as a function of $w$ for $\ell = 9$, $\mu = 0.03$. For a fixed value $a_f$ of $a$, the $w$ values for the fixed points can be found by intersecting the horizontal line $a = a_f$ with the graph (or by substituting $a_f$ for $a$ in equation (6) and solving for $w$). For example, for $a = 1$, there are fixed points at $0.0425$, $0.4478$, and $0.8287$.

The $(w, a)$ points where the curve has a zero slope can be critical values. As $a$ increases through such a value, a pair of fixed points can appear or disappear. The points $(w0, a0)$ and $(w1, a1)$ in figure 1 are such points. For $a < a1$, there is one fixed point in the interval $[0, 1]$; for $a1 < a < a0$ there are three fixed points; and for $a0 < a$ there is one fixed point.

Thus, it is useful to find those points where this curve has slope 0. We differentiate equation (9) with respect to $w$ and set the derivative to 0, and solve for $w$. The solutions are given by

$$w_c = \frac{-2\ell\mu + 2\mu + \ell - 1 \pm \sqrt{4\mu^2 - 4\mu + 16\ell\mu - 8\ell\mu^2 + 1 - 6\ell + \ell^2 - 4\ell^2\mu + 4\ell^2\mu^2}}{2\ell}$$

The critical values for $a$ can be found by substituting these critical values for $w$ into (9). For example, for $\ell = 9$, $\mu = 0.03$, the critical points shown in figure 1 are $(w0, a0) = (0.1530, 1.658)$, $(w1, a1) = (0.6825, 0.7536)$.

For a fixed value $a_f$ of $a$, the critical values for the mutation rate $\mu$ can be found by substituting the formula for $w_c$ written as a function of $\mu$ into (6), and also substituting $a_f$ for $a$, and solving for $\mu$. For example, for $\ell = 9$ and $a = 1$, the critical mutation values are $\mu_l = 0.01861$ and $\mu_u = 0.037540$. Below the lower critical mutation $\mu_l$ rate, there is one fixed point population near the needle (which is stable). Between the two critical mutation rates, there are three fixed point populations, one stable near the needle, one unstable, and one stable which is near a random population. In other words, there is bistability.

By analyzing equation (9), we can give a definitive characterization of the fixed points.

**Theorem 13** *If $0 < \mu < 1/2$, then there can be at most 3 fixed points. For $a$ and $\mu$ where there is one fixed point, it is stable. When there are three fixed points, they are stable, unstable, and stable when ordered by $w$ values. If there are 2 fixed points, then a small perturbation of either $a$ or $\mu$ can give one fixed point.*

**Proof:** For $w < 0$, the graph of (9) is below the $a = 0$ axis, so there are no fixed points under our assumption that $a > 0$.

The graph of (9) crosses the $a = 0$ axis only at $w = 0$. Thus, for fixed $\mu$ and sufficiently small $a > 0$, there must be a single fixed point near $w = 0$ (the center of the simplex). As $w$ approaches $1 - 2\mu$ from the left, the graph goes to positive infinity. Thus, for fixed $\mu$ and sufficiently large $a$, there must be a single fixed point near $1 - 2\mu$.

We have already seen that there are at most two points where the graph has zero slope. Thus, the graph can have at most one local maximum and one local minimum, and for fixed $\mu$, a horizontal line corresponding to a fixed $a$ value can intersect the curve in at most 3 points.

We have show earlier that the stability of the fixed points in the simplex is the same as the stability of equation (7). The graph of (7) starts above the diagonal and ends below the diagonal. Each intersection of the graph with the diagonal gives a fixed point, and if the slope of the graph at the intersection has absolute value less than 1, the fixed point is stable.

Thus, after a small perturbation of $a$, the graph must either cross the diagonal once from above the diagonal to below, or cross the diagonal three times, once from above to below, once from below to above, and once from above to below. Furthermore, we can assume that the derivative at the crossing point is not equal to 1.

Since $\frac{d\mathcal{G}}{dw} > 0$ from equation (7), at a fixed point where the graph crosses the diagonal from above to below must be stable. A fixed point where the graph crosses from below to above must be unstable. $\square$

Thus, we have exactly characterized the fixed points for the gene pool model on the NEEDLE fitness function. For fixed values of $a$ and $\mu$, the location of the fixed points can be found using equation (6). For a fixed value of $\mu$, the range of values of $a$ which give bistability can be found, and for a fixed value of $a$ the range of values of $\mu$ which give bistability can be found.

The whole system exhibits what is known as hysteresis. In a system with varying parameters, the state of the system depends on the history of how that state was reached in parameter space. For example, suppose that the system is started with a random population and a mutation rate between the upper and lower mutation rates. It will then converge to the random fixed point. If the mutation is lowered below the lower critical mutation rate, the system will jump to the needle fixed point, and if the mutation is then raised back to its original value, the system will stay on the needle fixed point.

Figure 2 shows how the critical values change for a fixed string length as the mutation changes. The horizontal axis is $w$, and the vertical axis is $a$. The curves correspond to mutation rates of

$0.02, 0.03, 0.04, 0.05$. For a given $a$ value $a_f$, intersecting the horizontal line $a = a_f$ with the curve for a particular $\mu$ value gives the fixed points for those $a$ and $\mu$ values.

Figure 3 shows the region of bistability in $(\mu, a)$ space for $\ell = 9$. The horizontal axis is the mutation rate $\mu$ and the vertical axis is the needle height parameter $a$. Between the two curves, the system is bistable. Underneath the lower curve, only the needle fixed point is stable. Above the upper curve, only the random fixed point is stable.

Figure 4 shows how the bistability phenomenon depends on the string length. There is a small bistability region for small $a$ for $\ell = 6$. There is no bistability for $\ell < 6$.) Figure 4 shows the upper and lower critical mutation rates for $a = 1/5$, $a = 1$, and $a = 5$.

We see that the upper critical mutation rate decreases relatively slowly as a function of string length. The lower critical mutation rate decreases relatively rapidly. The graph of lower critical mutation rate is concave down on the semilog plot, so this suggests that it is decreasing faster than exponentially.

## 7 A gene pool GA model with a recombination rate

In this section we develop a model where gene pool recombination is applied with a probability (or recombination rate) of $\alpha$. The general model is not as elegant since we can no longer assume that the population is in linkage equilibrium immediately after the recombination stage. Thus, the model must keep track of all $2^\ell$ strings rather than only the $\ell$ allele frequencies. We apply this model to the NEEDLE fitness function. For this fitness function we can reduce the fixed point equation to a single polynomial in a single variable.

In this section, we let $\mathcal{M}_\alpha : \Lambda \to \Lambda$ be defined by $\mathcal{M}_\alpha(x) = \alpha\mathcal{M}(x) + (1-\alpha)x$ where $\mathcal{M}$ denotes the gene pool recombination function and where $0 \le \alpha \le 1$. Thus, gene pool recombination is done with probability $\alpha$ and "cloning" is done with probability $1 - \alpha$.

This corresponds to modeling the following finite population algorithm with a population size $r$:

1 Choose a random population.
2 Calculate the order-1 schema averages $x_0^{(k)}$ for each $k \in \mathcal{L}$.
3 Construct a new population by:
  3.1 **for** $i$ **from** $0$ **to** $r - 1$ **do**
  3.1.1 Choose a random number $p$ from $[0, 1]$ by sampling from a uniform distribution.
  3.1.1 **if** $p < \alpha$ **then**
  3.1.1.1 **for** $k \in \mathcal{L}$ **do**
  3.1.1.1.1 Choose bit $k$ of individual $i$ to be a 0 with probability $x_0^{(k)}$ and a 1 otherwise.
  3.1.2 **else**
  3.1.2.1 Copy a randomly chosen individual from the current population to the new population.
4 Apply proportional selection to this population.
5 Apply mutation according to bitwise mutation rate $\mu$.
6 Test for termination.
7 Go to step 2.

First, we consider the case where there is no recombination ($\alpha = 0$).

**Theorem 14** *Let $\mathcal{G}(x) = \mathcal{U}(\mathcal{F}(x))$. Then for the NEEDLE fitness function and for each $k \in \Omega$,*

$$\widehat{\mathcal{G}(x)}_k = (1-2\mu)^{\#k} \frac{\widehat{x}_k + ax_0}{1 + ax_0} \tag{10}$$

*where $x_0 = 2^{-\ell} \sum_{j \in \Omega} \widehat{x}_j$.*

**Remark:** This is an exact formulation of the Eigen model.

**Proof:** Note that in the proof of Theorem 8, the derivation of equation (3) does not assume that $y$ is in linkage equilibrium. Substituting the NEEDLE fitness into this equation gives the result. □

**Theorem 15** *Let $\mathcal{G}_\alpha(x) = \mathcal{U}(\mathcal{F}(\mathcal{M}_\alpha(x)))$. For the NEEDLE fitness function and for each $k \in \Omega$,*

$$\widehat{\mathcal{G}_\alpha(x)}_k = (1-2\mu)^{\#k} \frac{\alpha \prod_{j \in \mathcal{L}_k} \widehat{x}_j + (1-\alpha)\widehat{x}_k + E}{1+E} \tag{11}$$

*where*

$$E = 2^{-\ell} a \left( \alpha \prod_{i \in \mathcal{L}} (1 + \widehat{x}_i) + (1-\alpha) \sum_{j \in \Omega} \widehat{x}_j \right)$$

**Proof:** Let $y = \mathcal{M}_\alpha(x)$. Then

$$\widehat{y}_k = \alpha \prod_{j \in \mathcal{L}_k} \widehat{x}_j + (1-\alpha)\widehat{x}_k$$

For the NEEDLE fitness, $\widehat{f}_i = a + 2^\ell \delta_{i,0}$ and $(\widehat{f}^T \sigma_k)_i = (\sigma_k \widehat{f})_i = a + 2^\ell \delta_{i,k}$. Thus,

$$\begin{aligned}
\widehat{f}^T \widehat{y} &= 2^\ell + a \left( \alpha \sum_{i \in \Omega} \prod_{j \in \mathcal{L}_i} \widehat{x}_j + (1-\alpha) \sum_{i \in \Omega} \widehat{x}_i \right) \\
&= 2^\ell + a \left( \alpha \prod_{j \in \mathcal{L}} (1 + \widehat{x}_j) + (1-\alpha) \sum_{i \in \Omega} \widehat{x}_i \right) \qquad \text{by corollary 3}
\end{aligned}$$

and

$$\widehat{f}^T \sigma_k \widehat{y} = 2^\ell \left( \alpha \prod_{j \in \mathcal{L}_k} \widehat{x}_j + (1-\alpha)\widehat{x}_k \right) + a \left( \alpha \prod_{j \in \mathcal{L}} (1 + \widehat{x}_j) + (1-\alpha) \sum_{i \in \Omega} \widehat{x}_i \right)$$

Then for $k \in \Omega$,

$$\begin{aligned}
&\widehat{\mathcal{G}(x)}_k = (1-2\mu)^{\#k} \frac{\widehat{f}^T \sigma_i \widehat{y}}{\widehat{f}^T \widehat{y}} \\
&= (1-2\mu)^{\#k} \frac{2^\ell \left( \alpha \prod_{j \in \mathcal{L}_k} \widehat{x}_j + (1-\alpha)\widehat{x}_k \right) + a \left( \alpha \prod_{j \in \mathcal{L}} (1 + \widehat{x}_j) + (1-\alpha) \sum_{i \in \Omega} \widehat{x}_i \right)}{2^\ell + a \left( \alpha \prod_{j \in \mathcal{L}} (1 + \widehat{x}_j) + (1-\alpha) \sum_{i \in \Omega} \widehat{x}_i \right)}
\end{aligned}$$

The theorem follows by multiplying numerator and denominator by $2^{-\ell}$. □

**Theorem 16** *The fixed points of $\mathcal{G}_\alpha$ are symmetric in unitation classes. In other words, if $x$ is such a fixed point, then $\#j = \#k$ implies $\widehat{x}_j = \widehat{x}_k$.*

**Proof:** First consider the case of $k \in \mathcal{L}$. Then $\widehat{x}_k = \prod_{j \in \mathcal{L}_k} \widehat{x}_j$. The fixed point equations can be "solved" for $\widehat{x}_k$ as follows:

$$\widehat{x}_k = \frac{(1-2\mu)E}{E+2\mu}$$

Since the right-hand-side is the same for any $k$ with $\#k = 1$, $\widehat{x}_k$ is independent of $k$.

In the case where $\#k > 1$, we can assume that $\alpha < 1$ since for $\alpha = 1$ we do not need to keep track of $\widehat{x}_k$ for $\#k > 1$. Since the $\widehat{x}_j$, $j \in \mathcal{L}$, do not depend on $j$, $\prod_{j \in \mathcal{L}_k} \widehat{x}_j$ depends only on $\#k$. So again the equations can be "solved" for $\widehat{x}_k$:

$$\widehat{x}_k = \frac{(1-2\mu)^{\#k}\left(\alpha \prod_{j \in \mathcal{L}} \widehat{x}_j + E\right)}{1 + E - (1-2\mu)^{\#k}(1-\alpha)}$$

Again, the right-hand-side is independent of the representative of the unitation class chosen. □

We derive fixed point equations. We will be able to reduce the equations to a single polynomial equation in one variable. In view of theorem 16, we only need keep track of one variable per unitation class. Thus, if $x$ is a point so that $\widehat{x}_k$ depends only on $\#k$, let $w_i = \widehat{x}_k$ for all $k$ with $\#k = i$. In terms of these variables,

$$E = 2^{-\ell} a \left( \alpha(1+w_1)^\ell + (1-\alpha)\sum_{i=0}^{\ell} w_i \right) \tag{12}$$

The fixed point equations are obtained from equation (11).

$$w_k = (1-2\mu)^k \frac{\alpha w_1^k + (1-\alpha)w_k + E}{1+E}$$

For $k = 1$ the equation is:

$$w_1 = (1-2\mu)\frac{w_1 + E}{1+E}$$

This can be solved for $E$:

$$E = \frac{2\mu w_1}{1 - 2\mu - w_1} \tag{13}$$

For $k > 1$ the equation is:

$$w_k = (1-2\mu)^k \frac{\alpha w_1^k + (1-\alpha)w_k + E}{1+E}$$

This equation can be solved for $w_k$:

$$\begin{aligned}
w_k &= (1-2\mu)^k \frac{\alpha w_1^k + E}{1 + E - (1-\alpha)(1-2\mu)^k} \\
&= (1-2\mu)^k \frac{\alpha w_1^k + \frac{2\mu w_1}{1-2\mu-w_1}}{1 + \frac{2\mu w_1}{1-2\mu-w_1} - (1-\alpha)(1-2\mu)^k} \\
&= (1-2\mu)^{k-1} \frac{\alpha(1-2\mu)w_1^k - \alpha w_1^{k+1} + 2\mu w_1}{1 - w_1 - (1-\alpha)(1-2\mu)^{k-1}(1-2\mu-w_1)}
\end{aligned}$$

Let

$$S = \sum_{k=0}^{\ell} w_k = 1 + \ell w_1 + \sum_{k=2}^{\ell} (1-2\mu)^{k-1} \frac{\alpha(1-2\mu)w_1^k - \alpha w_1^{k+1} + 2\mu w_1}{1 - w_1 - (1-\alpha)(1-2\mu)^{k-1}(1-2\mu-w_1)} \quad (14)$$

We now use equations (12), (13), and (14) to obtain a single fixed point equation in $w_1$.

$$2^{\ell+1}\mu w_1 = a\left(\alpha(1+w_1)^{\ell} + (1-\alpha)S\right)(1-2\mu-w_1) \quad (15)$$

This equation is sufficiently complex that it is hard to use it theoretically. However, it can be solved numerically using a mathematics software package such as Maple or Mathematica. (The package can symbolically simplify the summation in equation (14), and then solve the equation obtained by setting the numerator to zero. This is a polynomial equation of degree $2\ell$.)

As an application, we consider results obtained by Suzuki and Iwasa (1999). They use an approximate finite population model of a GA to analyze the time to "needle domination" on the NEEDLE fitness landscape. (Here "needle domination" means that the frequency of the needle string exceeds $1/2$.) Their model assumes linkage equilibrium. They consider two modes of crossover, FPAP (few-points major participants) and MPIP (many-points minor-participants). Their MPIP mode is similar to uniform or gene pool crossover, and their crossover rate is defined in the same way as our crossover rate. They found using both an approximate model and experimental techniques that the time to needle domination diverged as the crossover rate approached a critical value between 0 and 1. For example, in their Figure 2b they show model and experimental results for string length 20, mutation rate 0.002, needle height parameter $a = 1$, and population size approximately 4000. Their critical crossover rate from their model is about 0.48, and from experiment is about 0.5. By solving equation (15) with different rates $\alpha$, we can determine that for gene pool recombination, the critical crossover rate for bistability is between $\alpha = 0.4825$ and $\alpha = 0.4826$. Below this value, only the fixed point near the needle is stable, and above this value there is also a stable fixed point near the center of the simplex. This fixed point prevents the GA from converging in a reasonable time period.

# 8 Empirical Results

The paper Wright, Rowe, and Neil (2002) gives empirical results demonstrating bistability for two-parent crossover using both the Vose infinite population model and finite population GAs.

Figure 5 compares the gene pool GA with uniform and one-point crossover GAs on a NEEDLE fitness function with $a = 1$, $\ell = 14$, mutation rate $0.05$, crossover rate 1, starting with a homogeneous population of all-zeros strings. The results are the average of 10 runs with a population of 1,000,000 on each run. This mutation rate is above the upper critical mutation rate for these crossover types. Also shown is a GA with the same mutation rate and no crossover. We can show analytically using equation (10) that the limiting average fitness in this case is $1.021$. The effect of crossover is to decrease the fitness more quickly and to converge to a lower-fitness fixed point.

We can also show that bistability occurs on a single-peak fitness landscape where there is a more gradual increase in fitness as one approaches the peak. We consider a fitness function where the all-zeros string is the global optimum, and where the fitness of strings decreases monotonically as the Hamming distance from the optimum increases. In other words, the fitness decreases as the number of ones in the string increases. In particular, let the fitness function be defined by

$$f(k) = 1 + \frac{\ell}{5(\#k+1)}$$

This fitness function is graphed in Figure 6.

Formula (2) can be used to obtain a 1-variable fixed point equation for the gene pool GA for fitness functions of unitation (which depend only on the number of ones in the string). When this is solved for the above fitness function, $\mu = \frac{2}{3\ell}$, and $\ell = 50$, there are three real solutions: $w = \{.41341, .69616, .88452\}$. The first stable fixed point corresponds to an average fitness of $1.6685$, and the second stable fixed point (of $.88455$) corresponds to an average fitness of $4.2322$. These agree with the results of simulations. Uniform crossover with crossover rate 1 is also bistable, and the corresponding average fitnesses are: $1.804$ and $4.980$ (as determined by an average 10 runs of population size 100,000).

# 9 Conclusion

This paper has developed a dynamical system model of a GA that uses gene pool recombination, proportional selection, and mutation. The bistability phenomenon is shown using this model for the NEEDLE fitness function. Fixed points can be explicitly computed, and stability/instability shown for large string lengths. We also demonstrate bistability on a single-peak landscape which gives selective pressure toward the optimum at all points of the search space. This shows both that our techniques can be extended to other fitness functions, and that there is good qualitative agreement between the NEEDLE model and fitness functions that approximate the NEEDLE fitness.

Simulations of the Vose model and finite population simulations Wright, Rowe, and Neil (2002) suggest that the simple GA using two-parent crossover on the NEEDLE fitness landscape has similar behavior to the gene pool GA. Both types of GA have bistability on the NEEDLE fitness function, show similar population characteristics at fixed points, and similar behavior over time. Thus, the gene pool GA model can be considered to be a more tractable approximate model for the conventional GA for these fitness functions.

There is a lesson for the practitioner. When the GA is initialized with a random population, bistability is a phenomenon that should be avoided since it may prevent the GA from finding peaks in the fitness. One way to do this is to keep the strength of recombination down. This can be done by reducing the crossover rate, or by choosing a “weaker” crossover (such as one-point or two-point instead of uniform).

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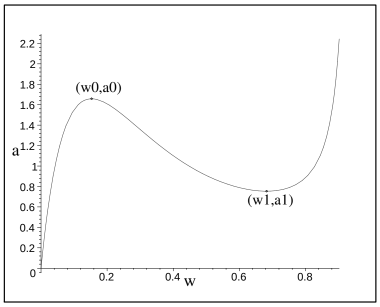


**Figure 1** Values of parameter $a$ that will give a fixed point as a function of $w$. $\ell = 9$, $\mu = 0.03$.

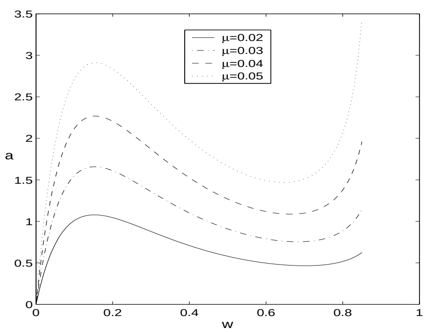


**Figure 2** Values of parameter $a$ that will give a fixed point as a function of $w$ for $\mu = 0.02, 0.03, 0.04, 0.05$ and $\ell = 9$.

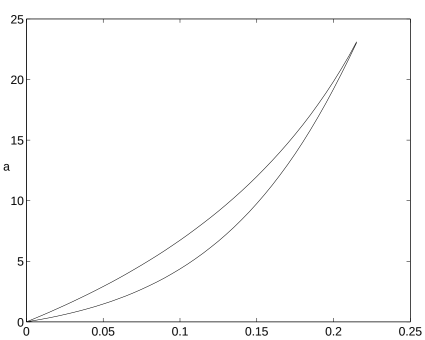


**Figure 3** Region of bistability in $(\mu, a)$ space for $\ell = 9$.

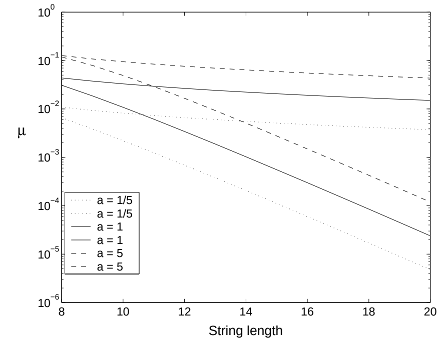


**Figure 4** Upper and lower critical mutation rates for different $a$ values.

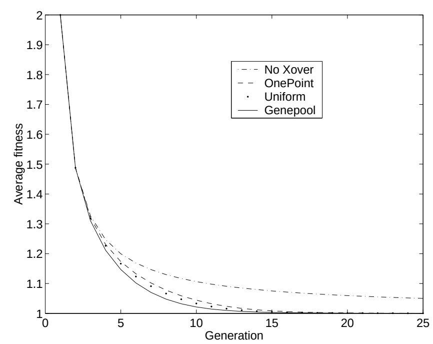


**Figure 5** NEEDLE fitness, $\mu = 0.05$, $\ell = 14$, $a = 1$.

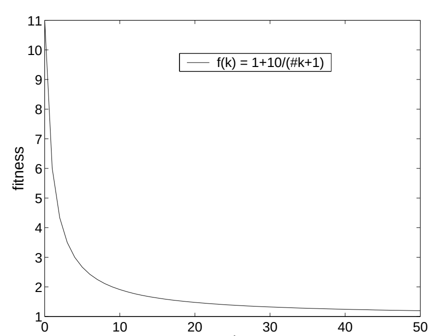


**Figure 6** Fitness that exhibits bistability, $\mu = 1/75$, $\ell = 50$.