On Elements of Evolution and Genetics in the Application of Genetic Algorithm to Optimization Mathematics

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Authors’ contributions

This work was carried out in collaboration among all authors. Author ECE designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors AAD and ECL managed the analyses of the study. Author ECL managed the literature searches. All authors read and approved the final manuscript.

ABSTRACT

This work is a major review of the existing on evolution and genetics. It was started by discussing the Charles Darwin theory of evolution i.e. by exploring patterns of bones in vertebrates showing typical pentadactyl limbs, vestigial structures, sorology, parasitology etc. with special attention in man and his races. Following was the existence theory of genetics in the development of man. The introduction of chromosomes was used to strengthen this resulting in the development of character. The occasional occurrences of mutation in the chromosomes due to some factors were also discussed together with the idea of sex linkage. Later, at the end, the mathematics of genetic algorithm was applied in the work to see how selection chromosomes could influence artificial intelligence and neural network training mostly seen in the area of optimization mathematics.

Keywords: Chromosomes; environment; evolution science; genetic behavior; mathematical genetic algorithm; mutation; variation.

1. EVOLUTION

Two hypothesis [Barton and Keightley [1]] have been advanced to explain (a) the considerable variation shown by individuals of species and by different off springs of the same parent (b) the similarities of anatomy and physiology of organisms with different habits such as between man and the gyraphs and the bath. The first and oldest of these two hypotheses is creations which believe that all living organism were created and that the creator determine the structural physiology of the organism.

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The second [Barton and Turelli [2]] theory believes that structural similarity is a result of genetic relationship while diversity is induced by time.

Evolution is testable scientifically but creation cannot be scientifically tested because of this evolution is more popular scientifically.

Diversity is due to a gradual change called Evolution.

Evolution [Bulmer [3]] is a term to describe the progressive changes in successive generations of living organism. It describes the production of new species of organisms from existing ones by series of gradual changes over a known period. Evolution helps to explain two things

1. The similarities between two related organisms as being due to descent of the common ancestors.
2. The differences between organisms are being due to variation inherited from an older generation the theory evolution postulate that life starts on earth in simple forms over several thousands of millions of year ago.

Theory depends on the facts: -

1. That genotype of organism may change in time because selection.
2. That the changes results in diversification or speciation.

1.1 Lamackism

A French biologist [Fisher [4]] considered changes due to activities of organisms themselves, and therefore proposed the theory of use and is used to explain the mechanism of Evolution. His theory states that when an organisms has the need for anatomical structure. Such structures is acquired by induction. His hypothesis was based on observation that use of a structure lead to the structure’s development and disuse leads to extinction. No evidence has been found to support the evidence of characteristics acquired during the life of an individual.

1.2 Charles Darwin’s Theory of Natural Selection

Charles Darwin sailed in [Gimelfarb [5]] and was surprised at the nature of Flora and Famine on the Galapagos Island in the Pacific Ocean. His observation made him to include that animals and plants are brought about by process of show and gradual change over successive generations which is caused by natural selection. Natural selection [Kimura [6]] postulates that poorly adapted individuals do not survive but adapted ones survives and pass down the survival trait to the next generation. It is also called the survival of the fittest. So Charles Darwins used the following argument to support his theory of natural selection:

a. There is variation among individual within one specie
b. The variation among individual within the species can be inherited
c. In all species of organism individual die before reaching maturity
d. Individual carrying certain genotypes are more likely to die during early mortality than others i.e. only the fittest will survive in the struggle for survival.
e. Those that survive are very fit and so will reach reproductive stage. The unfit ones [Lande and Arnold [7,8]] will be eliminated.

Fig. 1.1. Evolution tree

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Fig. 1.1. Evolution tree
Lastly, Darwin’s argues that those that survive will transmit the beneficial character to the offspring i.e. the selective advantage is heritable.

\[
\text{Survival struggle} \rightarrow \text{selection (fittest) of the best} \rightarrow \text{Enhances}
\]

Therefore, variation in a species is determined by the degree to which the individuals within that specie are adapted to their environment i.e. how fit they are in the strength for survival.

### 1.3 Some Selective Forces

1. Changing environment
2. Predator pressure: (a) Selection for protection (b) Mummery and this can be in form of:
   - (i) Form
   - (ii) colour
   - (iii) sound
   - (iv) pheromone etc.
3. Requirement for food: there is great or efficiency in predation.
4. Competition from other species.
5. Temporary absence of food resources: there will be selection for faster reproduction and more resistance in life history. Separation is canted first by selection thus by separation of two population in independence units Lessard [9].

### 1.4 Examples of Barriers

1. Enlargement of river or mountain. This will prevent mating between separated population in future due to development (pheromones and clapper).
2. Genetic barriers especially to plant.
3. Hybridization between existing species may result to a new specie eg Tetra phoid wheat pheromone No28 – used for making macaroni is a hybrid between a wild grass and a diploid wheat chromosome No 19. The above are due to Lin and Hajela [10].

### 1.5 Evidence of Evolution

If species have arised from a gradual change then, all organisms have a common origin and similarities and differences between them will enhance an understanding of their ancestry.

The study of fossil records is called paleontology. These are remains preserved in rocks e.g. bones. Fossils provide the greatest evidence that evolution has occurred. The following features of fossils support evolution [Lyubich [11]].

1. They indicate that some organism which are now extinct once existed are no longer there e.g. the Dinosaurs.
2. They show difference between a step by step evolution trend.
3. The older the fossil deposit the greater the difference between the fossilized species and existing once.
4. Fossils make the construction trends possible.

### 1.6 Comparative

Patterns of Bones in different vertebrates show a homologous relationship variation is Lyubich [12] deep to specialization.

**Typical Pentadactyl Limbs:**
Early developmental changes are very conservative. Studies of vertebrate embryos show striking similarities: 

(a) Gill slits in fish have equivalence in all vertebrate. This indicates that the ancestral vertebrate must have gill slits. 
(b) The possession of a tail even in human embryos indicate a common origin with chicken and fish.

1.7 Vestigial Structure

These Michalwicz [13] are structures which have no known functions: 

(a) Vermtomappendix in man is vestigial but in rat it play a role in cellulose digestion. 
(b) The feathers of ostriches are vestigial. 
(c) The hind limb bone of the snake is vestigial.

1.8 Geographical Distribution

This two aspects of biology Nagylaki [14] show evidence of evolution by natural selection when comparison are made between organism on the man island and these on an island e.g the finches on the galapago Island. Difference in the
finches’ evolved in isolation in such a way as to be adapted to a particular environment.

(a) Those finches due to land have beaks adapted for cracking nuts.
(b) Those that live on water flower have long stretch beak.
(c) Those that feed on insects have slender beaks.

Geographical distribution deals with dispersal from point of origin.

Geographical barriers, isolation and adaption environment show evidence of evolution by natural selection.

1.9 Biochemical Composition

The structure of a par creature enzyme, insulin indicates an ancestral link in different animal.

1.10 Scrology

This is the study of serouon. The chemicals and positions of bloods of different animals Nagylaki [15] show different degree of similarity and also show phylogenetic relationship. The following Nagylaki and Crow [16] show the degree of incompatibility between human serum and blood of other vertebrates.

<table>
<thead>
<tr>
<th>Animal</th>
<th>Similarity</th>
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<tbody>
<tr>
<td>Gorilla</td>
<td>39%</td>
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<tr>
<td>Baboreen</td>
<td>70%</td>
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<tr>
<td>Horse</td>
<td>98%</td>
</tr>
<tr>
<td>Rat</td>
<td>100%</td>
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1.11 Parasitology

Most parasites Nagylaki and Lou [17] retain evidence of specialization from an ancestral free living form from which they have evolved by becoming adapted to live on, in and at the expense of the host. The choice of host by parasites show some evolutionary relationship e.g. predators specie which is only found on man chimpanzee.

1.12 Behavior

All primates Okereke and Akogoun [18] exhibit hierarchy social order which enhances acculturation. An emotion or social behavior is similar. This could be affectionate. They show joy and they show aggression. Aggression well directed in other animals but man is the animals that will misuse a member of his own specie.

1.13 Mechanism of Evolution

1.13.1 Speciation

Evolution Otto [19] refers to gradual changes brought about by natural selection. Evolution may occur without speciation since specie are group of organism which interbreed, speciation then is a result of acquisition of enough genetic change in the course of adaption which makes interbreeding with ancestral species unusually difficult and eventually in profitable isolation enhanced speciation.

1.13.2 Evolution of man

Man’s morphology and physiology function Reeve [20] show similarity to other animals Apes and Man are similar in anatomical and physiological details, immunological responses, blood grouping, parasitic, investigation, susceptibility to obtain diseases and behavior (emotional and social).

1.13.3 Taxonomy

Class: Mammalia – rats, monkeys, Apes, apameny and man
Order: primates- monkeys
Sub-Order- Anthropoids- monkeys, Apes, Apenemny
Sub- Family – Homindea – Apes, Apemena, Man
Family – Hominea – Apes, apemena, Man
Sub- Family – Hominea – Apemen, Man
Genus- Homo – Modern and early
Species- Homo species – modern, man

The above is due to Schneider [21].

1.13.4 Mechanism of evolution

1. Natural selection: this on the change in genetic composition of a population through time. It Schneider [22] is the statistical selection of the most fit gene combination for a particular environment. Fitness refers to population’s ability to cope successfully with a particular environment at a particular time. Unfit gene combinations and elimination advantaged ones are preserved because of selection determined by predation diseases migration, conflict, behavior, competition, breeding species, mate, maid, nest and food, climate, natural disinterpolation of coil air and water. In man medicine and
surgery has showed down the rate of natural selection.

2. **Sexual selection:** some males ones more fit than other others in getting mates because of striking coloration structures (form or behavior) these contribute more or the gene pool them the less sexually selected ones. In this sense, selection then depends on the ability of the male to raise the physiological state of the female to a cereal at which she accepts to mate.

3. **Mutation:** Genetic information Schneider [23] is contained in two DNA strands, one of which way made the other (Dominant) change in the gene may be fit but variation in usually due to change in frequency of existence gene under diff environmental and social stimuli.

4. **Homeotic selection:** Homeostasis Shashahani [24] is the states of equilibrium of organism biochemistry which can be adapted to respond to changes through acceleration. The most fit homeostasis population becomes selected. Man occupies a wide range of habitat hence he is able to homeostatically adapt.

5. **Blood mechanism:** Advantageous blood groups Singiresu [25] are selected in the changing environment in a homozygous state; sickle anaemia is lethal, in a heterozygous state.

The individual Svirezhev and Yu [26] is insusceptible to malaria in tropical African where Malaria is endemic, heterozygous individuals are fit one. The lacks of sickle cell my result in being killed by Malaria.

Sickle cell is absent in arrabaria area. Certain blood groups are more susceptible to certain diseases for example group O is more susceptible to Syphilis. Group A is less susceptible to Plague; group B is less susceptible to streptococcal.

**Genetic Drift:** This is random to make of aeration gene frequency under special condition of isolation. This may explain the absence of blood group B among American-Indian.

These points Turelli and Barton [27] do not exempt man the effect of the environment pressure or Evolution. The following evidence shows that man like other animals still respond to stimuli.

i. Immunity to diseases, their pathogenesis Eg. Moades are highly pathogenic in Polynesian people. Epidermics may act as selective force especially if causes are yet unrecognized diseases agent.

ii. ABO blood grouping and sickle cell anemia is another point to show that man also responds to environmental selection. This means that some blood groups is common among certain tribe which demonstrate ancestral link.

1.13.5 Will man continue to evolve

Since evolution is enhanced by consistent response to environment overtime, man may be said to be evolving. The following, make man’s continued evolution of man difficult to appreciate. Those points Turelli and Barton [28] are:

1. Man’s adaptation is broad and generalized. This enables him to inhabit various types of ecological niche.

2. There is flexibility in human gene pool and they are very limited if any isolating barriers.

3. Intervention of culture: man and environment has resulted in skills such as tools, clothing, etc which have greatly reduced the effect of environment on man.

4. Evolution is easily in rapidly reproducing population but in man because of size of the gene pool and the length of a generation, the flow of favourable genotype takes a very long time to know it.

1.13.6 Races of man

The races of a man correspond to geographical barrier which functions to reduce free gene flow.

To change environment sufficiently, enough to bring about adaptive response.
2. GENETICS BEFORE 1900

1. **Spontaneous Generation**: Biologists once believed that an organism could give rise to young ones spontaneously; e.g., maggots are spontaneously generated from faeces. Pasterns, however, showed that new organisms arise through the continuity of life. Aristotle had earlier taught the transfer of formulated theory of genetics.

2. **Preformationism**: Wright [30] is the second theory of genetics and this believes that either male or female sex cells have a miniature forms and that only nutritional factor will develop to correct proportions.

3. **Epigenesis**: Coolf gave the concept of epigenesis in which he proposed that many factors such as organism and tissues appear during development of an organism even though not originally present in the perordial form. This theory believes that the transfer of information of one generation to the other was mystical.

4. **Pangenesis**: (blending theory) arose with the advent of the evolutionary theory of Charles Darwin. This school of thought Gimelfarb [5] believes that small particles of each body organ are conveyed by the school stream to the sex organ and are assembled into gametes. This particles are mixed on fertilization and the minute elements are rearranged hence giving rise to an organism which consists of blended material from both parent organism. This is also called the blending theory.

Weisman later disproved pangenesis by cutting the tale of mice for 22 generations. The mice of course still inherited a complete tale thereby showing that other factors rather than blending was from one generation to the other.

2.1 Mendel's Contribution to Genetics

Gregory Mendel an Austrian monk and regarded as the father of genetics further works on genes are corned on chromosomes. He started his experiment in 1857 using the garden pea.

Advantages of his derived from using garden pea

i. The plant was easy to cultivate.

ii. Characters are sharply defined

a. Mendel chose units of characters called traits.

b. E.g. of these characters are tall or short, red or white.

c. He considered each character separately to avoid confusing them.

iv. Mendel analyzed his results in mathematical ratios thereby showing the importance of quantitative expression.

v. Mendel draws a mathematical conclusion from his result.

2.2 Monohybid Inheritance

This Lande and Arnold [8] is the consideration of one pair of contrasting characters examples of this kind of character are tall or short, yellow or green etc. an initial cross between two varieties is called parental generation P.

The first generation is called F₁, or first Fijian. The 2nd generation is called F₂. For all the characters Mendel tested there was a pattern:

a) Only one trait was shown in the F₁ generation.

b) It did not matter which parent provided the ova i.e. each one contributed equally.

c) The hidden trait in F₁ reappeared in F₂ in the ratio of 1:3.

From the result Mendel formulated a theory called particulate theory which states that inheritance is a process whereby definite threats which may or may not show themselves in outward appearance of the organism are transmitted from parent to offspring. This theory points an end to the blending theory Lessard [9].

Two possible conclusions can be drawn from this:

i. Although F₁ are (white) the gene for red must be present

ii. That the gene for red fails to show itself because it was dominated by the gene for whiteness in the F₁.

A biologist studying the inheritance of color in rabbits mated a brown female rabbits (heterozygous) with a white male rabbit (homozygous) (a) what is the F₁ generation i.e. what phenotype and genotype does F₁ possess.
(b) If brown is dominant over red what will be the result of mating one brown rabbit with a heterozygous rabbit. (c) A heterozygous black N/B brown is dominant over white black and red.

2.3 Terminologies

i. Variation: (a) continuous variation: is brought about by Lin and Hajela [10] environmental factors such as food supply, humidity climate, soil, light, attitude but this continuous generation can be genetically controlled. (b) Discontinuous variation: does not produce intermediate result i.e. no gradation e.g. sex in man, blood groups (this type of variation expresses that you are either tall or short and not intermediate. Mendelianian inheritance in discontinuous. The phenotype of an organism can Lyubich [11] be affected by the environment but the genotype cannot be affected by the environment.

ii. Gene: this is an inherited Factor that determines a biological character.

iii. Allele: this a pair of character in a gene e.g. white, red, e.g. T,t, W,w.

iv. Homozygous: it is used to describe a gene pair with two identical alleles e.g. ww, WW.

v. Heterozygous: is used to describe a gene pair with different alleles e.g. Ww, Tt.

vi. Dominant Gene: the gene that marks or hinders the other phenotypic e.g. Ww (the big W indorminant).

vii. Recessive gene: is a hidden gene the gene that is marking phenotypic Ww (w is the recessive gene.

viii. Phenotype: this is the outer and appearance of the gene i.e. the expression you see or show.e.g. tallness in phenotype in both Tt and TT.

ix. Genotype: describes the genetic constriction of the organism e.g. Tt, TT are genotypes.

2.4 Monohybrid Inheritance

This is consideration of or a pair of contrasting character e.g. smooth or wrinkle, tall and short, yellow or not yellow.

![Monohybrid Inheritance Diagram](image)

**Fig. 2.1. Monohybrid inheritance**
**Fig. 2.2. Inheritance**

T (Tall) dominant or t (short)

\[
P:\quad TT \times tt \\
\downarrow \quad \downarrow \\
\text{Segregation} \\
\hline
T \quad T \\
\hline
\text{Fusion of gametes} \\
\hline
F_1 \quad Tt, Tt, Tt, Tt, (Tall) \\
\hline
\text{Selfing} \\
\hline
T \quad T \\
\hline
\hline
\text{TT, Tt, Tt, Tt, tt} \quad 3:1
\]

**Fig. 2.3. Trait inheritance**
The second genetic is called the F$_2$ or 2$^{nd}$ filial.

For all character tested Mendel got the same pattern Lyubich [12] as follow.

i. Only one trait was shown in F$_1$ generation.

ii. It did not matter which parents provide the patterns or the orals, this indicate that each one contributed equally.

iii. The hidden trait in the F$_1$ generation replace in the F$_2$ generation in a ratio of 1:3. From this results Mendel's formulated the particulate theory which states that: inheritance is a process by which definite trait (character) which may or may not show them self in outward appearance of the organism are transmitted from parent to offspring. This theory put to rest the blending theory. Thus two possible conclusions Michalwicz [13] can be drawn from the theory.

a) That although F$_1$ are tall, the gene for dwarfness must be present.

b) That the gene for dwarfness fails to show because it was dominated by the gene for tallness in the F$_1$ generation.

Mendel formulated his first law which is called the law of segregation the law states that:
- A hybrid between two different varieties process both types of parental factor which separates or segregates in the gametes since only one of the pairs can be carried in a single gamete.

2.5 Backcrossing

Selfing in a way of finding an observed phenotype e.g. tall is homozygous or heterozygous. The ration of the F$_1$ generation is used to determine the genotype of the parents:-

a. 3:1 ration: one parent is heterozygous and the other double recessive (Tt and Tt).

b. 1:1 ration: one the other double recessive (Tt and tt).

c. If all are that same 1:4, 4:0 ration. The one parent is homozygous and the other double recessive (TT and tt).

2.6 Dihybrid Inheritance

Suppose the plant is tall and had red flower and is crossed with a short plant with white flower. This Nagylaki [14] deals with two characteristics and is called dihybrid crossing.

The possible number of gametes Nagylaki [15] is determined by the number of chromosomes pair’s n as $2^n$ from this Mendel concluded that the two pair of genes – height and color are independently transmitted from parents and that they assort freely i.e. each of a pair of alleles may randomly combine with either of another pair.

2.7 The Law of Independent Assignment

States that two pairs of genes are independently transmitted from parents and they assort freely it is the same thing as on and its second law.

2.8 Problems on the Law of Segregation

The main problem in Mendel’s second law Nagylaki and Crow [16] is that mutant genes are transmitted the same way as other genes. What is mutation? It is an accidental change in genetic material e.g. achondroplasia (which is due to mutation in reproduction of cells which results in dwarfness and which exert itself in either homo or heterozygous conditions.

The second problem is that genotype does not charge i.e constant and pure, but phenotype may change with time.

2.9 Co Dominance or Incomplete Dominance

Some exceptions to Mendel’s law that a dominant gene R Nagylaki and Lou [16] will hide a recessive gene were found and protagonist of the blending theory rejoiced these exceptions however were shown to be due to co dominance. Co dominance occurs when the two alleles in a pair is fully expressed in heterogeneous condition.

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2.10 Examples of Co Dominance

A. 4’oclock flower. When Red RR is crossed with rr. The F₁ is pink instead of being Red (Rr). This was initially thought to be due to incomplete dominance of the color over the color white as the experiment above.

B. Fizzle feather

N = normal feather dom n fizzle
Fig. 2.6. Fizzle trait

F₁ NnNnNnNnNn (mild fizzle)

Fig. 2.7. Dominance trait

C. ABO blood Group

F₁ I^A I^B I^A I^B (characteristics of both A&B)

Fig. 2.8. Recessive trait

Fig. 2.9. Mutation
Fizzle feather NN are normal homozygous feather when crossed results in mild feather which is also a results of co dominance.

2.11 Mutations

These are Otto [19] alterations (aberrations) in the structure of chromosomes. Chromosomes become interlocked during meiosis and this interlocking may create a charge due to structural alteration (mutation). This mutation will alter the character under its control.

2.12 Types of Mutation

(1) Deletion:- here the middle piece of chromosome break off and this change the nature of genetic information and results in an abnormality.

(2) Inversion:- this results after breaking off the piece rejoin at a different location giving an inverse information.

(3) Translocation: This occur when a section of Xsomes breaks off and translocate on another Xsomes.

Fig. 2.10. Deletion

Fig. 2.11. Inversion

Fig. 2.12. Translocation
(4) Duplication: This is a reputation of a set of genes.

\[
\begin{array}{c}
\text{Duplication} \\
\end{array}
\]

\[
\begin{array}{c}
\text{Disjunction} \\
\end{array}
\]

Homologous xsome are supposed to segregate into separate cells at meiosis so that half of xsome is in each cell. If this fails both xsome enter one cell and this is known as non disjunction one of the two guaranties has two xsome but the other has non. Non disjunction cells have serious offences on the organism development. Egmonoglis/ down syndrome (the idiot): This is caused by the extra xsome (xsome = 21).

(3) Klinelfelter’s Syndrome xxy

This is due to non-disjunction “X” Xsome during _____ genesis. The individual has male feature phenotypically. They cannot produce spermatozoa and they do infact have some woman characteristic.

(3) Turners Syndrome

This is the reverse klinelfelter’s syndrome, the cell will be missing ‘X’ Xsome as sterile.

(4) xyy – syndrome: extra masculin syndrome

They are giants and have exaggerate male features.

Mutant gene Price [32] are passed onto the next generation but may be lost to the species since organism carrying them often die.
Chromosomes and heredity: Genes are heritable factors which are located on chromosome. Therefore, their transmission is effected to the next offspring. Different genes are located on specific size along the length of the chromosome.

Sex determination: In some plants e.g paw paw. The chromosomes of both female and male are identical and sex is determined by invisible differences in genes.

Differences in xsome structure accounts for sex differences in other organisms. Reeve [20].

In drosophylia, the males has two pairs of chromosome of different shape, while the female has pairs of chromosomes of similar shape.

In man there are 23 pairs of xchromosomes in female members of each pair are alike in shape. In male one chromosome is bigger than the other but of the malexsome is similar in shape those of female. The two chromosomes of the female are designated as x while the distinctly bigger one is called ychromosome for convenience. This mean that the femalegenet poses a pair of homologous (xx) chromosomes while the male appearances of the y dominated the x and results in a male. (♀ 23 of xchromosomes) all 23 pairs are exactly alike. O 23 pair of x and one pair is exactly like those of formally. The other is larger and bigger than the female.

2.13 Sex Linkage

Genes which Reeve [20] are carried on the sex xsome are transmitted along the determining sex and are sad to be sex linked. Any recessive gene located on the X xsome of the male is phenotypically expressed (X X). Since male has only one ‘x’ xsome. In female this may not be so since heterozygous characteristic are possible X‘X e.g sickle cell anemia. Certain characteristics are due to genes on the sex xsome e.g hemophilia, colour blindness. There are common in male and rare in female.
3. THE GENETIC ALGORITHM IN OPTIMIZATION

In this section, we discuss genetic algorithms and their application to solving optimization problems. Genetic algorithms do not use gradients or hessians. Consequently, they are applicable to a much wider class of optimization problems.

A genetic algorithm is a probabilistic search technique that has its roots in the principles of genetics. The beginning of genetic algorithms is credited to John Holland, who developed the basic ideas in the late 1960s and early 1970s. Since its conception, genetic algorithms have been used widely as a tool in computer programming and artificial intelligence optimization, neural network training, and many other areas. Suppose that we wish to solve an optimization problem of the form

\[
\begin{align*}
\text{Maximize} & \quad f(x) \\
\text{Subject to} & \quad x \in \Omega.
\end{align*}
\]

The underlying idea of initial set of points in \( \Omega \), denoted \( P(0) \). We call \( P(0) \) the initial population. We then evaluate the objective function at points in \( P(0) \). Based on this evaluation, we create a new set of points \( P(1) \). The creation of \( P(1) \) involves certain operations on points in \( P(0) \), called crossover and mutation. We repeat the above procedure iteratively, generating populations \( P(2) \), \( P(3) \), \ldots, until an appropriate stopping criterion is reached. The purpose of the crossover and mutation operations is to create a new population with an average objective function value that is higher than the previous population. To summarize, the genetic algorithm iteratively performs the operations of crossover and mutation on each population to produce a new population until a chosen termination criterion is met.
The terminology used in describing genetic algorithms is Schneider [21] adopted from genetics. To proceed with describing the algorithm, we need the additional ideas and terms described below.

### 3.1 Chromosomes and Representation Schemes

First, we Schneider [22] point out that, in fact, genetic algorithms do not work directly with points in the set \( \Omega \). Specifically, we need first to map \( \Omega \) onto a set consisting of strings of symbols, all of equal length. These strings are called chromosomes. Each chromosome consists of elements from a chosen set of symbols, called the alphabet. For example, a common alphabet is the set \{0,1\}, of chromosomes (i.e., the number of symbols in the strings). To each chromosome there corresponds a value of the objective function, referred to as the fitness of the chromosome. For each chromosome \( x \), we write \( f(x) \) for its fitness. Note that, for convenience, we Schneider [23] use \( f \) to denote both the original objective function as well as the fitness measure on the set of chromosomes.

The choice of chromosome length, alphabet, and encoding (i.e., the mapping from \( \Omega \) onto the set of chromosomes), is called the representation scheme for the problem. Identification of an appropriate representation scheme is the first step in using genetic algorithms to solve a given optimization problem.

Once a suitable representation scheme has been chosen, the next phase is to initialize the first population \( P(0) \) of chromosomes. After we form the initial population of chromosomes, we then apply the operations of crossover and mutation on the population. During each iteration \( k \) of the process, we evaluate the fitness \( f(x^{(k)}) \) of each member \( x^{(k)} \) of the population \( P(k+1) \) in two stages.

### 3.2 Selection and Evolution

In first stage, we Shashahani [24] apply an operation called selection, where we form a set \( M(k) \) with the same number of elements as \( P(k) \). This number is called the population size, which we denote by \( N \), and \( M(k) \), called the mating pool, is formed from \( P(k) \) using a random procedure as follows: each point \( m^{(k)} \) in \( M(k) \) is equal to \( x^{(k)} \) in \( P(k) \) with probability \( p_c \).

\[
f(x^{(k)}) / F(k)
\]

Where, \( f(k) = \sum f(x^{(k)}) \)

And the sum is taken over the whole of \( P(k) \). In other words, we select chromosomes into the mating pool with probabilities proportional to their fitness.

The above selection scheme is also called the roulette-wheel scheme, for the following reason. Imagine a roulette wheel in which each slot is assigned to a chromosome in \( P(k) \); some chromosomes may be assigned multiple slots. The number of slots associated with each chromosome is in proportion to its fitness. We then spin the roulette wheel and select (for inclusion in \( M(k) \)) the chromosome on whose slot the ball comes to rest. This procedure is repeated \( N \) times, so that the mating pool \( M(k) \) contains \( N \) chromosomes.

An alternative selection scheme is the tournament scheme, which proceeds as follows. First, we select a pair of chromosomes at random from \( P(k) \). Then we compare the fitness values of these two chromosomes, and place the fitter of the two into \( M(k) \). We repeat this operation until the mating pool \( M(k) \) contains chromosomes. The second stage is called evolution: in this stage, we Singiresu [25] apply the crossover and mutation operations. The crossover operation takes a pair of chromosomes, called the parents and gives a pair of offspring chromosomes, described below.

Pairs of parents for crossover are chosen from the mating pool randomly, such that the probability that a chromosome is chosen or not is independent of whether or not any other chromosome is chosen for crossover.

We can pick parents for crossover in several ways. For example, we may randomly choose two chromosomes from the mating pool as parents. In this case, if \( N \) is the number of chromosomes in the mating pool, then \( p_c = 2/N \). Similarly, if we randomly pick \( 2k \) chromosomes from the mating pool (where \( k < N/2 \)), forming \( k \) pairs of parents, we have \( p_c = 2k/N \). In the above two examples, the number of pairs of parents is fixed and the value of \( p_c \) is dependent on this number. Yet another way of choosing parents is as follows: given a value of \( p_c \), we pick a random number of pairs of parents such that the average number of pairs is \( p_c N/2 \).
Once the parents for crossover have been determined, we Svirezhev and Yu [26] apply the crossover operation to the parents. There are many types of possible crossover operations. The simplest crossover operation is the one-point crossover. In this operation, we first chose a number randomly between 1 and L – 1 according to a uniform distribution where L is the length of chromosomes. We refer to this number as the crossing site. Crossover then involves exchanging substrings of the parents to the left of the crossover site, as illustrated in Fig. 2.16 and in the following example.

Example 3.1 Suppose that we have chromosomes of length L = 6 over the binary alphabet {0, 1}, consider the pair of parents 000000 and 111111. Suppose that the crossing site is 4. Then, the crossover operation applied to the above parent chromosomes yields the two offspring 000011 and 111100.

We can also have crossover operations with multiple crossing sites, as illustrated in Fig. 3.1 and in the following example.

Example 3.2 consider two chromosomes, 000000000 and 1111111111, of length L = 9. Suppose that we have two crossing sites, at 3 and 7. Then, the crossover operation applied to the above parent chromosomes yields the two offspring 000111100 and 111000011.

After the crossover operation, we replace the parent in the mating pool by their offspring. The mating pool has therefore been modified, but still maintains the same number of elements.

Next, we apply the mutation operation. The mutation operation takes each chromosome from the mating pool and randomly changes each symbol of the chromosome with a given probability $p_m$. In the case of the binary alphabet, this change corresponds to complementing the corresponding bits; that is, we replace each bit with probability $p_m$ from 0 to 1, or vice versa. If the alphabet contains more than two symbols, then the change involves randomly substituting the symbol with another symbol from the alphabet. Typically, the value of $p_m$ is very small (e.g., 0.01), so that only a few chromosomes will undergo a change due to mutation, and of those that are affected, only a few of the symbols are modified. Therefore, the mutation operation plays only a minor role in the genetic algorithm relative to the crossover operation.

![Fig. 3.1a. Illustration of basic crossover operation](image)

![Fig. 3.1b. Illustration of two – point crossover operation](image)
After applying the crossover and mutation operations to the mating pool $M(k)$, we obtain the population $P(k+1)$. We then repeat the procedure of evaluation, selection, and evolution, iteratively. We summarize the genetic algorithm as follows.

### 3.3 Genetic Algorithm

1. Set $k := 0$; form initial population $P(0)$;
2. Evaluate $P(k)$;
3. If stopping criterion satisfied, then stop;
4. Select $M(k)$ from $P(k)$;
5. Evolve $M(k)$ to from $P(k+1)$;
6. Set $k := k+1$, go to step 2.

The above is due to Turelli and Barton [27].

A flow chart illustrating the algorithm is shown in Fig. 3.2.

During the execution of the genetic algorithm, we keep track of the best-so-far chromosome; that is, the chromosome with the highest fitness of all the chromosomes evaluated. After each iteration,
best-so-far chromosome serves as the candidate for the solution to the original problem. Indeed, we may even copy the best-so-far chromosome into each new population, a practice referred to as elitism. The elitist strategy may result in domination of the population by “super chromosomes.” However, practical experience suggests that elitism often improves the performance of the algorithm.

The stopping criterion can be implemented in a number of ways. For example, a simple stopping criterion is to stop after a prespecified number of iteration. Another possible criterion is to stop when the fitness for the best-so-far chromosome does not change significantly from iteration to iteration.

The genetic algorithm differs from the algorithms discussed in previous chapters in several respects:

1. It works with an encoding of the feasible set rather than the set itself;
2. It searches from a set of points rather than a single point at each iteration;
3. It does not use derivatives of the objective function;
4. It uses operations that are random within each iteration.

Application of the genetic algorithm to an optimization problem is illustrated in the following example.

**Example 3.3** consider the MATLAB “peaks” function $f \mathbb{R}^2 \rightarrow \mathbb{R}$ given by

$$f(x, y) = 3(1 - x)^2 e^{-x^2 - (y+1)^2} - 10 \left(\frac{x}{5} - x^3 - y^5\right) e^{-x^2 - y^2} - \frac{e^{-(x+1)^2 - y^2}}{3}$$

(see Turelli and Barton [28], pp. 178-180) for an example involving the same function). We wish to maximize $f$ over the set $\Omega = \{(x, y) \in \mathbb{R}^2 : -3 \leq x, y \leq 3\}$. A plot of the objective function $f$ over the feasible set $\Omega$ is shown in Fig. 3.3. using the MATLAB function `fminunc` (from the Optimization Toolbox), we found the optimal point to be $[-0.0093, 1.5814]^T$, with objective function value 8.1062.

To apply the genetic algorithm to solve the above optimization problem, we use a simple binary representation scheme with length $L = 32$, where the first 16 bits of each chromosome encode the $x$ component, whereas the remaining 16 bits encode the $y$ component. Recall that $x$ and $y$ take values in the interval $[-3, 3]$. We first map the interval $[-3, 3]$ onto the interval $[0, 2^{16} - 1]$, via asimple translation and scaling. The integers in the interval $[0, 2^{16} - 1]$ are then expressed as binary 16 bit strings. This defines the encoding of each component and $y$. The chromosome is obtained by juxtaposing the two 8 bit strings. For example, the point $(x, y)^T = [-1, 3]^T$ is encoded as (for a simple algorithm for converting from decimal into binary)

Using a population size of 20, we apply 50 iteration of the genetic algorithm on the above problem. We used parameter values of $p_c = 0.75$ and $p_m = 0.0075$.

**Fig. 3.3.** Plot of $f$ for example 3.3
3.4 Analysis of Genetic Algorithms

In this section, we Wright [29] use heuristic arguments to describe why genetic algorithms work. As pointed out before, the genetic algorithm was motivated by ideas from natural genetics. Specifically, the notion of “survival of the fittest” plays a central role. The mechanism used in the genetic algorithms mimic this principle. We start with a population of chromosomes, we form the new generation by combining information encoded in them. In this way, the goal is to ensure that the fittest members of the population survive, and their information content is preserved and combined to produce even better offspring.

To further analyze the genetic algorithm in a more quantitative fashion, we need to define a few terms. For convenience, we only consider chromosomes over the binary alphabet. We introduce the notion of a scheme (plural: schemata) as a set of chromosomes with certain common features. Specifically, a schema is a set of chromosomes that contain 1s and 0s in particular locations. We represent a schema using a string notation over an extended alphabet \{0,1,*\}. For example, the notation 1*01 represents the schema

\[ 1^*01 = \{1001,1101\}, \]

and the notation 0*101* = \{001010,001011, 011010,011011\}.

In the schema notation, the numbers 0 and 1 denote the fix binary values in the chromosomes that belong to the schema. The symbol '*', meaning “don’t care”, matches either 0 or 1 at the positions it occupies. Thus, a schema describes a set of chromosomes that have certain specified similarities. A chromosome belongs to a particular schema if for all positions \(j = 1,\ldots, L\) the symbol found in the \(j^{th}\) position of the understanding that any symbols, then it contains 2^L chromosomes. Moreover, any chromosome of length \(L\) belongs to 2^L schemata.

Fig. 3.4 shows plot of the best, average and worst objective function values in the population for every iteration (generation) of the genetic algorithm in example 3.3. The best-so-far solution obtained at the end of the 50 iteration is [0.0615,1.5827]^T, with objective function value 8.1013. Note that this solution and objective function value are very close to those obtained using MATLAB.

3.4.1 Introduction to Schemata

Using a string notation over an extended alphabet \{0,1,*\}. For example, the notation 1*01 represents the schema

\[ 1^*01 = \{1001,1101\}, \]

and the notation 0*101* = \{001010,001011, 011010,011011\}.

In the schema notation, the numbers 0 and 1 denote the fix binary values in the chromosomes that belong to the schema. The symbol '*', meaning “don’t care”, matches either 0 or 1 at the positions it occupies. Thus, a schema describes a set of chromosomes that have certain specified similarities. A chromosome belongs to a particular schema if for all positions \(j = 1,\ldots, L\) the symbol found in the \(j^{th}\) position of the understanding that any symbols, then it contains 2^L chromosomes. Moreover, any chromosome of length \(L\) belongs to 2^L schemata.
Given a schema that represents good solutions to our optimization problem, we would like the number of matching chromosomes in the population $P(k)$ to grow as $k$ increases. This growth is affected by several factors, which we analyze in the following discussions. We assume throughout that we are using the roulette-wheel selection method.

The first key idea in explaining why the genetic algorithm works is the observation that is schema has chromosomes with better than average fitness, then the expected (mean) number of chromosomes matching this schema in the mating pool $M(k)$ is larger than the number of chromosomes matching this schema in the population $P(k)$. To qualify this assertion, we need some additional notation. Let $H$ be a given schema, and let $e(H,k)$ be the number of chromosomes in $P(k)$ that match $H$; that is $e(H,k)$ is the number of elements in the set $P(k) \cap H$. This means that if $H \cap H$, then $f(H,k)$ be average fitness of chromosomes in $P(k)$ that match schema $H$, this means that $f(H,k) = \{x_1, \ldots, x_{e(H,k)}\}$, then

\[ f(H,k) = f(x_1) + \ldots + f(x_{e(H,k)}) = e(H,k) \]

Let $N$ be the number of chromosomes in the population and $F(k)$ be the sum of the fitness values of chromosomes in $P(k)$, as the before. Denote by $F(k)$ the average fitness of chromosomes in the population; that is,

\[ F(k) = \frac{F(k)}{N} = \frac{\sum f(x_i)}{N} \]

Finally, let $m(H,k)$ be the number of chromosomes in $M(k)$ that match $H$, in other words, the number of elements in the set $M(k) \cap H$.

**LEMMA 3.1 [Wright [30]]:** Let $H$ be a given schema, and $M(H,k)$ the expected value of $m(H,k)$ given $P(k)$, then,

\[ M(H,k) = \frac{f(H,k)e(H,k)}{F(k)} \]

**Proof:** Let $P(k) \cap H = \{x_1, \ldots, x_{e(H,k)}\}$. In the remainder of the proof, the term "expected" should be taken to mean "expected, given $P(k)$." for each element $m^{(k)} \in M(k)$ and each $i = 1, \ldots, e(H,k)$, the probability that $m^{(k)} = x_i$ is given by $f(x_i)$ \mid F(k). Thus, the expected number of chromosomes in $M(k)$ equal to $x_i$ is

\[ \sum_{i=1}^{e(H,k)} f(x_i) \]

Hence, the expected number of chromosomes in $P(k) \cap H$ that are selected $M(k)$ is

\[ M(H,k) = \frac{f(H,k)e(H,k)}{F(k)} \]

Because any chromosomes in $M(k)$ is also a chromosomes in $P(k)$, the chromosomes in $M(k) \cap H$ are simply those in $P(k) \cap H$ that are selected into $M(k)$. Hence

\[ M(H,k) = \frac{f(H,k)e(H,k)}{F(k)} \]

The above lemma quantifies our assertion that if a schema $H$ has chromosomes with better than average fitness (i.e., $f(H,k) > 1$), then the expected number of chromosomes matching $H$ in the mating pool $M(k)$ is larger than the number of chromosomes matching $H$ in the population $P(k)$.

We now analyze the effects of the evolution operations on the chromosomes in the mating pool. For this, we need to introduce two parameters that are useful in the characterization of a schema, namely, is order and length. The order $o(S)$ of a schema $S$ is the number of fixed symbols (non-* symbols) in its representation (the notation $o(S)$ is standard in the literature on genetic algorithms, and should not be confused with the “little-oh” symbol defined in Section 3.3). If the length of chromosomes in $S$ is then $o(S)$ is $L$ minus the number of * symbols in $S$. For example,

\[ o(1*01) = 4 \]

Whereas $o(0*1*01) = 6$. The length of $l(S)$ of a schema $S$ is the distance between the first and last fixed symbols (i.e., the difference between the positions of the rightmost fixed symbol and the leftmost fixed symbol). For example,

\[ l(1*01) = 4 \]

whereas $l(0*101*) = 5$.

Note that for a schema $S$ with chromosomes of length $L$, the orders $o(S)$ is a number between 0 and $L$, and the length $l(S)$ is a number between $L - 1$. The order of a schema with all * symbols
is 0; its length is also 0. The order of a schema containing only a single element (i.e., its representation has no * symbols) is L e.g., o (1011) = 4 · 0 = 4. The length of a schema with fixed symbols in its first and last positions is L – e.g., / (0**1) = 4 – 1 = 3.

We first consider the effect of the crossover operation on the mating pool. The basic observation in the following lemma is that given a chromosome in M (k) ∩ H, the probability that it leaves H after crossover is bounded above by a quantity that is proportional to p_c and l (H).

**LEMMA 3.2 [Zhang, Wang and Hill [31]]:** Given a chromosome in M (k) ∩ H, the probability that is it chosen for crossover and neither of its offspring is in H is bounded above by

\[ p_c \frac{H}{L−1}. \]

**Proof:** Consider a given chromosome in M(k) ∩ H. the probability that it is chosen for crossover is p_c. If neither of its offspring is in H, then the crossover point must be between the corresponding first and last fixed symbols of H. the probability of this is l (H) / (L -1). Hence, the probability that the given chromosome is chosen for crossover and neither of its offspring is in H is bounded above by

\[ p_c \frac{H}{L−1}. \]

From the above lemma, we conclude that given a chromosome in M (k) ∩ H, the probability that either it is not selected for crossover, or at least one of its offspring is in H after the crossover operation, is bounded below by

\[ L − p_c \frac{H}{L−1}. \]

Note that if chromosome in H is chosen for crossover, and the other parent chromosome is also in H, then both offspring are automatically in H.

Hence, for each chromosome in M (k) ∩ H, there is a certain probability that it will result in an associated chromosome in H (either itself or one of its offspring) after going through crossover (including selection for crossover), and that probability is bounded below by the above expression.

We next consider the effect if the mutation operation on the mating pool M (k).

**LEMMA 3.3 [Zhang, Wang and Hill [31]]:** Given a chromosome in M (k) ∩ H, the probability that it remains in H after the mutation operation is given by

\[ (1-p_m)^{\ell (H)} \]

above is approximately equal to

\[ (1-p_m)^{\ell (H)}. \]

**Proof:** Given a chromosome in M (k) ∩ H, it remains in H after the mutation operation of and only if none of the symbols in this chromosome that correspond to fixed symbol in H is changes by the mutation operation. The probability of this event is (1 – p_m)^{\ell (H)}.

Note that if p_m is small, the expression \((1-p_m)^{\ell (H)}\) above is approximately equal to

\[ 1 - p_m \ell (H). \]

The following theorem combines the results of the preceding lemmas.

**THEOREM 3.1 [Zhang, Wang and Hill [31]]:** Let \( H \) be a given schema, and \( \varepsilon (H, k + 1) \) the expected value of \( e (H, k + 1) \) given \( P (k) \). Then,

\[ \varepsilon (H, k + 1) \geq (1 - p_c) \left( \frac{1}{L−1} \left( 1 - p_m \right)^{\ell (H)} \right) \frac{1}{F(k)} \varepsilon (H, k) \]

**Proof:** Consider a given chromosome in M (k) ∩ H, if, after the evaluation operation it has a resulting chromosome that is in H, then that chromosome is in P (k +1) ∩ H.

By Lemmas 3.2 and 3.2, the probability of this event is bounded below by

\[ (1 - p_c) \left( \frac{1}{L−1} \left( 1 - p_m \right)^{\ell (H)} \right) \]

Therefore, because each chromosome in M (k) H results in a chromosome in P (k +1) H with a probability bounded below by the above expression, the expected values of \( e (H, k +1) \) given is bounded below by

\[ (1 - p_c) \left( \frac{1}{L−1} \left( 1 - p_m \right)^{\ell (H)} \right) m(H, k) \]

Taking the expectation given P (k), we get

\[ \varepsilon (H, k + 1) \geq (1 - p_c) \left( \frac{1}{L−1} \left( 1 - p_m \right)^{\ell (H)} \right) M(H, k) \]
Finally, using Lemma 3.3 we arrive at the desired result.

The above theorem indicates how the number of chromosomes in a given schema changes from one population to the next. Three factors influence this change, reflected by three terms on the right hand side of inequality in the above theorem, namely, $1 - pcf(H)/(L-1) (1-p_m)^{o(H)}$ and $f(H,k)/f(k)$. Note that the larger the values of these terms, the higher the expected number of matches of the schema $H$ in the nest population. The effect of each term is summarized as follows:

- The term $f(H,k)/f(k)$ reflects the role of average fitness of the given schema $H$ – the higher the average fitness, the higher the expected number of matches in the next population.
- The term $1 - pcf(H)/(L-1)$ reflects the effect of crossover – the smaller the term $pcf(H)/(L-1)$, the higher the expected number of matches in the next population.
- The term $(1-p_m)^{o(H)}$ reflects the effect of mutation – the longer term, the higher the expected number of matches in the next population.

In summary, we see that a schema that is short, low order, and has above average fitness will have on average an increasing number of its representatives in the population from iteration to iteration. Observe that the encoding is relevant on the performance of the algorithm. Specifically, a good encoding is one that results in high fitness schemata having small lengths and orders.

### 3.5 Real–Number Genetic Algorithms

The genetic algorithms described thus far operate on binary strings, representing elements of the feasible set $\Omega$. Binary encodings allows us to use schema theory, describe in the previous section, to analyze genetic algorithms. However, there are some disadvantages to operating on binary strings. To see this, let $g: \{0,1\}^n \to \Omega$ represent the binary “decoding” function; that is binary chromosome, $g(x) \in \Omega$ is the point in the feasible set $\Omega \subset \mathbb{R}^n$ whose encoding is $x$. Therefore, the objective function being maximized by the genetic algorithm is not $f$ itself but rather the composition of $f$ and the decoding function $g$. In other words, the optimization problems being solved by the genetic algorithm is

Maximize $f(g(x))$
Subject to $x \in \{y \in \{0,1\}^L: g(y) \in \Omega\}$.

This optimization problem may be more complex than the original optimization problem. For example, it may have extra maximizer, making the search for a global maximizer more difficult.

The above motivates a consideration of genetic algorithms that operate directly on the original optimization problem. In other words we wish to implement, a genetic algorithm that operates directly on $\mathbb{R}^n$. The steps of this algorithm will be the same as before (see Fig. 3.4) expect that the elements of the population are points in the feasible set $\Omega$, rather than binary string. We will need to define appropriate crossover and mutation operations for this case.

For crossover, we have several options. The simplest is to use averaging: for a pair of parents $x$ and $y$, the offspring is $z = (x + y) / 2$ (this type of crossover operation is used, e.g., in [75]). This offspring can then replace one of the parents. Alternatively, we may produce two offspring as follows $z_1 = (x + y) / 2 + w_1$ and $z_2 = (x + y) / 2 + w_2$, where $w_1$ and $w_2$ are two randomly generated vectors (with zero mean). If either offspring lies outside $\Omega$, we have to bring the offspring back into $\Omega$, using for example a projection (see section 22.2) a third option for crossover is to take random convex combinations of parents. Specifically, given a pair of parents $x$ and $y$, we generated a random number $\alpha \in (0,1)$ and then produce two offspring $z_1 = \alpha x + (1-\alpha)y$ and $z_2 = (1-\alpha) x + \alpha y$. This method of crossover ensures that the offspring are always in the feasible set, proved the feasible set is convex. A forth option is to perturb the above two points by some random amount: $z_1 = \alpha x + (1-\alpha)y + w_1$ and $z_2 = (1-\alpha) x + \alpha y + w_2$ where $w_1$ and $w_2$ are two randomly of the offspring, and vectors (with zero mean). In this case, we have to check for feasibility of the offspring, and use projections if needed.

For mutation, a simple implementation is to add a random vector to the chromosome. Specifically, given a chromosome $x$, we produce it mutation as $x' = x + w$ where $w$ is a random vector with zero mean. This mutation operation is also called a “real number creep” [see, e.g.,... As before, we have to ensure that the mutated chromosome is feasible. If not, we may use a projection. An alternative method for mutation is to replace the chosen chromosome with a random convex combination of the chromosome with a random
The best, average and worst objective function values in the population every iteration (generation) of the real number genetic algorithm in example 3.4.

Example 3.4: Consider again the function $f: \mathbb{R}^2 \rightarrow \mathbb{R}$ from example 14.3 apply a real number genetic algorithm to find a maximizer of $f$, using a cross operation of the fourth type described above, and a mutation operation of these type above. With a population size of 20, we apply iterations of the age algorithm. As before, we use parameter values of $p_c = 0.75$ and $p_m = 0.00$.

Fig. 3.5 shows plots of the best, average, and worst objective function $y_i$ in the population for every iteration (generation) of the algorithm. The best-solution obtained at the end of the 50 iterations is $[-0.0096, 1.5845]$, with objective function value 8.1061, which is close with the result of example 3.3.

4. CONCLUSION

The work reviewed the elementary theory of evolution and genetics after which its mathematics in the form of genetic algorithm was developed and well defined. The analysis revealed that the best, average and worst objective function values in the population for every iteration (generation) of the genetic algorithm is optimally feasible as shown in Fig. 3.4.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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