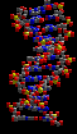


# The Biology of Creation

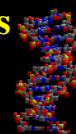
Studying God's World (Science)  
in the Light of God's Word (Scripture)  
Mr. Galloway



## Chapters 10 DNA & Protein Synthesis



© Designed Not  
Accidental



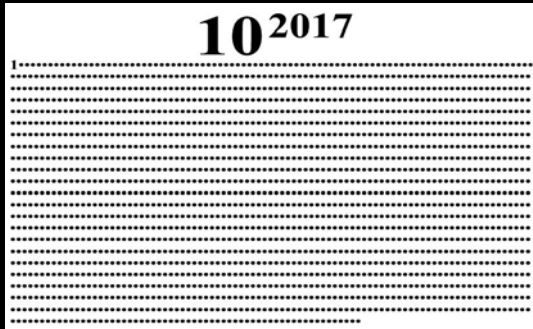
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Number of electrons that can be fitted  
into the known Universe:  $10^{130}$

**10<sup>130</sup>**  
10,000,000,000,000,000,  
000,000,000,000,000,000,  
000,000,000,000,000,000,  
000,000,000,000,000,000,  
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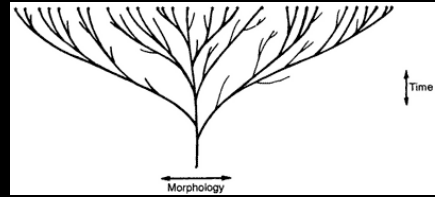
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Number of children from one couple  
without two exactly the same:  $10^{2017}$



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## Evolutionary (Imaginary) "Tree"



This evolutionary 'tree' claims that:  
- all modern species are descended from a common ancestor  
- the first living cell arose from non-living chemicals

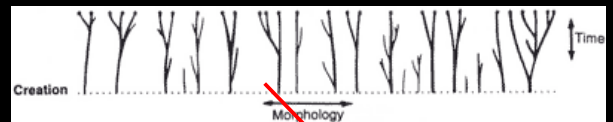
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## Genesis 1 "Kinds"

- Repeated phrase (10 x's in Gen 1):
  - "... according to its **kind** ..."
  - "... according to its their **kind** ..."
- Fits genetic facts known today:
  - breeding only possible within a **kind**
  - vast yet limited variation in each **kind**
  - breed forever yet stays same "**kind**"

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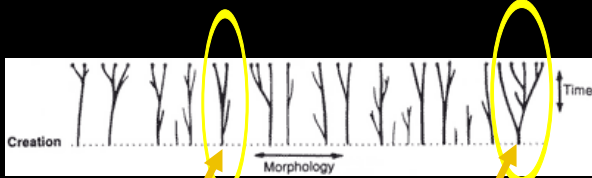
## Correct Creation "Orchard"



However, the Bible seems to teach that God created a certain number of distinct 'kinds' of organisms. Each of these Biblical 'kinds' would be unrelated to other Biblical 'kinds.' Instead of an evolutionary 'tree,' we would have an orchard of trees. Within each tree in the orchard would be a variety of related species. Creationists are currently working on a new classification system that would reflect this understanding. The study of created kinds is known as 'baraminology.' The name is taken from two Hebrew words found in Genesis 1: 'bara,' which means 'create,' and 'min,' which means 'kind.' **Baraminology = Bara + min + ology**

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## An Orchard of Holobaramins



This could represent the  
**FELINE Holobaramin**

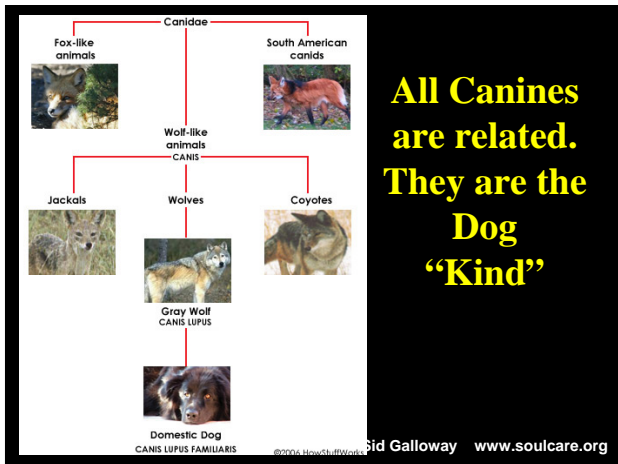
This could represent the  
**CANINE Holobaramin**

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This is a PAINTING by Carl Brenders  
Why are arctic wolves white & fluffy?



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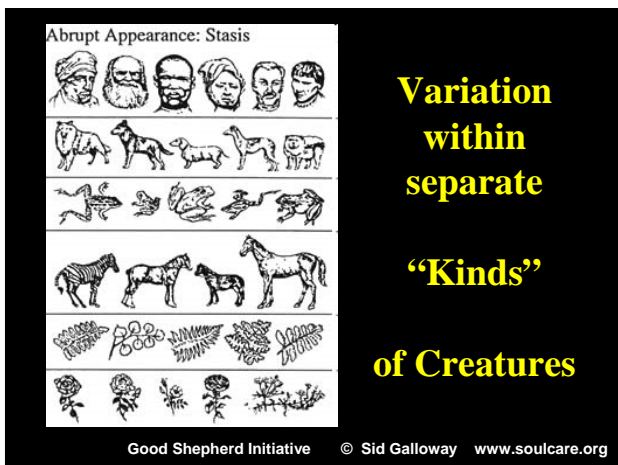


**All Canines  
are related.  
They are the  
Dog  
“Kind”**

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**Variation  
within  
separate  
“Kinds”  
of Creatures**

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**Ligers: lion/tiger hybrid @ 900 lbs**  
Such hybrids support the biblical “Kind” categories



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### Seven (7) Levels of Classification:

- Kingdom –
- Phylum –
- Class –
- Order –
- \*Family – level**
- Genus –
- Species –

Genetic *breeding* evidence shows the biblical “*Kind*” likely referred to the “*family*” level, not “*species*”.

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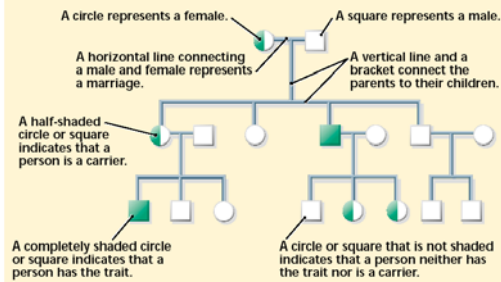
## Easy Outline of Ch 10

- **DNA**
  - **Transcription** of DNA into RNA
  - Into same language (nucleic acid to nucleic acid)
- **RNA**
  - **Translation** of RNA into a Protein
  - Into different lang. (nucleic acid to amino acid)
- **Protein**
  - A chain or sequence of **amino acids**.

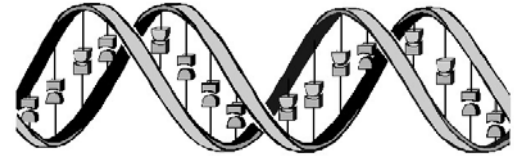
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**Pedigree** is a chart or “family tree” that tracks which members of a family have a particular trait.

### 17 Exploring a Pedigree



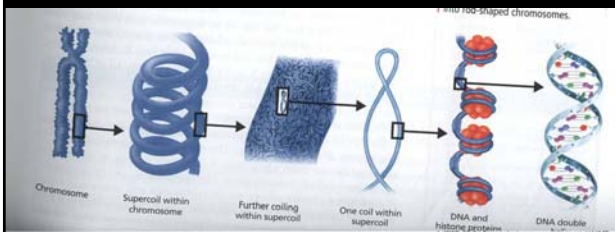
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DNA DOUBLE HELIX

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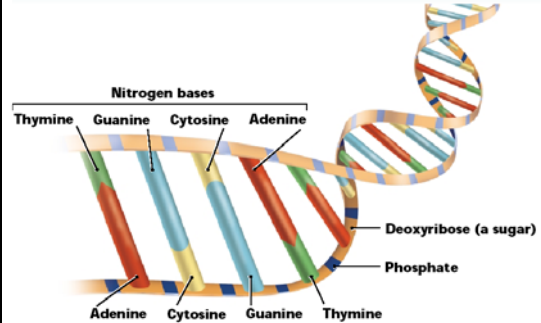
## DNA Complexity Designed Not Accidental (DNA)



DNA is a long thin molecule in a human cell with over 6 billion nucleotides. If a cell was the size of a basketball, the DNA would be 40 miles long.

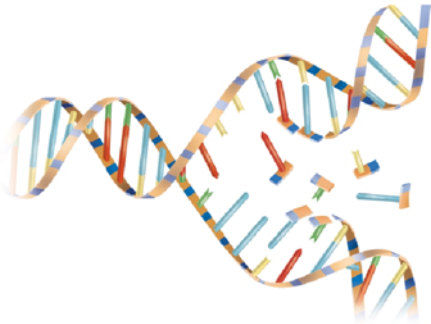
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### 10 DNA Structure



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## 11 DNA Replication



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## Nucleic Acids

**Nucleic Acids** = very large and complex molecules (polymers / macromolecules) that store information in the cell.

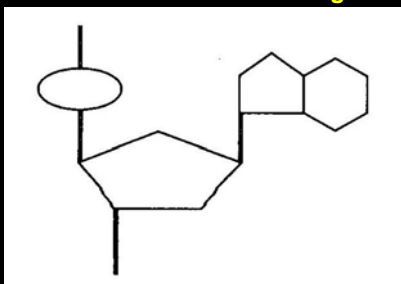
- Similar to a computer's binary code of zeros and ones.
- Nucleic acids use four compounds to store hereditary information.
- The order of arrangement of these four determines the code.
- **DNA** = Deoxyribonucleic Acid, contains the information for all cell activities, including cell division. (Designed Not Accidental)
- **RNA** = Ribonucleic Acid, stores and transfers information for making proteins.
- Both DNA and RNA are polymers, composed of thousands of linked *monomers*.
- **Nucleotides** are the *monomers*. Each is composed of three main components:
  - a. Phosphate group
  - b. Five-carbon sugar
  - c. Ring-shaped nitrogen base

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## DNA Nucleotide

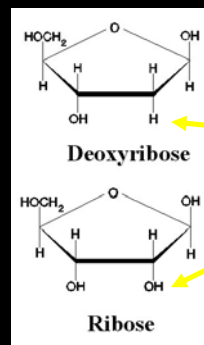
Phosphate

Nitrogen Base



Deoxyribose (sugar)

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## Structure of DNA

### A. Nucleotide Data

1. DNA contains four different nucleotide:

- a. **Purine** bases = **adenine** (A) and **guanine** (G);  
(a **purine** is a type of nitrogen-containing) base having a double-ring structure.
- b. **Pyrimidine** bases = **thymine** (T) & **cytosine** (C);  
(a **pyrimidine** is a type of nitrogen-containing) base having a single-ring structure.

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A = Adenine — *purine*  
G = Guanine — **Double-ring**

C = Cytosine — *pyrimidine*  
T = Thymine — **Single-ring**

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# DNA

Adenine  
 Guanine  
 Thymine  
 Cytosine

QUANINE  
 THYMINE  
 ADENINE  
 CYTOSINE

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Adenine      Cytosine      Guanine  
 Thymine      Uracil

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5'      3'

3'      5'

nucleotide

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### DNA Replication Process

Copy or Duplicate  
Using the same language:  
Nucleic Acid to Nucleic Acid language

DNA polymerase III  
 complementary #2  
 helicase  
 complementary #1  
 DNA polymerase III

C. Ophardt, c. 2003

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### RNA Transcription

A Scribe makes a copy using the same language:  
Nucleic Acid to Nucleic Acid language

Non coding DNA  
 Template DNA  
 new RNA  
 uracil nucleotide  
 RNA Polymerase

C. Ophardt, c. 2003

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### Elongation (translation)

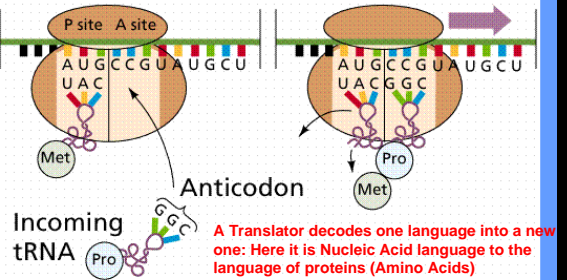


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### Elongation continues

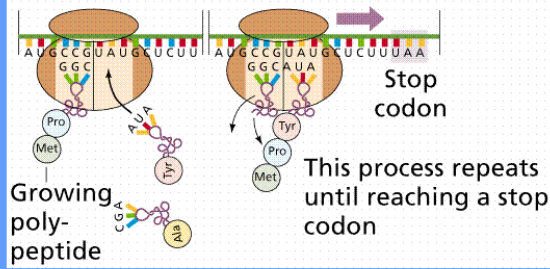


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### Termination

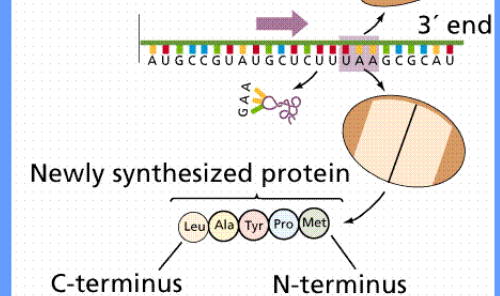


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### Codons to Amino Acids

- The **language** of nucleic acids is made up of only 4 "**letters**" (A, T, C, G).
- A "**sentence**" (a gene which is a DNA segment) is composed of "**words**" (codes).
- Each **word** is 3 "**letters**" (nitrogen bases) long. [Such as AAT, CTG, GTT]
- A **chromosome** is a long DNA macromolecule.
- The genetic **coding** on the DNA = BILLIONS of bases (A, T, C, G).
- **Transcription** converts a segment of DNA to an mRNA inside the nucleus.
- **Translation** then translates the mRNA.
- Each **tRNA** transfers a specific amino acid to a **codon** on the mRNA.
- Since there are 4 letters and each **word / code** is made of 3 of the **letters**, then the number of possible **codes** is  $4 \times 4 \times 4 = 64$
- **20** amino acids used in living organisms make 1,000's of **proteins**.
- More than one codon can code for some amino acids.

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**tRNA's,  
and coding  
for  
amino acids**

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		Second Letter					
		U	C	A	G		
1st letter	U	UUU   Phe UUC UUA   Leu UUG	UCU   Ser UCC UCA UCG	UAU   Tyr UAC   Stop UAA   Stop UAG	UGU   Cys UCC UGA   Stop UGG   Trp	U C A G	
	C	CUU   Leu CUC CUA CUG	CCU   Pro CCC CCA CCG	CAU   His CAC CAA CAG	CGU   Arg CGC CGA CGG	U C A G	
	A	AUU   Ile AUC AUA AUG   Met	ACU   Thr ACC ACA ACG	AAU   Asn AAC AAA AAG   Lys	AGU   Ser AGC AGA AGG   Arg	U C A G	
	G	GUU   Val GUC GUA GUG	GCU   Ala GCC GCA GCG	GAU   Asp GAC GAA GAG   Glu	GGU   Gly GGC GGA GGG	U C A G	

**Coding for amino acids**

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## Mutations, Myths, and Macroevolution

- Any **change / error** in the original genome.
  - Somatic** (body) cell mutations only affect the individual and can cause cancer, etc.
  - Germline** (gamete) cell mutations are passed on to the next generation.
- Most are destructive and usually recessive, so they pass on in the heterozygous form.

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## Mutations continued . . .

- Three major **causes** of mutational change:
  - High** energy radiation such as X-rays
    - Cause physical breakage
  - Low** energy radiation such as UV light
    - Cause DNA cross-links
  - Chemical** modification of the DNA bases
- Or a copying error in mitosis or meiosis

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## Mutations and Evolution

Lee Spetner (Ph.D. Physics – MIT, taught information and communications at Johns Hopkins University), *Not By Chance*, 1997, pp. 131, 138.


“But in all the reading I’ve done in the life-sciences literature, I’ve never found a mutation that added information. . . . All point mutations that have been studied on the molecular level turn out to reduce the genetic information and not increase it.”

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## Lee Spetner, PhD

- Jewish Creationist Biophysics Professor
- Former professor and researcher with Johns Hopkins University
  - One of the most respected scientists in the world.
- Author of a new book: **NOT BY CHANCE: SHATTERING THE THEORY OF EVOLUTION**. His focus is on the fact that mutations never add information.
- “It is certainly the most rational attack on evolution that I have ever read.”
  - Professor E. Simon, Dept. of Biology, Purdue University



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**Dr Lee Spetner** p.159-160

“**Not even one mutation** has been observed that adds a little information to the genome. .... The **failure** to observe even one mutation that adds information is **more than just a failure to find support** for the theory. It is **evidence against** the theory. We have here a **serious challenge** to neo-Darwinian theory.” [Emphasis added]

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**John Sanford, PhD**

Professor of genetics at Cornell University for 25 years.  
*Genetic Entropy & The Mystery of the Genome* 2005  
 Over 70 scientific publications & over 25 patents

“Modern **Darwinism** is built, most fundamentally, upon what I will be calling “**The Primary Axiom**”. . . . [It] is that man is merely the product of *random mutations* plus *natural selection*. . . . It is for this reason that the overwhelming **majority of youth** who start out with a belief that there is more to life than mere chemistry – **will lose their faith while at college**. I believe this is also the **cause** of the widespread **self-destructive** and **self-denigrating** behaviors we see throughout our culture.”

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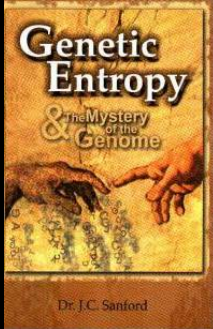
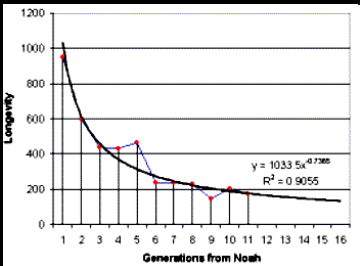
**Dr. Sanford & his “Gene Gun”**




Co-developer of the computer program: MENDEL'S ACCOUNTANT

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**Book: Genetic Entropy, by John Sanford, PhD**


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**Werner Gitt, PhD**

- Six Day / Young Earth Creationist
- Retired director and professor at the German Federal Institute of Physics and Technology
- Former Head of the Department of Information Technology
- Developed the best “scientific” definition of “information” useful for the evaluation of DNA mutations.

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**John Baumgardner, PhD**

- Six Day / Young Earth Creationist (YEC)
- Famous Los Alamos National Laboratory
- M.S., Electrical Engineering, Princeton University, Princeton, NJ, 1970
- M.S., Ph.D Geophysics and Space Physics, University of California, Los Angeles
- Called by U.S. News & World Report:
  - “the world's pre-eminent expert in the design of computer models for geophysical convection.”

Co-developer of the computer program: MENDEL'S ACCOUNTANT

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## DNA Information Team

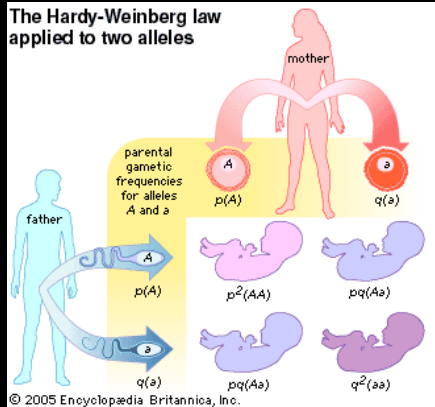


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## Hardy-Weinberg Law

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### The Hardy-Weinberg law applied to two alleles



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## Hardy-Weinberg Law

- The Hardy-Weinberg law of genetic equilibrium provides a mathematical model for studying evolutionary changes in allelic frequency within a population. In this laboratory, you will apply this model by using your class as a sample population.

[http://www.phschool.com/science/biology\\_place/labbench/lab8/intro.html](http://www.phschool.com/science/biology_place/labbench/lab8/intro.html)

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## Mutation Mechanism ??

- \*\* Note that mutations cannot add sufficient NEW genetic information to have generated the massive amount hypothesized by macroevolutionists for microbes to monkeys to man. Mutations cannot generate new KINDS of creatures. They can only degrade a genome over time. (see the book, *GENETIC ENTROPY*, by Dr. John Sanford of Cornell University)

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- In 1908 G. Hardy and W. Weinberg independently proposed that the frequency of alleles and genotypes in a population will remain constant from generation to generation if the population is stable and in genetic equilibrium. Five conditions are required in order for a population to remain at Hardy-Weinberg equilibrium:

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## 5 conditions for H-W Equilibrium

- 1. A large breeding population
- 2. Random mating
- 3. No change in allelic frequency due to mutation
- 4. No immigration or emigration
- 5. No natural selection

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## 1. A large breeding population

- A large breeding population helps to ensure that chance alone does not disrupt genetic equilibrium. In a small population, only a few copies of a certain allele may exist. If for some chance reason the organisms with that allele do not reproduce successfully, the allelic frequency will change. This random, nonselective change is what happens in genetic drift or a bottleneck event.
- genetic drift** = Changes in the gene pool of a small population due to chance.

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### LARGE BREEDING POPULATION



An earthquake that kills three people out of a population of 10 million has little effect on the composition of the gene pool.

### SMALL BREEDING POPULATION



An earthquake that kills three people out of a band of 20 individuals has a significant effect on the composition of the gene pool.

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## 2. Random mating

- In a population at equilibrium, mating must be random. In assortative mating, individuals tend to choose mates similar to themselves; for example, large blister beetles tend to choose mates of large size and small blister beetles tend to choose small mates. Though this does not alter allelic frequencies, it results in fewer heterozygous individuals than you would expect in a population where mating is random.

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### RANDOM MATING



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### ASSORTATIVE MATING




## 3. No change in allelic frequency due to mutation

- For a population to be at Hardy-Weinberg equilibrium, there can be no change in allelic frequency due to mutation. Any mutation in a particular gene would change the balance of alleles in the gene pool. Mutations may remain hidden in large populations for a number of generations, but may show more quickly in a small population.

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**NO MUTATIONS**



With no mutations, the composition of the gene pool remains the same generation after generation, if the other conditions for Hardy-Weinberg equilibrium are also met.

**MUTATIONS**



Mutations change the composition of the gene pool. New alleles are introduced, and allelic frequencies change.

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### 4. No immigration or emigration

- For the allelic frequency to remain constant in a population at equilibrium, no new alleles can come into the population, and no alleles can be lost. Both immigration and emigration can alter allelic frequency.


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**NO MIGRATION**



Isolation of a population of trees prevents changes in the gene pool due to immigration and emigration.

**MIGRATION**



Immigration of alleles in pollen from a neighboring population of trees can cause a change in the composition of the gene pool.

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### 5. No natural selection

- In a population at equilibrium, no alleles are selected over other alleles. If selection occurs, those alleles that are selected for will become more common. For example, if resistance to a particular herbicide allows weeds to live in an environment that has been sprayed with that herbicide, the allele for resistance may become more frequent in the population.

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**NO SELECTION**



Herbicide-resistant weed      Herbicide-sensitive weed

In an environment without herbicide, both herbicide-resistant weeds and herbicide-sensitive weeds can live and reproduce.

**SELECTION**



In an environment containing herbicide, weeds that are sensitive to herbicide die and thus do not pass on their genes. Weeds with the allele for herbicide resistance are selected for.

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### Estimating Allelic Frequency

- If a trait is controlled by two alternate alleles, how can we calculate the frequency of each allele? For example, let us look at a sample population of pigs.
- The allele for black coat is recessive to the allele for white coat. Can you count the number of recessive alleles in this population?

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## The Hardy-Weinberg Equation

- $p$  = frequency of the dominant allele (represented here by  $A$ )
- $q$  = frequency of the recessive allele (represented here by  $a$ )
- For a population in genetic equilibrium:  
 $p + q = 1.0$  (Sum of frequencies of alleles is 100%.)  
 $(p + q)^2 = 1 \dots$  so  $\dots p^2 + 2pq + q^2 = 1$
- The three terms of this binomial expansion indicate frequencies of three genotypes:
- $p^2$  = frequency of  $AA$  (homozygous dominant)
- $2pq$  = frequency of  $Aa$  (heterozygous)
- $q^2$  = frequency of  $aa$  (homozygous recessive)

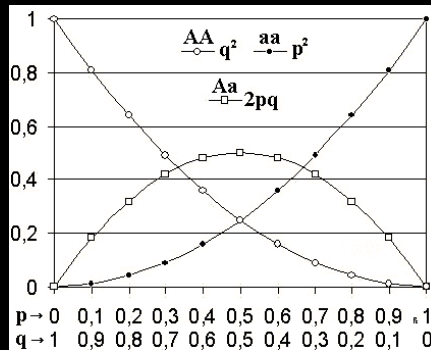
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## Basic formulations (again) . . .

- $p^2 + 2pq + q^2 = 1 \dots \dots$  and  $\dots p + q = 1$
- 1 = 100% of alleles in the population, when the fractions are converted to %
- $p$  = frequency of the dominant allele in the population (Ex =  $A$ )
- $q$  = frequency of the recessive allele in the population (Ex =  $a$ )
- $p^2$  = percentage of homozygous dominant individuals (Ex =  $AA$ )
- $q^2$  = percentage of homozygous recessive individuals (Ex =  $aa$ )
- $2pq$  = percentage of heterozygous individuals (Ex =  $Aa$ )
- [Genotype frequencies:  $AA = p^2$ ,  $aa = q^2$ ,  $Aa = 2pq$ ]
- [Phenotype frequencies: Freq Dom =  $AA + Aa$  or  $p^2 + 2pq$ , Freq Rec =  $aa = q^2$ ]

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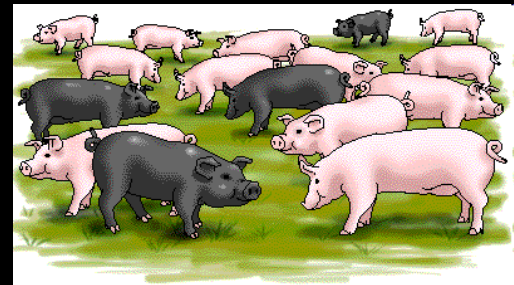
Y axis =  
genotype frequencies



X axis = allele frequencies

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## See sample problems . . .

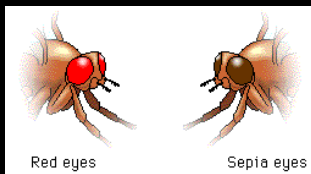


[http://www.phschool.com/science/biology\\_place/labbench/lab8/intro.html](http://www.phschool.com/science/biology_place/labbench/lab8/intro.html)

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## H-W Sample Problem:

- “In a certain population of 1000 fruit flies, 640 have red eyes while the remainders have sepia eyes. The sepia eye trait is recessive to red eyes.”



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## H-W Sample Problem cont . . .

- **How many individuals would you expect to be homozygous for red eye color?**
- **Hint:** “The first step is always to calculate  $q^2$ ! Start by determining the number of fruit flies that are homozygous recessive. If you need help doing the calculation, look back at the Hardy-Weinberg equation.”

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## H-W Sample Answer:

- You should expect 160 to be homozygous dominant.
- Calculations:  $q^2$  for this population is  $360/1000 = 0.36$
- $q = \sqrt{0.36} = 0.6$
- $p = 1 - q = 1 - 0.6 = 0.4$
- The homozygous dominant frequency =  $p^2 = (0.4)(0.4) = 0.16$ .
- Therefore, you can expect 16% of 1000, or 160 individuals, to be homozygous dominant.

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## Mendel's Accountant

<http://mendelsaccount.sourceforge.net/>

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## “Mendel's Accountant”

- “Mendel's Accountant (MENDEL) is an advanced numerical simulation program for modeling genetic change over time and was developed collaboratively by Sanford, Baumgardner, Brewer, Gibson and ReMine.”
- <http://mendelsaccount.sourceforge.net/>

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<http://mendelsaccount.sourceforge.net/>

- “MENDEL is a genetic accounting program that allows realistic numerical simulation of the mutation/selection process over time. MENDEL is applicable to either haploid or diploid organisms, having either sexual or clonal reproduction. Each mutation that enters the simulated population is tracked from generation to generation to the end of the experiment - or until that mutation is lost either as a result of selection or random drift. Using a standard personal computer, the MENDEL program can be used to generate and track millions of mutations within a single population.”

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- “MENDEL's input variables include such things as mutation rate, distribution specifications for mutation effects, extent of dominance, mating characteristics, selection method, average fertility, heritability, non-scaling noise, linkage block properties, chromosome number, genome size, population size, population sub-structure, and number of generations.”

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- “The MENDEL program outputs, both in tabular and graphic form, provide several types of data including: deleterious and beneficial mutation counts per individual, mean individual fitness as a function of generation count, distribution of mutation effects, and allele frequencies.”

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- “MENDEL provides biologists with a new tool for research and teaching, and allows for the modeling of complex biological scenarios that would have previously been impossible.”
- **You can**
  - [Run the program](#)
  - [View screen shots](#)
  - [Download Mendel's Accountant from Sourceforge](#) (see download instructions [here](#))
  - [Read about the authors](#)
  - Download the [User's Manual](#) and [Linux How-to](#) (requires [Adobe Reader](#))
  - [Join the discussion group](#)

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## References

- J. Sanford, J. Baumgardner, W. Brewer, P. Gibson, and W. Remine. [Mendel's Accountant: A biologically realistic forward-time population genetics program](#). SCPE. 8(2), July 2007, pp. 147-165.
- J. Sanford, J. Baumgardner, W. Brewer, P. Gibson, and W. Remine. [Using computer simulation to understand mutation accumulation dynamics and genetic load](#), in Y. Shi et al. (eds.), ICCS 2007, Part II, LNCS 4488, Springer-Verlag, Berlin, Heidelberg, pp. 386-392.

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## Sample Trial: Human 1

• Generation	Fitness	#del mut	#fav mut
• 500	.7644E+00	4852	0

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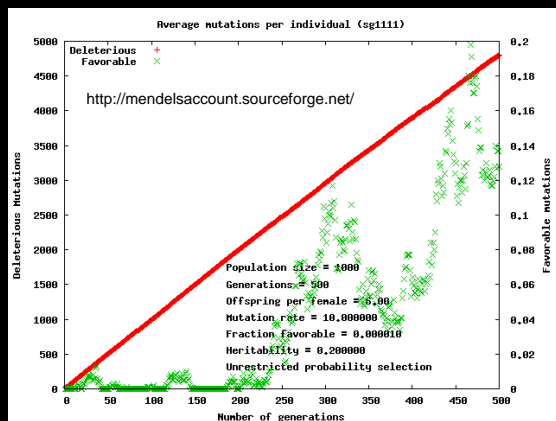
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## 1a. Generational History

- *updated every generation*
- [Average mutations](#)
- **Figure 1a** plots the number of both deleterious and beneficial mutations per individual. The scale for deleterious mutations is on the left and the scale for the beneficial mutations is on the right.

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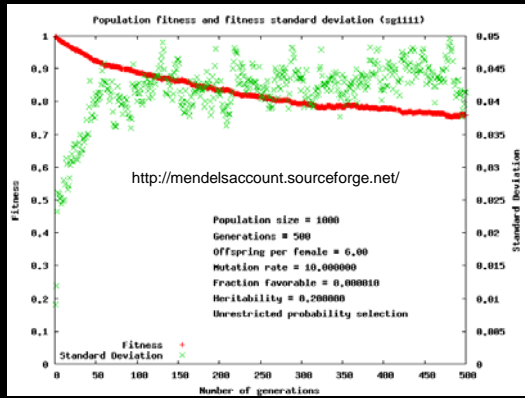
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## 1b. Generational History

- *updated every generation*
- [Fitness](#)
- **Figure 1b** shows average individual fitness (left scale) and the standard deviation for fitness (right scale), plotted versus generation count. The initial fitness is always assumed to be 1.0. For both 1a and 1b, the y-axis is self-scaling, since mutation counts and fitness can change dramatically over time.

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## 2. Distribution of accumulated mutations

- updated every 20 generations
- **Deleterious**
- **Figure 2:** This figure is designed to reveal how selection is altering the distribution of mutation effects in the population. Typically it shows that the frequencies in the population of mutations with larger effects are affected by the selection process much more strongly than the frequencies of mutations with small effects.

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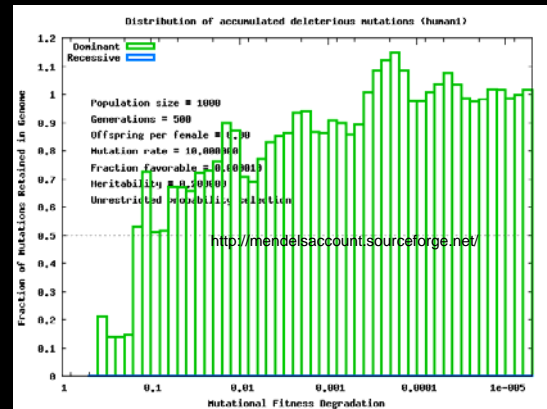
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### Fig. 2a Deleterious Mutations

- Figure 2a is **histogram** plot that shows the mutation effect distribution for deleterious mutations. The x-axis uses a log scale to represent the magnitude of the mutation effects and ranges from lethal (-1.0) on the left to the minimal mutation effect tracked on the right. The y-axis uses a linear scale, and the height of the bar reflects the fraction of each class of mutations that was not eliminated by selection. A value of 1.0 is the expected height for all bars when there is no selection. Perfect selection for a given mutation effect interval will result in bars of zero height.

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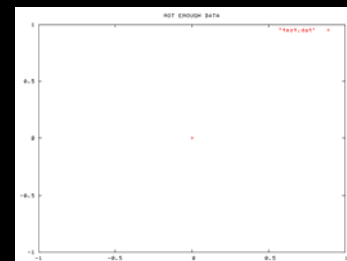
### Fig. 2b Beneficial Mutations

<http://mendelsaccount.sourceforge.net/>

- Figure 2b shows the beneficial half of this same distribution. The distribution extends from the left with the smallest mutational effects tracked (tracking threshold) to the maximal beneficial effect (on the right), and any bar significantly higher than 1.0 reflects positive selection. Note: These figures are not generated until there are a "sufficient" number of mutations. Even so, plots can initially show significant noise associated with sampling error until larger data sets have built up. This is especially true for beneficial mutations (Figure 2b), because they are usually relatively rare. The user can know that the number of accumulated mutations has become large enough to give reliable plots when there is little random fluctuation in bar heights and the transition from selectable to near-neutral becomes smooth and unambiguous.

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### Beneficial Mutations: "Not Enough Data"



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### 3. Selection threshold history

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- updated every 20 generations
- Threshold at generation =
- Figure 3: This figure plots the selection threshold for dominant alleles as a function of the generation count, beginning with generation 200. The selection threshold, described more completely in the glossary below, is the value of absolute fitness effect for which the deleterious allele frequency is 50% of what it would be, if there had been no selection.

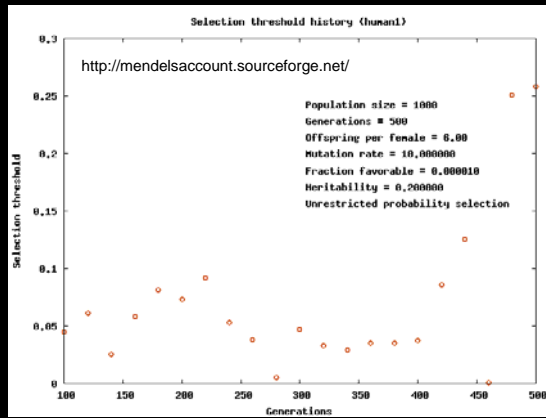
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### 3. Selection threshold history . . . .

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- For values of absolute fitness effect smaller than this threshold, alleles frequencies are governed more by drift and less by selection, while for values larger than the threshold, the opposite is true. Data for recessive alleles is not plotted, but is available in the data file. At any given generation, one can readily estimate the selection threshold visually using Figure 2, by observing the fitness value on the horizontal scale corresponding to a bar height of 0.5.

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### 4. Distribution of near-neutrals

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- updated every 20 generations
- Deleterious
- Figure 4: This figure, like figure 2, is also designed to reveal how selection is altering the distribution of mutation effects in the population. By using a linear (but truncated) scale for the x-axis, it shows more closely just the lower impact mutations. It displays, in greater detail the differences between the theoretical (red) distribution of mutation effects (as would accumulate apart from selection) and the actual distribution of accumulating mutations (blue = recessive, green = dominant).

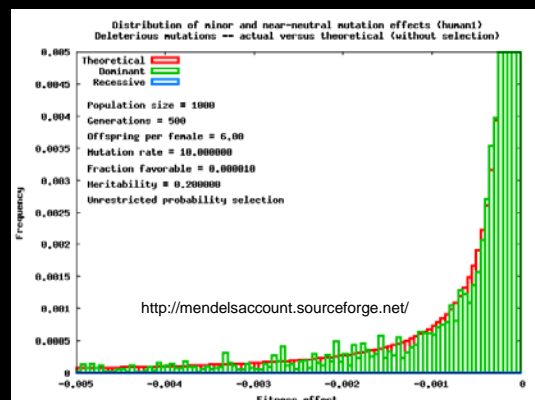
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### 4. Distribution of near-neutrals - - -

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- Like figure 2, this figure reveals the variable effectiveness of selection over a range of mutation effects. Unlike Figure 2, the viewer can readily see that small-effect mutations are always much more numerous than large-effect ones, both before (red), and after (blue/green) selection. Figure 4a displays deleterious mutations, and figure 4b displays beneficial mutations. Like figure 2, the plotting of accumulated mutations is only reliable when enough mutations are present, evident when the distribution visually transitions from jagged to smooth.

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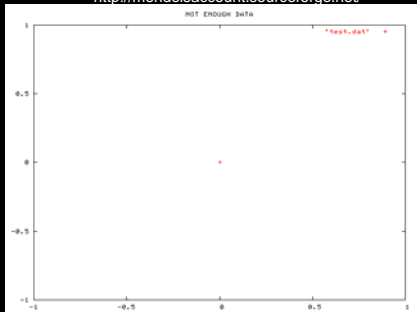


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## Beneficial Mutations:

“Not Enough Data”

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## 5. Linkage block effects

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- updated every 20 generations
- Average linkage block effect =  $-1.178e-004$
- Linkage blocks which have a positive fitness value = 0.00%
- .....

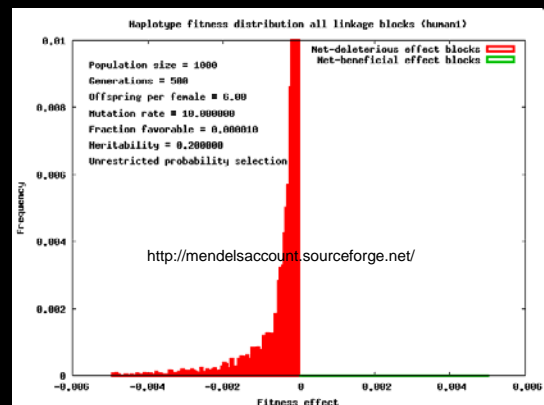
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## 5. Linkage block effects.....

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- Figure 5: This figure displays the frequency distribution of the fitnesses of the accumulating haplotypes versus their composite fitness effect. Those haplotypes with a net deleterious effect are plotted in red (left of zero) and those with a net beneficial effect in green (right of zero). This figure shows the distribution of net linkage blocks fitness effects (as opposed to individual mutation effects).

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## 6. Selection effects

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- updated every 20 generations
- # pre selection fitness = .76176
- # post selection fitness = .76442

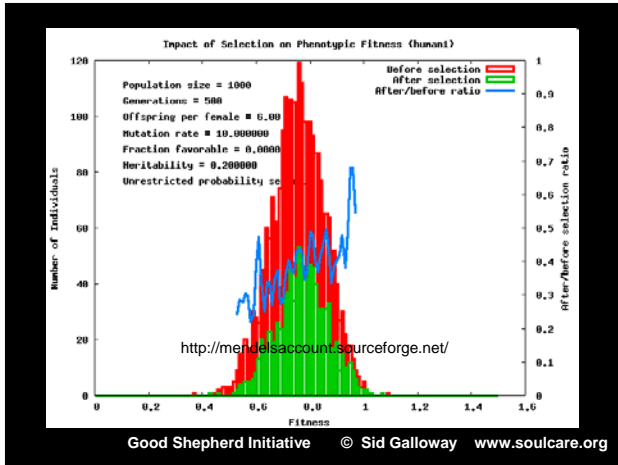
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## 6. Selection effects.....

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- Figure 6: This figure superimposes the distribution of phenotypic fitness values of the individuals in the population before (red), and after (green), selection. A histogram format is used. The blue line plots the ratio of the number of surviving (selected) individuals within a given phenotypic class versus the number of offspring in that fitness category prior to selection (scale shown on the right). The portion of the red distribution not covered by the green represents those offspring that are selected away (those offspring that will not reproduce to create the next generation).

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## 7. Allele Frequency updated

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- at generation 200, every 1000 generations starting from generation 0, and at end of run.
- | Number of alleles                    | Deleterious | Favorable |
|--------------------------------------|-------------|-----------|
| very rare (0-1%)                     | 15107       | 0         |
| polymorphic (1-99%)                  | 8202        | 0         |
| fixed or very nearly fixed (99-100%) | 0           | 0         |

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## 7. Allele Frequency updated .....

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- Figure 7: This figure displays the frequencies of all tracked individual mutant alleles in the population. It is plotted at generation 200, at generation 1000, then every 1000 generations, and finally at the end of the run.
- The figure provides the number of very rare alleles, in the population (having frequencies of less than 1%), the number of polymorphic alleles (having frequencies between 1- 99%), and the number of fixed alleles (having frequencies greater than 99%).

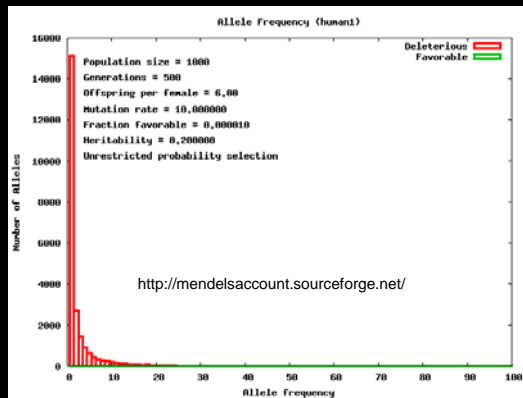
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## 7. Allele Frequency updated .....

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- The data button on this plot provides access to the data for the current and all the previous plots. The file with suffix .pmd provides even more detailed information on the distribution of polymorphisms at each of these plot times. Results are in table format with 500 values of polymorphism frequency (50 intervals) vs. fitness effect (10 intervals). Caution – this data represents only tracked mutations, so the tracking threshold must be set to be less than the lowest-impact mutation effect for this data to be fully accurate.

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## Applications of MENDEL.

- Teaching:**
- The MENDEL program is a useful teaching tool to demonstrate to students in a concrete and visual manner the fate of mutations once they enter a population, and how they increase in frequency, are eliminated, or simply drift randomly. MENDEL shows how these dynamics play out over many generations under a wide range of conditions. The student can see how this process affects average mutation count per individual, average fitness, allelic frequencies, and mutational fixations over time.

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## Applications of MENDEL...

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- The student can experiment with the biological parameters that alter the rates of these processes. MENDEL also allows the student to see exactly what happens during a population bottleneck, what happens in a mutational meltdown scenario, how genes can circulate between sub-populations, and what happens when key biological parameters are modified during a run.

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## Applications of MENDEL ... Research:

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- MENDEL's Accountant can function as a sophisticated research tool. To our knowledge, there is no simulation program comparable that provides genetic researchers with such a realistic and flexible research simulation capability. Highly specific scenarios can be run that have bearing on extinction of species, management of endangered species, germplasm preservation, epidemiology, ecology, etc. Likewise, simulations can also be run which have bearing on more basic questions, including the relative importance of the different variables that affect selection efficiency.

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## MENDEL Glossary

<http://mendelsaccount.sourceforge.net/>

- Mendel's Accountant (MENDEL).
- An advanced numerical simulation program that acts as a genetic accounting system, and realistically models how genomes change over time in response to mutation and selection.

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## MENDEL Glossary ...

<http://mendelsaccount.sourceforge.net/>

- Genome. The entire genetic content of an organism. In MENDEL, the initial genome is not specified except in terms of genome size, chromosome number, and number of linkage blocks. A functional genome is simply assumed as a backdrop for the accumulating mutations, which are individually being tracked.

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## MENDEL Glossary ...

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- Functional genome. The entire physical genome, minus any portions that have no biological expression or consequences relating to biological fitness.

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## MENDEL Glossary ...

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- Population. All the individuals that constitute an inter-breeding group. In MENDEL, the population is specified in terms of number of reproducing individuals, mating pattern, fertility level, and sub-structure (tribes).

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## MENDEL Glossary ...

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- **Mutation.** Any heritable change in the genome that was not present in the previous generation. In MENDEL, mutations can have a range of effect from lethal to beneficial, and a range of expression from entirely dominant to entirely recessive. MENDEL tracks the effect of every mutation from the time that the mutation enters the population until it may be lost due to selection or random drift.

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## MENDEL Glossary ...

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- **Mutant locus** - The location of a mutation, in terms of its position within a linkage block within a chromosome. In MENDEL, all loci are assumed to be non-mutant except where a mutation has been added. Once a single mutation arises within an individual at a specific location, the same corresponding location in all the other individuals not carrying this mutation, by definition, becomes the non-mutant allele.

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## MENDEL Glossary ...

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- **Mutant allele.** All the derived copies of an initial mutation, which are being passed from generation to generation. A mutant allele can increase or decrease in its frequency within the population.

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## MENDEL Glossary ...

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- **Mutant allele frequency** - The mutant allele frequency for a given locus is determined by number of copies of a given mutation in the population, compared to how many copies there would be if every individual was homozygous for that mutation. (For diploids the total number of possible mutant copies is two times the population size.). If there are 2 copies of a mutation in a diploid population of 100, the mutant allele frequency is 1%, so the non-mutant allele frequency is 99%.

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## MENDEL Glossary ...

- **Mutation fitness effect.** We refer to the biological impact of a mutation on individual fitness as the mutation's "fitness effect". In MENDEL a given mutation fitness effect can be small or large. The mutation effect is expressed as the relative change in an individual's total *biological functionality*, as reflected by a corresponding change in an individual's *genotype value* (see below). A deleterious mutation with an effect of -0.01 decreases an individual's genotype value by 1%. Crudely speaking, one percent of the genomic information is lost, or more accurately, total biological functionality is reduced by 1%. Another way of saying this is that such a mutation decreases genotypic fitness by 1%.

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## Glossary Cont . . . "Mutation fitness effect" . . .

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- . . . "These are all just alternative ways of describing the biological effect of a mutation. Mutation effect is independent of environmental variation (phenotypic noise), and random aspects of reproduction (reproductive noise). Mutation effect is similar but not identical to the traditional concept of a *selection coefficient*. See Sanford et al. (SCPE 8(2), 147-165) for the mathematical function we use to generate the distribution of mutation effects, and for an explanation of the difference between fitness effect and selection coefficient."

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## MENDEL Glossary ...

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- **Genotype.** The genotype is the specific collection of genetic alleles present in a specific individual within the population. In MENDEL, the starting genotype for all individuals is an unspecified and invariant genome for the organism. MENDEL specifies only the mutational deviations from this non-mutant starting genotype. In MENDEL, the specified genotype is simply the sum total of all the mutant alleles (including their chromosomal locations), within an individual.

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## MENDEL Glossary ...

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- **Genotype value.** The genotype value is that portion of an individual's total biological functionality that is derived exclusively from that individual's genetic makeup. The genotype value is different from the phenotype value because environmental factors (phenotypic noise) also contribute to an individual's biological functionality.
- In MENDEL, the initial genotypic value of all individuals is defined as 1.0. Beneficial mutations increase this value, and deleterious mutations decrease this value. The extent to which a mutation alters genotypic value is a function of its specific mutation effect (see above).

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## ... Genotype value ...

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- Genotype value can be understood as being synonymous with the term *genotype fitness*. However, the concept of genotype value (genetic fitness) is distinct from what most population geneticists formally define as "fitness". For clarity, we will use the term *reproductive fitness* (see below), to refer to the traditional population geneticist's definition of fitness, as distinct from *genetic fitness*. Genetic fitness is what is actually plotted in MENDEL's figure 1b.

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## MENDEL Glossary ...

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- **Linkage block (haplotype).** Mutations are not inherited independently but are passed from generation to generation in clusters or blocks. These clusters are physically linked together, within "linkage blocks", which represent specific regions of linear chromosomes. Although chromosomes recombine something like cutting a deck of cards, some cards consistently stick together, due to recombinational "cold" spots. Points of frequent recombination (hot spots) separate linkage blocks (cold spots) from each other. A specific set of mutations which is physically being inherited as a single unit is called a *haplotype*.

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## MENDEL Glossary ...

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- **Phenotype.** The actual biological functionality of an individual. The phenotype is affected both by the genotype and by the environment in which the individual develops. The genotype and phenotype are correlated, but they are not identical. In MENDEL, the phenotype value (or fitness) is created by adding to each genotype value (or fitness) a random "environmental noise value" based upon the specified "heritability" and using a random number generator. This environmental noise value is not heritable and so is not passed on to the next generation.

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## MENDEL Glossary ...

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- **Phenotype value.** This is the actual biological functionality of an individual, arising due to the combination of genotypic effects and environmental effects. Phenotypic value is what selection actually acts on - it is what "Mother Nature" actually "sees". In MENDEL, the initial *mean* phenotypic value is always 1.0. In the initial first generation, all individuals will have an identical genotype, but there will still be variance around the population's mean phenotypic value - due to environmental effects. ....

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## ... Phenotype value ...

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- ... “The amount of environmental noise is controlled by specifying a “heritability value”, which is the input parameter that defines the ratio of genotypic variance to environmental variance. Because the phenotypic contribution from heritability scales with genotype value and therefore becomes small when the genotypic value becomes small, an additional non-scaling noise factor can also be used to modify phenotypic values.”

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## MENDEL Glossary ...

<http://mendelsaccount.sourceforge.net/>

- . . . . Phenotype value is synonymous with the terms *phenotypic fitness* or *biological fitness* as reflected by the common use of these terms among biologists. However, the concept of phenotype value (phenotypic fitness) is distinct from what population geneticists formally define as “fitness”. For clarity, we will use the term *reproductive fitness* (see below), to refer to the traditional population geneticist’s definition of fitness, which is distinct from phenotypic fitness.

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## MENDEL Glossary ...

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- **Reproductive fitness.** We define this term as the phenotypic value (phenotypic fitness), plus “reproductive noise”. Reproductive noise arises because actual success in reproduction is not just determined by biological functionality, but also by random reproductive factors. So phenotypic fitness and reproductive fitness are correlated, but not identical. The strength of correlation between phenotype value and reproductive fitness depends upon the selection scheme employed. Artificial truncation will yield the highest possible correlation, while classical probability selection will yield the lowest correlation of the various schemes implemented in MENDEL.

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## .... Reproductive fitness ....

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- What we are calling “reproductive fitness” is sometimes called “Darwinian fitness” or “Wrightian fitness” after Sewell Wright, the first to formulate “Darwinian fitness” mathematically. We recognize that “Darwinian fitness” encompasses elements beyond “reproductive fitness”, but have not identified a more precise term that is still understood intuitively by a broad audience.

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## MENDEL Glossary ...

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- **Selection threshold.** The selection process eliminates deleterious mutations with large negative fitness effect values more effectively that it does for deleterious mutations with smaller ones. Similarly, selection enhances the frequencies of favorable mutations with large positive fitness effect values more effectively than it does for favorable mutations with smaller values. For fitness effect values sufficiently small, selection plays essentially no role in altering the frequencies of such alleles.

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## . . . Selection threshold . . . 2

- Mutations in this range, both deleterious and favorable, are generally referred to as “effectively neutral”. The fate of these mutations is governed essentially entirely by drift. By contrast, deleterious mutations that have large impacts on genetic fitness are eliminated very effectively by selection such that their frequencies in the population are maintained at nearly zero. Typically, there is a very broad transitional zone (representing several orders of magnitude of fitness effect), between the zone of highly effective selection and the zone of essentially no selection.

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### ... Selection threshold ... 3

- The mutations in this transition zone have been termed “nearly neutral”. We define the term ‘selection threshold’ as the absolute value of fitness effect at the midpoint of this transition region - wherein the allele frequency is precisely 50% of what it would be if there had been no selection. This means that above this threshold value in absolute fitness effect there is more than 50% elimination of deleterious mutations as a result of the selection process, while below this threshold value there is less than 50% elimination.

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### ... Selection threshold ... 4

- Typically, mutations more than an order of magnitude smaller in absolute fitness effect below the threshold are not significantly influenced by selection, while deleterious alleles more than an order of magnitude larger than the threshold are entirely eliminated by selection. For the case of favorable alleles, the same absolute threshold value tends to apply.

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### MENDEL Glossary ...

- **Mutation/selection chain.** In the real biological world, this is the chain of events that links a mutational event to a selection event. A single mutation affects a linkage block, which affects a chromosome, which affects a genotype, which affects a phenotype, which affects the reproductive fitness of an individual, which affects the actual transmission of a mutation into the next generation. There is biological noise at each link in this chain, and so each link of this chain is associated with an imperfect correlation.

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### ... Mutation/selection chain ... 2

- MENDEL is designed to accurately reflect this chain of events, so that a given mutant value actually has a limited effect on a linkage value, which has a limited effect on a chromosome value, which has a limited effect on a genotype value, which has a limited effect on a phenotype value, which has a limited effect on the reproductive fitness value of a given individual - which defines the actual transmission of the mutation. The strength of the correlation at each stage depends on the values chosen by the user for the various input parameters.

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### Now download “Mendel” ...

- Play
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