Mendelian speciation: part 1—what is the abundant source of significant biodiversity?

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Information in the genome required to produce new species (or phenotypes) must either mutate into existence or be already encoded as alternate genetic programs. Mendel demonstrated the latter to be correct. Three fundamental Mendelian principles explain how new species arise rapidly: latent information, exponential trait combinations, and loss of heterozygosity; and these neither require any new genes nor a multitude of mutations. Latent information can remain unexpressed through three mechanisms: dominance, epistasis and transposition. Upon suitable mating, latent traits can become visible in the progeny. Pre-existing genetic programs encoding alternative traits, shuffled at meiosis and maintained by reproductive isolation, is the only observed and empirically tested source of speciation in eukaryotes. Mendelian speciation can produce significant multi-feature phenotype change; a distinction typically displayed even by close species. It can achieve this rapidly, obviating the need for very long evolutionary time.

For many decades creation scientists have reflected on how so many species with so much biological variety could have arisen quickly; in particular, among the animals which descended from Noah's Ark. Old-Earth creationists have been heavily influenced by evolutionist beliefs on how new traits (and species) arise, and these mechanisms seem to require millions of years. The mistake has been to assume Adam and Eve (along with other organisms) were created without internal genetic diversity, their phenotype fixed, and endless random mutations were needed to produce change.[1]

As Carter pointed out, the notion of 'the fixity of species' is a relic of Aristotle's influence, which Darwin wrongly assumed the Bible taught.[2] Young Earth Creationists (YECs) agree that "organisms are designed to vary" as Williams phrased it.[3] This consensus view was well-expressed by YEC researcher Borger, who wrote, "life on Earth thrived due to frontloaded baranomes—pluripotent, undifferentiated genomes with an intrinsic ability for rapid adaptation and speciation."[4–7]

In a series of papers, Carter documented various mechanisms YEC scientists have been evaluating for the origin of genetic variability.[2,8,9] These include: distinct germ cells in sexually reproducing, created organisms; a variety of individual animals within a created 'kind'; heterozygous alleles; recombination to split alleles in the new chromosomes formed; retrotransposons which modified gene expression; gene gain and loss (e.g. for bacteria); and various forms of epigenetic inheritance (which includes imprinting).

Williams noted that biological information, which guides all aspects of an organism's life processes, must reflect the intention of the Creator. Species-level DNA-encoded differences can include point mutations, crossing over, indels, transpositions, jumping genes, and any other enzyme mediated process.[3]

In this 3-part series, we refer to some of the sources of biological diversity mentioned above. The short-coming in all of these is clearly *microspeciation*: cause DNA change in a fruit fly, it remains a fruit fly. However, speciation is typically far greater than this, involving the expression of *multiple significant traits simultaneously*. We examine how Mendel's findings provide an explanation for such holistic genetic change, and how this can occur rapidly.[10]

Mendel's seminal insights: the most promising explanation for rapid speciation

Where does biodiversity come from? How can diversity arise within genetic families or basic types? Could new species have arisen quickly following the Genesis Flood? Gregor Mendel, famed for his laws of inheritance, discovered a solution whose significance has been underestimated. Observations of rapid speciation are strong indicators that speciation does not result from Darwin's natural selection and mutations, but rather as a result of Mendel's law of exponential trait combinations[11] employing pre-existing genetic programs.

The question of how speciation occurs, i.e. the splitting of one species into two or more daughter species, is one of the central questions in biology. Charles Darwin's epoch-making work, *On the Origin of Species*, addresses this question explicitly in its title.[12]

According to the Modern Synthesis of Evolution, species are said to have diverged and fragmented by the gradual accumulation of many mutations; populations being separated geographically, and differential selection acting on the separated subpopulations. Eventually, over time, reproductive barriers are said to have arisen (genetic isolation). It also includes the possibility of gradual sympatric speciation. On

this basis, large periods of time for the formation of new species are claimed to be necessary. This is now mainstream evolutionary biology.

A contemporary of Darwin's, the Augustinian friar Gregor Mendel, considered the father of modern genetics, had a very different take on the origin of species. It is based on pre-existing diversity encoded in genetic programs which can produce speciation within a few generations; biological novelties do not need to arise via lengthy mutation–selection cycles.

Mendel's work provides a clear understanding for how species can appear within genetic families. Mendel's concept—Mendelian speciation—will be presented in this 3-part series and compared to the mainstream evolutionist view. Crompton has drawn attention to this topic for many years,[13–16] directly communicating with members of the US creationist community, during the mid-2000s. Much of the content in this current series has also been discussed, since 2004,[17] during conferences sponsored by the German creation science organization Wort und Wissen.

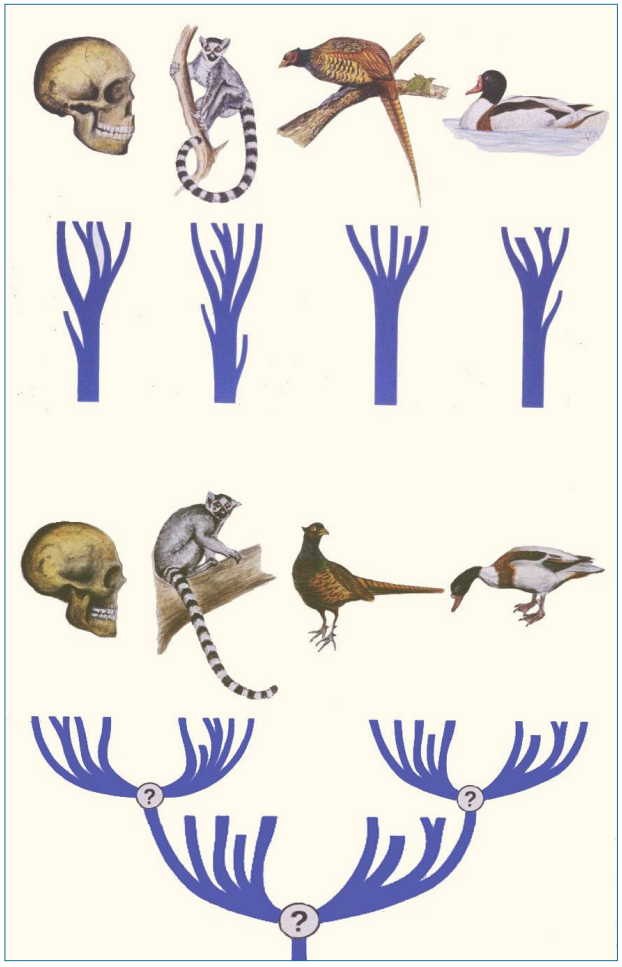


Figure 1. *Cis*-evolution versus *trans*-evolution. Top: *Cis*-evolution. Each independent family is shaped like a tree, displaying *cis*-evolution. The branches (species) are kept apart by reproductive isolation, but may occasionally reunite by sporadic hybridization. Such diversification is proposed to result from pre-existing genetic programs and Mendelian speciation. Bottom: *Trans*-evolution. All families are claimed to be ancestrally linked, and these links are claimed to have arisen through mutations and selection. From Junker and Scherer with permission.[23]

Where does the biological diversity in nature come from?

Why are lions and tigers similar and yet significantly different? Why are Red Admiral and Painted Lady butterflies similar and yet significantly different? Why are wolves relatively homogeneous as a species and yet dogs, their artificially bred offspring, represent such a plethora of different breeds? Why can some organisms produce a wide variety of new species when colonizing sea islands, yet others do not? Explanation of speciation must be able to go beyond limited biological change, restricted to traits or genes; and must encompass significant biological change, affecting many features of an organism.

Darwin was a passionate biologist and claimed that natural selection was the means by which new species arose. Many today believe he solved the mystery of speciation. Before Darwin, most scientists attributed biodiversity to divine agency, but afterwards the credit was given to natural selection. A fundamental shift in scientific thinking resulted which ignored any divine contribution and required eons of time. In fact, Darwin's premise was not correct. Selection, whether natural or through deliberate breeding is not the true mechanism that produces new characters or new species. Only after the phenotype of a novel species has appeared (i.e. after the origin of species) can natural selection act to favour the new phenotype, which should be fitter.

In 1905 de Vries (one of the fathers of mutation theory) recognized that

> "… natural selection may explain the survival of the fittest, but it cannot explain the arrival of the fittest."[18]

Professor Andreas Wagner at the University of Zurich used this insight to write a book, *Arrival of the Fittest*.[19] He noted correctly:

> "And if we do not know what explains its arrival, then we do not understand the very origins of life's diversity."[20]

Remarkably, considering the subtitle of his book, *Solving Evolution's Greatest Puzzle*, Wagner did not offer any new explanation for the origin of novel phenotypes either.[21]

Natural selection, it is claimed, produces *de novo* novelty but through use of nebulous and misleading phrases like, '*caused by environmental pressures*' and '*convergent evolution*'.

Single common ancestor or multiple independent lineages?

The eukaryotic world is extremely diverse, encompassing millions of species.[22] It could be interpreted as a mono-arboreal single lineage tree of life or sylvan, characterized by a multi-lineage forest (see figure 1).

Both models assume an ancestral species, but they have very different views as to the 'how' new species arise and the magnitude of the changes. How species arise addresses what is arguably the most important question in biology: Are species fixed within certain limits beyond which they cannot change (leading to a forest-like phylogenetic pattern), or can species vary without limit (allowing all living things to have derived from a single organism)? It was a question discussed by Mendel.[24]

Mendel's sidelined experiments

Gregor Mendel (figure 2) investigated seven *characters* of the garden pea, including seed colour, seed shape, flower colour, pod colour, pod shape, stem length, and flower position.[10] Each of these characters was represented by two *traits*; for instance, seed colour can be either yellow or green, and seed shape can be either round or wrinkled. (The three terms: trait, character, and phenotype, are crucial to a correct understanding of Mendelian speciation. They are explained in the glossary).

Mendel's publication provided an explanation for the rapid origin and vast number of new species.[10,25] But his ground-breaking discoveries had no influence until the significance of his work became recognized thanks to three independent articles in the March, April, and June 1900 editions of the *Report of the German Botanical Society*. The authors were de Vries,[26] Correns,[27] and Tschermak.[28]

Correns recognized the significance of Mendel's publication clearly:

> "It hardly needs to be said that this behaviour is of importance for the question of whether hybrids can become species."[27]

Mendel had discovered the source of diversity in eukaryotic organisms. The underlying physiological process would later become known as meiotic recombination, and with this discovery Mendel had unravelled the mystery of the origin of (eukaryotic) species.

Mendel recognized the *Law of Exponential Trait Combinations* at least three times in his famous paper on plant hybrids.[11] It is the only proven mechanism able to generate (non-trivial) new species within eukaryotic families. Biology and genetics textbooks ascribe two, sometimes three, other laws to Mendel: the *uniformity rule*, *splitting rule* (figure 3), and *independence rule*, as discussed in the Appendix.

Figure 2. Gregor Johann Mendel (1822–1884), the father of modern genetics, was born in Heinzendorf, Schlesien (today Hynčice in the Czech Republic). He was an Augustinian friar (not a monk) in St. Thomas' Abbey, Brünn, Austria (now Brno, Czech Republic). He conducted his experiments with plant hybrids between 1856 and 1863, presented his findings at the Brünn Society for Natural Sciences in February and March of 1865, and published his famous paper in their proceedings journal in 1866.[10] (Courtesy of St. Thomas' Abbey, Brno)

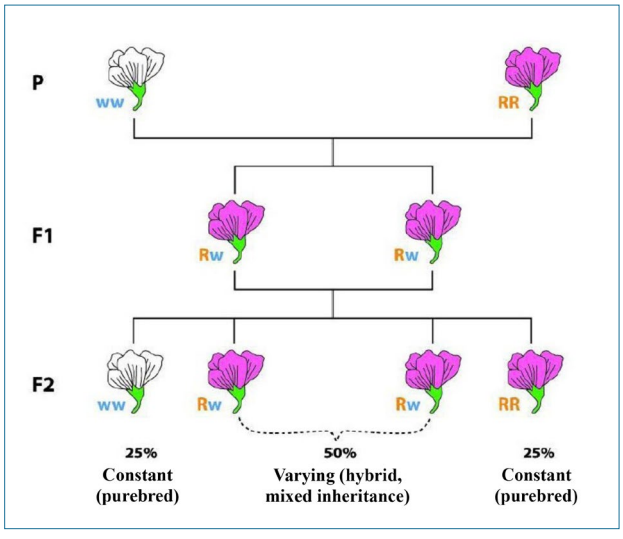


Figure 3. Illustration of the uniformity rule (at generation F1) and the splitting rule (at generation F2) in monohybrid dominant-recessive inheritance. Below: difference between constant (homozygous, purebred) and varying (heterozygous, mixed inheritance) offspring.

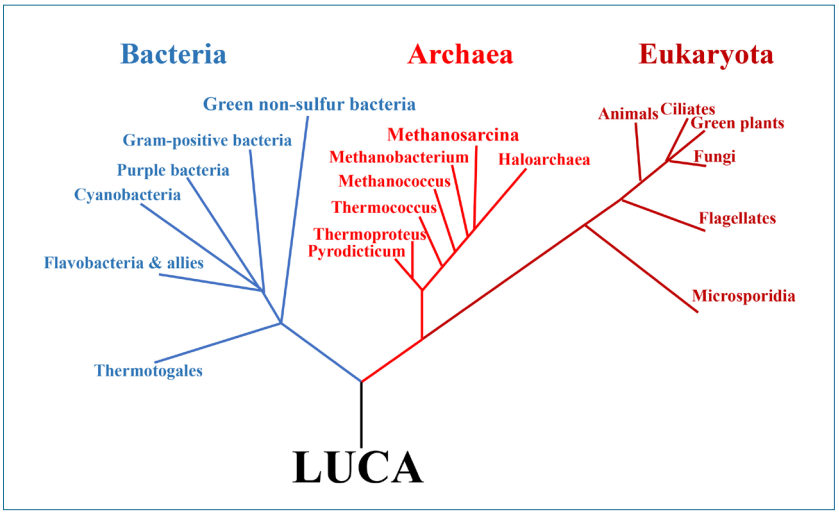
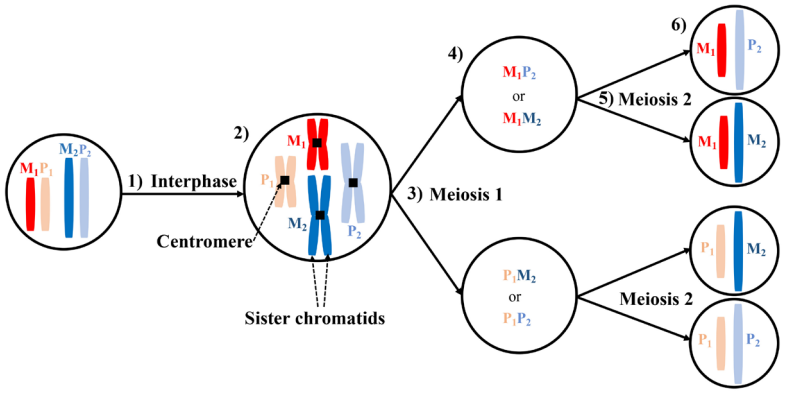


Figure 4. High-level tree of life rooted on a Last Universal Common Ancestor (LUCA), according to Woese *et al.*[31]



M_1 = Maternal Chromosome 1; P_1 = Paternal Chromosome 1;
M_2 = Maternal Chromosome 2: P_2 = Paternal Chromosome 2
1) DNA duplicates and each chromosome creates a copy of itself.
2) The chromosomes line up at the equator in pairs, one maternal one paternal.
3) First division. Maternal and paternal chromosomes are randomly distributed into the new cells.
4) The chromosomes line up at the equator in both cells.
5) Second division for both cells. The chromatids are separated.
6) Haploid (one chromosome copy) daughter gametes are made.

Figure 5. During meiosis, gametes (sex cells) are formed. The gamete mother cells are diploid. They have two sets of DNA, one from the maternal and one from the paternal parent. After meiosis 1 at 4), both daughter cells inherit, in a random manner, one chromosome from each chromosome pair, each having two chromatids. After meiosis 2, the four gametes inherit those same chromosomes, but each now has one chromatid. The gametes are haploid and inherit just one set of chromosomes. During gamete formation, the DNA is recombined in segments, each segment being inherited either from the original maternal or the original paternal DNA. At prophase 1, chromosome segments; at metaphase 1, whole chromosomes are exchanged. For a more detailed explanation, see Meiosis—Khan Academy. Exchange of DNA segments (cross-over) at prophase 1 is not displayed in the figure.[33] Figure by R. Truman. Original diagram based on Meiosis: Its Stages—Study Mind.

Using current knowledge of molecular biology, we call these rules Mendel's Law of Segregation (rule 1 and 2) and Mendel's Law of Independent Assortment (rule 3). Mendel's research provided the evidence for these two laws, but he never formulated them explicitly. The second law is actually deficient; the possibility of gene linkage only realized later by geneticists.

Mendel demonstrated that combining independent alternate traits could produce an exponential number of phenotypes (different plants). This was pointed out in an excellent review by Ellis *et al.*, who noted that Mendel's emphasis was on speciation, the transformation of one species into another.[29] Mendel's Law of Exponential Trait Combinations confirms that much information is already encoded in genomes. This fact is sufficient to explain most of the diversity observed within eukaryotic families.

Darwin's *On the Origin of Species by Natural Selection* was published in 1859, while Mendel was simultaneously conducting his experiments (1854–1865). Mendel had a copy of the 1863 first German translation of Darwin's book and understood that Darwin was suggesting all life could have arisen from a single common ancestor.[30] A modern version of Darwin's view is shown in figure 4.[31]

Based on his results, and discussions with others involved in breeding plants, Mendel thought there were many independent lines of descent in nature. However, as a good scientist, he recognized the limited sample coverage of his experiments; mostly with the Garden Pea, *Pisum sativum*; a few with hybrids of two Common Bean varieties, *Phaseolus vulgaris* and *P. vulgaris nanus;* and a few with hybrids of two separate species, the Common and Runner Beans, *P. vulagris* and *P. coccineus*; but was unable to interest others to experiment with other organisms. He was therefore cautious about overgeneralizing his results (the

cautionary approach being a good practice many origin-of-life researchers are encouraged to imitate).

Fundamental principles in Mendelian speciation

Mendel's experiments on seven plant characters reveal three fundamental principles that together result in Mendelian speciation:

- I. Latent information
- II. The law of exponential trait combinations
- III. Loss of heterozygosity.

I. Latent information

Mendel deduced the presence of latent information during his experiments with hybrids when introducing the terms '*dominant*' and '*recessive*' expression.[32] He called a plant trait recessive if it was not expressed for a generation but remained potentially available to be expressed in subsequent generations.

As will be elaborated on in part 2 of this series, the genetic information is held in a latent state by at least three mechanisms:

- dominance
- epistasis
- transposition.

II. The Law of Exponential Trait Combinations

Mendel's studies of plant hybrids revealed a satisfactory explanation for how novel trait combinations arise. The mechanism at work in eukaryotes is now known as meiotic recombination, as shown in figure 5.[33]

Pairs of the same chromosome (one paternal, one maternal) are called 'homologous chromosomes'. The Law of Independent Assortment states that during meiosis all genes are randomly distributed among the four daughter cells; see stage 6) of figure 5.

Meiosis will be discussed further in part 2 of this series. Non-meiotic organisms such as prokaryotes use other mechanisms to exchange genetic information. For example, plasmids and viruses provide a reservoir of genes that can be transferred to prokaryotes,[34] and antimicrobial resistance can be conferred by viral genes.[35] There are alternative methods to effectively transfer genes between prokaryotes.[36] However, eukaryotes undergo Mendelian speciation.

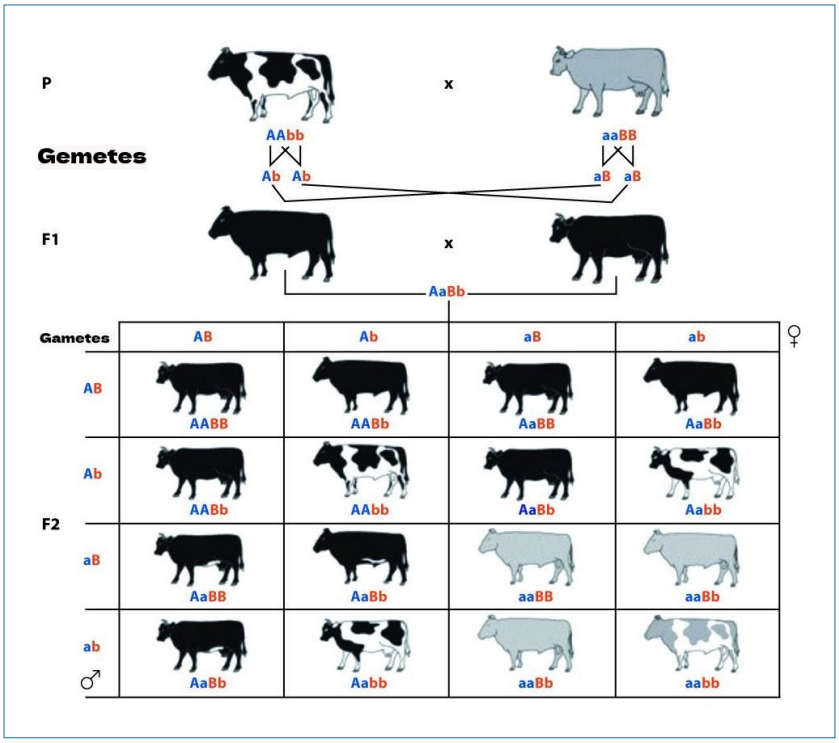


Figure 6. Illustration of the uniformity rule (at generation F1) and the splitting rule (at generation F2) for dihybrid (doubly heterozygous) inheritance. Single-colour (solid) is dominant over spotted; black is dominant over grey. In the F1 generation, the recessive traits (for grey and piebald) are hidden (latent).

Using the Law of Exponential Trait Combinations, Mendel was able to predict the expected ratio of genotypes in monohybrid, dihybrid, and trihybrid crosses. He discovered the 3:1 phenotype ratio of dominant to recessive trait expression (figure 3). But Mendel's research went even further. He recognized that the dominant phenotype could be either constant (homozygous) or varying (heterozygous). He showed that, when heterozygous, progeny could display dominant or recessive traits; but, when homozygous, progeny would only ever display that one trait. Today we understand this is because genes are present as two copies, called 'alleles' (one from each parent). If the alleles are identical (the gene is homozygous) the progeny can only inherit one allele and so can display only one trait; however, if the alleles differ (the gene is heterozygous), the progeny can display either of the traits, depending on which pair of alleles they inherited.

Mendel showed that the dihybrid genotype (two heterozygous genes) leads to 9 unique genotypes with a relative proportion of 1:1:1:1:2:2:2:2:4 (viewing a Punnett square from top left to bottom right, see figure 6, this ratio can be written 1:2:1:2:4:2:1:2:1) as shown in figure 6. In genetics textbooks, the ratio typically described is 9:3:3:1, referring to the phenotypes that are seen, because the genotypes are usually unseen.

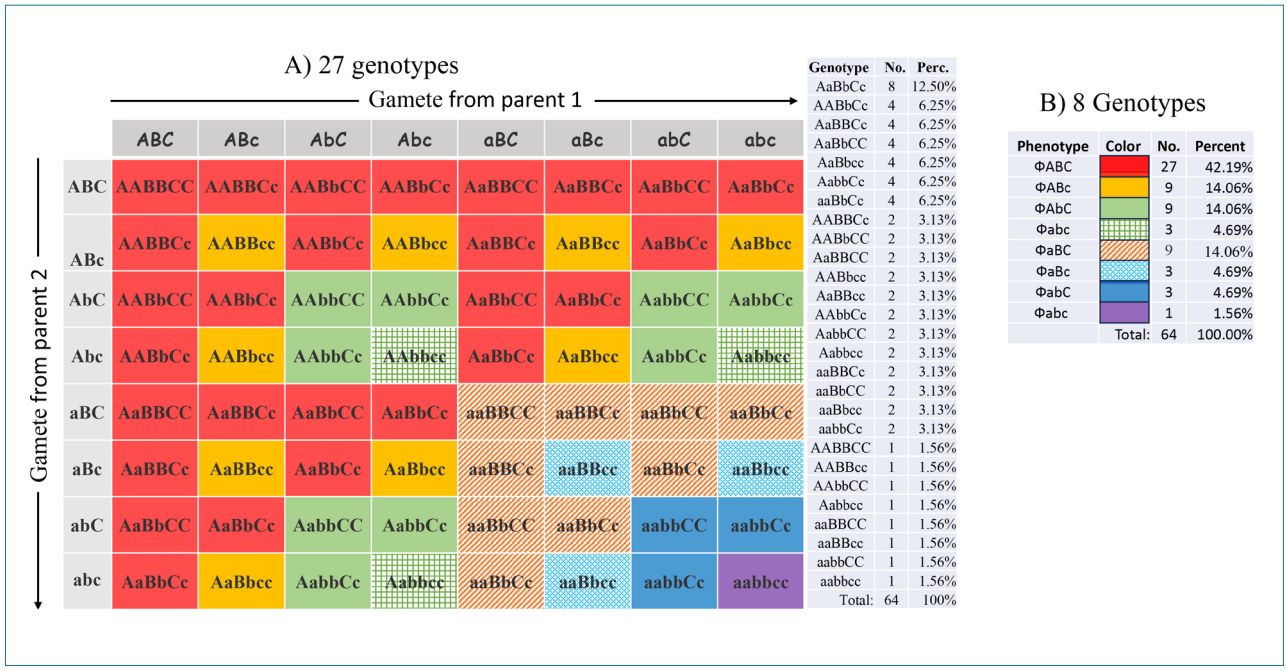


Image: Redrawn by R. Truman from information generated by the Punnett Square Calculator (Punnett Square Calculator - Idealcalculator.com)

Figure 7. Punnett square with three genes showing the possible outcomes of a trihybrid self-cross. None of these traits are linked. A) 27 different genotypes (letter combinations) can form. The 8 unique phenotypes are shown in different colours. B) The frequency of the 8 different phenotypes (f). Capital letters are used to designate a dominant allele, which dictates the phenotype.

The British biologist Reginald Punnett developed a compact method to determine the phenotype and genotype ratios in breeding experiments, known as the Punnett square. If drawn correctly, these squares display a high degree of internal symmetry that is revealed in the patterns of phenotype and genotype ratios. Figure 7 shows the Punnett square revealing the trihybrid (triply heterozygous) self-cross.[37]

Figure 7 shows the 8 (= 2^3) distinct phenotypes which can form, and their expected frequencies. The 27 (= 3^3) unique genotypes are also labelled. Note that there are as many unique gametes as unique phenotypes. The leading diagonal (top left to bottom right) reveals each of the 8 possible fixed phenotypes, which are the purely homozygous states; Mendel's 'constant' plants. All other genotypes are heterozygous; Mendel's 'varying'plants. The trailing diagonal (top right to bottom left) reveals the pan-heterozygous genotypes, which are able to generate any of the trait combinations. The pan-heterozygous plants possess the maximum phenotype information. Mendel's 'constant' and 'varying' plants are now referred to as 'pure-breeding' and 'hybrid' plants.

That two traits exist for each of the 3 characters explains the 8 (2^3) different phenotypes and the 27 (3^3) different genotypes observed by Mendel. Even though some phenotypes (trait combinations) look the same, Mendel recognized that dominant traits could be either 'constant' or 'varying'; however, recessive traits are always 'constant'.

Mendel demonstrated experimentally that hybrid organisms followed his law of exponential trait combinations. Diversity increases exponentially with the number of characters. Plants possessing *n* (hybrid) characters can generate 2^n different phenotypes and 3^n different genotypes.[38] *As a side-note, 2n phenotypes arise when dominance is complete. If dominance is incomplete, intermediate phenotypes arise in the heterozygotes. The classic example of this is homozygous red and white carnations, and heterozygous pink carnations. In cases of co-dominance, double phenotypes arise in the heterozygotes. The classic example of this is homozygous blood groups A and B, and heterozygous blood group AB. In these circumstances more than 2n different phenotypes can arise. However, during speciation, heterozygosity is typically lost, and these additional phenotypes will also be lost.* Mendel examined seven varying characters and eventually observed all 128 (= 2^7) unique phenotypes. Since his law is mathematically exponential, it readily explains the emergence of a multitude of phenotypes. Just 10 varying characters (heterozygous genes) can produce 2^{10} >1,000 different homozygous phenotypes; 20 varying traits (heterozygous genes) 2^{20} >1,000,000 different homozygous (pure-breeding) phenotypes. This is where the various species, and the diversity of nature, have their primary origin.

III. Loss of heterozygosity

The third principle described by Mendel is a trend towards fixed, homozygous phenotypes, which is referred to as the loss of heterozygosity. This means that eventually only one trait of each character is displayed by all individuals in a subpopulation. This is crucial to the production of new species. Loss of heterozygosity's vital biological role in speciation often goes completely unappreciated, even by Mendel's biographers,[39] despite Mendel referring to the observation as a developmental law, 'Entwicklungsgesetz'.

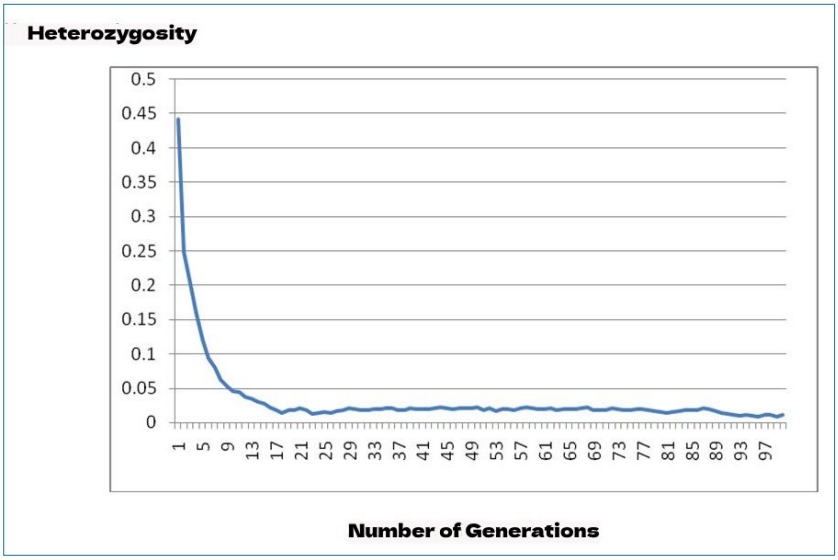


Figure 8. Proportion of heterozygous genes plotted against the number of generations in a typical population using a Monte Carlo simulation. Loss of heterozygosity, which corresponds to complete adaptive radiation of a genetic family with many new species and genera, took less than twenty generations. In this simulation, organisms were at liberty to occasionally interbreed, unlike Mendel's peas, which self-pollinated.

Mendel was familiar with the work of various other plant breeders such as German professors Kölreuter[40] and Gärtner.[41] They had observed that hybrids have a tendency to revert to the parent (pure-breeding) forms. Meiosis, shown in figure 5, is the reason for the loss of heterozygosity and for the well-documented reversion to pure breeds.

Suppose that trait *A* is dominant and trait *a* is recessive, and both parents carry both alleles. This can produce the combinations AA, Aa, aA, and aa in their offspring. Since traits Aa and aA are phenotypically equivalent, it leads to the expected genotypic ratio of 1:2:1. In this example, although both parents were heterozygous, only half of the children are. The other half are homozygous; either homozygous dominant or homozygous recessive. In the next generation, those 50% homozygous children give rise to only homozygous grandchildren; they are said to be 'fixed'. However, the 50% heterozygous children will once again give rise to half heterozygous grandchildren (25%) and to half homozygous grandchildren (25%). This is an unwavering feature of meiosis; each generation loses 50% of whatever heterozygosity the parents possessed, and the remaining children are homozygous. It is an important finding, first numerically explained by Mendel.[42]

If one starts with a situation where any individual can cross with any other individual, when left to naturally reproduce, every successive generation will retain the 1:2:1 genotype ratio; with its high proportion of heterozygotes. However, Mendel worked with Garden peas, which self-cross (self-fertilize). This is a crucial difference. In this situation, the ratio quickly changes. The starting, or P, generation are all hybrids; the F1 generation displays the genotype ratio 1:2:1; the F2 generation, the ratio 3:2:3; the F3 generation, the ratio 7:2:7; and the F5 generation, the ratio 15:2:15. The outer two values are the homozygous-dominant and homozygous-recessive values, and the central value is for the heterozygotes. Within just a few generations the homozygous genotypes rule the roost. Of course, as with Mendel's peas, this assumes strict reproductive isolation.

The two-character (gene) case is much more complex, because there are increasingly more genotypes. The P generation begins with the dihybrid, AaBb. The F1 generation has the genotype ratio 1:2:1:2:4:2:1:2:1 and genotypes AABB, AABb, AAbb, AaBB, AaBb, Aabb, aaBB, aaBb, aabb. The F2 generation has the genotype ratio 9:6:9:6:4:6:9:6:9; the F3 generation, the genotype ratio 49:14:49:14:4:14:49:14:49; and the F4 generation, the genotype ratio 225:30:225:30:4:30:225:30:225. The '4' in the middle indicates the dihybrids, which get exponentially overwhelmed by the homozygotes (both the single homozygotes and especially the double homozygotes) at each successive generation. The two-gene self-cross overwhelms its hybrids faster than the single-gene self-cross. The three-gene self-cross is faster still. For a population to retain significant heterozygosity, it must have mechanisms in place to avoid self-crossing (reproductive isolation). Where this is not the case, populations quickly become homozygous, a situation that promotes speciation.

How quickly is heterozygosity lost in a population? Using an example based on one character (gene), Mendel predicted that within 10 generations a pan-heterozygous starting population (i.e. all genes fully heterozygous) would become

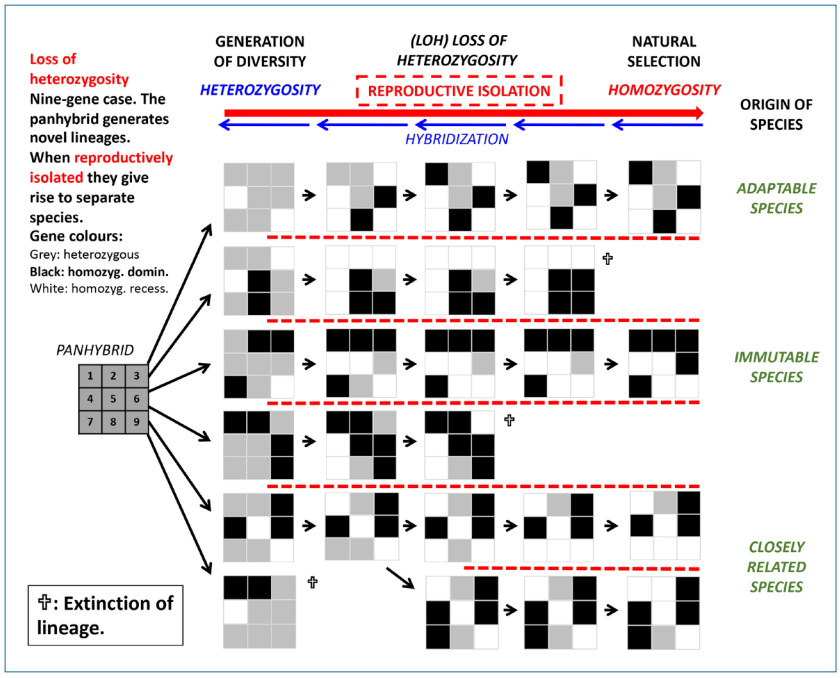


Figure 9. Example of potential evolution of a basic type using nine characters (see text for details). Graphic from N. Crompton.[14]

constant (homozygous), 50% dominant and 50% recessive; any hybrids would be reduced more than 1,000 fold.[43]

Buri studied 107 populations of (single gene) heterozygous fruit flies. After generation 6, populations appeared that were 100% homozygous.[44] By generation 19, almost all the populations were either 100% homozygous dominant or 100% homozygous recessive. Populations with more than one heterozygous gene show an equally rapid loss of heterozygosity.

Usually, species differ in more than one gene. Mendel discusses this in his paper.[45] He refers to workers with 'sharp definitions' of the term 'species', where even a single character difference is considered sufficient to confer species status, e.g. *P. quadratum* are pure-breeding garden peas with wrinkled seeds; *P. saccharatum* are pure-breeding garden peas with constricted pods; and *P. umbellatum* are pure-breeding garden peas with flower heads at the end of their stems. However, Mendel and most of his contemporaries preferred to refer to such groups as 'varieties'.

The Oxford definition of a variety is "a group of individuals that differ distinctly from but can interbreed with other varieties of the same species. The characteristics of a variety are genetically inherited."[46] Because Mendelian speciation employs meiosis, all the genes of an organism (not just one or a few) are involved; and this typically results in sister species varying in many characters.

Meiosis is a very different situation to that which results from the mechanisms usually proposed as causes of speciation. Alternative mechanisms of genetic change based on mutations, or even transpositions, cause limited phenotypic change to organisms. If one mutates a fruit fly, it remains a fruit fly. If one triggers a transposition event in a peppered moth or a garden pea, you still have a peppered moth and a garden pea. No significant speciation has occurred. One's organism simply displays a new trait or so. Mendelian speciation, however, is based on meiosis and therefore global genomic change. It can account for comprehensive phenotypic change, which readily accounts for new species.

We programmed a Monte Carlo mathematical model of populations based on 25 heterozygous genes and found that within about 19 generations almost all the genes had become either homozygous dominant or recessive, as shown in figure 8. The reason the curve levels off 'above zero' in the simulation (after 20 generations) is that the organisms occasionally interbreed (not so with Mendel's peas, which, after the first hybrid cross, only ever self-pollinate and which would quickly tend to zero).

The description above illustrates the fundamental processes at work. But natural populations are far more complex than the simplifying assumptions used for the model. For example: populations can be of different sizes; subpopulations may be only partially isolated; individuals carrying rare alleles can live beyond a single breeding cycle, passing it on through multiple generations. To more accurately model the richness of natural processes, we programmed the Monte Carlo simulation with several semi-isolated populations.

The model allows occasional random hybridization between groups (populations). The separated groups become, as expected, nearly pan-homozygous, but each with a different combination of alleles. The separate groups, therefore, form sources of new alleles for neighbouring groups whenever hybridization between the two occurs. Typically, these alleles persist briefly in the new population, but are lost again within a few breeding cycles. A more complete description of our models and their predictions is in a manuscript in preparation.

Perspectives

The second paper of this three-part series takes a closer look at how genetic information is kept latent in

chromosomes. Dominance, epistasis, and transposition are all means of achieving this. A pan-heterozygous ancestor can hold a large pool of latent traits, sufficient to help produce all the existing species in a family of organisms. This completely circumvents the need for any new genes or for a vast number of random mutations.

The third paper of this three-part series takes a closer look at the crucial role of reproductive isolation in speciation. If progeny can freely mate among themselves, a single, highly variable species arises. However, if reproductive isolation interferes with this, and progeny are constrained in their mating; groups of unique, less variable, separate species arise. This mechanism of speciation can occur within tens of generations. Under appropriate conditions, the process gives rise to a species swarm, an adaptive radiation.

These processes provide a comprehensible and testable mechanism for rapid speciation based on pre-existing genetic programs, unlike Darwinian processes. Figure 9 illustrates how diversity can develop from a pan-heterozygous founding pair, in which all characters are initially heterozygous (i.e. both traits are present) under self-crossing conditions, and how different species arise.

In figure 9, population or species genotypes are represented as 3 × 3 matrices. Each column of 3 × 3 matrices represents a consecutive generation and each row an evolutionary lineage. Each cell within a matrix represents one character: black = homozygous dominant; grey = heterozygous; white = homozygous recessive. Each grey cell will express the dominant phenotype, but can pass on a recessive allele to the next generation.

The evolution of traits can be viewed as a stochastic process. Because subpopulations are reproductively isolated (dashed lines), homozygous characters (black or white) remain permanently fixed. The creation of alternative combinations of fixed traits (separate species) through the loss of heterozygosity is an ongoing process.

The loss of heterozygosity leads to unique, unvarying constellations of homozygous-dominant and recessive traits, thereby creating new species. Figure 9 indicates some populations (species) become extinct for various reasons (✞). The last column represents extant populations or species. Most have retained some heterozygosity and therefore the potential for diversification. One species (3rd row) is fully homozygous. As such, it is an immutable species (lineage 3); like the cheetah or the northern elephant-seal. Only hybridization with a sister lineage (species) can restore heterozygosity, but with this comes the potential to form new species (see right-to-left pointing (blue) arrows).

Hybridization with other lineages has the potential to partially restore the original heterozygosity. In figure 9, some heterozygosity is still observed in the final generation of many of the lineages. A recent branching event is illustrated in the two lowest lineages, and the offspring remain very similar. Hybridization between these two would restore the phenotype of their most recent common ancestor, but heterozygosity would be present only in characters 2, 7, and 8.

In the scenario shown in figure 9, no hybridization between existing lineages (populations or species) can restore the dominant trait of gene 9, or the recessive trait of gene 6, since the necessary genomic information has been lost from all of the various lineages.

Concluding remarks

Mendel's experiments demonstrated how new phenotypes could become manifest, and quite rapidly, due to pre-existing genomic capabilities. Individual phenotypes are rapidly fixed in small, reproductively isolated subpopulations. New genes are not required for this process, nor multiple beneficial mutation events, nor long time periods.

These insights are profoundly relevant today to understand the genetic basis of rapid adaptability and adaptive radiations. For young-earth creationists, Mendelian speciation offers an empirically sound explanation for rapid speciation.

We propose that our Creator-God originally endowed basic kinds (genetic families) with genetic programs able to activate latent traits already encoded in the DNA relatively rapidly, through a variety of mechanisms, including loss of heterozygosity, but not all of the details of this process can be satisfactorily discussed, even in a three-part series.[47]

Glossary

Allele: variant of the same gene, having a unique nucleotide sequence.

Character: particular feature of an organism, typically produced by one or more genes. Example: flower colour (the traits could then be, for example, purple or white).

Dihybrid: mixture of two characters. The individual is heterozygous at two different genes.

Dominant: An allele is dominant if it suppresses the trait caused by the other allele.

Epistasis: One (epistatic) gene masks the effects of a different (hypostatic) gene. Instead of the expected two traits, only one is observed. *Dominant epistasis* occurs when the dominant allele of the epistatic gene masks the effects of the hypostatic gene. *Recessive epistasis* occurs when the presence of both recessive alleles of the epistatic gene masks the effects of the hypostatic gene.

Eukaryotes: organisms with cells having a nucleus. They are normally capable of meiosis.

Expression (gene expression): making usable the information encoded on a gene.

Genetic family: biological family whose member species possess different combinations of dominant and recessive traits, and the pre-existing genetic programs that code for them.

Genome: all of the DNA present in a cell.

Genotype: an organism's set of alleles at one, several, or all its genes.

Heterozygous: when different alleles of a gene are present in an organism.

Homozygous: when the same alleles of a gene are present in an organism.

Hybrid: of mixed parentage.

Isolation: separation of subpopulations and suppression of gene exchange outside the group.

Latent: hidden, concealed (here: in the genome). A trait or trait state is latent if it is genetically present but not phenotypically expressed.

Meiosis: the cellular process of chromosome exchange and inheritance by gametes (germ or sex cells).

Monohybrid inheritance: when a heritable character is controlled by a single gene.

Mutation: change in DNA, including: replacing nucleotides, deletions, insertions, and rearrangements.

Phenotype: the external appearance, relating to morphology, anatomy or physiology. It is used in two senses in this three-part series. 1. The sum of the observed traits. 2. The observed species, which is simply the sum of its traits.

Pre-existing genetic program: genetic information encoded in the chromosomes. The information codes for proteins, and regulation of their expression, which interact to produce the alternative traits of any given phenotypic character. The term 'pre-existing' refers to the fact that this information was present, prior to any speciation event; and it has been passed down through successive generations to the present.

Recessive: when the effect of a trait or an allele is suppressed by the presence of another (dominant) trait or allele.

Recombination: mixing of DNA segments or homologous chromosomes obtained from the parents during gamete formation.

Speciation: process by which a species splits into two daughter species.

Trait: the actual observed phenotype of a character. If the character of interest is flower position, then its traits might be terminal or axial. Typically, a trait is coded for by one specific allele of a gene.

Trihybrid: comprised of three characters. The individual is heterozygous at three different genes.

Zygote: the cell of eukaryotes formed after the fusion of an egg and sperm.

Appendix: Various formulations of Mendel's Laws

Various laws have been attributed to Mendel which he did not actually formulate. Genetics textbooks in the English-speaking world generally have the following two (three) laws.

- Mendel's first law, *The Law of Segregation*, states that traits (or alleles) are randomly passed to the next generation due to meiosis.
- Mendel's second law, *The Law of Independent Assortment,* states that the genes are independently passed (sorted) to the next generation at meiosis. However, when multiple genes necessary to produce a character are physically nearby on the same chromosome, they could be 'linked' and thereby not truly independent.
- Mendel's third law, *The Law of Dominance,* is only rarely cited. It states that dominant traits (caused by alleles) mask (or hide) recessive traits.

German textbooks on genetics usually mention the following three rules or laws:

- Rule 1, the *uniformity rule* states that parents who are differently homozygous (e.g. one has aa and the other AA alleles) produce uniform heterozygous offspring.
- Rule 2, the *splitting rule*, states that parents who are equally heterozygous (e.g. Aa and Aa) split in the offspring both phenotypically and genotypically.
- Rule 3, the *independence rule*, corresponds to the Law of Independent Assortment, above.

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