Different characteristics will be **advantageous** on each side so the **allele frequency** will change in each population

- Frequency of the **advantageous** allele will **increase** on one side of the barrier

Mutations will take place **independently** in each population, changing the **allele frequencies** and **phenotype frequencies.** The **longer** the groups are isolated, the **more allele frequency change**, increasing the chance of speciation.

Eventually the different populations will have become **genetically distinct** and **won't** be able to **breed** or produce **fertile** offspring = ***reproductively isolated.***

They are now a separate species.

HARDY-WEINBERG EQUATION

Evolution is a **change** in allele frequency. **New alleles** are usually generated by **mutations** in **genes**.

the H-W equation are used to estimate the **frequency** of particular **alleles, phenotypes** and **genotypes** in a population.

The frequency of **all possible alleles** is **1** so the **individual** alleles must **add up to 1.**

frequency of homozygous dominant individuals	+	frequency of heterozygous individuals	+	frequency of homozygous recessive individuals	= 1
p^2		$2pq$		q^2	

H-W predicts that allele frequencies **won't change** from one generation to the next but this is only true under **certain conditions:**

- **Large** population
- **No** immigration, emigration, mutations or natural selection
- **Random** mating

If allele frequency **does change** then all of these have happened.

When a gene has two alleles, you can figure out the frequency of one of the alleles of the gene if you know the frequency of the other allele, using this equation:

$p + q = 1$

Where: p = the frequency of the **dominant** allele
q = the frequency of the **recessive** allele

Make sure you learn this equation — you might not get given it in the exams.

The total frequency of all possible alleles for a characteristic in a certain population is 1.0. So the frequencies of the individual alleles (e.g. the dominant one and the recessive one) must add up to 1.0.

E.g. a species of plant has either **red** or **white** flowers. Allele **R** (red) is **dominant** and allele **r** (white) is **recessive**. If the frequency of **R** is **0.4**, then the frequency of **r** is: 1 – 0.4 = **0.6**.

... and to *Predict Genotype and Phenotype Frequency*

You can figure out the frequency of one genotype if you know the frequencies of the others, using this equation:

$p^2 + 2pq + q^2 = 1$

Where: p^2 = the frequency of the homozygous dominant genotype
$2pq$ = the frequency of the heterozygous genotype
q^2 = the frequency of the homozygous recessive genotype

The total frequency of all possible genotypes for one characteristic in a certain population is 1.0.
So the frequencies of the individual genotypes must add up to 1.0.

E.g. if there are two alleles for flower colour (R and r), there are three possible genotypes — RR, Rr and rr.
If the frequency of genotype RR (p^2) is 0.34 and the frequency of genotype Rr ($2pq$) is 0.27,
the frequency of genotype rr (q^2) must be: 1 – 0.34 – 0.27 = **0.39**.

Genotype frequencies can then be used to work out phenotype frequencies (the frequencies of observable traits).

E.g. the frequency of red flowers is equal to the genotype frequencies of RR and Rr added together (0.34 + 0.27 = **0.61**) and the frequency of white flowers is equal to the genotype frequency of rr (**0.39**).

Processing new findings

- Findings are reported in a **scientific journal, conference papers** or the **internet.**
- Scientists can comment on **data, methods** and the **interpretations**
- **Peer review** detects **invalid claims** and **adds weight** to valid ones

Woese proposed a new category of organisms- **Archaea** which survive in **anaerobic environments**, *e.g. hot springs or salty lakes.* Many **ignored** this idea but more papers were published by other scientists and there were **conferences** held. Woese proposed the phylogenetic tree above based on **three domains** (Archaea, Bacteria and Eukaryota).

- Organisms in each of the **three domains** contain **RNA sequencing** that is **unique** to their domain.

Biodiversity within a **species**

Each individual has a **unique combination** of **alleles** = variety of genotypes = **genetic diversity** which allows the population to **adapt** to changing conditions and so should be **conserved**. If the **population declines** some alleles may be **lost** and the genetic diversity **decreases**.

Random mutationscan create genetic variation by changing the **base sequence of DNA** in the cells of an organisms, creating **new alleles** which increases the **gene pool.**E.g:

- Single point mutation (alteration of **one base**) resulting in **sickle cell anaemia**
- **Deletion** of **3 nucleotides** resulting in **cystic fibrosis**

Mutations can have **no effect** on phenotype, have **harmful effects** or have **benefits** e.g. mutations in **houseflies** making them **resistant** to **pesticide.**

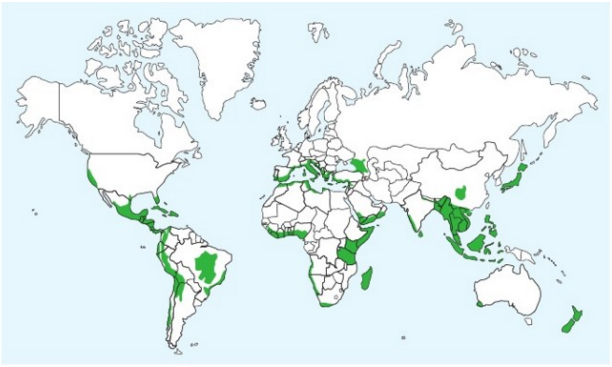
Genetic diversity can be **visible** through **phenotypes** but can also be measured by **species richness** and **species evenness.**

Species evenness:

- Takes **no account** of **population size** of each species
- A community in which most species have **similar abundances** has **high evenness**

A very **common species** in a habitat is sometimes called the **dominant organism.** In **species-rich** ecosystems, such as **rainforests,** habitats will show **more even** species composition.

Biodiversity hotspot



Areas with **particularly high biodiversity.** The idea was adopted as a way of **focusing conservation effort** on the most critical places.

Measuring biodiversity

A **direct** method is **DNA sequencing** to determine the **bases** in a segment of DNA to determine **which alleles** are present.

DNA can be cut into **fragments** and then **separated** using **electrophoresis**. Different alleles can be identified because they produce fragments of **different lengths.**

- **Trial and error** method

Drug testing today

It can take **over 10 years** and cost over **1 billion dollars.**

1. Substances are **analysed** and the **active ingredient**is **identified** and **copied** so it can be manufactured synthetically
2. **Slight variations** of the chemical **structure** are made in case they have a **better effect**(can be computer modelled)

Then…

Pre-clinical testing	- Tests on **animals** and **isolated cell/tissue** - Assess **safety** and **effectiveness** - Can take **years** - Authorised by **independent scientists, doctors** and **members of the public** to avoid any **bias**
Clinical trials Phase I	- **Small** group of **healthy volunteers** are given **different doses** - Tests confirm whether the compound is **used** by the body in the way **predicted** by **laboratory tests** - Tests for **safety**
Clinical trials Phase II	- 100-300 patients with the **disease** are treated to test **effectiveness**
Clinical trials Phase III	***Double-blind randomised controlled trial*** - **Large** group (1000-3000) is divided into **2 groups** - One group is given the compound, other group given the **placebo** or an **existing standardised treatment** - Tests for **side effects** and **effectiveness** - Can be **licensed** and **sold**
After licensing	- Trials continue to **collect data** on the **effectiveness/safety** of a **new drug** after this one has been licensed.

SEEDS FOR **SURVIVAL**

Seeds are adapted to:

- **Protect** the **embryo**
- Aid **dispersal**
- Provide **nutrition** for the **new plant**

The **ovule** is fertilised by the **nucleus** from a **pollen grain** and develops into a seed. The **outer** layers of the seed become **lignified** to form the seed coat which protects the embryo. The surrounding **ovary** develops into the **fruit** which aids in **dispersal** of seeds.

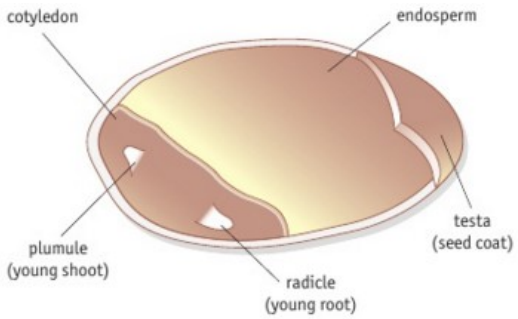


Figure 4.63 The internal structure of an endospermic seed.

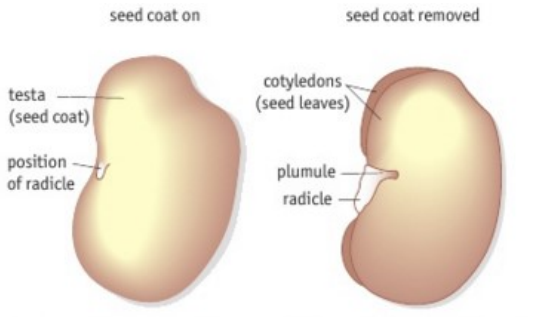


Figure 4.64 Opening up a broad bean reveals the cotyledons (seed leaves), plumule (young shoot) and radicle (young root) that make up the embryo.