

UPSC CSE MAINS

| ANTHROPOLOGY OPTIONAL

PAPER I

Physical and Archaeological Anthropology Notes

Part B of Paper I: human evolution and the theories of organic evolution, primates, fossil hominids, the biological basis of life, prehistoric archaeology and dating, human genetics, race, ecological and epidemiological anthropology, growth and development, demography and applied anthropology, with previous year questions after every chapter.

Compiled by Arvind KrS

HUMAN EVOLUTION

PRIMATES AND FOSSIL MAN

PREHISTORIC ARCHAEOLOGY

HUMAN GENETICS

GROWTH AND DEMOGRAPHY

APPLIED ANTHROPOLOGY

25

Chapters

5

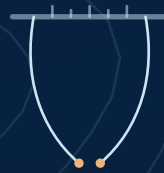
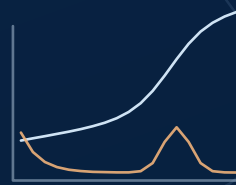
Units

790

Previous year questions

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Diagrams



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01 Human Evolution, Primates, Fossil Man and Prehistoric Archaeology

10 Human Growth and Development

12 Applications of Anthropology

09 Human Genetics, Race and Ecological Anthropology

11 Bioevents, Fertility and Demography



Anthropology Optional — Paper I

Part B: Physical and Archaeological Anthropology

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UNIT 01

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Human Evolution, Primates, Fossil Man and Prehistoric Archaeology

Human Evolution and emergence of Man: (a) Biological and Cultural factors in human evolution. (b) Theories of Organic Evolution (Pre-Darwinian, Darwinian and Post-Darwinian). (c) Synthetic theory of evolution; Brief outline of terms and concepts of evolutionary biology (Doll's rule, Cope's rule, Gause's rule, parallelism, convergence, adaptive radiation, and mosaic evolution). Characteristics of Primates; Evolutionary Trend and Primate Taxonomy; Primate Adaptations; (Arboreal and Terrestrial) Primate Taxonomy; Primate Behaviour; Tertiary and Quaternary fossil primates; Living Major Primates; Comparative Anatomy of Man and Apes; Skeletal changes due to erect posture and its implications. Phylogenetic status, characteristics and geographical distribution of the following: (a) Plio-preleistocene hominids in South and East Africa—Australopithecines. (b) Homo erectus: Africa (Paranthropus), Europe (Homo erectus heidelbergensis), Asia (Homo erectus javanicus, Homo erectus pekinensis. (c) Neanderthal man—La-chapelle-aux-saints (Classical type), Mt. Carmel (Progressive type). (d) Rhodesian man. (e) Homo sapiens—Cromagnon, Grimaldi and Chancelade. The biological basis of Life: The Cell, DNA structure and replication, Protein Synthesis, Gene, Mutation, Chromosomes, and Cell Division. (a) Principles of Prehistoric Archaeology. Chronology : Relative and Absolute Dating methods. (b) Cultural Evolution—Broad Outlines of Prehistoric cultures : (i) Paleolithic (ii) Mesolithic (iii) Neolithic (iv) Chalcolithic (v) Copper-Bronze age (vi) Iron Age

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ANTHROPOLOGY OPTIONAL

PAPER I · PART B

Human Evolution and Theories of Organic Evolution: Hominization, Lamarck and Darwin

PYQ RADAR 43 questions · last asked 2026


This article covers the **biological and cultural factors** in human evolution and the **pre-Darwinian, Darwinian and post-Darwinian theories**. The **synthetic theory** and the **terms of evolutionary biology** are in Unit 1.4b.

SYLLABUS 1.4 Human Evolution and emergence of Man: Biological and Cultural factors in human evolution. Theories of Organic Evolution (Pre-Darwinian, Darwinian and Post-Darwinian). Synthetic theory of evolution; Brief outline of terms and concepts of evolutionary biology (Doll's rule, Cope's rule, Gause's rule, parallelism, convergence, adaptive radiation, and mosaic evolution).

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ANTHROPOLOGY · HUMAN EVOLUTION

How Humans Evolved, and How We Explain It

Body and culture shaping each other; theories from Lamarck to the synthesis

TOPIC MAP

HOMINIZATION

ape-like ancestor (Africa, 6–7 mya) to *Homo sapiens*

Biological factors

Bipedalism: S-shaped spine, bowl pelvis, arched foot

Hand: opposable thumb, precision grip

Brain: about 400 cc to 1,350 cc; language areas

Face and teeth: smaller jaws, parabolic arch, chin

Life history: helpless newborn, long childhood

→
biocultural feedback
←

Cultural factors

Tools: Lomekwi 3.3 mya; Oldowan; Acheulean

Fire and cooking: softer food, smaller gut

Food-sharing: hunting and gathering

Language and symbols: cumulative culture

Gene-culture: lactase in north-west India

Theories of organic evolution

Pre-Darwinian

fixity of species; **Lamarck 1809:** use, disuse, acquired traits

→

Darwinian

Darwin, Wallace 1858–59: variation plus natural selection

→

Post-Darwinian

Weismann, Mendel, de Vries: germ plasm, genes, mutation

In simple words: **the body made culture possible; culture remade the body.**

Anthropology

01

1. Human Evolution and the Process of Hominization

IN SIMPLE WORDS

Human evolution is the long story of how an ape-like ancestor living in Africa about seven million years ago slowly became the modern human. It did not happen in one step: first our ancestors walked upright, much later the brain grew large, and language and culture came last. Biology and culture pushed each other forward throughout.

At **Laetoli** in Tanzania, volcanic ash preserved a trail of footprints about **3.66 million years old**. The prints show a raised arch, a big toe in line with the others and a heel strike: their makers walked **upright like us**. Yet those makers, almost certainly *Australopithecus afarensis*, had a **brain the size of a chimpanzee's** (about

400–500 cc). One trail shows the central fact of human evolution: the human body was assembled **part by part, at different times**.

- **Human evolution** is the history of the human lineage after it **separated from the chimpanzee line**, about **6–7 million years ago (mya)** on fossil and molecular evidence. The oldest claimed member, *Sahelanthropus* from Chad (about 7 mya), is still debated.
- **Hominization** is the **set of biological and cultural changes** that turned an ape-like ancestor into *Homo sapiens*: upright walking, a skilful hand, a large reorganised brain, a smaller face and teeth, speech, a long childhood and **dependence on culture**.
- **Why “process” matters**: hominization was **not a ladder** with a goal but **many branching species**, several living at once; only one survives.
- **Evidence: fossils and artefacts**, and **living and ancient DNA**, which dates splits and detects interbreeding.

COMMON CONFUSION

Hominid or hominin: which word means “the human line”? In older books and many past questions, **hominid** (family Hominidae) meant humans and their upright ancestors only. Genes later showed humans are closer to chimpanzees than chimpanzees are to orangutans, so Hominidae now includes all great apes, and the human line is called **hominin**. Both usages are correct in their own scheme; this note uses “hominin”.

1.1 Major trends in hominization

The trends did not appear together. The table lists them in the order the fossil record shows them, and that order is itself the main lesson.

TREND	WHAT CHANGED	EARLIEST CLEAR EVIDENCE	WHY IT MATTERED
Bipedalism	Upright posture , two-legged walking	<i>Ardipithecus</i> 4.4 mya; Laetoli 3.66 mya	Freed the hands ; reshaped spine, pelvis, legs, foot
Tool-making hand	Long, fully opposable thumb ; precision grip	Lomekwi tools 3.3 mya; Oldowan 2.6 mya	Made planned tool-making possible
Facial reduction	Smaller canines, no diastema, parabolic arch , flatter face	Australopithecines onward; chin only in <i>H. sapiens</i>	Tools and cooking took over the jaws' work
Encephalization	Brain grows faster than body; cortex reorganised	Early <i>Homo</i> , about 2 mya	Planning, language, social intelligence

TREND	WHAT CHANGED	EARLIEST CLEAR EVIDENCE	WHY IT MATTERED
Long life history	Slow growth, long childhood , helpless newborn	<i>Homo erectus</i> onward	Time to learn culture
Speech and symbol	Lowered larynx, language areas, art and ornament	Clear symbols from about 100,000 years ago	Culture becomes cumulative

Read down the third column: **feet before brains**. Upright walking appears millions of years before the brain expands, and symbolic culture comes last. This uneven pace is **mosaic evolution** (different parts evolving at different rates; Unit 1.4b). It also corrects the old assumption, behind the acceptance of the faked **Pittdown** skull (exposed 1953), that a **big brain came first**. Older textbooks also placed **Ramapithecus**, jaws from the **Siwalik Hills** of India and Pakistan (about 12–8 mya), at the base of the human line; fuller finds in the 1980s showed it belongs with **Sivapithecus**, an **ancestor of the orangutan** (Unit 1.5).

1.2 Why Africa is central

- **Every early stage is African:** the earliest hominins (*Sahelanthropus*, Chad; *Orrorin*, Kenya; *Ardipithecus*, Ethiopia), all australopithecines, the **earliest Homo** (Ledi-Geraru jaw, Ethiopia, about 2.8 mya), the **earliest tools** (Lomekwi, 3.3 mya) and the **earliest Homo sapiens** (**Jebel Irhoud**, Morocco, about 315,000 years ago).
- **Genes agree:** Africans carry the **greatest genetic diversity**, and the mitochondrial DNA of all living humans traces to an **African ancestor** (Rebecca Cann, Mark Stoneking and Allan Wilson, American geneticists, 1987). Diversity is highest where a species has lived longest.
- **Darwin predicted it** (*The Descent of Man*, 1871), because chimpanzees and gorillas live in Africa.
- **But Asia is not a mere receiver.** Hominins and tools reached **Dmanisi**, Georgia (about 1.8 mya) and **Shangchen**, China (about 2.1 mya). In India, **Attirampakkam** (Tamil Nadu) has **Acheulean tools about 1.5 million years old** (Shanti Pappu and colleagues, 2011). Asia held Denisovans and *Homo floresiensis*, and modern humans **interbred** with Neanderthals and Denisovans after leaving Africa.
- **Balanced view:** Africa is the **cradle**; Eurasia was a **theatre** where lineages evolved, met and mixed (fossils in Unit 1.6).

02 2. Biological Factors in Human Evolution

IN SIMPLE WORDS

The human body changed in a few key ways: we walk upright, our hands grip finely, our brains became very large, our faces and teeth became smaller, and our babies are born helpless and grow slowly. Each change made the next one possible, and together they prepared the body for a life based on culture.

A human newborn's head barely passes through its mother's pelvis; the baby must **rotate** during birth, and most mothers are helped by others, which is rare among primates. Two defining traits pull in opposite directions: **upright walking** favours a **narrow pelvis**, a **large brain** needs a **wide birth canal**. The American biological anthropologist **Sherwood Washburn** called this the **obstetrical dilemma** (1960). The compromise was a baby born with a brain only about **30 per cent** of adult size, so most brain growth happens **after birth, in a cultural world**. One example ties bipedalism, brain and culture together.

2.1 Erect posture and bipedal locomotion

Bipedalism (habitual walking on two legs) is the **earliest hominin trait**, and it reshaped almost every bone.

- **Skull:** the **foramen magnum** (the hole for the spinal cord) moved **forward and down** to the centre of the skull base, so the head **balances on the spine**. Apes have it further back and need heavy neck muscles.
- **Spine:** a C-shaped curve became an **S-shaped curve** with a forward bend in the lower back (**lumbar lordosis**), placing the body's weight over the hips. Humans usually have **five lumbar vertebrae**, great apes three or four.
- **Pelvis:** the **ilium** (upper blade of the hip bone) became **short, broad and bowl-shaped**. Two buttock muscles moved to the side, where they **stop the pelvis tilting** when one foot leaves the ground.
- **Leg:** the thigh bone (**femur**) **slopes inward to the knee** (the **valgus** or bicondylar angle), bringing the feet under the centre of gravity; legs became **longer than arms**.
- **Foot:** a **big toe in line** with the others, **two arches** acting as springs, and a large heel.

Why did it evolve? The main hypotheses (treated fully in Unit 1.5) are **freeing the hands** for carrying and tools (Darwin, 1871), **energy efficiency** over knuckle-walking (Peter Rodman and Henry McHenry, 1980), **keeping cool** by exposing less of the body to the midday sun (Peter Wheeler, 1991), **provisioning** of mates and young (C.O. Lovejoy, 1981) and **endurance running** (Dennis Bramble and Daniel Lieberman, 2004). They are **not exclusive**: early bipedalism was probably about **efficient walking between woodland patches**, while carrying and running mattered later, in *Homo erectus*.

2.2 The hand and the precision grip

- Hominins added a **long, strong thumb** that can be **fully opposed** to every finger tip.
- The British primatologist **John Napier** distinguished the **power grip** (whole hand round an object, as with a hammer) from the **precision grip** (a small object between thumb and finger tips, as with a needle) (1956). Only humans **combine both fully**.
- **Why it mattered:** making a stone tool needs **both grips at once**, one hand holding the core and the other striking at a controlled angle. A precise hand, a brain that plans the blows, and hands freed by bipedalism made **the first tools possible**.

2.3 Expansion and reorganisation of the brain

Encephalization is brain growth **beyond what body size predicts**. Brain size roughly **tripled** in two million years:

HOMININ	APPROX. BRAIN SIZE (CC)
Chimpanzee (comparison)	350–400
<i>Australopithecus afarensis</i>	400–500
<i>Homo habilis</i>	about 600
<i>Homo erectus</i>	750–1,250
Neanderthals	about 1,450
<i>Homo sapiens</i>	about 1,350

Neanderthals had **larger brains than ours**, so **organisation**, not volume alone, explains human abilities.

- **Association areas:** growth was mainly in the **cerebral cortex**. Around each sensory area lie **association areas**, which match new input with memory and make recognition possible. In humans, large fibre bundles **link association areas directly** (the basis of thinking) and connect them to the areas that **control the hand** (the basis of skilled tool use).
- **Language areas:** **Broca's area** (frontal lobe; produces speech) and **Wernicke's area** (temporal lobe; understands speech), joined by the **arcuate fasciculus**, plus the **angular gyrus**, which links sight, hearing and touch to words. Being in the cortex, they let humans **speak without passing through the older emotional centres**.
- **The cost:** the brain is about **2 per cent** of body weight but uses about **20 per cent** of resting energy. The **expensive-tissue hypothesis** (Leslie Aiello and Peter Wheeler, British anthropologists, 1995) argues that the **gut shrank** as the brain grew, which needed **high-quality food**: meat, marrow and later cooked food.
- **The social brain hypothesis** (Robin Dunbar, British evolutionary psychologist, 1998): primates in larger groups have larger neocortices, so the brain grew mainly to manage **complex social life**.

2.4 Reduction of the face, jaws and teeth

The craniofacial changes form a clear sequence.

FEATURE	EARLIER HOMININS	MODERN HUMANS
Prognathism (projecting jaws)	Marked	Reduced ; face tucked under the brain case
Canines and diastema	Large canines; gap in the opposite jaw	Small canines; no gap
Dental arcade	U-shaped	Parabolic
Brow ridges, forehead	Heavy ridges; low sloping forehead	Small ridges; vertical forehead

FEATURE	EARLIER HOMININS	MODERN HUMANS
Chin	Absent, even in Neanderthals	Present: unique to <i>H. sapiens</i>
Sagittal crest	Present in robust forms	Absent

Three causes worked together. A **larger brain** under the obstetric limit made the snout shrink to make room. **Tools and cooking** took over cutting and softening food, so the mouth stopped being a “**fifth hand**” and big jaws were no longer favoured. A **shorter face** helped **speech**.

2.5 Speech and a long childhood

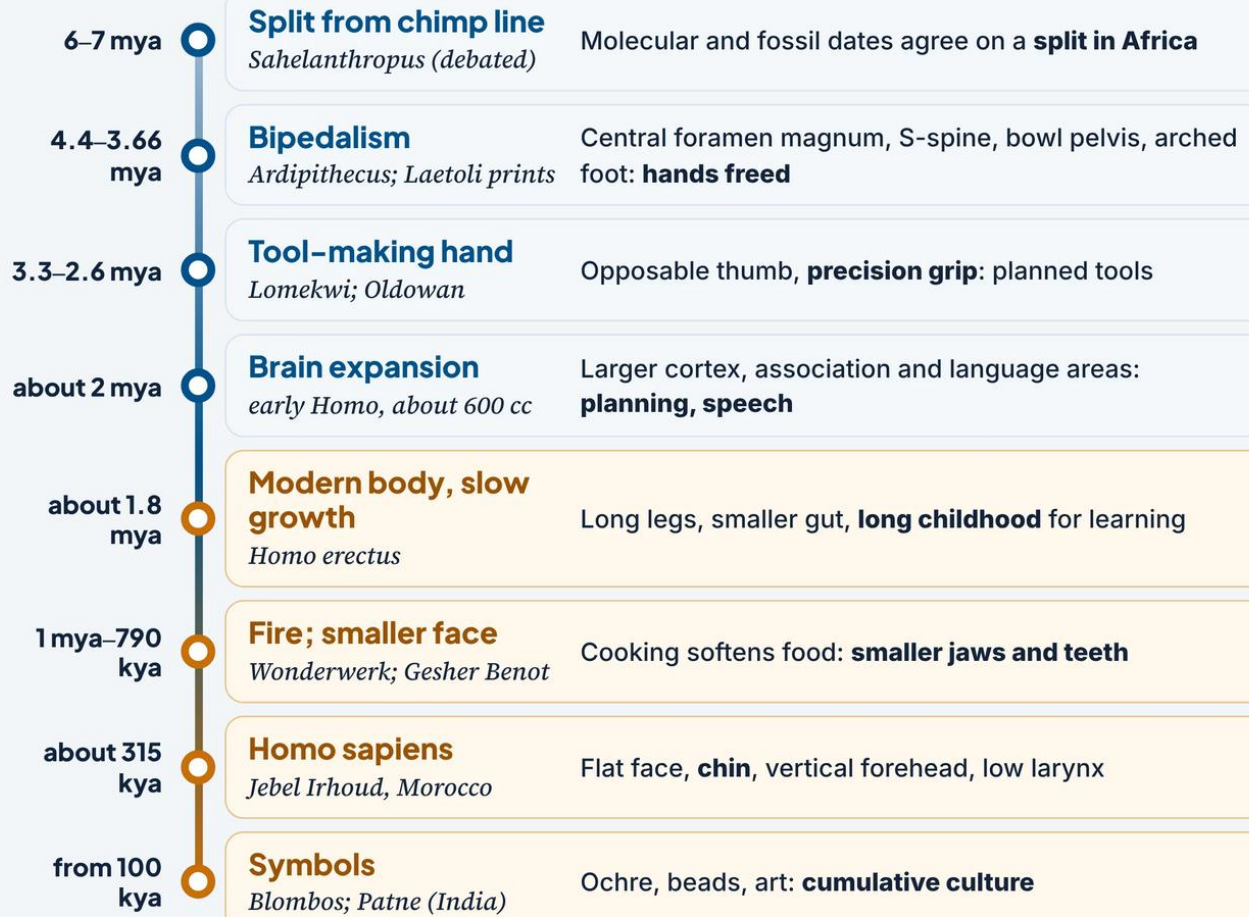
- **Vocal tract:** the **larynx** sits **low in the throat**, so throat and mouth form a two-part sound chamber that produces the full range of vowels (at the cost of a higher risk of choking).
- The **hyoid bone** of a Neanderthal from **Kebara**, Israel (about 60,000 years ago) is almost identical to ours; the speech-related gene **FOXP2** has a **human-specific form that Neanderthals shared**. Language has **deep roots**.
- **Slow growth:** helpless infancy and a **long childhood**. Washburn explained the newborn’s helplessness (**secondary altriciality**) by the obstetrical dilemma; the **metabolic hypothesis** (Holly Dunsworth and colleagues, 2012) adds that birth occurs when the foetus’s energy needs **exceed what the mother can supply**.
- **Why it matters:** a long childhood is the **period of learning culture**, and it needs helpers. The **grandmother hypothesis** (Kristen Hawkes, 1998) argues that older women feeding grandchildren favoured **long life after menopause**.

ANTHROPOLOGY · HOMINIZATION

The Human Body, Built in Stages

Main biological changes in fossil order, with what each made possible

SEQUENCE



PATTERN

Feet before brains: upright walking about 2 million years before brain growth

TRADE-OFF

Obstetrical dilemma: narrow pelvis, big head, so most brain growth comes after birth

In simple words: **our body was assembled part by part.**

Anthropology

3. Cultural Factors and the Biocultural Feedback

IN SIMPLE WORDS

Culture is not something humans added after their bodies were finished. Tools, fire, food-sharing and language changed which bodies survived, so culture shaped human biology. In return, each biological change made more culture possible. This two-way process is called biocultural evolution.

In north-western India, many people of herding and dairying communities digest milk all their lives, while in much of southern and north-eastern India most adults cannot. This ability, **lactase persistence**, depends on a variant (**-13910*T**) that keeps the milk-digesting enzyme working after childhood. Irene Gallego Romero and colleagues (2012) found the variant **common where cattle were herded and milked**, reaching levels that could make up to about **73 per cent** of adults lactase-persistent in some north Indian populations, and **rare or absent** in many southern and north-eastern groups. **Dairying made the gene useful, and the gene spread:** culture changed biology.

3.1 Tools: “man makes himself”

- **The sequence:** the earliest tools are from **Lomekwi 3**, Kenya (**3.3 mya**), **older than Homo**. The **Oldowan** (simple flakes and choppers) begins about **2.6 mya**; the **Acheulean** (symmetrical hand-axes) about **1.76 mya** in Kenya and **1.5 mya at Attirampakkam**.
- **Tools shaped the body:** Washburn argued (1960) that the success of even the simplest tools **started the trend of human evolution:** once tools were used, selection favoured **better hands, larger brains and smaller teeth**. This **reverses the older order** in which the brain came first and then invented tools.
- **“Man makes himself”:** the archaeologist **V. Gordon Childe** used this as the title of his book (*Man Makes Himself*, 1936): by creating their own means of living (tools, farming, cities), humans **created the conditions of their own development**.
- **Tools need foresight:** a tool helps only if it is **carried until needed**, which requires **planning** and **free hands**. The British palaeontologist **Kenneth Oakley** called humans **“man the tool-maker”** (1949) because making tools to a set pattern for future use requires **abstract thought**.

3.2 Fire and cooking

- **Evidence:** burnt bone and ash at **Wonderwerk Cave**, South Africa (about **1 mya**), and repeated hearths at **Gesher Benot Ya'aqov**, Israel (about **790,000 years ago**).
- **The cooking hypothesis** of the British primatologist **Richard Wrangham** (*Catching Fire*, 2009): cooking made food **softer and more digestible**, giving more energy from the same food. This explains the **smaller teeth and gut** of *H. erectus* and helped pay for a **larger brain**.
- Fire also gave **warmth, protection** and a **hearth** for sharing and talking after dark.

3.3 Hunting, sharing, language and social life

- **Man the Hunter** (Richard Lee and Irven DeVore, eds., 1968) argued that **cooperative hunting** drove intelligence, cooperation and the sexual division of labour.

- The feminist critique “**Woman the Gatherer**” (Sally Slocum, 1975) showed that among foragers such as the **!Kung San** of the Kalahari, **gathered plant food** supplies most calories, and that **sharing and child care** mattered as much as hunting. The current view stresses **food-sharing at a home base**, which needs **trust, memory and communication**.
- **Language** made culture **cumulative**: knowledge is taught and improved across generations instead of being rediscovered. **Symbols** appear from about 100,000 years ago (engraved ochre and shell beads, **Blombos Cave**, South Africa); in India, ostrich-eggshell beads come from **Patne** (Maharashtra), about 25,000 years ago.

3.4 Gene-culture co-evolution

Gene-culture co-evolution (or **dual inheritance theory**) holds that humans inherit **genes and culture**, and that **each changes the other**. Its main formulations came from **Luigi Luca Cavalli-Sforza and Marcus Feldman** (1981), **Robert Boyd and Peter Richerson** (*Culture and the Evolutionary Process*, 1985) and **William Durham** (*Coevolution*, 1991). **Niche construction** (John Odling-Smee, Kevin Laland and Feldman, 2003) adds that organisms **change their own environment** and so the selection acting on them.

Every case below follows one mechanism: a **cultural practice** changes the environment, and the new environment changes **which genes succeed**.

CULTURAL PRACTICE	ENVIRONMENTAL CHANGE	GENETIC RESPONSE	POPULATION AND STUDY
Dairying	Milk available to adults	Lactase persistence spreads	North-west India , Europe, East Africa (Gallego Romero, 2012)
Forest clearing for yam farming	Standing water breeds malaria mosquitoes	Sickle-cell gene protects carriers	West Africa (Frank Livingstone, 1958); central Indian tribal belts
Starchy diets	More starch to digest	More copies of the amylase gene	Farming peoples (George Perry, 2007)
Breath-hold diving	Long dives for food	Larger spleens	Bajau sea nomads, Indonesia (Melissa Ilardo, 2018)
Mortuary cannibalism	Spread of the prion disease kuru	Protective prion-gene variant	Fore , Papua New Guinea (Simon Mead, 2009)
Caste and community endogamy	Small closed breeding groups	Founder effects ; more recessive disease	Many Indian groups (Nathan Nakatsuka, 2017)

In every row **culture comes first** and the genetic change follows. The sickle-cell gene protects carriers against malaria but causes disease in those with two copies, which is why it is common in malarial regions and why India’s **National Sickle Cell Anaemia Elimination Mission** (2023) concentrates on tribal districts. In the endogamy row, a **social rule** studied by social anthropologists has **measurable genetic effects**: the two branches need each other.

ANTHROPOLOGY · BIOCULTURAL EVOLUTION

Body and Culture: A Feedback Loop

Each biological change made new culture possible; culture then changed which bodies survived

PROCESS



Culture first, genes follow

Dairying

Lactase persistence

common in north-west India,
rare in the south

Yam farming

Clearings breed malaria;
sickle-cell carriers survive

Endogamy

Closed marriage groups:
founder effects in India

In simple words: **we made culture, and culture made us.**

Anthropology

3.5 Biological and cultural evolution compared

Human evolution has two strands that work differently. The table shows why cultural change now **outpaces** biological change.

BASIS	BIOLOGICAL EVOLUTION	CULTURAL EVOLUTION
What is inherited	Genes (DNA)	Learned ideas, skills, values, artefacts
Route	Parents to offspring only	Parents, peers, teachers, media
Novelty from	Random mutation, recombination	Invention , often purposeful
Acquired traits passed on?	No	Yes ("Lamarckian")

BASIS	BIOLOGICAL EVOLUTION	CULTURAL EVOLUTION
Speed	Slow: many generations	Fast: within a lifetime
Pattern	Branching only	Branches and merges (borrowing)
Reversibility	Practically irreversible	Traits abandoned or revived

Because culture passes on **what is learned**, it responds to a problem in years, while genes need generations. That is why **sociocultural evolution is not parallel** to biological evolution: the body has changed little in 40,000 years, while societies moved from foraging bands to industrial states. The American anthropologist **Alfred Kroeber** called culture “**superorganic**” (1917), a level with its own laws. The Russian-American geneticist **Theodosius Dobzhansky** took the middle path (*Mankind Evolving*, 1962): culture is the **most powerful adaptation biology has produced**, yet it has **not stopped biological evolution** (Unit 1.4b shows human genes are still changing).

3.6 The bio-cultural approach in biological anthropology

Biological anthropology treats human **biology and culture as one system**; this bio-cultural approach is its defining feature.

- **Adaptability perspective:** the **Human Adaptability** section of the International Biological Programme (1964–74) studied how populations cope with altitude, cold and heat. The American biological anthropologist **Gabriel Lasker** (1969) showed humans adapt at **several levels: genetic** (natural selection), **developmental** (during growth), short-term **acclimatisation**, and **cultural** (clothing, housing, technology) (Unit 9.7).
- **Political-economy perspective:** **Alan Goodman and Thomas Leatherman** (*Building a New Biocultural Synthesis*, 1998) argued that the environment humans adapt to is largely **made by society**: poverty, unequal land, wage labour, discrimination. **Inequality shows up in the body** as stunting, anaemia and disease.
- **Integration:** the newer synthesis keeps the adaptability and **ecological** tools (variation as a fit to environment) but asks **who is exposed to which stress, and why**. In India, **NFHS-5** (2019–21) recorded **stunting** in **35.5 per cent** of children under five overall, but **40.9 per cent** among Scheduled Tribe and **39.2 per cent** among Scheduled Caste children. An ecological reading looks for adaptation; the political-economy reading explains the gap by **unequal access** to land, food, sanitation and care.

4. Organic Evolution: Meaning and Evidence

IN SIMPLE WORDS

Organic evolution means that living things change over generations, and that new kinds of plants and animals arise from older ones. We know this from fossils, from similar bone plans in different animals, from embryos, from where species live, from DNA, and from changes we can watch today.

Every human has a **coccyx** (tailbone) of fused tail vertebrae, a small **appendix**, and ear muscles most people cannot use. These parts make no sense in a species designed from scratch, but perfect sense if humans **descend from tailed, ear-moving ancestors**. Such **vestigial organs** (reduced remnants of organs that worked in ancestors) are one of several **independent lines of evidence** for evolution.

- **Meaning:** the English philosopher **Herbert Spencer** popularised "evolution" for the development of the universe, life and society (1857). **Darwin** preferred "**descent with modification**": new species **descend** from ancestral ones and are **modified** along the way.
- **Organic evolution** is the **change in heritable characteristics of populations over generations**, leading to **new species** from earlier ones. Change in societies is **cultural evolution**.
- **Ontogeny and phylogeny** (both coined by the German zoologist **Ernst Haeckel**, 1866): **ontogeny** is the development of **one individual**; **phylogeny** the evolutionary history of a **group of species**. Evolution concerns phylogeny.

The evidence comes from **independent sciences**, which is why the conclusion is secure.

LINE OF EVIDENCE	WHAT IT SHOWS	EXAMPLE
Fossils	Ordered sequences, transitional forms	<i>Archaeopteryx</i> (reptile-bird); horse and hominin series
Comparative anatomy	Homologous organs: same plan, different use	Human arm, bat wing, whale flipper
Vestigial organs	Remnants of ancestral organs	Coccyx, appendix, ear muscles
Embryology	Related species look alike as embryos	Human embryo has pharyngeal arches and a tail
Biogeography	Species resemble those of the nearest mainland	Galápagos finches; Australia's marsupials
Molecular biology	Shared code ; similarity tracks relationship	Humans and chimpanzees differ at about 1.2 per cent of DNA letters

LINE OF EVIDENCE	WHAT IT SHOWS	EXAMPLE
Direct observation	Evolution happening now	Finch beaks after the 1977 drought; antibiotic resistance

Molecular evidence is strong because it is **independent of anatomy**: humans share with other monkeys and apes a **broken vitamin-C gene (GULO)** with the same defects, best explained by **a common ancestor** in which it first broke.

05

5. Pre-Darwinian Theories

IN SIMPLE WORDS

Before Darwin, most people believed that each species was created separately and never changed. Thinkers gradually found fossils of extinct animals, learned that the earth is very old, and began to suspect that species change. Lamarck gave the first full theory of how they change, but his mechanism was wrong.

For centuries, women in many Indian communities have had their **ears and noses pierced**, and Jewish and Muslim boys have been **circumcised**. Yet no baby is born with pierced ears or without a foreskin. If changes made during life were inherited, these would long since have become inborn. This everyday fact is the simplest refutation of the best-known pre-Darwinian theory, the **inheritance of acquired characters**.

5.1 From fixity of species to the idea of change

The period moved from belief in **fixed, separately created species** towards the idea that species **change over a very long time**.

IDEA	THINKER (DATE)	CORE CLAIM	SIGNIFICANCE OR WEAKNESS
Spontaneous generation	Aristotle; Greek tradition	Life arises from non-living matter	Disproved by Redi (1668) and Pasteur (1859–62)
Great chain of being	Aristotle; Augustine, Aquinas	All beings in a fixed ladder up to God	A ladder, not a tree
Special creation	Biblical tradition; Francisco Suárez	God created each species separately	Fixity of species
Classification	Linnaeus , <i>Systema Naturae</i> (1735)	Fixed species, binomial names	Nested groups later read as common descent

IDEA	THINKER (DATE)	CORE CLAIM	SIGNIFICANCE OR WEAKNESS
Preformation	Malpighi; Hartsoeker's homunculus (1694); Bonnet's encasement	A miniature adult in egg or sperm	Disproved by embryology
Epigenesis	C.F. Wolff (1759)	Organs form gradually	Correct; basis of embryology
Mutability	Buffon , <i>Histoire naturelle</i> (from 1749)	Species change with environment	First serious challenge to fixity
Common origin	Erasmus Darwin , <i>Zoonomia</i> (1794–96)	All life from one source	Charles Darwin's grandfather; no mechanism
Catastrophism	Cuvier (early 1800s)	Extinction is real ; repeated catastrophes	Founded palaeontology; opposed evolution
Uniformitarianism	Hutton (1788); Lyell (1830–33)	Slow present processes over vast time	Gave Darwin deep time
Population pressure	Malthus (1798)	Population outgrows food	Gave Darwin the struggle for existence

The last three rows matter most. **Cuvier** proved that species go **extinct**, **Lyell** that the earth is **very old**, and **Malthus** that **more are born than can survive**. Darwin combined all three. Lamarck, meanwhile, offered the first complete theory of **how** species change.

5.2 Lamarckism: the inheritance of acquired characters

Jean-Baptiste Lamarck (1744–1829), a French naturalist, set out his theory in *Philosophie Zoologique* (1809). He was the **first to argue that evolution is a general fact covering all life**, against the fixity defended by Cuvier.

The four propositions:

1. **Internal tendency to growth:** organisms and their parts tend to **increase in size** and complexity.
2. **New needs produce new organs:** a changed environment creates **new needs** (*besoins*), new habits and, through them, new organs.
3. **Use and disuse:** organs used constantly **develop**; unused organs **weaken and vanish**.
4. **Inheritance of acquired characters:** changes acquired in life are **passed to offspring**.

His examples: the **giraffe**, whose ancestors stretched for high leaves, lengthening the neck a little each generation; **webbed feet** of ducks from stretching the toes; **limbless snakes** from creeping through narrow places; **blind moles and cave animals** from disuse. It was **simple**, matched experience (exercise builds muscle) and could be **tested**.

Critique, proposition by proposition:

- **Growth in size** is not universal: giant tree-ferns gave way to **smaller flowering plants**.
- **Need does not create organs**: humans have always wished to fly but **never grew wings**.
- **Use and disuse** works **within a lifetime** (a blacksmith's arm) but proves nothing unless **inherited**; the heart works constantly yet does not keep growing.
- **Inheritance of acquired characters** failed every test:
 - **August Weismann**, a German zoologist, cut off the tails of mice for **five generations (901 young)** and reported in **1889** that **none** was born with a shorter tail.
 - **Ear and nose piercing** in India, circumcision and Chinese **foot-binding** produced **no inherited change**.
 - **William Castle and John Phillips** transplanted the ovaries of a **black guinea pig** into a white female (1909); mated to a white male, she bore **only black young**. Offspring followed the **genes in the ovary**, not the body carrying it.
- **Why it fails**: changes in body cells **do not alter the sex cells** that carry heredity (§7.1).

5.3 Neo-Lamarckism and its modern echoes

- **Neo-Lamarckism** (term coined by Alpheus Packard, 1885) revived Lamarck in the late 19th century, especially in America (**Edward Drinker Cope**, Packard, Alpheus Hyatt) and in **Herbert Spencer**. Its followers held that **environment and habit**, not selection, drive evolution.
- **Failed or false experiments**: **Paul Kammerer's** midwife toads, whose inherited "nuptial pads" were shown in **1926** to have been **injected with ink**; **William McDougall's** rats, said to learn a water maze faster each generation (1920s–30s), **not replicated**; and **Trofim Lysenko**, whose state-backed Lamarckism **suppressed Soviet genetics** (the geneticist **Nikolai Vavilov** died in prison, 1943), a warning about **ideology overriding evidence**.
- **Epigenetics**: chemical tags on DNA (such as **methylation**) change how genes are **expressed** without changing the sequence, and the environment can set them. People conceived in the **Dutch Hunger Winter** (1944–45) still showed altered methylation of the **IGF2** gene six decades later (Bastiaan Heijmans and colleagues, 2008). But most tags are **erased each generation**, and inheritance over several human generations is **not established**. Epigenetics **qualifies** Weismann's rule; it **does not restore** Lamarckism.
- **Baldwin effect** (James Mark Baldwin, American psychologist, 1896): a **learned behaviour** can change which genes are favoured, so learning **guides** genetic evolution without inheriting acquired traits. Lactase persistence (§3) is a human case.

COMMON CONFUSION

Did Lamarck and Darwin disagree about whether evolution happens? No. Both accepted evolution; they disagreed about its **mechanism**. For Lamarck, a giraffe **acquired** a longer neck by stretching and passed it on. For Darwin, giraffes already **varied** in neck length; those born with longer necks survived and bred more. Darwin himself did not reject inheritance of acquired characters; that came later, with Weismann.

6. Darwinian Theory: Natural Selection

IN SIMPLE WORDS

Darwin noticed that living things produce more young than can survive, that the young differ, and that some differences are inherited. Those whose differences suit their surroundings survive and breed more, so useful differences become common. Over very long periods, this natural selection can turn one species into another.

On **Daphne Major**, a tiny Galápagos island, the biologists **Peter and Rosemary Grant** measured every finch for decades. In the **drought of 1977**, small soft seeds ran out and only large hard seeds remained. Medium ground finches with **deeper beaks** could crack them; most others starved. Because beak depth is **inherited**, the next generation had **measurably deeper beaks** (Peter Boag and Peter Grant, 1981). This is **natural selection observed directly**, on the birds Darwin collected.

6.1 How the theory was formed

- **Charles Darwin** (1809–1882), son of a Shrewsbury doctor, sailed as naturalist on **HMS Beagle (1831–36)**.
- **What he saw:** fossils of **giant extinct mammals** in Argentina resembling living South American forms; and on the **Galápagos**, finches, mockingbirds and tortoises that **differed from island to island** yet resembled mainland species, suggesting **descent from mainland colonists** with change on each island.
- **What he read and learned:** **Lyell's** deep time; **Malthus** (read in **1838**); and breeders' **artificial selection** of pigeons and cattle.
- **Wallace:** in 1858 **Alfred Russel Wallace**, a British naturalist in the Malay Archipelago, sent Darwin an essay with the **same theory**. Their papers were read at the **Linnean Society** on **1 July 1858**, and ***On the Origin of Species*** appeared on **24 November 1859**.
- **"Survival of the fittest"** is **Spencer's** phrase (1864); Darwin adopted it in 1869.

6.2 The logic: five facts, three inferences

The German-American biologist **Ernst Mayr** (*The Growth of Biological Thought*, 1982) reduced Darwin's argument to **five facts and three inferences**. Each conclusion follows from observation.

OBSERVED FACTS

1. Overproduction: populations could grow geometrically (even the slow-breeding elephant, Darwin calculated, would leave nearly nineteen million descendants from one pair in about 750 years). **2. Yet populations stay roughly stable.** **3. Resources are limited.**

INFERENCE

I. Struggle for existence: most offspring die young. The struggle is within species, between species, and against drought or cold.

OBSERVED FACTS	INFERENCE
4. Variation: no two individuals are alike. 5. Much variation is heritable.	II. Natural selection: those with favourable variations survive and reproduce more (differential reproduction).
All five, over many generations	III. Evolution and origin of species: favourable traits accumulate; populations in different conditions diverge into species.

Every step rests on something **observable**, which is the argument's strength. "**Fittest**" means not the strongest but the one that **leaves most surviving offspring** in that environment; a drought-tolerant plant or a cooperative animal can be the fittest.

6.3 Darwin's supplementary theories

- **Artificial selection:** selection **under human control**, which produced breeds as different as greyhound and bulldog from one wolf stock; Darwin's **model** for natural selection.
- **Sexual selection** (*The Descent of Man*, 1871): traits that help in **winning mates**, through male competition (antlers) or **female choice** (the peacock's tail). It explains traits that seem **costly for survival**.
- **Pangenesis** (1868): every body part sheds particles (**gemmules**) that travel to the sex organs, so bodily changes could be inherited. His cousin **Francis Galton** transfused blood between rabbits of different colours (1871) and found **no effect** on the young; the idea was abandoned.

6.4 Critique of Darwinism

Each objection was real in Darwin's time, and each was answered by a later discovery.

OBJECTION	WHY IT HELD	HOW IT WAS ANSWERED
No source of variation: it explains the survival of the fittest, not their arrival	Heredity unknown	Mutation and recombination
Blending inheritance would dilute new variants (Fleeming Jenkin, 1867)	Offspring thought a blend of parents	Mendel: genes do not blend
Useless beginnings: half a wing cannot fly (St George Mivart, 1871)	Early stages seem valueless	Early stages had other uses (feathers for warmth): exaptation (Gould and Vrba, 1982)
Overspecialisation: giant antlers of the Irish elk	Selection should not create burdens	Sexual selection and growth scaling (Gould, 1974)
Pangenesis was Lamarckian	Darwin needed a theory of heredity	Rejected by Galton and Weismann

OBJECTION	WHY IT HELD	HOW IT WAS ANSWERED
Not all change is adaptive	Darwin stressed selection almost alone	Genetic drift and the neutral theory (Unit 1.4b)
"Tooth and claw"	Struggle read as fighting	Struggle includes drought; cooperation aids survival (Peter Kropotkin, <i>Mutual Aid</i> , 1902)

Darwin's **core idea, selection acting on heritable variation, survived every objection**; what was missing was a **theory of heredity**, which genetics supplied. The theory was also **misused**: **Social Darwinism** used "survival of the fittest" to justify inequality and empire, and **eugenics** (Galton's word, 1883) led to forced sterilisation. Both are rejected as **scientifically wrong and morally indefensible**.

6.5 Lamarckism and Darwinism compared

Both accept evolution and both explain adaptation. They differ in the **order of events**: for Lamarck, the **change comes first** and is then inherited; for Darwin, **variation comes first** and is then selected.

BASIS	LAMARCKISM	DARWINISM
Source of change	Need and effort of the organism	Chance variation already present
Role of environment	Directly causes change	Selects among variants
What changes	The individual , in its life	The population , over generations
Inheritance	Acquired characters inherited	Inborn variation inherited
Direction	Towards perfection	No goal
Status today	Rejected for genes; echoes in epigenetics and cultural inheritance	Core of modern theory , completed by genetics

Contribution to the synthetic theory: Darwin supplied its **mechanism** (natural selection) and its **population thinking**. Lamarck's lasting contribution was that **evolution is a general fact** and that **adaptation to the environment** is what needs explaining. Debates on epigenetics, the Baldwin effect and niche construction revisit his questions while rejecting his mechanism.

ANTHROPOLOGY · THEORIES OF EVOLUTION

Why Is the Giraffe's Neck Long?

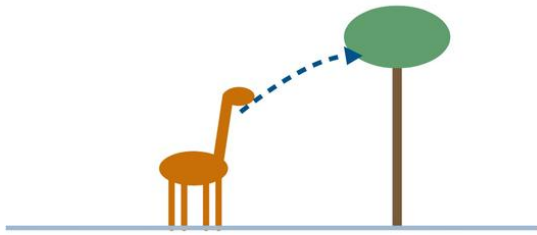
Lamarck: change first, then inheritance. Darwin: variation first, then selection

COMPARISON

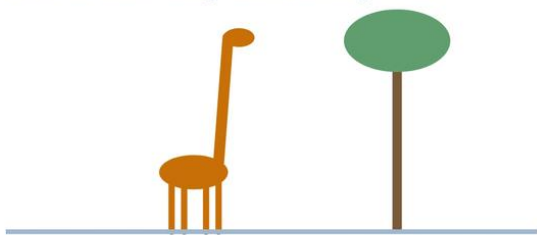
Lamarck (1809)

use, disuse and acquired characters

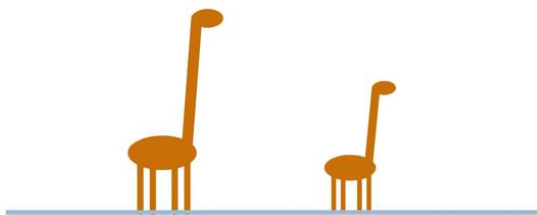
1. Need: the ancestor stretches for high leaves



2. Use: its neck lengthens during its life



3. Inheritance: the young are born with the longer neck

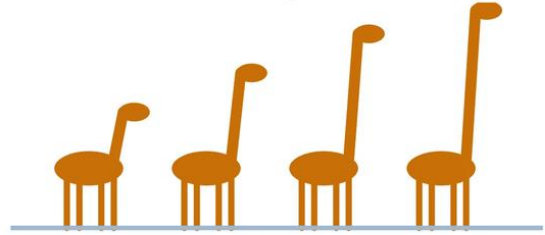


Disproved: acquired changes do not reach the sex cells (Weismann)

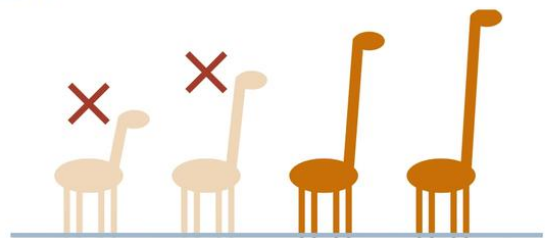
Darwin (1859)

variation and natural selection

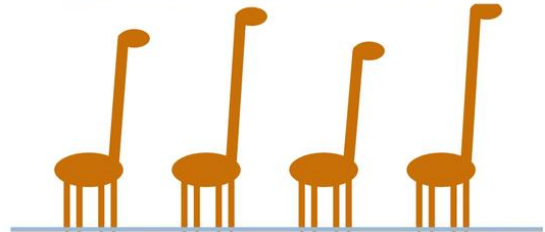
1. Variation: necks already differ at birth



2. Selection: in drought, short-necked animals starve



3. Next generation: longer necks on average



Supported: core of modern theory, completed by genetics

In simple words: **the environment selects; it does not create.**

Anthropology

7. Post-Darwinian Theories

IN SIMPLE WORDS

After Darwin, scientists argued about what his theory could not explain: how traits are inherited and where new variations come from. Some found that heredity runs only through the sex cells, some rediscovered Mendel's laws, and some thought new species appear suddenly through big mutations. These debates prepared the way for the synthetic theory.

In **1886**, in a field near Hilversum in the Netherlands, the Dutch botanist **Hugo de Vries** found **evening primroses** (*Oenothera lamarckiana*) with new forms that **bred true at once**: giants, dwarfs, plants with odd leaves. He concluded that new species arise **suddenly, in one step**. The example captures the period: critics now looked for the **source of variation**, and some believed they had found a **rival to natural selection**.

7.1 Weismann's germ-plasm theory

- **August Weismann** (*Das Keimplasma*, 1892) divided the body into the **germ plasm** (hereditary material of the sex cells) and the **somatoplasm** (all other cells).
- **Continuity of the germ plasm**: the germ plasm passes **unbroken** between generations; the body is its **temporary carrier**, and bodily changes **cannot reach it** (the **Weismann barrier**).
- **Why it mattered**: it **ruled out Lamarckism and pangenesis**, leaving selection of **inborn variation**. George Romanes called this purified theory **neo-Darwinism** (1890s).
- **Limits**: in plants and many simple animals the germ line separates late, and epigenetics shows some effects can cross (§5.3).

7.2 Mendel and the rediscovery of heredity

- **Gregor Mendel**, an Austrian monk, showed with garden peas (**1866**) that traits are carried by **separate factors** that **segregate** in forming sex cells and **assort independently**.
- Ignored for decades, his work was **rediscovered in 1900** by **de Vries, Carl Correns and Erich von Tschermak**.
- **Why it mattered**: inheritance is **particulate, not blending**, so a rare new variant is **not diluted** and can be selected, answering Jenkin (§6.4).
- **New vocabulary**: **Bateson** named **genetics** (1905); **Johannsen** coined **gene, genotype and phenotype** (1909); **Morgan's** fruit flies (from 1910) placed genes **on chromosomes**.

7.3 Mutation theory

- **Hugo de Vries** (*Die Mutationstheorie*, 1901–03): new species arise by **large, sudden, heritable changes (mutations)**; evolution proceeds by **saltation** (jumps), and selection only removes the unfit.
- **Strength**: he made **mutation the source of new variation**, which Darwin lacked.
- **Weaknesses**: most of his *Oenothera* "mutations" were later shown to be **chromosomal irregularities** (doubled chromosome sets, unusual chromosome rings), not ordinary gene changes. Real mutations are

mostly **small and often harmful or neutral**, and new species rarely arise in one jump. Richard Goldschmidt's later "**hopeful monsters**" (1940) found little support.

7.4 Isolation, orthogenesis and recapitulation

- **Isolation theory: Moritz Wagner** (1868) argued that **geographical isolation** is needed for new species, since interbreeding would otherwise erase local differences; Mayr later made it central (Unit 1.4b).
- **Orthogenesis** ("straight-line evolution"; term by Wilhelm Haacke, 1893, popularised by **Theodor Eimer** and later **H.F. Osborn**): evolution moves in **fixed directions** driven by an **inner force**, as in a supposed straight-line growth of the horse or the human brain. **Critique: George Gaylord Simpson** (*Horses*, 1951) showed horse evolution was a **branching bush** with reversals; long trends reflect **consistent selection**, not an inner drive.
- **Recapitulation (Haeckel's biogenetic law, 1866): "ontogeny recapitulates phylogeny"**, an embryo passing through its ancestors' adult stages. **Critique:** embryos resemble the **embryos** of related species, not their adults, as **Karl Ernst von Baer** had observed (1828); Haeckel's drawings exaggerated. Only a modest version survives, in **evolutionary developmental biology** ("evo-devo").

7.5 The "eclipse of Darwinism" and the road to synthesis

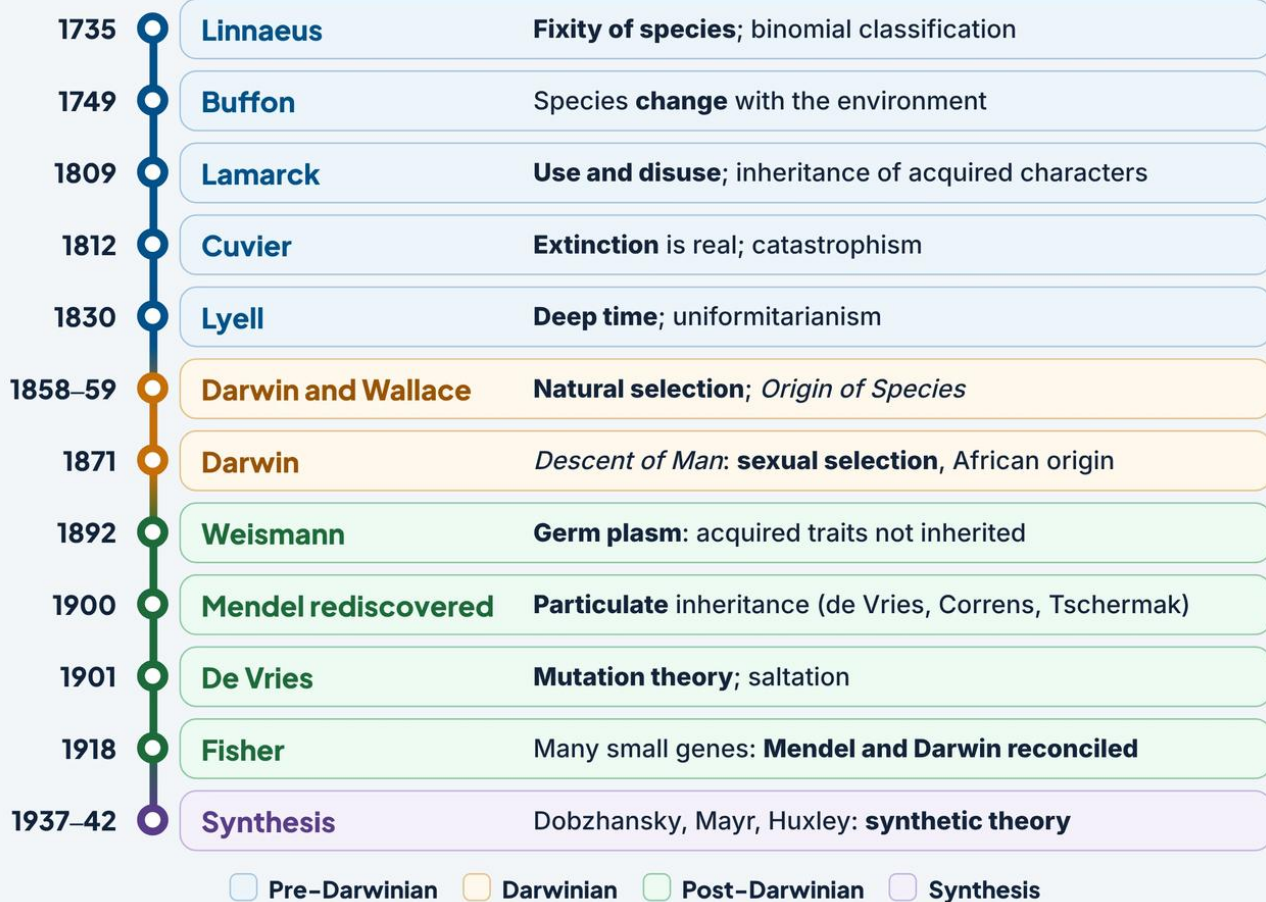
- From about **1880 to 1920** natural selection lost support: Julian Huxley's "**eclipse of Darwinism**".
- **Mutationists** (Bateson, de Vries) held that evolution proceeds by discrete jumps; **biometricians** (Karl Pearson, W.F.R. Weldon, followers of Galton) measured **continuous variation** such as height and defended gradual selection.
- **Resolution:** the British statistician **R.A. Fisher** showed (1918) that **many genes of small effect** produce smooth, continuous variation, so **Mendel and Darwin were compatible**. Population genetics, and then the **synthetic theory** of the 1930s–40s, followed (Unit 1.4b).

ANTHROPOLOGY · THEORIES OF EVOLUTION

From Fixed Species to the Synthesis

How explanations of organic evolution developed

TIMELINE



In simple words: **first change itself, then its cause, then its genetics.**

Anthropology

08 Key Terms

TERM	MEANING IN SIMPLE WORDS
Hominization	All the biological and cultural changes that turned an ape-like ancestor into modern humans
Hominin	A member of the human line after the split from chimpanzees (older books: "hominid")
Bipedalism	Habitually walking upright on two legs

TERM	MEANING IN SIMPLE WORDS
Encephalization	Brain growth beyond what body size predicts
Obstetrical dilemma	Conflict between a narrow pelvis for walking and a big-headed baby
Gene-culture co-evolution	A cultural practice changes which genes are favoured
Organic evolution	Change in inherited traits of populations over generations, producing new species
Homologous organs	Same basic structure and origin, different uses
Vestigial organ	A reduced remnant of an organ that worked in ancestors
Inheritance of acquired characters	Lamarck's idea that changes made in life pass to offspring
Natural selection	Individuals with favourable inherited variations leave more offspring
Pangenesis	Darwin's disproved idea that body particles carry acquired traits to sex cells
Weismann barrier	Bodily changes cannot reach the hereditary material of the sex cells
Orthogenesis	The rejected idea of straight-line evolution driven by an inner force

09 Contemporary Developments

- **Selection is still at work (2026):** nearly **16,000 ancient genomes** from West Eurasia showed **479 gene variants** strongly selected over the last 10,000 years, with selection **accelerating after farming** (Ali Akbari, David Reich and colleagues, *Nature*, April 2026): a cultural change intensified biological evolution.
- **Denisovans get a face (June 2025):** proteins and mitochondrial DNA from dental plaque identified the **Harbin cranium** (north-east China, at least **146,000 years old**) as **Denisovan** (*Science, Cell*).
- **Interbreeding dated (December 2024):** early modern human genomes showed **Neanderthal gene flow** lasting about **7,000 years**, centred on about **47,000 years ago** (*Nature, Science*).

10 Diagrams to Master

1. **Topic map: biological and cultural factors** around human evolution; below, the three phases of theory.
2. **Hominization sequence: bipedalism** (Laetoli 3.66 mya) → **tools** (Lomekwi 3.3 mya) → **brain expansion** (2 mya) → **long childhood** → **symbols**; label "feet before brains".
3. **Biocultural loop:** free hands → tools → better diet → larger brain → culture → new selection; mark **lactase (north-west India)** and **sickle cell (malaria)**.

4. **Lamarck vs Darwin giraffe:** stretch → longer neck → inherited against variation → selection → more long-necked young.
5. **Timeline:** Linnaeus 1735 → Lamarck 1809 → Lyell 1830 → Darwin and Wallace 1858–59 → Weismann 1892 → Mendel rediscovered 1900 → de Vries 1901 → Fisher 1918 → synthesis.

11

Revision Grid

SCHOLAR	WORK (YEAR)	KEY IDEA
Lamarck	<i>Philosophie Zoologique</i> (1809)	Use and disuse; acquired characters inherited
Darwin	<i>Origin of Species</i> (1859); <i>Descent of Man</i> (1871)	Natural selection; sexual selection; African origin
Galton	Transfusions (1871)	Disproved pangenesis
Weismann	Mouse tails (1889); <i>Das Keimplasma</i> (1892)	Germ plasm; Weismann barrier
Mendel	1866; rediscovered 1900	Particulate inheritance
De Vries	<i>Die Mutationstheorie</i> (1901–03)	Mutation; saltation
Fisher	1918	Many small genes reconcile Mendel and Darwin
Washburn	"Tools and Human Evolution" (1960)	Tools shaped the body; obstetrical dilemma
Childe	<i>Man Makes Himself</i> (1936)	Humans create their own conditions
Wrangham	<i>Catching Fire</i> (2009)	Cooking hypothesis
Boyd and Richerson	1985	Dual inheritance
Livingstone	1958	Farming, malaria, sickle cell
Lasker	1969	Levels of adaptability
Goodman and Leatherman	1998	Political economy in human biology



Previous Year Questions



- 2026 Human evolution is a product of both biological and cultural factors. Elaborate.
- 2025 How political economy is integrated with ecological and adaptability perspectives in bio-cultural anthropology?
- 2025 Critically discuss the centrality of the African continent in the narrative of human evolution.
- 2024 Critically discuss the synergistic effect of biological and cultural factors in human evolution.
- 2023 What is hominization process? Discuss the major trends in human evolution with the help of suitable examples and illustrations.
- 2021 "The bio-cultural approach is the hallmark of Biological Anthropology." Explain. (in about 150 words).
- 2018 Explain the biological changes that made human beings capable of making culture.
- 2016 Elucidate the biological and cultural factors in human evolution.
- 2015 Elucidate the biological and cultural factors in Human Evolution.
- 2015 Critically examine Darwin's theory of evolution in understanding evolution.
- 2010 What are the major craniofacial changes that occurred during Hominid evolution ? Discuss.
- 2009 Distinguish between 'chronological age' and 'biological age'.
- 2002 What is Organic Evolution ? Explain the theories of evolution.
- 1997 Write a note on Biological evolution of early man.
- 1996 Discuss the geographic distribution and taxonomic issues concerning early hominids. Elucidate evidence in support of alternative positions.
- 1992 What is organic evolution ? Give evidences for human evolution.
- 1991 Write a note on Social-cultural evolution.

- 1991 What is organic evolution ? Describe the theories of organic evolution.
- 1990 Evolution is biological and sociocultural. Discuss.
- 1989 Elucidate the Theory of Evolution as put forward by Lamarck.
- 1989 Write a note on Meaning of Biological and Socio-cultural Evolution.
- 1987 'Evolution is biological and cultural'. Discuss.
- 1987 How is Darwinism different from Lamarckism ? Critically evaluate their contribution to synthetic theory of evolution.
- 1986 Write a note on Biological evolution and concept of new physical anthropology.
- 1986 Write a note on Lamarckism and Darwinism.
- 1986 Write a note on Relationship between cultural evolution and biological evolution.
- 1985 Write a note on Theory of organic evolution.
- 1984 Distinguish between : Lamarckism and Darwinism.
- 1984 Discuss the inter-relation of biological and cultural dimensions in Human Evolution.
- 1983 Sociocultural evolution is not parallel to biological evolution in man. Substantiate.
- 1983 Darwinism is not adequate to explain [evolution. Give your] view. (wording garbled in all available copies)
- 1982 Man has made himself. Justify.
- 1982 Human evolution has cultural dimensions. Elaborate.
- 1981 Has biological evolution caused [ceased] in man? Briefly substantiate your answer. (200 words).
- 2025 Unit 1.7: Write notes on: Mitochondrial DNA and human evolution.
- 2025 Unit 1.5: Discuss the Miocene hominoid remains and their significance in evolution.

2020

Unit 1.4: Elucidate how Darwin and Post-Darwin theories of evolution resulted in the development of Synthetic theory of evolution.

2019

Unit 1.5: Discuss the evolutionary significance of bipedalism and erect posture.

2017

Unit 1.5: Bring out the comparative anatomical features of man and apes. Discuss their evolutionary significance.

2013

Unit 1.4: Discuss how Neo-Darwinism integrated the fields of Genetics and Evolution. (250 words).

2012

Unit 9.3: Write a note in 150 words on How 'natural selection' acts on variation?

2009

Unit 9.3: Write a note on Natural selection in humans.

1994

Unit 1.4: How Darwinism differs from the synthetic theory of organic evolution ?

LOTUSARISE IAS

UNIT 09

8 CHAPTERS

Human Genetics, Race and Ecological Anthropology

Human Genetics: Methods and Application: Methods for study of genetic principles in man-family study (pedigree analysis, twin study, foster child, co-twin method, cytogenetic method, chromosomal and karyo-type analysis), biochemical methods, immunological methods, D.N.A. technology and recombinant technologies. Mendelian genetics in man-family study, single factor, multifactor, lethal, sub-lethal and polygenic inheritance in man. Concept of genetic polymorphism and selection, Mendelian population, Hardy-Weinberg law; causes and changes which bring down frequency-mutation, isolation, migration, selection, inbreeding and genetic drift. Consanguineous and non-consanguineous mating, genetic load, genetic effect of consanguineous and cousin marriages. Chromosomes and chromosomal aberrations in man, methodology. (a) Numerical and structural aberrations (disorders). (b) Sex chromosomal aberration- Klinefelter (XXY), Turner (XO), Super female (XXX), intersex and other syndromic disorders. (c) Autosomal aberrations- Down syndrome, Patau, Edward and Cri-du-chat syndromes. (d) Genetic imprints in human disease, genetic screening, genetic counseling, human DNA profiling, gene mapping and genome study. Race and racism, biological basis of morphological variation of non-metric and characters. Racial criteria, racial traits in relation to heredity and environment; biological basis of racial classification, racial differentiation and race crossing in man. Age, sex and population variation as genetic marker: ABO, Rh blood groups, HLA Hp, transferrin, Gm, blood enzymes. Physiological characteristics-Hb level, body fat, pulse rate, respiratory functions and sensory perceptions in different cultural and socio-economic groups. Concepts and methods of Ecological Anthropology: Bio-cultural Adaptations—Genetic and Nongenetic factors. Man's physiological responses to environmental stresses: hot desert, cold, high altitude climate. Epidemiological Anthropology: Health and disease. Infectious and non-infectious diseases, Nutritional deficiency related diseases.

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ANTHROPOLOGY OPTIONAL

PAPER I · PART B

Hardy–Weinberg Law and Genetic Polymorphism: Gene Frequency, Drift and Consanguinity


PYQ RADAR 48 questions · last asked 2026

Unit 9.2 followed genes through **families**; this unit follows them through **populations**. A caste, a tribe or an island community can be treated as one pool of genes, and the question becomes: what keeps the proportions of alleles steady, and what changes them? The answer joins Mendel to Darwin. The forces of evolution were introduced in Unit 1.4b (in brief: mutation and gene flow bring in variation, drift and selection sort it, isolation lets populations diverge); here they are measured, applied to Indian populations, and linked to marriage rules.

SYLLABUS 9.3 Concept of genetic polymorphism and selection, Mendelian population, Hardy-Weinberg law; causes and changes which bring down frequency – mutation, isolation, migration, selection, inbreeding and genetic drift. Consanguineous and nonconsanguineous mating, genetic load, genetic effect of consanguineous and cousin marriages.

CONTENTS

01	1. Mendelian Population and Gene Pool	02	2. The Hardy–Weinberg Law
03	3. Genetic Polymorphism and Selection	04	4. Forces That Change Gene Frequency
05	5. Consanguineous and Non-consanguineous Mating	06	6. Coefficient of Inbreeding and the Genetic Effects of Cousin Marriage
07	7. Genetic Load	08	8. Worked Examples
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ANTHROPOLOGY · POPULATION GENETICS

Genes in Populations

What keeps gene frequencies steady, and what changes them

TOPIC MAP

GENE POOL · EQUILIBRIUM · FORCES · MARRIAGE

Mendel's rules applied to whole communities

Mendelian population

Shared **gene pool**; deme; panmixiaEndogamous **castes and tribes****81** Indian founder groups (2017)

Hardy–Weinberg law

 $p^2 + 2pq + q^2 = 1$ (1908)Five **conditions**; null modelCarrier frequency; **chi-square**

Polymorphism and selection

Ford 1940; the **1 per cent** rule**Balanced**: sickle-cell and malaria**Transient**; neutral (Kimura)

Forces of change

Mutation, **gene flow**, isolation**Selection**, **drift** (founder, bottleneck)**Inbreeding** changes genotypes only

Consanguinity

India **10.8%**; Tamil Nadu **28%****F**: cousins 1/16, uncle–niece 1/8About **3.5%** excess child deaths

Genetic load

Muller 1950; **Crow** 1958**Mutational**, **segregational**, incompatibilityAbout **1.4 lethal equivalents**In simple words: **populations keep or lose gene forms through chance, choice and survival.**

Anthropology

01

1. Mendelian Population and Gene Pool

IN SIMPLE WORDS

A Mendelian population is a group of people who marry among themselves and so share one common stock of genes. Population genetics counts how often each form of a gene occurs in that stock. In India, an endogamous caste or tribe often behaves as such a population.

An example first. The **Vysya**, a trading community of Andhra Pradesh and Telangana of over 3 million people, have married within their community for centuries. A rare allele carried by a few early ancestors has therefore stayed inside the group and multiplied: the Vysya show about a **100-fold higher** rate of **butyrylcholinesterase deficiency** (a dangerous reaction to certain anaesthetics) than other Indians (**Nathan Nakatsuka** and

colleagues, 2017). The Vysya are a **Mendelian population**: their marriages define the boundary of a gene pool.

Definitions.

- **Mendelian population**: the term of the Russian-American geneticist **Theodosius Dobzhansky**: a **reproductive community** of sexual, cross-fertilising individuals who **share a common gene pool**. The largest Mendelian population is the **species**; the smallest is the local breeding unit.
- **Deme**: a **local** interbreeding population, the smallest Mendelian unit. A deme in which every individual is equally likely to mate with any other is **panmictic** (the American geneticist **Sewall Wright**'s term **panmixia**).
- **Gene pool**: the **sum of all alleles** of all genes carried by the members of a population at one time. Each generation draws its genes from this pool, so the pool, not the individual, is what **evolves**.
- **Allele frequency** (gene frequency): the **proportion** of all copies of a gene in the pool that are of one type.
Genotype frequency: the proportion of **individuals** with a given genotype.

Properties of a Mendelian population: **continuity in time** (the pool persists while individuals die), a **boundary set by mating** (in humans by culture as well as geography), and a **measurable structure** of allele and genotype frequencies.

The Indian application. Because caste and tribal **endogamy** restricts marriage, Indian groups living side by side can differ genetically.

- The Indian anthropologist **Irawati Karve** and **K.C. Malhotra** compared **eight endogamous groups of the same rank** in Maharashtra (*Current Anthropology*, 1968) and found significant differences between them, a pattern Malhotra later documented among the **Dhangar** (shepherd) castes.
- On a national scale, **Nakatsuka** and colleagues (*Nature Genetics*, 2017) studied over **2,800** people from over **260** South Asian groups and found **81** groups that descend from **founder events** stronger than those of Finns and Ashkenazi Jews; **14** of them number over a million people.
- **Why it matters**: each endogamous group can carry its **own recessive diseases**, which calls for **community-specific** carrier screening.

02 2. The Hardy–Weinberg Law

IN SIMPLE WORDS

The Hardy–Weinberg law says that if nothing disturbs a large, randomly mating population, the proportions of each gene form stay the same in every generation. It is the “no-change” baseline: when a real population departs from it, some force is at work.

2.1 Origin and statement

In **1908** the English mathematician **G.H. Hardy** (answering the geneticist **R.C. Punnett**) and the German physician **Wilhelm Weinberg** independently showed why a **dominant** allele does not automatically spread.

The geneticist **Curt Stern** (1943) drew attention to Weinberg's priority, and the rule came to be called the **Hardy-Weinberg law**.

Statement: in a **large, randomly mating** population, with **no mutation, migration or selection**, (1) **allele frequencies remain constant** from generation to generation, and (2) **genotype frequencies** reach **p^2 , $2pq$ and q^2** after **one** generation of random mating and stay there.

2.2 How it works

Let a gene have two alleles, **A** with frequency **p** and **a** with frequency **q**, so that **$p + q = 1$** .

- With random mating, every gamete is drawn at random from the pool: a sperm carries A with probability p and a with probability q, and so does an egg.
- The chance that a child gets **A from both** is $p \times p = p^2$; **a from both**, q^2 ; **one of each**, $pq + qp = 2pq$.
- So **$(p + q)^2 = p^2 + 2pq + q^2 = 1$** .
- **Why frequencies do not change:** the next generation's A alleles come from AA (all) and Aa (half), so the new $p = p^2 + \frac{1}{2}(2pq) = p(p + q) = p$. Mendelian segregation only **reshuffles** alleles; it does not create or destroy them.

ANTHROPOLOGY · POPULATION GENETICS

The Hardy–Weinberg Principle

Random union of gametes from a pool with $p = 0.7$ and $q = 0.3$

WORKED

egg \ sperm	A ($p = 0.7$)	a ($q = 0.3$)
A (0.7)	AA $p^2 = 0.49$	Aa $pq = 0.21$
a (0.3)	Aa $pq = 0.21$	aa $q^2 = 0.09$

Why nothing changes

Next generation's $A = AA + \frac{1}{2} Aa$
 $= 0.49 + 0.21 = \mathbf{0.70}$
 Same p : segregation only **reshuffles** alleles.

READING IT BACKWARDS

If 9% show a recessive trait, $q = \sqrt{0.09} = 0.3$,
 and **42%** are hidden carriers.

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

$$0.49 + 0.42 + 0.09 = 1$$

CONDITIONS FOR EQUILIBRIUM (AND THE FORCE THAT BREAKS EACH)

Large population	broken by genetic drift
Random mating	broken by inbreeding, endogamy
No mutation	broken by mutation
No migration	broken by gene flow
No selection	broken by natural selection

In simple words: **without disturbing forces, gene proportions stay the same each generation.**

Anthropology

Extensions:

- **Three alleles (ABO):** with frequencies p (I^A), q (I^B) and r (i), $(p + q + r)^2 = 1$; group O = r^2 , A = $p^2 + 2pr$, B = $q^2 + 2qr$, AB = $2pq$ (Worked Example 5).
- **X-linked genes:** in males the **genotype frequency equals the allele frequency** (q), in females it is q^2 . If 8 per cent of men are colour-blind ($q = 0.08$), only about **0.64 per cent** of women are, which matches observation.

2.3 Conditions for genetic equilibrium

The table lists the assumptions; the last column shows which evolutionary force breaks each one.

ASSUMPTION	WHY IT IS NEEDED	FORCE THAT BREAKS IT
Large population	Chance cannot change frequencies much	Genetic drift
Random mating (panmixia)	Gametes combine by chance	Inbreeding, assortative mating, endogamy
No mutation	No allele is converted into another	Mutation
No migration	No alleles enter or leave	Gene flow
No selection	All genotypes survive and reproduce equally	Natural selection
Sexual, diploid, non-overlapping generations	Simplifies the arithmetic	Real generations overlap (minor effect)

The table shows why the law is the **foundation of population genetics**: each condition corresponds to one evolutionary force, so a population that departs from equilibrium tells us which force to look for. No real population meets all the conditions, yet most human populations come **close** for most genes, because the forces act slowly.

2.4 Importance and uses

- **A null model:** a population stays in equilibrium unless a force acts, so the law tells us when **evolution** (a change in allele frequency) has occurred.
- **Estimating hidden carriers:** from **affected** people (q^2) we calculate **carriers** ($2pq$), whom the eye cannot see (Worked Example 2); this guides screening, as in India's sickle-cell mission.
- **Testing populations:** observed counts are compared with expected ones by a **chi-square test** (Worked Examples 3 and 4); an **excess of homozygotes** points to inbreeding, endogamous subgroups or selection.
- **Anthropology:** allele frequencies of blood groups, haemoglobins or DNA markers measure **genetic distance** between castes and tribes.
- **Why a dominant allele does not spread:** dominance concerns how an allele **shows**, not how it is **transmitted**; without selection, a dominant allele at 10 per cent stays at 10 per cent, the point Hardy made in 1908.

3. Genetic Polymorphism and Selection

IN SIMPLE WORDS

A gene is polymorphic when two or more of its forms are each fairly common in a population, too common to be explained by fresh mutation alone. Something, usually natural selection, keeps them. Blood groups, haemoglobin types and the ability to taste PTC are familiar human examples.

An example first. In the **Nilgiri Hills** of Tamil Nadu in **1952**, **Hermann Lehmann** and **Marie Cutbush** found the **sickle-cell gene** (HbS) among tribal people. It has since been found across central India, where the **sickle-cell trait** reaches **1 to 40 per cent** of people in different tribal groups. HbS is harmful in double dose, yet it is common. Its frequency is too high to be renewed by mutation: it is kept by **selection**. This is the essence of **genetic polymorphism**.

3.1 Definition and levels

- **Definition** (the English geneticist **E.B. Ford**, 1940): “the occurrence together in the same locality of two or more discontinuous forms of a species in such proportions that the rarest of them cannot be maintained merely by recurrent mutation.”
- **The 1 per cent rule of thumb:** a locus is called **polymorphic** when its **rarest common allele exceeds 1 per cent**; rarer variants are attributed to mutation. A population with only one form is **monomorphic**.
- **Polymorphic traits are discrete** (taster or non-taster; group A or O) and largely genetic; continuous traits such as stature show **variation**, not polymorphism in Ford’s sense.

Levels at which polymorphism is found:

- **Morphological** (PTC tasting, ear-lobe attachment) and **cellular:** **ABO, Rh, MN** blood groups and **HLA** (human leucocyte antigens), the most polymorphic system known.
- **Protein:** **haemoglobin** variants (HbS, HbE, HbD), **G6PD**, **haptoglobin**. In **1966 Harry Harris** (Britain) and **Richard Lewontin and J.L. Hubby** (USA) used **electrophoresis** (separating proteins in an electric field) to show that about **a third** of protein loci are polymorphic.
- **Chromosomal:** inversions and heteromorphisms visible under the microscope.
- **DNA:** **single-nucleotide polymorphisms** (SNPs), **VNTRs** (variable numbers of tandem repeats) and **copy-number variants:** the basis of DNA fingerprinting (Unit 9.1).

3.2 Types of polymorphism

Balanced polymorphism is kept **stable** by selection.

- **Heterozygote advantage** (overdominance): the heterozygote is **fitter than both homozygotes**, so both alleles are kept. **Sickle-cell** is the classic case (§3.3).
- **Other malaria-linked cases:** **β -thalassaemia** (proposed as malaria-protective by the British geneticist **J.B.S. Haldane**, 1949); **HbE** in **north-east India**; **G6PD deficiency**.

- **Frequency-dependent selection** (a form favoured **while rare**, as rare HLA types may resist local pathogens) and **varying environments** also keep polymorphisms stable. **Sex** itself is the most stable polymorphism: selection keeps the two sexes near 1 : 1.
- **Disease and ABO**: people of group **O** suffer more **severe cholera**, which may explain the low frequency of O in the cholera-endemic **Ganges delta** (Roger Glass and colleagues, 1985).

Transient polymorphism is a **temporary** stage while one allele **replaces** another under **directional selection**.

- **Peppered moth** (*Biston betularia*): dark forms rose in sooty industrial England (**H.B.D. Kettlewell's** experiments, 1950s) and fell again after clean-air laws.
- **Lactase persistence** (digesting milk sugar as adults) spread among dairying peoples over the last few thousand years.

Neutral polymorphism: the Japanese geneticist **Motoo Kimura** (1968) argued that **most molecular variation** is **selectively neutral** and changes by **drift**; many DNA polymorphisms are of this kind.

The table compares the three types; the last row gives the quickest way to tell them apart.

FEATURE	BALANCED	TRANSIENT	NEUTRAL
Cause	Selection keeps both alleles	Selection replaces one allele	Drift ; no selection
Duration	Stable , long-lasting	Temporary	Variable
Mechanism	Heterozygote advantage; frequency dependence	Directional selection	Random sampling
Examples	Sickle-cell , thalassaemia, HbE, G6PD	Peppered moth , lactase persistence	Most SNPs , many blood-group differences
Test	Frequencies steady over generations	Frequencies moving one way	Frequencies wander, linked to population size

The table shows that the same visible pattern, two common forms, can arise from very different causes. Only **long-term data** or knowledge of **fitness** reveals which kind a polymorphism is.

COMMON CONFUSION

Is a balanced polymorphism a sign that the harmful allele is “useful”? Only in a **particular environment**. HbS helps carriers **where falciparum malaria is common**; where malaria is controlled, the advantage vanishes and the allele slowly declines (**relaxed selection**). African Americans, removed from malaria for centuries, carry HbS at lower frequencies than West Africans, partly for this reason and partly through admixture.

3.3 Sickle-cell: a balanced polymorphism worked out

ANTHROPOLOGY · POPULATION GENETICS

Sickle-Cell: A Balanced Polymorphism

Malaria removes one homozygote, anaemia the other; the carrier wins

SELECTION

HbAA

Normal haemoglobin
Dies more often of falciparum malaria

fitness $1 - s$
e.g. 0.85

HbAS (carrier)

Healthy
Protected against severe malaria

fitness 1
the fittest

HbSS

Sickle-cell anaemia
Historically died young

fitness $1 - t$
e.g. 0.15

$$\text{Equilibrium } q = s / (s + t) = 0.15 / 1.00 = 0.15$$

carriers $2pq = 25.5\%$ · patients $q^2 = 2.25\%$ · segregational load $\approx 13\%$

India

Found in the **Nilgiris**, 1952

Trait in **1-40%** of tribal groups

Mission: **20.6 lakh** carriers found by 2026

Relaxed selection

Malaria controlled → **no carrier advantage**

HbS then **declines slowly**

Allison 1954: HbS highest where malaria worst

In simple words: **a harmful gene stays common when carriers survive malaria best.**

Anthropology

- **The three genotypes:**
 - **HbAA:** normal haemoglobin but **vulnerable to falciparum malaria**.
 - **HbSS:** **sickle-cell anaemia**, historically fatal in childhood without care.
 - **HbAS:** **sickle-cell trait**, healthy and **protected against severe malaria**, because infected cells sickle and are removed by the spleen before the parasite multiplies.
- **The proof:** the British physician **Anthony Allison** (1954) showed in East Africa that HbS frequencies were **highest where malaria was intense** and that carriers had **fewer parasites**.
- **The arithmetic:** if the fitness of AA is reduced by s (malaria) and that of SS by t (anaemia), selection reaches equilibrium when $q = s / (s + t)$. With $s = 0.15$ and $t = 0.85$, $q = 0.15$ and **25.5 per cent** of people are carriers (Worked Example 8), close to the levels in parts of central India's tribal belt.
- **Natural selection on variation:** this is how selection acts on genetic variety. It does not create alleles; it **changes their frequencies** through the **differential reproduction** of genotypes, measured by **fitness (w)** and the **selection coefficient ($s = 1 - w$)**.
 - **Against a dominant** harmful allele it is **fast**; **against a rare recessive**, **very slow**, because almost all copies hide in carriers (worked in Unit 1.4b).

- For heterozygotes it produces a **stable equilibrium**; against heterozygotes an **unstable** one, as in Rh incompatibility between mother and foetus.
- **Other human cases: skin colour** and ultraviolet light (Unit 9.2); the **EPAS1** variant protecting **Tibetans** at altitude, inherited from **Denisovans** (Huerta-Sánchez and colleagues, 2014).

04 4. Forces That Change Gene Frequency

IN SIMPLE WORDS

Six forces can push a population away from Hardy–Weinberg equilibrium: mutation, migration, isolation, selection, inbreeding and genetic drift. Some change how often an allele occurs; inbreeding changes only how alleles are paired. Together they explain why human populations differ.

ANTHROPOLOGY · POPULATION GENETICS

Forces That Change Gene Frequency

What each force does, how fast, and an example

FORCES

FORCE	WHAT IT DOES	EXAMPLE
Mutation	Creates new alleles ; very slow $p_n = p_0(1 - u)^n$	Sickle-cell: one base change, GAG → GTG
Gene flow	Mixes pools; fast $\Delta q = m(q_m - q_0)$	Siddi : about 67% African ancestry
Isolation	Blocks gene flow; lets groups diverge	Caste endogamy ; Onge, Jarawa
Selection	Directional ; the only force that adapts	HbS and malaria; EPAS1 in Tibetans
Drift	Random ; strong when N is small $\sigma = \sqrt{pq / 2N}$	Vysya founder effect; Pingelap
Inbreeding	Changes genotypes , not alleles; more homozygotes	Cousin marriage: $F = 1/16$

Pull populations together

Gene flow (and mutation adds variety within)

Push populations apart

Drift, isolation, local selection: India's thousands of endogamous groups

In simple words: **new genes come from mutation; chance, choice and survival sort them.**

Anthropology

4.1 Mutation

- **What it is:** a heritable change in DNA, the **only source of new alleles** (kinds in Unit 1.7).
- **Mutation pressure:** if A mutates to a at rate u per generation, after n generations $p_n = p_0(1 - u)^n$. Rates are about **1 in 100,000 to 1 in a million** per gene, so $p = 0.90$ falls only to **0.891** in **1,000 generations** (about 25,000 years) at $u = 10^{-5}$.
- **Two-way mutation:** if a also mutates back to A at rate v , an **equilibrium** is reached at $q = u / (u + v)$.
- **Mutation–selection balance:** a harmful recessive is removed by selection as fast as mutation creates it when $q \approx \sqrt{(u/s)}$, which explains why most disease alleles stay **rare but never vanish**.

4.2 Migration and gene flow

- **What it is:** movement of individuals **and their genes** between populations followed by interbreeding; called **admixture** or **hybridisation** when distinct populations merge.
- **The formula:** if a fraction m of a population's parents are migrants with allele frequency q_m , the new frequency is $q_1 = (1 - m)q_0 + m q_m$, so the change is $\Delta q = m(q_m - q_0)$. Gene flow is **fast** compared with mutation: $m = 0.2$ can move q from 0.10 to **0.082** in one generation (Worked Example 7).
- **Effects:** gene flow makes populations **more alike** (reduces differences **between** them) and adds variety **within** each. It is the main **counterweight to drift and isolation**.
- **Clines:** gradual change in allele frequency across geography, such as the fall of **blood group B** from Central Asia westwards across Europe.
- **Indian cases:**
 - The **Siddi** of Karnataka and Gujarat, descendants of Africans brought by the Portuguese, carry about **67 per cent African** and 33 per cent Indian ancestry, from admixture about **8 generations** (some 200 years) ago (**Anish Shah** and colleagues, 2011).
 - Most Indian groups descend from mixture of **Ancestral North Indians** and **Ancestral South Indians** (David Reich and colleagues, 2009), followed by **endogamy** that froze the mixture about **2,000 years** ago (Moorjani and colleagues, 2013).

4.3 Isolation

- **What it is:** any barrier that **prevents gene flow**: geographical (mountains, seas, forests), ecological, and in humans **social and cultural** (caste and tribal endogamy, religion, language).
- **Effect:** isolated populations **diverge**, because drift and local selection act on each separately; prolonged **reproductive isolation** in other species leads to **speciation** (isolating mechanisms in Unit 1.4b).
- **Human isolates** such as the **Onge** and **Jarawa** of the Andamans show **low diversity**; and Indian castes in one village behave as partly **isolated gene pools**, social distance replacing geographical distance.

4.4 Natural selection

Selection is the **only force that adapts** a population to its environment (forms and the sickle-cell case in §3). Its human agents are **disease**, **climate** and **diet**; modern medicine **relaxes** it.

4.5 Genetic drift

- **What it is:** **random** change in allele frequency due to **chance** in which individuals happen to reproduce; named the **Sewall Wright effect** after the geneticist who modelled it (1931).
- **Size matters:** the spread of chance change per generation is $\sigma = \sqrt{pq / 2N}$. With $p = q = 0.5$, σ is **0.05** in a population of 50 but **0.005** in one of 5,000 (Worked Example 9). Drift is strong only in **small** populations.
- **Outcomes:** an allele is eventually **lost** or **fixed**, with a chance of fixation **equal to its present frequency**; drift **reduces variation within** populations and **increases differences between** them.
- **Founder effect** (the German-American biologist **Ernst Mayr's** "founder principle", 1942): a new population started by a **few founders** carries only their alleles.
 - The **Old Order Amish** of Lancaster County, Pennsylvania, have a high frequency of **Ellis-van Creveld syndrome** (dwarfism with extra fingers), traced to one founding couple (Victor McKusick, 1964).
 - **Tristan da Cunha** in the **South Atlantic**, settled by a few families in the nineteenth century, has high **retinitis pigmentosa** and **asthma**.
 - In India, the **81 founder groups** of Nakatsuka and colleagues (§1) are the largest known set; the **Vysya** case shows the medical consequence.
- **Bottleneck:** a population **shrinks sharply** and recovers from a few survivors. A typhoon about **1775** left some 20 survivors on **Pingelap** atoll in Micronesia; today about **1 in 10** islanders has **achromatopsia** (total colour blindness). The Andamanese groups, reduced to a few hundred after colonial contact, show bottleneck effects.

4.6 Inbreeding and non-random mating

Inbreeding (mating of relatives) and **assortative mating** do **not change allele frequencies**; they change **how alleles are combined**, raising **homozygotes** at the expense of heterozygotes. Inbreeding thus **exposes hidden recessives** to selection, which can then change allele frequencies. It is treated in §5 and §6.

The table summarises how each force acts; notice that only mutation creates new alleles and only selection is directional.

FORCE	CHANGES ALLELE FREQUENCY?	VARIATION WITHIN A POPULATION	DIFFERENCES BETWEEN POPULATIONS	SPEED
Mutation	Yes, slightly	Increases	Increases	Very slow
Gene flow	Yes	Increases	Decreases	Fast
Isolation	Indirectly (lets others act)	—	Increases	Slow
Selection	Yes, directionally	Decreases (or keeps, if balancing)	Increases (different environments)	Depends on s
Drift	Yes, randomly	Decreases	Increases	Fast if N small

FORCE	CHANGES ALLELE FREQUENCY?	VARIATION WITHIN A POPULATION	DIFFERENCES BETWEEN POPULATIONS	SPEED
Inbreeding	No (genotypes only)	Fewer heterozygotes	Increases	Fast in close kin

The table explains human diversity in one line: **gene flow pulls populations together while drift, isolation and local selection push them apart**, and the balance between these sets how different two populations become. India, with thousands of endogamous groups, is an extreme case of the “pushing apart” side.

05

5. Consanguineous and Non-consanguineous Mating

IN SIMPLE WORDS

Consanguineous marriage is marriage between relatives, such as cousins or an uncle and niece. Because relatives share genes from a common ancestor, their children are more likely to inherit two copies of the same harmful recessive gene. In South India such marriages are a long-standing preferred custom.

An example first. In much of **Tamil Nadu and Karnataka**, a man's preferred bride has long been his **mother's brother's daughter** or, in some communities, his **elder sister's daughter**. According to the **National Family Health Survey 2019–21** (NFHS-5), about **28 per cent** of marriages in **Tamil Nadu** and about **27 per cent** in **Karnataka** were between blood relatives, against **10.8 per cent** for India as a whole and only **4.4 per cent** in Kerala (Tamil Nadu *Economic Survey 2025–26*). The same custom that builds alliances between families also concentrates genes.

5.1 Definitions

- **Consanguineous mating:** marriage between people who share at least one **recent common ancestor**; in clinical genetics, couples related as **second cousins or closer** (coefficient of inbreeding $F \geq 1/64 = 0.0156$).
- **Non-consanguineous mating:** marriage between people **not known** to be related. It may be **random** (panmixia) or **non-random**:
 - **Positive assortative mating:** like with like in **phenotype**: by stature, skin colour, education, or deafness among the deaf. It increases **homozygosity** for the genes behind the chosen trait only.
 - **Negative assortative (disassortative) mating:** unlike with unlike; rare in humans as a conscious rule, though it may occur for some immune genes.
- **Forms of consanguineous marriage in India** (Unit 2.3 gives the social side: cross-cousin marriage builds alliances between descent groups, as in Dravidian kinship, while parallel-cousin marriage keeps property inside the lineage):
 - **Cross-cousin** (MBD, FZD) and **uncle–niece** (elder sister's daughter) marriage in the **south**; **parallel-cousin** (FBD) marriage among many **Muslim** communities.

- **Incest** (parent–child, brother–sister) is prohibited almost everywhere, though sibling marriage occurred in some royal lines (Egyptian pharaohs; Inca rulers).

The Indian pattern in numbers:

- **NFHS-4** (2015–16): **9.9 per cent** of marriages consanguineous; **South 23 per cent, North-East 3.1 per cent**; first cousins **8.7 per cent**, second cousins 0.7, uncle–niece **0.6 per cent** (Santosh Kumar Sharma and colleagues, 2020).
- **Trend**: prevalence fell from **14.4 per cent** (1992–93) to **10.8 per cent** (2019–21); **97 of 707 districts** exceed 20 per cent, the top five in Karnataka and Tamil Nadu (Kumari and colleagues, 2026).
- **Who**: commoner among **Muslims** (about 16 per cent) than Hindus (about 10), and among the **less educated** and **younger** women, though in the south it cuts across class and caste. The Indian geneticist **L.D. Sanghvi** (1966) had documented high inbreeding in **Andhra Pradesh**.
- **Reasons**: keeping **property** in the family, **lower dowry**, **security for the bride** among known in-laws, and **Dravidian kinship terms** that mark cross cousins as marriageable.

COMMON CONFUSION

Is inbreeding the same as consanguinity? Not exactly. **Consanguinity** is a **social and genealogical** fact: the couple are known relatives. **Inbreeding** is the **genetic** result: the chance that a child receives two copies of an ancestor's allele. A small **isolate** or an endogamous caste can be inbred **without any known consanguinity**, simply because all its members descend from few founders.

06 6. Coefficient of Inbreeding and the Genetic Effects of Cousin Marriage

IN SIMPLE WORDS

The coefficient of inbreeding measures how closely the parents are related: the chance that their child gets two identical copies of a gene from one ancestor. For first cousins it is 1/16; for uncle and niece 1/8. The closer the relationship, the higher the risk of recessive disease.

6.1 The coefficient of inbreeding (F)

- **Definition** (Sewall Wright, 1922): **F** is the **probability that the two alleles at a locus in an individual are identical by descent**, that is, copies of the **same** ancestral allele.
- **The path method**: draw the path from the child's father up to a **common ancestor** and down to the mother. If **n** is the number of people in the path (counting both parents and the ancestor), that ancestor contributes $(\frac{1}{2})^n$. Add the contributions of all common ancestors: **$F = \Sigma (\frac{1}{2})^n$** .
- **First cousins**: father → his parent → **grandparent** → her parent → mother = **5** people, so each grandparent gives $(\frac{1}{2})^5 = 1/32$; two shared grandparents give **$1/16 = 0.0625$** .

- **Uncle–niece:** uncle → **his parent** → niece's parent → niece = **4 people**; $(\frac{1}{2})^4 = 1/16$ per ancestor; two ancestors give $1/8 = 0.125$.

ANTHROPOLOGY · POPULATION GENETICS

The Coefficient of Inbreeding (F)

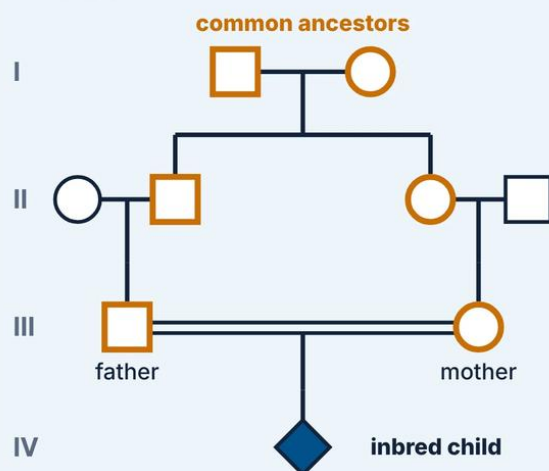
Count the people on each path through a common ancestor

WORKED

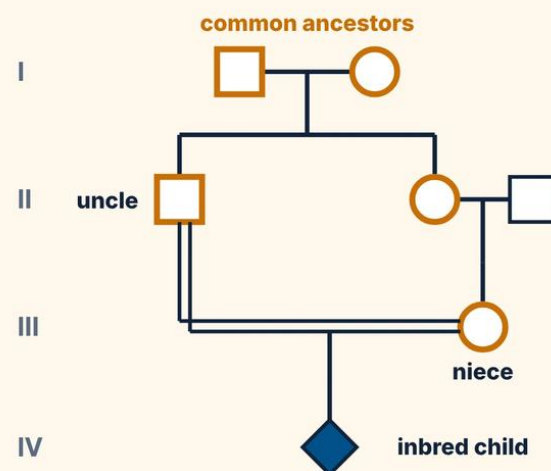
$$F = \sum (\frac{1}{2})^n$$

n = people on the path from father, up to a common ancestor, down to mother

First cousins



Uncle and niece

**1/4**

brother–sister

1/8

uncle–niece

1/16

first cousins

1/64

second cousins

In simple words: **shorter paths mean closer kin and a higher chance of two identical genes.**

Anthropology

The table gives the values most often needed; note how each step away from close kinship halves or quarters the risk.

RELATIONSHIP OF PARENTS	F	AS A DECIMAL
Brother–sister; parent–child	1/4	0.25

RELATIONSHIP OF PARENTS	F	AS A DECIMAL
Uncle–niece; double first cousins; half-siblings	1/8	0.125
First cousins	1/16	0.0625
First cousins once removed	1/32	0.031
Second cousins	1/64	0.0156
Unrelated (random mating)	0	0

The table shows why **uncle–niece** marriage, common in parts of South India, carries **twice** the genetic risk of first-cousin marriage, and why second-cousin marriage adds very little.

6.2 What inbreeding does to genotypes

- With inbreeding, genotype frequencies become $AA = p^2 + Fpq$, $Aa = 2pq(1 - F)$, $aa = q^2 + Fpq$. Heterozygotes fall by the fraction F ; both homozygotes rise. Under the extreme of **self-fertilisation** in plants, heterozygosity halves every generation, from 0.5 to about 0.001 in ten generations.
- **The risk of a recessive disease** in a child of related parents is therefore $q^2(1 - F) + qF$. The extra term qF is large compared with q^2 when **q is small**:
 - For $q = 0.01$, first-cousin children are affected **7 times** as often as children of unrelated parents; uncle–niece children **13 times**.
 - For $q = 0.001$, the ratios are **63** and **126** (Worked Example 10).
- **Hence the rule:** the **rarer** a recessive disease, the **larger the share** of cases from consanguineous marriages. **Garrod** noticed this for alkaptonuria in 1902.

6.3 The observed effects

- **Mortality:** a meta-analysis of **69 populations** in 15 countries (**2.14 million** people) found **about 3.5 per cent more deaths** from late pregnancy to about 10 years among the children of first cousins than among children of unrelated parents (**Alan Bittles and Michael Black**, *PNAS*, 2010). An earlier estimate was **4.4 per cent** (Bittles and James Neel, 1994).
- **Morbidity and inbreeding depression:** more **autosomal recessive disorders** (thalassaemia, spinal muscular atrophy, hearing and metabolic disorders), some malformations, and reduced growth or fitness in inbred offspring.
- **Classic studies:** **William Schull and James Neel** (*The Effects of Inbreeding on Japanese Children*, 1965) found small but real increases in mortality in Hiroshima and Nagasaki; in **Karnataka**, Bittles and Indian colleagues (*Science*, 1991) found raised **stillbirth and infant death**, though smaller than once feared.

6.4 “Inbreeding is not necessarily deleterious”

The statement, set by UPSC in **1981**, holds in four senses:

- **Inbreeding creates no harmful allele**; it only **reveals** those already present. In a family with **no deleterious recessives**, cousin marriage carries **no extra risk**.
- **The absolute extra risk is small**: first-cousin children carry about **2–3 per cent** additional risk of serious birth defects or early death, above a background of about 3 per cent for all births.
- **Purging**: where inbreeding has continued for many generations, harmful recessives exposed in homozygotes are **removed by selection**, so long-inbred populations may suffer less than expected.
- **Social benefits** (property, support, lower dowry) are real for the families concerned.

The opposite, **outbreeding** (crossbreeding or **hybridisation**), raises heterozygosity and can give **hybrid vigour** (heterosis) in crops and livestock; in humans it lowers recessive disease but confers no “superiority”.

07 7. Genetic Load

IN SIMPLE WORDS

Genetic load is the burden of harmful genes that every population carries. Because of it, the average person is a little less fit than the best possible genotype. Mutation, balanced polymorphism and inbreeding all add to the load.

Definitions. The American geneticist **H.J. Muller** introduced the idea in “**Our Load of Mutations**” (1950). **James F. Crow** (1958) defined **genetic load** as “the proportion by which the **average fitness** of a population is reduced in comparison with the **best genotype**”: $L = (w_{\max} - \bar{w}) / w_{\max}$.

Types of genetic load:

- **Mutational load**: harmful alleles kept in the pool by **recurrent mutation**, balanced by selection. The **Haldane–Muller principle** states that each harmful mutation eventually causes **one “genetic death”** (failure to reproduce), whether mild or severe.
- **Segregational (balanced) load**: in a **balanced polymorphism**, the less fit **homozygotes** are produced in every generation by segregation. In the sickle-cell example, the population’s mean fitness is **0.8725** of the carriers’ fitness: a load of about **13 per cent** (Worked Example 8).
- **Incompatibility load**: loss due to mother–foetus incompatibility, as in **Rh haemolytic disease** (*erythroblastosis fetalis*) when an **Rh-negative** mother carries an **Rh-positive** child.

How it is measured. **Newton Morton, James Crow and H.J. Muller** (1956) compared mortality in children of **related and unrelated** parents. The increase in death with F gives the number of **lethal equivalents**: sets of genes that, if all were homozygous, would cause one death. Human estimates are about **1.4 lethal equivalents** per person acting between late pregnancy and age 10 (Bittles and Neel, 1994): each of us carries a few hidden recessive alleles that would be lethal in double dose.

Factors that influence genetic load:

- **Mutation rate:** raised by radiation, mutagenic chemicals and advanced paternal age.
- **Inbreeding: exposes** hidden load, converting it into deaths and disease.
- **Medicine:** relaxed selection lets carriers of once-lethal genes reproduce, so load **accumulates** slowly.
- **Population size and environment:** small groups fix harmful alleles by drift; an allele useful in one setting (HbS under malaria) becomes a load in another.

08

8. Worked Examples

IN SIMPLE WORDS

These examples show how allele, genotype and carrier frequencies are calculated, how equilibrium is tested, and how inbreeding raises risk. Each needs only simple arithmetic.

1. From non-tasters to all frequencies (PTC). In a sample, 30 per cent are non-tasters (*tt*).

- $q^2 = 0.30$, so $q = \sqrt{0.30} = 0.548$ and $p = 0.452$.
- Homozygous tasters $TT = p^2 = 0.205$; heterozygous tasters $Tt = 2pq = 0.495$.
- Half of the population are **hidden carriers** of the non-taster allele.

2. Carrier frequency of a rare recessive. A disease affects 1 in 10,000 births.

- $q^2 = 0.0001$, so $q = 0.01$ and $p = 0.99$.
- Carriers $2pq = 2 \times 0.99 \times 0.01 = 0.0198$, about **1 in 50** people.
- **Lesson:** carriers outnumber patients **200 to 1**, and **99 per cent** of the disease alleles sit in healthy carriers.

3. Testing equilibrium with chi-square (MN blood groups). Observed in 1,000 people: MM 298, MN 489, NN 213.

- Allele counting: $p(M) = (2 \times 298 + 489) / 2,000 = 0.5425$; $q(N) = 0.4575$.
- Expected: $MM = p^2 \times 1,000 = 294.3$; $MN = 2pq \times 1,000 = 496.4$; $NN = q^2 \times 1,000 = 209.3$.
- $\chi^2 = \sum (O - E)^2 / E = 0.046 + 0.110 + 0.065 = 0.22$.
- **Degrees of freedom** = 3 classes – 1 – 1 (one allele frequency estimated) = **1**; the critical value at $P = 0.05$ is **3.84**.
- Since $0.22 < 3.84$, the sample is **in Hardy–Weinberg equilibrium**.

4. A real departure (sickle-cell screening, Gadchiroli, Maharashtra). Under the national mission, 4,92,884 people were screened: 24,056 carriers (AS) and 2,654 patients (SS), so 4,66,174 were AA (Lok Sabha reply, 31 July 2026).

- $q(S) = (2 \times 2,654 + 24,056) / (2 \times 4,92,884) = 0.0298$.
- Expected $SS = q^2 \times N = 437$; observed **2,654**, about **six times** more. Expected $AS = 28,489$; observed **24,056**, fewer.

- χ^2 is about **11,900** (1 d.f.): a massive **excess of homozygotes**. An inbreeding-type coefficient $F = 1 - (\text{observed AS} / \text{expected AS}) = 1 - 24,056/28,489 \approx \mathbf{0.16}$.
- **Why:** (a) the district pools **endogamous tribes** with different frequencies (the **Wahlund effect**, after the Swedish geneticist Sten Wahlund, 1928: pooled groups always show fewer heterozygotes than expected); (b) **consanguinity**; (c) screening is **not a random sample**, since known patients come forward. The district is not one Mendelian population.

5. ABO with three alleles. Phenotypes: **O 30, A 25, B 35, AB 10 per cent.**

- $r(i) = \sqrt{0.30} = \mathbf{0.548}$.
- $p(I^A) = 1 - \sqrt{(B + O)} = 1 - \sqrt{0.65} = \mathbf{0.194}$.
- $q(I^B) = 1 - \sqrt{(A + O)} = 1 - \sqrt{0.55} = \mathbf{0.258}$.
- Check: $0.548 + 0.194 + 0.258 \approx \mathbf{1.00}$. A high I^B , as here, is typical of much of India.

6. Mutation pressure. $p = 0.90$, $u = 10^{-5}$ per generation. After 1,000 generations, $p = 0.90 \times (1 - 10^{-5})^{1000} = \mathbf{0.891}$; with $u = 10^{-6}$ it is still **0.899**. Mutation alone changes populations almost imperceptibly.

7. Gene flow. A tribal village has HbS frequency $q = \mathbf{0.10}$. In one generation **20 per cent** of parents come from a plains population with $q = 0.01$.

- $q_1 = 0.8 \times 0.10 + 0.2 \times 0.01 = \mathbf{0.082}$.
- One generation of gene flow outweighs thousands of generations of mutation.

8. Balanced polymorphism and segregational load. Fitness: $AA = 1 - s = \mathbf{0.85}$ (malaria), $AS = 1$, $SS = 1 - t = \mathbf{0.15}$ (anaemia).

- Equilibrium $q = s / (s + t) = 0.15 / 1.00 = \mathbf{0.15}$; carriers $2pq = \mathbf{0.255}$; patients $q^2 = \mathbf{0.0225}$ (2.25 per cent of births).
- Mean fitness $\bar{w} = 1 - s p^2 - t q^2 = 1 - 0.15 \times 0.7225 - 0.85 \times 0.0225 = \mathbf{0.8725}$.
- Segregational **load** $L = 1 - 0.8725 = \mathbf{0.1275}$, about 13 per cent: the price of malaria protection.

9. Genetic drift and population size. With $p = q = 0.5$, $\sigma = \sqrt{(pq / 2N)}$ is **0.05** for $N = 50$ (so 0.40–0.60 is common next generation), **0.016** for $N = 500$ and **0.005** for $N = 5,000$, where drift is almost negligible.

10. Risk from consanguinity. Risk of an affected child = $q^2(1 - F) + qF$.

ALLELE FREQUENCY (q)	UNRELATED PARENTS ($F = 0$)	FIRST COUSINS ($F = 1/16$)	UNCLE-NIECE ($F = 1/8$)
0.01	1 in 10,000	7.2 in 10,000 (7 ×)	13.4 in 10,000 (13 ×)
0.001	1 in 1,000,000	63 in 1,000,000 (63 ×)	126 in 1,000,000 (126 ×)

The table shows the key consequence of cousin marriage: it raises the risk of **rare** recessive diseases far more, in relative terms, than of common ones.

Key Terms

TERM	MEANING IN SIMPLE WORDS
Mendelian population	A group that marries within itself and shares one gene pool
Gene pool	All the alleles carried by a population
Allele (gene) frequency	The share of all copies of a gene that are of one type
Hardy–Weinberg equilibrium	Unchanging allele and genotype frequencies without disturbing forces
Genetic polymorphism	Two or more common forms of a gene, the rarer above about 1 per cent
Balanced polymorphism	Two forms kept stable by selection, often because carriers are fitter
Transient polymorphism	Two forms present only while one is replacing the other
Genetic drift	Random change in allele frequency, strongest in small populations
Wahlund effect	Fewer heterozygotes than expected when separate groups are pooled
Consanguinity	Marriage between known blood relatives
Coefficient of inbreeding (F)	The chance that a child's two alleles are copies of one ancestral allele
Genetic load	The reduction in average fitness caused by harmful genes
Lethal equivalent	A set of genes that would cause one death if all were homozygous

Contemporary Developments

- **Consanguinity mapped (August 2026):** a district-level analysis of NFHS-5 found consanguinity fell from **14.4 per cent** (1992–93) to **10.8 per cent** (2019–21), with **97 of 707 districts** above 20 per cent, the highest in Karnataka and Tamil Nadu (Kumari, Noolvi and Hallad).
- **Sickle-cell screening (September 2026):** over **7.3 crore** people screened in tribal areas, with about **20.6 lakh carriers** and **2.5 lakh** patients identified; carriers receive cards and **pre-marital counselling**, a population-scale use of Hardy–Weinberg reasoning.
- **GenomeIndia (April 2025):** **9,772** genomes from **83** populations (30 tribal, 53 non-tribal) yielded about **180 million** variants, most of them rare, supporting community-specific screening for founder diseases (*Nature Genetics*).

11 Diagrams to Master

1. **Topic map:** Mendelian population → Hardy–Weinberg → polymorphism and selection → six forces → consanguinity and F → genetic load.
2. **Hardy–Weinberg square:** gametes p and q on both sides; cells p^2 , pq , pq , q^2 ; write $p^2 + 2pq + q^2 = 1$ and the five conditions.
3. **Sickle-cell balance:** three genotypes with **fitness**, malaria versus anaemia, $q = s/(s + t)$.
4. **Forces:** mutation, gene flow, isolation, selection, drift, inbreeding, each with its **effect** and one **Indian example**.
5. **Inbreeding paths:** first-cousin pedigree with **five-person paths** through **two grandparents** ($F = 1/16$); uncle–niece with **four-person paths** ($F = 1/8$); mark the **double marriage line**.

12 Revision Grid

SCHOLAR	WORK (YEAR)	KEY IDEA
Hardy; Weinberg	1908	Equilibrium of allele and genotype frequencies
Sewall Wright	1922; 1931	Coefficient of inbreeding; genetic drift
E.B. Ford	1940	Definition of genetic polymorphism
J.B.S. Haldane	1949	Malaria hypothesis for thalassaemia
H.J. Muller	"Our Load of Mutations" (1950)	Genetic load
Anthony Allison	1954	Sickle-cell trait protects against malaria
Morton, Crow and Muller	1956	Lethal equivalents
James F. Crow	1958	Formal definition of genetic load
Schull and Neel	<i>The Effects of Inbreeding on Japanese Children</i> (1965)	Effects of consanguinity
L.D. Sanghvi	1966	Inbreeding in India
Karve and Malhotra	1968	Genetic differences among endogamous groups

SCHOLAR	WORK (YEAR)	KEY IDEA
Motoo Kimura	1968	Neutral theory
Bittles and Black	PNAS (2010)	3.5 per cent excess mortality in first-cousin offspring
Nakatsuka and colleagues	Nature Genetics (2017)	Strong founder events in 81 South Asian groups



Previous Year Questions



- 2026** Write notes on: Genetic polymorphism and natural selection.
- 2025** What are genetic markers? Discuss their applications in understanding population variation, disease association and forensics.
- 2023** What assumptions must be met for a population to be in genetic equilibrium? Explain the importance of genetic equilibrium.
- 2022** Write notes on: Balanced and transient genetic polymorphism.
- 2022** Discuss the role of evolutionary forces in creating human diversity.
- 2020** Write notes on the Genetic drift in about 150 words.
- 2020** How do marriage rules impact the gene pool of populations?
- 2019** Differentiate between transient and balanced genetic polymorphism. Illustrate your answer with suitable examples from human populations.
- 2017** Write a note in 150 words on Hardy-Weinberg Law.
- 2015** Define genetic polymorphism. Give details of its types with suitable examples.
- 2015** What do you understand by Immunogenetics? Explain with suitable examples.
- 2014** Discuss the factors affecting gene frequencies among human populations.

- 2013 What do you understand by 'Genetic Load' in a population? How is it measured and what are the important factors that can influence it? (250 words).
- 2012 Write a note in 150 words on How 'natural selection' acts on variation?
- 2012 What are the genetic effects of consanguinity? Give examples.
- 2011 Write a note in 150 words on Conditions necessary for the operation of Hardy. Weinberg Law.
- 2011 Write a note in 150 words on Genetic polymorphism.
- 2010 What is Balanced Genetic Polymorphism? How is it maintained in a population?
- 2009 Write a note on Natural selection in humans.
- 2009 Write a note on Anthropological relevance of population genetics.
- 2009 What is 'genetic load' & what factors influence it ?
- 2008 Write a note on Inbreeding.
- 2008 What do you understand by Hardy-Weinberg equilibrium? Discuss the factors that produce and redistribute variations.
- 2006 Write a note on Genetic Polymorphism.
- 2005 Write a note on Problems of Inbreeding.
- 2003 Write a note on Genetic Polymorphism and Selection.
- 2003 Describe in brief the major causes of change in gene frequency of a population.
- 2000 Write a note on Genetic Adaptation.
- 1998 Write a note on Mutation.
- 1998 Define Hardy-Weinberg law and state briefly how Hardy-Weinberg law is applicable in population human genetics ?

- 1997 Discuss the concept of 'Mendelian population' and its application in the study of anthropo-genetic variations in India.
- 1996 Discuss the concepts of balanced polymorphism and relaxed selection with special reference to Malaria dependent polymorphisms in man.
- 1993 Write a note on Inbreeding and crossbreeding.
- 1988 Write a note on Inbreeding and hybridization.
- 1987 Is inbreeding different from consanguinity? Give an account of inbreeding studies in India and comment on their social relevance.
- 1986 What are the various forms of preferential mating? Explain with Indian examples.
- 1985 Discuss the role of genetic drift, mutation and migration as the causes of variation.
- 1981 'Inbreeding is not necessarily deleterious in effect.' Comment (200 words).
- 2026 *Unit 2.3:* Discuss 'incest taboo' as the basis of development of kinship systems in human societies.
- 2024 *Unit 9.6:* Write notes on: Hemoglobin in health and disease.
- 2021 *Unit 9.5:* Write notes on : Is race a valid and biologically meaningful concept ?(in about 150 words).
- 2020 *Unit 1.4:* Elucidate how Darwin and Post-Darwin theories of evolution resulted in the development of Synthetic theory of evolution.
- 2019 *Unit 1.7:* Write a note in 150 words on Implications of mutation in evolution.
- 2018 *Unit 10:* Explain the mechanisms of human variation in gene frequencies.
- 2015 *Unit 2.3:* Write a note in 150 words on Incest Taboos.
- 2013 *Unit 9.6:* What are genetic markers and what is their usefulness? Why are blood groups considered as good genetic markers? Illustrate with examples. (250 words).
- 1991 *Unit 10:* Describe the stages of human physical growth and development and discuss the effect of nutrition and inbreeding on growth and development.

1988

Unit 1.4: Give a critical account of the processes of organic evolution, and explain how they contribute to speciation.

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