CHAPTER 4
GENETICS, EVOLUTION, DEVELOPMENT, AND PLASTICITY

Chapter Outline

I.Genetics and Evolution of Behavior

A. Mendelian Genetics

1. During the nineteenth century, Gregor Mendel demonstrated that inheritance occurs through **genes** (units of heredity that maintain their identity from one generation to the next).
2. As a rule, genes come in pairs, as they are aligned along **chromosomes** (strands of genes). One exception to this rule is male sex chromosomes, which do not come in pairs.
3. A gene is a portion of a chromosome, which is composed of a double-stranded chemical called **deoxyribonucleic acid** (**DNA**).
4. A strand of DNA serves as a template (model) for the synthesis of **ribonucleic acid** (**RNA**).
    a. RNA is a single-stranded chemical: One type of RNA molecule—messenger RNA—serves as a template for the synthesis of protein molecules.
    b. DNA contains four "bases"—adenine, guanine, cytosine, and thymine.
    c. The order of those bases determines the order of corresponding bases along an RNA molecule—adenine, guanine, cytosine, and uracil.
5. Proteins can be part of the structure of the body or serve as *enzymes* (biological catalysts that regulate chemical reactions in the body).
6. If individuals have an identical pair of genes on the two chromosomes, they are **homozygous** for that gene; if they instead have an unmatched pair of genes, they are **heterozygous** for that gene (e.g., a gene for black hair on one chromosome and a gene for brown hair on the other).
7. Genes are dominant, recessive, or intermediate.
    a. **Dominant** genes show a strong effect in either homozygous or heterozygous conditions
    b. **Recessive** genes show their effects only in homozygous conditions (e.g., a carrier for both a dominant black hair gene and a recessive brown hair gene will have black hair).
8. Often, more than one single gene contributes to the appearance of certain characteristics (e.g., at least 10 genes contribute to variations in eye color).
9. Partial expression of a gene is also possible.
10. Sex-Linked and Sex-Limited Genes

a. Genes located on sex chromosomes are known as **sex-linked genes.** All other chromosomes are referred to as autosomal chromosomes. Genes located on autosomal chromosomes are **autosomal genes.**
b. A female mammal has two X chromosomes; a male has one X and one Y chromosome.
c. When biologists speak of sex-linked genes, they usually mean X-linked genes.
d. **Sex-limited genes**: Genes present in both sexes but active mainly in one sex.

11. Genetic Changes
    a. Genes change in several ways.
    b. **Mutation:** A heritable change in a DNA molecule. Changing one base in DNA to any of the other three types means that the mutant gene will code for a protein with a different amino acid at one location in the molecule.
        i. Mutations are rarely advantageous. One rare example: FOXP2 gene, which facilitates language development.
        ii. Other types of mutations: duplication or deletion; microduplication or microdeletion. Scientists believe microduplications and microdeletions may be responsible for schizophrenia.
12. **Epigenetics**: Changes in gene expression without modification of the DNA sequence (e.g., brain changes resulting from drug addiction).
    a. How could an experience modify gene expression?
        i. Proteins called histones bind DNA into a shape that is more like string wound around a ball.
        ii. To activate a gene, the DNA must partially unwind from the histones.
        iii. The result of an experience—maternal deprivation, cocaine exposure, new learning, or whatever—in some way alters the chemical environment within a cell.
        iv. In some cases, the outcome adds acetyl groups (COCH3) to the histone tails near a gene, causing the histones to loosen their grip on the DNA, and facilitating the expression of that gene.
        v. Another possibility is to add or remove methyl groups from DNA, usually at the promoter regions at the beginning of a gene.
    b. Experiences act by altering the activity of genes.

B. Heredity and Environment
1. Most variations in behavior depend on the combined influence of many genes and environmental influences.
2. If the variations in some characteristic depend largely on genetic differences, the characteristic has high **heritability**.
3. Heritability in humans is studied in three ways:
    a. Comparing **monozygotic** (identical) twins and dizygotic (fraternal) twins
    b. Studying adopted children and their biological parents
    c. Identifying specific genes linked to some behavior
4. Environmental Modification

a. Even a trait with high heritability can be modified by environmental interventions.
b. **Phenylketonuria**, or **PKU**: A genetic inability to metabolize the amino acid phenylalanine. If PKU is not treated, phenylalanine accumulates to toxic levels, impairing brain development and leaving a child mentally retarded, restless, and irritable. Although PKU is a hereditary condition, environmental interventions can modify it.

5. How Genes Affect Behavior
   a. If we speak of a "gene for alcoholism," we should not imagine that the gene itself causes alcoholism. Rather, it produces a protein that under certain circumstances increases the probability of alcoholism.
   b. Exactly how a gene increases the probability of a given behavior is a complex issue.

C. The Evolution of Behavior
1. **Evolution**: Change over generations in the frequencies of various genes in a population.
2. Offspring generally resemble their parents, but because of mutations and recombinations, new inheritable variations in the gene pool can occur.
3. **Artificial selection**: Breeding animals for desirable individual characteristics (this causes changes in various genetic frequencies in a population).
4. Common Misunderstandings about Evolution
   a. *Does the use or disuse of some structure or behavior cause an evolutionary increase or decrease in that feature?* This idea is a carryover of biologist Jean-Baptiste Lamarck's theory of evolution through the inheritance of acquired characteristics, known as **Lamarckian evolution**.
   b. *Have humans stopped evolving?*
   c. *Does "evolution" mean "improvement"?* The flaw in this argument is that evolution depends on reproduction, not just survival. Evolution improves fitness, which is operationally defined as the number of copies of one's genes that endure in later generations. The genes of the current generation evolved because they were fit for previous generations. They may or may not be adaptive in the future.
   d. *Does evolution benefit the individual or the species?* Neither: It benefits the genes!
5. Brain Evolution
   a. Humans have bigger and better brains (at least we think so) than other species.
   b. Humans may have been able to evolve such big brains without sacrificing other functions, because of our diet.
   c. We devote more energy to our brains and less to physical strength.
6. **Evolutionary psychology**: Concerns how behaviors evolved.
   a. The emphasis is on evolutionary and functional explanations (how a behavior may be useful to a population and why natural selection would favor it).
   b. Some proposed evolutionary explanations are speculative and controversial.

c. **Altruistic behavior**: Behaviors that benefit others rather than the individual committing the behavior. This is in contrast to the belief that genes evolve for their own benefit.
    i. Perhaps a better explanation is **kin selection**—selection for a gene that benefits the individual's relatives. A gene spreads if it causes you to risk your life to protect your children, who share many of your genes, including perhaps a gene for altruism.
    ii. Another explanation is **reciprocal altruism**, the idea that individuals help those who will return the favor.
    iii. A more controversial hypothesis is group selection. According to this idea, altruistic groups thrive better than less cooperative ones.

II.The Development of the Brain

A. Maturation of the Vertebrate Brain
1. The human central nervous system begins to form when the embryo is about two weeks old.
2. A neural tube forms around a fluid-filled cavity; this structure eventually sinks under the skin surface and develops into the hindbrain, midbrain, and forebrain. The fluid-filled cavity becomes the central canal and the four ventricles.
3. The human brain weighs approximately 350 grams at birth and around 1,000 grams at one year of age. The average adult brain weighs between 1,200 and 1,400 grams.
4. Growth and Development of Neurons
   The five steps of neuron development:
    a. **Proliferation**: Production of new cells; cells along the ventricles of the brain divide. Some cells remain where they are as **stem cells**, continuing to divide. Others become primitive neurons and glia that migrate to other locations.
    b. Early in development, the primitive neurons begin to **migrate** (move). Chemicals known as *immunoglobins* and *chemokines* guide the new cells to their eventual destination in the brain.
    c. At first, a primitive neuron looks like any other cell. Gradually, the neuron **differentiates**, forming its axon and dendrites; the axon grows before the dendrites.
    d. A later and slower stage of neuronal development is **myelination**, the process by which glia produce the insulating fatty sheaths that accelerate transmission in many vertebrate axons. Myelin forms first in the spinal cord and then in the hindbrain, midbrain, and forebrain.
    e. The final stage is **synaptogenesis**, or the formation of synapses. Although this process begins before birth, it continues throughout life, as neurons form new synapses and discard old ones.
5. New Neurons Later in Life
    a. The traditional belief was that adult vertebrate brains gain all their neurons during early development and could only lose neurons later in life.

b. The differentiation of stem cells (undifferentiated cells) is an exception to the traditional belief. For example, stems cells in the nose remain undifferentiated throughout life, and periodically divide to replace a dying olfactory receptor.
c. This phenomenon is also seen in animals, like songbirds, who lose neurons in areas necessary for singing in the fall and winter, only to regain neurons in those areas in the spring.
d. In general, new neurons do not form in other parts of the adult mammalian brain. This is evidenced by the age of a radioactive isotope of carbon in one's brain and heart cells.

B. Pathfinding by Axons
1. Axons travel over long distances to precise locations. How do they find their way?
2. Chemical Pathfinding by Axons
a. Weiss (1924) grafted an extra leg to a salamander and eventually axons grew into it so that the leg moved in sync with the salamander's other legs. Weiss suggested that the nerves attached to muscles at random and then sent a variety of messages, each one tuned to a different muscle.
b. Specificity of Axon Connections
i. Evidence suggests Weiss was wrong—sensory axons find their way to their correct targets.
ii. Sperry (1943) discovered that severed optic nerve axons will grow back to their original targets in the *tectum*. He showed that this process was dependent on chemical gradients in the target cells by severing the optic nerve and rotating the eye by 180°.
c. Chemical Gradients
i. A growing axon follows a path of cell-surface molecules, attracted by some chemicals and repelled by others, in a process that steers the axon in the correct direction.
ii. Eventually, axons sort themselves over the surface of their target area by following a gradient of chemicals.
3. Competition Among Axons as a General Principle
a. Postsynaptic cells strengthen the synapses of some cells and weaken synapses with others.
b. **Neural Darwinism**: During development of the nervous system, synapses form randomly before a selection process keeps some and rejects others (this is only partly accurate since synapse formation is also influenced by chemical guidance and neurotrophic factors).

C. Determinants of Neuronal Survival
1. While working on the sympathetic ganglion, Rita Levi-Montalcini discovered that muscles that synapse with the axons from the ganglia don't determine how many neurons are produced but which synapses survive.
2. Levi-Montalcini discovered that muscles produce and release **nerve growth factor** (**NGF**), which promotes the survival and growth of axons.

3. Axons that don't receive enough NGF degenerate and their cell bodies die. All neurons are born with this suicide program and will automatically die if the right synaptic connection is not made. This programmed cell death is called **apoptosis**.
4. **Neurotrophin**: a chemical (like NGF) that promotes the survival and activity of neurons. In addition to NGF, the brain also uses *brain-derived neurotrophic factor* (*BDNF*) as a neurotrophin. BDNF is the most abundant neurotrophin in the adult mammalian cortex.
5. Initially, all areas of the developing nervous system produce far more neurons than will survive into adulthood. This loss of cells is a natural part of development.
6. After maturity, the apoptotic process becomes dormant and neurons do not need neurotrophins to survive. Neurotrophins are used in adult brains to increase branching of axons and dendrites throughout life. Deficiencies of neurotrophins lead to cortical shrinking and are linked to several brain diseases.

D. The Vulnerable Developing Brain
1. The earliest stages of development are remarkably similar across species. A series of genes known as *homeobox genes*—found in vertebrates, insects, plants, even fungi and yeast—regulate the expression of other genes and control the start of anatomical development
2. During early development, the brain is highly vulnerable to malnutrition, toxic chemicals, and infections that would produce only mild problems at later ages.
3. **Fetal alcohol syndrome** (**FAS**): Caused by alcoholic consumption during pregnancy. Symptoms include decreased alertness, hyperactivity, facial abnormalities, mental retardation, motor problems, and heart defects.
4. Infant brains are especially sensitive to alcohol because it suppressed the release of glutamate, the brain's main excitatory transmitter. Thus, neurons receive less excitation and undergo apoptosis.
5. Social influences also affect the developing brain. Children of impoverished or abused mothers have increased problems in both academic and social functioning.

E. Differentiation of the Cortex
1. Neurons differ in shape and chemistry.
2. Ultimate shape of neurons and functions of regions depend on input received.
3. Immature neurons experimentally transplanted from one part of the developing cortex to another develop the properties characteristic of their new location. Neurons transplanted at a later stage develop some characteristics of the new location while retaining others of the initial location.
4. In immature ferrets, researchers rerouted the optic nerve on one side of the brain away from its normal thalamic target onto a thalamic target that usually gets input from the ears. They found that the parts of the thalamus and cortex that formerly received auditory information reorganized to process visual information.

F. Fine-Tuning by Experience
1. Because of the unpredictability of life, we have evolved the ability to redesign our brain (within limits) in response to experience.
2. Experience and Dendritic Branching

a. Environmental enrichment leads to a thicker cortex, more dendritic branching, and improved performance on learning tasks in rats.
b. Much of the benefit of enriched environments in rats is simply due to activity. Increased size expansion of neurons has also been demonstrated in humans as a function of physical activity.
c. Enriched environments enhance sprouting of axons and dendrites in a wide variety of species including humans. Some believe this is evidence of the psychological term "far transfer," which suggests enhanced capacity in one task leads to enhanced capacity of other tasks.
d. However, enriching environments produce enhanced capacity of only the tasks that are relevant in those environments. One researcher looked at a computer program designed to "train your brain." After six weeks of using the program several times a week, much of the 11,000 who participated saw substantial improvements in their ability to complete the computer task. Yet this improvement does not extend to other tasks.
e. One of the best ways to maintain intellectual vigor is physical activity.

3. Effects of Special Experiences
    a. Extensive practice of a particular skill makes a person more adept at that skill. In a few cases, researchers have identified brain changes that are associated with increased expertise at a particular skill.
    b. Brain Adaptations in People Blind Since Infancy
        i. People blind from birth are better at discriminating between objects by touch and have increased activation in their occipital cortex (visual cortex) while performing touch tasks.
        ii. Further research using magnetic stimulation to inactivate brain areas demonstrated that blind people use the occipital cortex to discriminate between tactile stimuli and Braille symbols but sighted people do not. Similar results are also found using verbal stimuli.
    c. Music Training
        i. The auditory cortex response to pure tones is twice as large for professional musicians as for nonmusicians. Moreover, a part of the temporal cortex was found to be 30% larger in professional musicians.
        ii. Violin players have a larger area devoted to the left fingers in the postcentral gyrus than nonmusicians.
    d. Special Training in Adulthood
        i. Might adult experiences modify brain anatomy also? In a sense, the answer is, "Yes, of course."
        ii. The issue is whether an adult experience produces a big enough effect that we might observe it with MRI or similar technology.
        iii. Many studies have in fact reported changes in adult brain anatomy from tasks such as learning to juggle three balls, 16 hours of playing a complex video game, or 40 hours of playing golf for the first time. However, skeptics raise serious objections.

iv. The only replicated finding is that physical exercise expands part of the hippocampus in older people.
v. In short, we should reserve judgment about most of the reported effects of brief experiences on the adult brain.

e. When Brain Reorganization Goes Too Far
i. Typically expanded cortical representation of personally important information is beneficial. However, in extreme cases the reorganization creates problems.
ii. **Focal hand dystonia** (musician's cramp): this happens in musicians who practice extensively when the expanded representation of each finger overlaps its neighbor. The fingers become clumsy, fatigue easily, and make involuntary movements that interfere with the desired task. A similar condition called "writer's cramp" can happen to people who spend all day writing.

G. Brain Development and Behavioral Development
1. Adolescence
a. Adolescents are widely regarded as impulsive and prone to seek immediate pleasure.
b. Research shows adolescents are able to make reasonable, mature decisions when they have had time to consider the options carefully. However, they are impulsive when making quick decisions, especially in the face of peer pressure.

2. Old Age
a. On average, older people's memory and reasoning begin to fade. Many neurons lose some of their synapses, and the remaining synapses change more slowly.
b. The frontal cortex begins thinning at age 30. However, there is great variance in the level of deterioration in different people.
c. The volume of the hippocampus also gradually declines.
d. In old age, even the blood contains chemicals that impair cognitive function.

II. Plasticity After Brain Damage

A. Brain Damage and Short-Term Recovery
1. Brain damage can result from a number of causes, including tumors, infections, exposure to radiation or toxic substances, and degenerative conditions such as Parkinson's and Alzheimer's disease.
2. **Closed head injury**: A sharp blow to the head that does not actually puncture the brain. The most common cause of brain damage in young people. Closed head injuries damage the brain because of rotational forces that drive the brain tissue against the inside of the skull.
3. Reducing the Harm from a Stroke

a. **Stroke** (**cerebrovascular accident**): A temporary loss of blood flow to the brain. This is a common cause of brain damage, especially in the elderly.
b. **Ischemia**: The most common type of stroke; loss of blood flow caused by a blood clot or other obstruction of an artery.
c. **Hemorrhage**: A less common type of stroke; bleeding due to the rupture of an artery.
d. Ischemia and hemorrhage lead to common problems including **edema** (fluid accumulation), increased potassium levels due to dysfunctional sodium-potassium pumps, and increased release of glutamate.
e. Immediate Treatments
    i. Decreasing cell death after a stroke can be accomplished by administering **tissue plasminogen activator** (**tPA**) clot-busting drugs, which restore blood flow following ischemia, or by using drugs that antagonize glutamate activity.
    ii. The most effective known method of preventing brain damage after strokes in laboratory animals is to cool the brain. Cooling protects the brain after ischemia by reducing overstimulation, apoptosis, and inflammation.
    iii. Exposure to cannabinoids (the chemicals found in marijuana) minimizes the damage caused by strokes in laboratory animals.

B. Later Mechanisms of Recovery
1. Increased Brain Stimulation
    a. **Diaschisis**: Decreased activity of surviving neurons after other neurons are destroyed. Behavioral deficits due to diaschisis can sometimes be improved with the use of stimulant drugs.
2. Regrowth of Axons
    a. Under certain circumstances, damaged axons can grow back. However, regeneration is minimal in the mature mammalian central nervous system, possibly because of a large amount of scar tissue or the secretion of growth-inhibiting chemicals.
3. Axon Sprouting
    a. Ordinarily, the surface of dendrites and cell bodies is covered with synapses, and a vacant spot doesn't stay vacant for long.
    b. After a cell loses input from an axon, it secretes neurotrophins that induce other axons to form new branches, or **collateral sprouts**, which take over the vacant synapses.
4. Denervation Supersensitivity
    a. If a certain set of synapses becomes inactive—perhaps because of damage elsewhere in the brain—the remaining synapses become more responsive, more easily stimulated. This process of enhanced response is known as **denervation supersensitivity** or receptor supersensitivity.

    b. Denervation supersensitivity is a way of compensating for decreased input. However, the increased sensitivity can lead to intense responses in normal inputs, which can result in prolonged pain.
5. Reorganized Sensory Representations and the Phantom Limb
    a. If a brain area loses a set of incoming axons, we can expect some combination of increased response (denervation supersensitivity) by the surviving axons and collateral sprouting by axons that ordinarily attach to some other target.
    b. Physicians have long noted that most people with amputations experience a **phantom limb**, a continuing sensation of an amputated body part.
        i. Modern methods show that phantom limbs develop when the relevant portion of the somatosensory cortex reorganizes and becomes responsive to alternative inputs.
6. Learned Adjustments in Behavior
    a. Much recovery from brain damage depends on learning to make better use of the abilities that were spared. A brain-damaged person or animal may also learn to use abilities that at first appear lost, but are only impaired.
        i. For example: Monkeys with a **deafferented** (loss of sensory or afferent input) limb fail to use it because walking on three limbs is apparently easier than trying to move the impaired limb. However, if forced, they can learn to use the deafferented limb.
    b. One treatment for people recovering from a stroke is to force them to use the weaker limb by preventing them from using the normal limb.