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The Study of How Drugs Affect the Nervous System's Cellular Function and the Neural Mechanisms

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Description

The study of how drugs affect the nervous system's cellular function and the neural mechanisms by which they influence behavior is known as neuropharmacology. There are two main subfields of neuro pharmacology: Molecular and behavioral. Neuro psychopharmacology, also known as behavioral neuropharmacology, is the study of how drugs affect human behavior. Molecular neuropharmacology, on the other hand, is the study of neurons and the neurochemical interactions between them, with the ultimate goal of developing drugs that improve neurological function. The interactions of neurotransmitters, neuropeptides, neuro hormones, neuromodulators, enzymes, second messengers, co-transporters, ion channels, and receptor proteins in the central and peripheral nervous systems are the focus of both of these fields, which are inextricably linked. Drugs to treat pain, neurodegenerative diseases like Parkinson's and Alzheimer's, psychological disorders, addiction and many other neurological conditions are being developed by researchers by studying these interactions. In the early 20th century, scientists were able to acquire a fundamental understanding of the nervous system and the mechanisms by which nerves communicate with one another, which was the beginning of the field of neuropharmacology.

Central and Peripheral Nervous Systems

Prior to this discovery, a number of drugs had been discovered to have some kind of influence on the nervous system. In the hope of developing a malaria-fighting medication, French scientists began working with a substance called phenothiazine in the 1930s. It was discovered to have sedative effects and what appeared to be beneficial effects for Parkinson's disease patients, despite the fact that this drug showed very little promise for use against malaria-infected individuals. Before scientists were able to identify specific neurotransmitters, such as norepinephrine (involved in the constriction of blood vessels and the increase in heart rate and blood pressure), dopamine (the chemical whose shortage is involved in Parkinson's disease), and serotonin (soon to be recognized as deeply connected to depression, this black box method, in which an investigator would administer a drug and examine the response without knowing how to relate drug action to patient response, was the voltage clamp was invented in 1949, which made it possible to study ion channels and the nerve action potential. In the 1950s, scientists also improved their ability to measure levels of particular neurochemicals in the body and thus correlate these levels with behavior. Scientists were able to study not only how information is transferred from one neuron to another but also how a neuron processes this information within itself thanks to these two significant occurrences in the history of neuropharmacology. Neurons are referred to as excitable cells due to the abundance of ion-channel-containing proteins on their surface membrane, which allows for the passage of small charged particles into and out of the cell.

Drug Action Communication between Neurons

Chemical information can be received by the dendrites of the neuron, transmitted down the axon through the perikaryon, or cell body, and finally to other neurons through the axon terminal. The cell can undergo rapid depolarization thanks to these voltage-gated ion channels. If this depolarization reaches a certain threshold, an action potential will occur. Calcium ions will enter the cell as soon as the action potential reaches the axon terminal. The calcium ions will then trigger the release of neurotransmitter-filled vesicles into the synapse by binding to the cell membrane. The cell that interacts with the released neurotransmitters is referred to as the post-synaptic neuron, and this cell is referred to as the pre-synaptic neuron. The neurotransmitter can either be broken down by enzymes in the synapse that are specific to that particular neurotransmitter or it can bind to receptors on the post-synaptic cell, allowing the pre-synaptic cell to re-uptake it and save it for later transmission. There are two types of receptors that neurotransmitters interact with on a post-synaptic neuron, and these three distinct actions are major areas where drug action can affect communication between neurons. LGICs, also known as ligand-gated ion channels, are the first class of receptors. The fastest way from a chemical signal to an electrical signal is through LGIC receptors.

When the neurotransmitter binds to the receptor, it will alter its conformation, allowing ions to enter the cell directly. G-protein-coupled receptors, also known as GPCRs, are the second category. Due to the increased number of intracellular biochemical reactions, these are significantly slower than LGICs. When the neurotransmitter binds to the GPCR protein, it initiates a series of intracellular interactions that can result in a wide range of alterations to the physiology, biochemistry, and gene expression of the cell. In the field of neuropharmacology, interactions between neurotransmitters and receptors are of the utmost significance because a great number of drugs that are currently in development involve disrupting this binding process. The central nervous system's rapid synaptic inhibition is mediated by the GABA neurotransmitter. GABA binds to a receptor, most likely the GABAA receptor, when it is released from its presynaptic cell, causing the postsynaptic cell to hyperpolarize (below its action potential threshold).Any excitatory manipulation caused by other interactions between neurotransmitters and receptors will be countered by this. By binding to five distinct GPCRs, the dopamine neurotransmitter mediates synaptic transmission. Depending on whether the response causes the post-synaptic cell to respond in an excitatory or inhibitory manner, these five receptor proteins are divided into two groups. Dopamine and the brain's interactions with it are affected by a wide variety of legal and illegal drugs. Due to the fact that L-dopa and dopamine can cross the blood–brain barrier, the dopamine precursor Levodopa is given to patients with Parkinson's disease, a disease that causes a decrease in the amount of dopamine in the brain. Parkinson's disease patients with Restless Leg Syndrome (RLS) are also given some dopamine agonists. These include ropinirole and pramipexole. Drugs like methylphenidate (also known as Ritalin), which prevent the pre-synaptic cell from re-up taking dopamine and increase the amount of dopamine remaining in the synaptic gap, can be used to treat psychological disorders like Attention Deficit Hyperactivity Disorder (ADHD). Dopamine binding to post-synaptic cell receptors will rise as a result of this increase in synaptic dopamine. Other illegal stimulants with greater potency, like cocaine, employ the same mechanism. Psychological disorders like that of Attention Deficit Hyperactivity Disorder (ADHD) can be treated with drugs like methylphenidate (also known as Ritalin), which block the re-uptake of dopamine by the pre-synaptic cell, thereby providing an increase of dopamine left in the synaptic gap. This increase in synaptic dopamine will increase binding to receptors of the post-synaptic cell. This same mechanism is also used by other illegal and more potent stimulant drugs such as cocaine.