Purkinje Cells [1]


Purkinje cells [5], also called Purkinje neurons, are neurons in vertebrate animals located in the cerebellar cortex of the brain. Purkinje cell bodies are shaped like a flask and have many threadlike extensions called dendrites, which receive impulses from other neurons called granule cells [6]. Each cell also has a single projection called an axon, which transmits impulses to the part of the brain that controls movement, the cerebellum [7]. Purkinje cells [8] are inhibitory neurons: they secrete neurotransmitters that bind to receptors that inhibit or reduce the firing of other neurons. Purkinje cells [9] were the first neuronal cells identified. Researchers study the embryonic development of Purkinje cells [5] to elucidate how they function in various mechanisms in the body.

Jan Evangelista Purkyné (Purkinje), working at the University of Breslau [8] in Breslau, Prussia, discovered these cells in the mid-nineteenth century. In 1832, he obtained a Plössl achromatic microscope [9], which brought two colors into focus at the same time, and he examined the structure of the cells in sheep [10]. He used alcohol to fix his preparations and made thin sections of sheep [10] brain tissues to examine them microscopically. Purkyné described the cells, which were later named after him, in his paper on the histology [11] of the nervous system, “Neueste Untersuchungen aus der Nerven-und Hirnanatomie” (Recent Studies from the Nervé and Brain Anatomy), which he presented in September 1837 in Prague, Bohemia, which later became the Czech Republic.

In the centuries after Purkyné´s discovery, researchers studied the structure and functions of Purkinje cells [5]. In the late nineteenth century, Camillo Golgi, at the University of Pavia in Lombardy, Italy, examined Purkinje cells [9] by staining them with silver nitrate. The silver nitrate stain enabled him to describe the cell body and its extensions. Santiago Ramón y Cajal [12] at the University of Barcelona in Barcelona, Spain, refined Golgi’s technique and discovered that Purkinje cells [9] have dendritic spines, which are small, door knob-like protrusions on the dendrites. Golgi and Ramón y Cajal shared the Nobel Prize in Physiology or Medicine [13] in 1906 for their research on the nervous system’s structure. Since then, research on these cells uncovered Purkinje cells’ relationships with other cells, such as Bergmann glial cells and granule cells [6] as well as the details of their functions in the cerebellum [7].

Purkinje cells [5] participate in the processes of motor control and learning. They are the only cells that emit signals from the cerebellar cortex that is the outer layer of the cerebellum [7], though they can receive input from hundreds of thousands of cells. Each cell body is eighty microns in diameter and inhibits the excitatory neurons of the spinal cord and other areas, from which they receive input. Purkinje cells [8] regulate the activation of excitatory neurons by interactions with their dendrites. Purkinje cells [5] release gama-aminobutyric acid (GABA), which is a neurotransmitter that inhibits certain neurons from transmitting impulses. The output of the nerve cells [14] is through the axon that carries electrical impulses.

Purkinje cells [5] inhibit the output centers called deep cerebellar nuclei and vestibular nuclei neurons in the cerebellum [7] by regulating the timing of the rising and falling of electrical signals (action potentials) down nuclei neurons’ axons. In turn, they control the output signals of the cerebellum [7]. Through synchronized signals, Purkinje cells [5] control the rate at which signals fire in the cerebellum [7] to produce precise output from the nuclei neurons, resulting in motor coordination such as hand movement. Studies on mammals revealed that Purkinje cells [5] also synthesize the hormones [15] progesterone [16] and estradiol [17] during the formation of the cerebellar circuits in developing embryos and fetuses. Progesterone and estradiol [17] promote the growth of dendrites, development of synapses (synaptogenesis [18]), and development of spines on the dendrites (spinogenesis) in the developing Purkinje cell.

Two kinds of neuronal fibers carry input to the Purkinje cells [5]: mossy fibers and climbing fibers. Mossy fibers, which originate in the spinal cord and brain stem influence Purkinje cells [5] by way of granule cells [6]. Mossy fibers along with granule cells [6] divide in two and form parallel fibers, analogous to telephone lines in a neighborhood. Each Purkinje cell receives input from roughly 200,000 parallel fibers. Climbing fibers originate in the inferior olivary nucleus [19] of the medulla oblongata, a region of the brainstem responsible for regulating respiration, heart rate, and digestive processes. The climbing fibers wrap around the body and dendrites of the Purkinje cell and make many synaptic contacts, but unlike mossy fibers, they contact only a few Purkinje cells [5]. Moreover, each of the Purkinje cells [5] receives input from at most one climbing fiber.

Embryonic research of mouse [20] and rat [21] brains showed the neurogenic aspects of Purkinje cells [5]. When vertebrates are embryos, Purkinje cells [5] arise in the ventricular zone in the neural tube [22], the nervous system’s precursor in the embryo. Purkinje cells [5] originate from a tissue called the cerebellar primordium. The cells that develop first are those of the cerebellum’s two hemispheres, or halves. Those cells generated in the cerebellar primordium form a cap over a diamond-shaped cavity of the developing brain called the fourth ventricle. The Purkinje cells [5] that develop later are those of the cerebellum’s center-lying
Purkinje cells [5] are susceptible to both genetic and environmental influences that may disrupt their regular functions. Embryonic examinations of the Ts65Dn strain of mice, which are a genetic model for Down syndrome [26] in humans [27] (trisomy twenty-one), show that the axons of Purkinje cells [6] are degenerated in the mice’s cerebellum [5]. Exposure of the fetus [28] to alcohol during embryonic growth can permanently destroy Purkinje cells [8] and lead to fetal alcohol syndrome. Individuals with autism have smaller than normal Purkinje cells [5]. Individuals with fewer than normal amounts of those cells often have Niemann-Pick disease type C, a lipid storage disease.

Sources

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