

# DOWNLOAD PDF INHIBITORY INTERNEURONS AND GLIAL CELLS IN THE CEREBELLAR CORTEX

## Chapter 1 : Changing Concepts of Cortical Development | Cerebral Cortex | Oxford Academic

5. *Inhibitory Interneurons and Glial Cells in the Cerebellar Cortex Introduction. The cerebellar cortex contains two types of inhibitory neurons in the molecular layer; basket and stellate cells.*

Suites of factors can convert non-neuronal cells into induced neuronal cells showing class-specific features. Pulsate Nature of Neural Network Activity Information flow in neurons and neuronal networks is not instantaneous. It takes time for action potential to propagate along axons and for neurotransmitters to span the synaptic cleft and activate the postsynaptic neuron. The state of individual synapses changes on the basis of about 4 ms. The propagation time and responses of neurons and synapses results in pulsate behavior of the neuronal network. Neurons are coincidence detectors – they respond to a population of input spikes above threshold. The neural network is characterized by reentrant signals positive feedback resulting in recursive updating of the synaptic state of the network on the basis of about to ms. Consciousness is mediated by the momentary state of active synapses in the neuronal network. The state of the dynamic core mediating an instantaneous thought changes and is updated via the recursive functionality of the neuronal network. Generally, the input signals comprising the quorum can occur on the synapses in any combination; no particular combination is required. Presynaptic neuron connections require that perhaps excitatory neurons fire together to produce a synaptic potential large enough to trigger an action potential. Kandel; Principles of Neural Science , Dendritic Tree Synaptic Efficacies Molded by Experience As a result of synaptic plasticity , the efficacies of synapses on a network of dendritic trees will achieve a pattern of efficacies corresponding to the input signal patterns the dendritic tree synapses have experienced in the past. This plasticity of the synapses and memory for input signal patterns contributes to an associative memory function. Note, however, the prevalent degeneracy in input signal sensitivity. No particular combination of active synapses is required. Each time a memory is reconstructed a slightly different set of many millions of efficacious synapses will be activated. Research study – Dendritic Spines and Memories – in the mouse cortex, learning and novel sensory experience lead to spine formation and elimination by a protracted process. Research Study – Dendritic Field Orientation Toward Active Axons in Developing Cortex – regulation of dendrite orientation is conserved across species and cortical areas and shows how high-acuity sensory function may be achieved by the tuning of subcellular polarity to sources of high sensory activity. Action Potential Triggered at Axon Hillock by Quorum Sensing of Dendritic Tree Synapses Action potential is generated in the initial part of the axon where it connects with the cell body. LeDoux; Synaptic Self , 47 A projection neuron typically has about 10, synapses on its dendritic tree; also, its axon may have a number of collateral branches. Each neuron receives about synapses and communicates with about other neurons [although not directly; the communication with other neurons will be via multi-synaptic connections in the neural network]. Consequently, a neuron will fire at a rate between its intrinsic quiescent low-level of a few Hertz to a maximum rate of about Hz. LeDoux; Synaptic Self, 64 Neurons in the sensory areas of the brain typically fire at high rates Hz , whereas neurons in the prefrontal cortex at a lesser rate Hz. Edelman; Universe of Consciousness , Almost Poisson nature of the spike trains of single neurons found in most brain areas. LeDoux; Synaptic Self, 64 Neuron Configuration, Size, Shape Unlike most of the cells of the body, which have a simple shape, nerve cells have highly irregular shapes and are surrounded by a multitude of exceedingly fine extensions of axons and dendrites. Kandel; Search of Memory , 61 Estimated to be many hundreds of different neuronal types, far more than in any other organ of the body. Kandel; Principles of Neural Science , Neurons show enormous variety in cellular anatomy, physiological function, neural chemistry, and conductivity. Sanes; Development of the Nervous System , 87 Axons and dendrites are extremely thin about the thickness of a human hair. Kandel; Search of Memory, 62 A neuron may be innervated by as many as 10, different synaptic endings. Kandel; Search of Memory, Some nerve connections are located nearly a meter from the cell bodies of origin. Kandel; Search of Memory, Postsynaptic Density

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PSD at Spines A prominent feature of almost all spines throughout the nervous system is a postsynaptic density PSD, an electron-dense thickening of the postsynaptic membrane. Andersen; Hippocampus Book, Functionally, the PSD is a biochemical specialization that allows numerous molecules e. Andersen; Hippocampus Book, NMDA receptors, which mediate a slow synaptic current, blocked in a voltage-dependent manner, occupy a disk-like space near the center of the PSD. Andersen; Hippocampus Book, Generally, each neuron lasts a lifetime Once a nerve cell has become differentiated, it does not divide anymore. Eichenbaum; Neuroscience of Memory, 31 A neuron receives many excitatory and inhibitory inputs from many other cells; the likelihood of firing at any one moment depends on the net balance between excitation and inhibition across all of the inputs at that particular time. Hobson; Dreaming as Delirium, 28 When nothing much is happening, a neuron usually sends spikes down its axon at a background rate between 1 and 5 Hz. When a neuron becomes excited, because it receives many excitatory signals, its firing rate increases to Hz or more. Crick; Astonishing Hypothesis, 92 Layer 5 neurons fire in bursts of a few spikes at rates at least as high as Hz, with intrinsic intrabursts firing rates on the order of 15 Hz. Most neurons in the brain are under the influence of as many as a dozen or more neuroactive substances. Shepherd, Synaptic Organization of the Brain; McCormick; Neurotransmitter Actions, 61 Neurons generate strong electrical impulses spikes when they received enough excitatory inputs from their connected neurons. These spikes cause a release of neurotransmitter molecules at the synaptic connections with other neurons, which triggers an electrical impulse in postsynaptic cells. Neurons can excite each other so that each spike in one neuron causes an increasing number of other neurons to degenerate spikes, even in the absence of external stimulation. This growth of activity in a population of strongly interacting neurons increases until some regulatory mechanisms stabilizes a constant average neuronal activity. Most Nerve Pulses about the Same Size and Shape Pulses throughout the CNS are pretty much the same size; amplitude carries little information beyond the presence or absence of a pulse. Holland; Emergence, 85 If enough pulses arrive at the surface of a neuron during a short interval of time, the neuron fires, propagating a new pulse down its own axon. Holland; Emergence, 85 Neurons are coincidence detectors: Zeman; Consciousness, The time it takes the body of a neuron to integrate incoming information is long compared with the time it takes a pulse to propagate between neurons. This time for processing of input information by each neuron sets the rate at which a network of neurons can function. Holland; Emergence, 85 Variable Discharge Bursting Properties of Hippocampal Neurons Electrophysiological behavior of the different neurons in the hippocampus is variable. Dentate granule and CA1 pyramidal neurons can fire repetitively at up to several hundred Hz. CA3 pyramidal neurons tend to fire in short bursts of action potentials. Bursting properties of CA3 hippocampal neurons are thought to be important for explaining the seizure susceptibility of the hippocampus.

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## Chapter 2 : Neurons and Support Cells

*Here, we investigate the role of *Ascl1* in the development of inhibitory interneurons and glial cells in the cerebellum. This gene is expressed by maturing oligodendrocytes and GABAergic interneurons and is required for the production of appropriate quantities of these cells, which are severely reduced in *Ascl1*  $\hat{\sim}/\hat{\sim}$  mouse cerebella.*

Open in a separate window Three markers account for nearly percent of the GABAergic cortical interneuron population: Outlined in this table are percentage of total GABA-expressing neurons, morphology, axonal targeting, firing pattern, and origin based on the type of marker expressed. In the recent years there has been a push to create a consistent nomenclature for the varying interneuronal subtypes; a conference in Petilla, Spain, was held to accomplish this task. A group of researchers known as the Petilla Interneuron Nomenclature Group PING convened to formulate a set of terminologies to describe the morphological, molecular, and physiological features of GABAergic cortical interneurons [ 16 ]. Morphologically speaking, cortical interneurons are described with regard to their soma, dendrites, axons, and the connections they make. Molecular features include transcription factors, neuropeptides, calcium-binding proteins, and receptors these interneurons express, among many others. Physiological characteristics include firing pattern, action potential measurements, passive or subthreshold parameters, and postsynaptic responses, to name a few [ 16 ]. The overarching goal of this conference and the resulting Petilla terminology is to create a uniform set of criteria by which interneurons can be described so as to reduce confusion between the findings by various research groups in this field. While results from the Petilla conference have shown that there are many criteria with which to define and distinguish classes of interneurons, this review will classify interneuron subtypes based on the markers they express, particularly PV, SST, or 5HT3aR, and elaborate upon each of these three groups. This population of interneurons possesses a fast-spiking pattern, and fire sustained high-frequency trains of brief action potentials [ 10 , 16 , 17 , 22 , 26 ]. Additionally, these interneurons possess the lowest input resistance and the fastest membrane time constant of all interneurons [ 10 , 16 , 17 , 22 , 23 , 27 , 28 ]. Two types of PV-interneurons make up the PV interneuron group: Far more is understood about basket cells, which are interneurons that make synapses at the soma and proximal dendrite of target neurons, and usually have multipolar morphology [ 16 , 22 ]. Several studies have shown that fast-spiking basket neurons are the dominant inhibitory system in the neocortex, where they mediate the fast inhibition of target neurons, among many other functions [ 29 - 35 ]. As will be discussed in a later section, fast-spiking basket neurons likely play a large role in regulating the delicate balance between excitatory and inhibitory inputs in the cerebral cortex [ 36 , 37 ]. Much less is known about the second subgroup of PV-expressing interneurons, the chandelier cells. Unlike basket neurons, chandelier cells target the axon initial segment of pyramidal neurons [ 16 , 22 ]. Both basket cells and chandelier cells are fast-spiking, but they differ in electrophysiological properties, as reviewed by Woodruff et al[ 38 ]. Several relatively recent studies have suggested that in contrast to other interneurons, chandelier cells may be excitatory rather than inhibitory due to their depolarizing effects on membrane potential [ 38 - 40 ], although the functions of this subgroup have yet to be elucidated. One research group has characterized a group of PV-expressing cells that is independent from chandelier and basket neurons in the mouse neocortex [ 41 ]. These interneurons were designated multipolar bursting cells, and differ from chandelier and basket cells in both electrophysiology and connectivity [ 41 ]. Multipolar bursting neurons possess synapses with pyramidal cells or other multipolar bursting cells that demonstrate a paired-pulse facilitation; in contrast, chandelier and basket cells are usually strongly depressing [ 41 ]. While this group of interneurons holds promise as a third subgroup within the PV-expressing interneuron type, further investigation is warranted, as no other research groups have characterized these neurons. SST-positive interneurons are known as Martinotti cells, and possess ascending axons that arborize layer I and establish synapses onto the dendritic tufts of pyramidal neurons [ 22 ]. Martinotti cells are found throughout cortical layers II-VI, but are most abundant in layer V [ 14 , 22 , 42 ]. These interneurons function by exhibiting a

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regular adapting firing pattern but also may initially fire bursts of two or more spikes on slow depolarizing humps when depolarized from hyperpolarized potentials. In contrast to PV-positive interneurons, excitatory inputs onto Martinotti cells are strongly facilitating [ 43 - 47 ]. More details regarding the electrophysiology and firing patterns of SST-expressing Martinotti cells can be found in a review by Rudy et al [ 8 ]. Results from several studies have suggested that the SST interneuron population is a heterogeneous one. These cells were located in layers IV and V that, unlike, Martinotti cells, targeted cells from cortical layer IV [ 48 ]. X94 cells also possessed a lower input resistance relative to Martinotti cells, with spikes of a shorter duration and a stuttering firing pattern [ 48 ]. This evidence clearly suggests that there are other interneuronal subgroups within the SST subtype than just Martinotti cells. Like the X94 cells, these two populations of interneurons differ electrophysiologically than Martinotti cells, although additional research must be performed to truly determine their presence, as other research groups have not been able to duplicate these findings [ 26 , 50 , 51 ]. It is clear that there are likely additional subpopulations of SST-expressing cortical interneurons. This is bolstered by the observed differences in firing properties, expression of molecular markers, and connectivity of different neurons within this population [ 8 , 49 , 50 , 52 , 53 ]. Due to its relatively recent discovery, this group has yet to be fully characterized, although it is evident that this population is a very heterogeneous one. Within the 5HT3aR interneuron group are several subsets of interneurons that also express other protein or neuropeptide markers, one of them being vasoactive intestinal peptide VIP [ 22 , 26 , 54 ]. VIP interneurons generally make synapses onto dendrites [ 25 , 55 , 56 ], and some have been observed to target other interneurons [ 57 , 58 ]. Relative to all cortical interneurons, VIP interneurons possess a very high input resistance and are among the most excitable of interneurons [ 25 , 55 , 56 ]. There are several types of VIP cortical interneurons that differ in electrophysiological properties, but in general they possess a bipolar, bitufted and multipolar morphology [ 22 , 26 , 54 , 56 ]. One VIP subtype in particular is commonly referred to as irregular-spiking neurons [ 17 , 25 , 55 , 56 , 59 - 61 ]. Irregular spiking interneurons possess a vertically oriented, descending axon that extends to deeper cortical layers, and have an irregular firing pattern that is characterized by action potentials occurring irregularly during depolarizations near threshold [ 17 , 25 , 55 , 56 , 59 - 61 ]. Additionally, irregular spiking cortical interneurons express the calcium-binding protein calretinin CR [ 56 , 61 ], which is a marker that some SST-positive interneurons also possess and interestingly, also a marker that neither VIP nor SST interneurons express [ 25 , 55 , 56 , 62 ]. There are several other subtypes of VIP-expressing 5HT3aR interneurons present in the cerebral cortex, which are nicely summarized by Rudy et al [ 8 ]. Briefly, among these are rapid-adapting [ 63 ], fast-adapting neurons [ 25 , 56 ] and IS2 [ 61 ], as well as a minor population of VIP-positive basket cells with regular, bursting, or irregular-spiking firing patterns [ 22 , 26 , 64 ]. Neurogliaform cells are a type of cortical interneuron that belongs to this category: Neurogliaform cells are unique relative to other GABAergic cortical interneurons because they are capable of forming synaptic connections with each other as well as with other interneuronal types as opposed to other interneurons that can only make synapses onto homologous neurons , thus solidifying their important role in regulating neural circuitry [ 66 - 68 ]. Furthermore, neurogliaform cells function by activating slow GABAA and GABAB receptors in order to provoke long lasting inhibitory postsynaptic potentials onto pyramidal neurons and other interneurons [ 65 , 69 ]. While progress has certainly been made in distinguishing interneuron groups from one another, further research is definitely warranted. The GE is a transitory brain structure located in the ventral area of the telencephalon, and is anatomically present during embryonic development. The GE becomes evident at approximately E In total there are three ganglionic eminences: The names of the different areas within the GE are based on their rostral-caudal location in the telencephalon. As embryonic development continues, the GEs grow and ultimately fuse, at which point they are no longer present in the mature brain.

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## Chapter 3 : Generation of Diverse Cortical Inhibitory Interneurons - Advanced Science News

Page 1 1. The three types of neurons are: A) excitatory, inhibitory, and myelinated. B) sensory, motor, and interneurons. C) interneurons, glial cells, and motor cells.

**Advanced Search** The search for truth is in one way hard and in another way easy, for it is evident that no one can master it fully nor miss it wholly. But each adds a little to our knowledge of nature, and from all the facts assembled there arises a certain grandeur. It is difficult to imagine that a cutting-edge assessment of any area of neurobiology could stand unchallenged for a few years, let alone decades, but the authors achieved this and more. At a recent international meeting held at Delphi, Greece, the current state of knowledge concerning cortical development was discussed intensively, and many of the contributions presented there form the basis for this issue of Cerebral Cortex. The terminology adopted by the Boulder Committee so many years ago was reconsidered by those in attendance, and found to be in most respects still current. Thus, the basic anatomical framework proposed by the Boulder Committee, including the terms used to describe the layers of the developing cortex, are still valid Fig. This is not to say that there has been no progress in our understanding of cortical development; quite the contrary, there has been a virtual sea change in our concepts of how cortical neurons are generated, where they are born, and how they migrate. In fact, our understanding of the basic developmental processes of neuronal induction, regionalization, neurogenesis and migration have undergone dramatic alterations within the last decade. The contributions presented in this issue touch upon many of these developments and may serve as an introduction to current concepts of cortical development.

**Cortical Arealization** How the cerebral cortex becomes parceled into areas, each with distinct morphological and functional traits, is one of the most fundamental questions in neuroscience. How do these distinct cortical areas acquire their identity during development? Two alternative hypotheses have been hotly debated since the late s: Recent evidence, however, favors the middle ground. Thus, it is now accepted that whereas late refinement of area differentiation relies on structural and functional integrity of thalamic afferents, the early cortical primordium has the intrinsic capability to undergo molecular regionalization presaging the later development of mature areal properties. Much work in recent years has focused on the role of transcription factor genes in early cortical arealization. Researchers have used mouse mutants to show that parcellation of cortical areas is regulated by gradients of homeodomain proteins, but the mechanisms that underlie these complex processes are presently under intensive investigation. Work has only recently begun to shed light on the role of diffusible ligands in establishing transcription factor gradients responsible for sculpting cortical areal profiles. The question of cortical arealization will undoubtedly remain in the forefront in the field of cortical development for some years to come.

**Merging of Glial and Neuronal Lineages: Neurogenesis by Radial Glial Cells** Since the time of the Boulder Committee meeting, a major advance has been in the methods that allow analysis of cell lineages both in vivo and in vitro. One of the intriguing new concepts discussed herein relates to the role of radial glial cells. Glial and neuronal lineages had been thought to be distinct, but results from a number of laboratories now indicate that these lineages are not as divergent as previously thought, and that radial glial cells are indeed neuronal precursor cells. Thus, in addition to their role in guiding migrating neurons, radial glia are now known to be neurogenic and have been shown to give rise to the pyramidal neurons of the cerebral cortex. Moreover, in the few niches where there is persistent neurogenesis in the adult forebrain, it appears that astroglial cells, lineally related to embryonic radial glia, function as neuronal precursor cells. An appreciation of the broadened role of the radial glial cell in brain development leaves many important questions unanswered. Among these are how prevalent neurogenic radial glia are in brain and cortical development, whether there is heterogeneity of precursor potential among cortical radial glia, and what molecular features distinguish them from their neuroepithelial cell precursors and the astrocytes into which they transform.

**Inhibitory Interneurons Arise from the Ventral Telencephalon** Given the segmental origin of the neuroaxis and the restriction of regionally expressed gene products at anatomical boundaries, it

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was a reasonable assumption that neurons destined for a particular segment would arise within that segment during development. The demonstration that significant numbers of neurons destined for the neocortex actually arise in the striatal anlage came as a surprise to many. More remarkable still, the cortical cells originating in the ventral telencephalon seem to consist entirely of inhibitory interneurons, while the excitatory principle neurons arise from the dorsal telencephalon. These cell types take markedly different routes of migration to converge in the developing neocortex and join together synaptically to form the canonical cortical circuit, where excitation is accompanied by feed-forward and feedback inhibition. Understanding the underlying molecular and cellular mechanisms that regulate regional neurogenesis and direct the migration of immature neurons poses a daunting challenge. Nonetheless, significant inroads have been made recently through the application of molecular biological and genetic tools. Some of these exciting new insights into the genetic control of neurogenesis and migration are presented here.

**Multiple Modes of Neuronal Migration**

Around the time of the Boulder Committee Report, the migration of cortical neurons along radial glial fibers had been recently described. It was subsequently widely assumed that glial-guided neuronal migration was the predominant method by which neurons were dispersed in the developing nervous system, despite a minority view that alternative modes of migration were also important. However, the traditional view of radial migration has recently been altered by convincing demonstrations that large numbers of cortical neurons migrate tangentially rather than radially, and that some radially migrating neurons do not appear to climb along radial glial fibers. The inhibitory interneurons that migrate to the developing cortex from the ventral telencephalon, for example, migrate tangentially, crossing generally at right angles to the predominant direction of radial glial fibers. Moreover, a subset of cells arising in the cortical ventricular zone may inherit the radial fiber of parent radial glial cells and migrate by somal translocation along their own radial fibers. These observations are producing a broader and more detailed view of cortical development, but one that will surely undergo further modification in time.

Schematic drawing of five stages in the development of the vertebrate central nervous system. CP, cortical plate; I, intermediate zone; M, marginal zone; S, subventricular zone; V, ventricular zone.

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## Chapter 4 : Development and specification of GABAergic cortical interneurons

*Purkinje cells send inhibitory messages to the deep cerebellar nuclei and are responsible for the sole output of motor coordination from the cerebellar cortex. With the work of the basket cell, Purkinje cells do not send the inhibitory response for motor coordination and motor movement occurs.*

Cells-R-Us illustrated text of a public lecture on the brain Histology textbooks are NOT recommended for the study of nervous tissue. Most histology textbooks begin with relatively insignificant, and often misleading, details rather than emphasizing features important for understanding nervous tissue function. The extended table of contents can be read, just as if it were a "capsule" textbook. In about two dozen pages following immediately after the chapter listing, all of the subheadings from every chapter are presented, each as a complete sentence. This extended table of contents offers a concise summary of major ideas. Your study through this entire unit can be usefully guided by this summary. You should, at a minimum, be fluent in the vocabulary of this summary so that every sentence here is meaningful to you. Chapter 1 provides an excellent narrative of how science and medicine have understood the brain. This chapter is available online at Amazon. Basic cellular organization of the nervous system is described in Chapter 2. Nerve cell structure is described in detail in Chapter 4 and subsequent chapters. Begin by reading the extended table of contents. Nerve cell structure is described in detail in Chapter 4. Begin by reading the extended table of contents p. Add more only as the need arises. Subcellular basis for neural function. Begin with the extended table of contents pp. Check out Chapters 3 genes , Chapter 5 proteins , and Chapters electrical activity. Dip into the main text for principal learning issues e. Nerve cell structure is described in detail in Chapter 3. Check out Chapters 4 proteins , and Chapters electrical activity. Nervous tissue has presented extraordinary challenges to science. Historically, a basic appreciation of the cellular composition of nervous tissue did not come until decades after other tissues were fairly well understood. One reason is that none of the cell types which comprise nervous tissue can be properly visualized in routine histological preparations. Do not expect examination of nervous system specimens to yield satisfying observations, at least not without an exceptional effort to understand why things look the way they do. Cajal introduced the four principles which comprise the neuron doctrine quotes are from Kandel , In Search of Memory , pp. Rather each nerve cell forms synapses and communicates with certain nerve cells and not with others. Information flows, from the dendrites of a given nerve cell to the cell body [then] along the axon to the presynaptic terminals and then across the synaptic cleft to the dendrites of the next cell, and so on. Axons, dendrites and synapses -- the most significant features of nerve cells -- cannot be readily seen without specialized techniques such as those used by Cajal. No other tissue in the body is characterized by cells whose cytoplasmic processes reach out for great distances away from the cell nuclei. This reality puts a special burden on the student as well as the researcher. You cannot simply look at a slide or micrograph, not even an electron micrograph, and truly "see" the most interesting features of the nerve cell. However, the length of axons and dendrites is wondrously great, far greater than ordinary cellular dimensions. Dendrites may extend several millimeters away from the cell body, into a volume the size of a pea. Axon length may exceed a meter for many sensory and motor axons , and commonly extends for several centimeters. As a simple consequence of this cellular geometry, the cell body of a neuron may comprise less than one percent of the entire cell volume. From this, you may deduce that the bulk of nervous tissue consists of nerve cell processes rather than nerve cell bodies. The study of neuroanatomy consists largely of understanding the routes travelled by nerve cell axons. Unfortunately, the organization of neural processes, most particularly the full length of axons and dendrites and the synaptic interactions between them, can seldom be visualized directly. In most other tissues of the body, what you can see in the microscope is directly informative. Consider skin , where a routine section of epidermis reveals almost everything interesting about the size, shape and growth sequence of epidermal cells. Electron microscopy of similar specimens simply adds more finely resolved detail. But making any sense at all of nervous tissue requires that you "see" with concepts

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acquired over decades of research with many special techniques. When you examine microscope slides or micrographs of nervous tissue, patterns of functional connection cannot usually be seen. Nevertheless, what you can observe should be interpreted in terms of neuronal functions and connectivity, including unseen axons, dendrites and synapses as well as associated supporting cells. Thus your job for comprehending nervous tissue is not just to look-and-learn, but to think rather deeply, to fit many different views and facts together. Most of the listed vocabulary terms for neuronal and glial structures are well defined in standard textbooks. You just have to make sense of it all. How to read this page. You might read this page straight down, from top to bottom. But it is written with hyperlinks to facilitate browsing. And return repeatedly to the outline at the top of this page to choose the topic that most closely engages your current curiosity. Nerve cells comprise the "enchanted loom" that is our brain. Here are three absolutely wonderful facts about nerve cells. The first and most wonderful fact is that, working together, nerve cells can perceive and think and dream. This is magic of the highest sort. See [Cells-R-Us](#) for extended discussion. The second wonderful fact is that nerve cells are much like other cells. Each is essentially a bag of water, surrounded by a fatty membrane and containing an assortment of molecules. There seems to be nothing about individual nerve cells that cannot be explained, at least in principle, by basic chemistry and biology. The third wonderful fact is that each nerve cell has a truly magnificent shape. Somehow, this third fact bridges the gap between the mystery that is our mind and the chemistry that is our cells. Somewhere in the shape of nerve cells, in the complexity of connections among billions of such cells, and in the intricate pattern of activity that plays upon those cells, our "self" emerges. Nerve cells come in extreme variety. In every region of the brain are several different nerve cell types, each distinguished by its own characteristic soma size, dendritic shape, source of synaptic input, destination of axonal output, and chemistry. Occasional nerve cell types may have characters which depart from the the typical description presented below. Because of this immense variety of nerve cell types, there is no "one-size-fits-all" description. So textbook descriptions of nerve cells tend to present overwhelmingly abundant detail. Although details of nerve cell shape and connectivity are usually insignificant for clinical practice, they can be quite beautiful and are essential for understanding research on brain function. It is also often necessary to learn some "irrelevant" detail in order to understand the particular examples used to demonstrate basic functional principles. Nerve cell bodies look more or less like other body cells, although they do have certain characteristic features. Extending out from each nerve cell body are long cytoplasmic processes, one axon and several dendrites. These processes usually cannot be distinguished in routine histological preparation. A typical nerve cell body contains only a small fraction of the total cell volume; the rest is contained in the axon and dendrites. The spaces between nerve cell bodies with a feltwork of these axonal and dendritic processes, called neuropil which also includes glial cell processes. The cell body of a nerve cell also called a soma, plural somata is basically a cell nucleus surrounded by cytoplasm. Nuclei of nerve cells are large, round and euchromatic with a single prominent nucleolus more below. Cytoplasm of nerve cell bodies is abundantly supplied with masses of rough endoplasmic reticulum traditionally called Nissl bodies , numerous Golgi bodies, lots of smooth endoplasmic reticulum, many mitochondria, and extensive cytoskeletal elements microtubules and various filaments. This metabolic machinery is needed for ongoing maintenance of extensive axonal and dendritic membranes. The axon is a process which is specialized for conducting signals from one nerve cell to another. Each nerve cell has one and only one axon. Typical axons have relatively few branches, except near the terminal end. The diameter of an axon is uniform along its entire length. The terminal branches of an axon make synaptic contacts onto other nerve cells or with peripheral effectors, i. Nerve signals travel along axons away from the cell body and toward synapses at the axonal terminal. Axonal nerve signals, called action potentials are initiated at the axon hillock, the site where an axon arises from the cell body. Action potentials are active, all-or-nothing signals which do not decline in strength as they travel along the axon.. Axons may be myelinated to increase the speed of signal conduction. A nerve cell typically has several dendrites, each with numerous branches. The word "dendrite" means "branch". The diameter of dendrites typically decreases away from the cell body, so that dendrites taper gradually to fine

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twigs. Dendrites typically receive synaptic contacts from axons of many other nerve cells. Synapses often occur on tiny dendritic spines. Nerve signals travel along dendrites toward the cell body. Dendritic nerve signals, called synaptic potentials, arise at synapses. Synaptic potentials are conducted passively fading with distance. Dendrites are not myelinated. Size and shape of dendritic spines influence synaptic strength. Plasticity of dendritic spine morphology is implicated in memory.

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## Chapter 5 : Basket cell - Wikipedia

*In the gamma-aminobutyric acid (GABA)-containing interneurons of the cerebral cortex and the hippocampus, as well as in the Purkinje cells of the cerebellum, parvalbumin only appears postnatally.*

Medium spiny neurons , most neurons in the corpus striatum. Purkinje cells , huge neurons in the cerebellum, a type of Golgi I multipolar neuron. Pyramidal cells , neurons with triangular soma, a type of Golgi I. Renshaw cells , neurons with both ends linked to alpha motor neurons. Unipolar brush cells , interneurons with unique dendrite ending in a brush-like tuft. Granule cells , a type of Golgi II neuron. Anterior horn cells, motoneurons located in the spinal cord. Spindle cells , interneurons that connect widely separated areas of the brain

**Direction[ edit ]** Afferent neurons convey information from tissues and organs into the central nervous system and are also called sensory neurons. Efferent neurons transmit signals from the central nervous system to the effector cells and are also called motor neurons. Interneurons connect neurons within specific regions of the central nervous system. Afferent and efferent also refer generally to neurons that, respectively, bring information to or send information from the brain. Action on other neurons[ edit ] A neuron affects other neurons by releasing a neurotransmitter that binds to chemical receptors. The effect upon the postsynaptic neuron is determined not by the presynaptic neuron or by the neurotransmitter, but by the type of receptor that is activated. A neurotransmitter can be thought of as a key, and a receptor as a lock: Receptors can be classified broadly as excitatory causing an increase in firing rate , inhibitory causing a decrease in firing rate , or modulatory causing long-lasting effects not directly related to firing rate. The two most common neurotransmitters in the brain, glutamate and GABA , have actions that are largely consistent. Glutamate acts on several different types of receptors, and have effects that are excitatory at ionotropic receptors and a modulatory effect at metabotropic receptors. Similarly, GABA acts on several different types of receptors, but all of them have effects in adult animals, at least that are inhibitory. Because of this consistency, it is common for neuroscientists to simplify the terminology by referring to cells that release glutamate as "excitatory neurons", and cells that release GABA as "inhibitory neurons". There are also other types of neurons that have consistent effects on their targets, for example, "excitatory" motor neurons in the spinal cord that release acetylcholine , and "inhibitory" spinal neurons that release glycine. The distinction between excitatory and inhibitory neurotransmitters is not absolute, however. Rather, it depends on the class of chemical receptors present on the postsynaptic neuron. In principle, a single neuron, releasing a single neurotransmitter, can have excitatory effects on some targets, inhibitory effects on others, and modulatory effects on others still. For example, photoreceptor cells in the retina constantly release the neurotransmitter glutamate in the absence of light. So-called OFF bipolar cells are, like most neurons, excited by the released glutamate. However, neighboring target neurons called ON bipolar cells are instead inhibited by glutamate, because they lack the typical ionotropic glutamate receptors and instead express a class of inhibitory metabotropic glutamate receptors. It is possible to identify the type of inhibitory effect a presynaptic neuron will have on a postsynaptic neuron, based on the proteins the presynaptic neuron expresses. Parvalbumin -expressing neurons typically dampen the output signal of the postsynaptic neuron in the visual cortex , whereas somatostatin -expressing neurons typically block dendritic inputs to the postsynaptic neuron. Tonic or regular spiking. Some neurons are typically constantly or tonically active, typically firing at a constant frequency. Neurons that fire in bursts are called phasic. Some neurons are notable for their high firing rates, for example some types of cortical inhibitory interneurons, cells in globus pallidus , retinal ganglion cells. Acetylcholine is released from presynaptic neurons into the synaptic cleft. It acts as a ligand for both ligand-gated ion channels and metabotropic GPCRs muscarinic receptors. Nicotinic receptors are pentameric ligand-gated ion channels composed of alpha and beta subunits that bind nicotine. Acetylcholine is synthesized from choline and acetyl coenzyme A. GABAergic neurons€” gamma aminobutyric acid. GABA is synthesized from glutamate neurotransmitters by the enzyme glutamate decarboxylase. Glutamate is one of two primary excitatory amino

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acid neurotransmitter, the other being Aspartate. Glutamate receptors are one of four categories, three of which are ligand-gated ion channels and one of which is a G-protein coupled receptor often referred to as GPCR. The function of NMDA receptors is dependant on Glycine receptor binding as a co-agonist within the channel pore. NMDA receptors do not function without both ligands present. Metabotropic receptors, GPCRs modulate synaptic transmission and postsynaptic excitability. Glutamate can cause excitotoxicity when blood flow to the brain is interrupted, resulting in brain damage. Glutamate is synthesized from the amino acid glutamine by the enzyme glutamate synthase. Dopamine is connected to mood and behavior and modulates both pre and post synaptic neurotransmission. Dopamine is synthesized from the amino acid tyrosine. Tyrosine is catalyzed into levadopa or L-DOPA by tyrosine hydroxylase, and levadopa is then converted into dopamine by amino acid decarboxylase. Serotonin 5-Hydroxytryptamine, 5-HT can act as excitatory or inhibitory. Serotonin is synthesized from tryptophan by tryptophan hydroxylase, and then further by aromatic acid decarboxylase. A lack of 5-HT at postsynaptic neurons has been linked to depression. Drugs that block the presynaptic serotonin transporter are used for treatment, such as Prozac and Zoloft. Neurons such as Purkinje cells in the cerebellum can have over dendritic branches, making connections with tens of thousands of other cells; other neurons, such as the magnocellular neurons of the supraoptic nucleus, have only one or two dendrites, each of which receives thousands of synapses. Synapses can be excitatory or inhibitory and either increase or decrease activity in the target neuron, respectively. Some neurons also communicate via electrical synapses, which are direct, electrically conductive junctions between cells. Calcium causes synaptic vesicles filled with neurotransmitter molecules to fuse with the membrane, releasing their contents into the synaptic cleft. The neurotransmitters diffuse across the synaptic cleft and activate receptors on the postsynaptic neuron. High cytosolic calcium in the axon terminal also triggers mitochondrial calcium uptake, which, in turn, activates mitochondrial energy metabolism to produce ATP to support continuous neurotransmission. The human brain has a huge number of synapses. Each of the one hundred billion neurons has on average 7, synaptic connections to other neurons. It has been estimated that the brain of a three-year-old child has about synapses 1 quadrillion. This number declines with age, stabilizing by adulthood. Estimates vary for an adult, ranging from to 5 x synapses to trillion. Mechanisms for propagating action potentials[ edit ] In , John Zachary Young suggested that the squid giant axon could be used to study neuronal electrical properties. By inserting electrodes into the giant squid axons, accurate measurements were made of the membrane potential. The cell membrane of the axon and soma contain voltage-gated ion channels that allow the neuron to generate and propagate an electrical signal an action potential. There are several stimuli that can activate a neuron leading to electrical activity, including pressure, stretch, chemical transmitters, and changes of the electric potential across the cell membrane. Neurons must maintain the specific electrical properties that define their neuron type. To minimize metabolic expense while maintaining rapid conduction, many neurons have insulating sheaths of myelin around their axons. The sheaths are formed by glial cells: The sheath enables action potentials to travel faster than in unmyelinated axons of the same diameter, whilst using less energy. Multiple sclerosis is a neurological disorder that results from demyelination of axons in the central nervous system. Some neurons do not generate action potentials, but instead generate a graded electrical signal, which in turn causes graded neurotransmitter release. Such non-spiking neurons tend to be sensory neurons or interneurons, because they cannot carry signals long distances. Neural coding[ edit ] Neural coding is concerned with how sensory and other information is represented in the brain by neurons. The main goal of studying neural coding is to characterize the relationship between the stimulus and the individual or ensemble neuronal responses, and the relationships amongst the electrical activities of the neurons within the ensemble. In other words, if a neuron responds at all, then it must respond completely. Greater intensity of stimulation does not produce a stronger signal but can produce a higher frequency of firing. There are different types of receptor responses to stimuli, slowly adapting or tonic receptors respond to steady stimulus and produce a steady rate of firing. These tonic receptors most often respond to increased intensity of stimulus by increasing their firing frequency, usually as a power function of stimulus plotted

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against impulses per second. There are a number of other receptor types that are called quickly adapting or phasic receptors, where firing decreases or stops with steady stimulus; examples include: The neurons of the skin and muscles that are responsive to pressure and vibration have filtering accessory structures that aid their function. The pacinian corpuscle is one such structure. It has concentric layers like an onion, which form around the axon terminal. When pressure is applied and the corpuscle is deformed, mechanical stimulus is transferred to the axon, which fires. If the pressure is steady, there is no more stimulus; thus, typically these neurons respond with a transient depolarization during the initial deformation and again when the pressure is removed, which causes the corpuscle to change shape again. Other types of adaptation are important in extending the function of a number of other neurons. In this paper, he tells he could not find evidence for anastomosis between axons and dendrites and calls each nervous element "an absolutely autonomous canton. It held that neurons are discrete cells not connected in a meshwork, acting as metabolically distinct units. Later discoveries yielded a few refinements to the simplest form of the doctrine. For example, glial cells, which are not considered neurons, play an essential role in information processing. In fact, there are examples of neurons forming even tighter coupling: The fruit fly *Drosophila melanogaster*, a common subject in biological experiments, has around 100,000 neurons and exhibits many complex behaviors. Many properties of neurons, from the type of neurotransmitters used to ion channel composition, are maintained across species, allowing scientists to study processes occurring in more complex organisms in much simpler experimental systems.

### Chapter 6 : Generation of diverse cortical inhibitory interneurons - WIREs Developmental Biology

*During development, inhibitory interneurons are generated by highly specialized stem cells called radial glial progenitor cells (RGPs) that reside in transient regions of the ventral forebrain. After birth, they undergo a long journey to reach their destination in the overlying cortex.*

### Chapter 7 : Neurons and glial cells | Clinical Gate

*First described by Ramon y Cajal as 'short-axon' cells over a century ago, inhibitory interneurons in the cerebral cortex make up ~10% of the neuronal milieu.*

### Chapter 8 : Neuron - Wikipedia

*The inhibitory interneurons that migrate to the developing cortex from the ventral telencephalon, for example, migrate tangentially, crossing generally at right angles to the predominant direction of radial glial fibers.*

### Chapter 9 : Neurons; Synapses; Glial Cells

*There are more neurons than glial cells in the nervous system. and interneurons. Damage to which portion of the cerebral cortex would most likely interfere.*