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## Chapter 1 : Neural Networks

*The Neurones And Supporting Elements Of The Brain Of A Selachian [Gilbert Logan Houser] on theinnatdunvilla.com*  
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Brains and nerves - Brain and nerve basics Summary The nervous system uses electrical and chemical means to help all parts of the body to communicate with each other. The brain and spinal cord make up the central nervous system. Nerves everywhere else in the body are part of the peripheral nervous system. The nervous system helps all the parts of the body to communicate with each other. It also reacts to changes both outside and inside the body. The nervous system uses both electrical and chemical means to send and receive messages. Neurones are the building blocks The basic building block of the nervous system is a nerve cell, or neurone. Neurones are shaped differently depending on where they are in the body and what role they play. All neurones have finger-like projections called dendrites and a long fibre called an axon. In many cases, the axon is coated by a specialised membrane called a myelin sheath. The axon feathers out and has a number of bumps on it. Each bump sits near to a dendrite from another neurone. The space between the bump and the dendrite is called a synapse. Messages jump the synapse from one neurone to the next, using special chemicals called neurotransmitters. Central nervous system The brain and the spinal cord make up the central nervous system. They are wrapped in a thin lining called meninges and bathed with cerebrospinal fluid CSF. This soft, jelly-like organ has countless billions of neural cross-connections. The brain oversees the workings of the body, while its higher functions give us consciousness and personality. The spinal cord The spinal cord is connected to the brain and runs the length of the body. It is protected by the bones of the spine vertebrae. Nerves branch off from the spinal cord into the arms, legs and torso. The peripheral nervous system Nerves connect the brain and spinal cord to the peripheral nervous system, which is what nerve tissue outside of the central nervous system is called. It is made up of two main parts: The autonomic nervous system The autonomic nervous system is part of the peripheral nervous system. One of its main roles is to regulate glands and organs without any effort from our conscious minds. The autonomic nervous system is made up of two parts: These systems act on the body in opposite ways. Together, they coordinate a multitude of adjustments required for our changing personal needs as we move through our environment. For example, the size of our pupils is adjusted automatically to allow the correct amount of light into our eyes for optimum vision, our sweat glands are turned on when we get too hot and our salivary glands produce saliva when we eat food or even think about it! The somatic nervous system The somatic nervous system is also a part of the peripheral nervous system. One of its roles is to relay information from the eyes, ears, skin and muscle to the central nervous system brain and spinal cord. It also obeys commands from the central nervous system and makes muscles contract or relax, allowing us to move. Problems of the nervous system Some common problems of the nervous system include: Symptoms include shaking and problems with movement Sciatica – pressure on a nerve caused by a slipped disc in the spine or arthritis of the spine and, sometimes, other factors Shingles – infection of sensory nerves caused by the varicella-zoster virus Stroke – a lack of blood to part of the brain. Where to get help Your doctor Things to remember The nervous system uses electrical and chemical means to help all parts of the body to communicate with each other.

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## Chapter 2 : Dissociation between sensing and metabolism of glucose in sugar sensing neurones

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There is no feedback loops i. Feed-forward ANNs tend to be straight forward networks that associate inputs with outputs. They are extensively used in pattern recognition. This type of organisation is also referred to as bottom-up or top-down. Feedback networks are very powerful and can get extremely complicated. They remain at the equilibrium point until the input changes and a new equilibrium needs to be found. Feedback architectures are also referred to as interactive or recurrent, although the latter term is often used to denote feedback connections in single-layer organisations. The activity of each hidden unit is determined by the activities of the input units and the weights on the connections between the input and the hidden units. The behaviour of the output units depends on the activity of the hidden units and the weights between the hidden and output units. This simple type of network is interesting because the hidden units are free to construct their own representations of the input. The weights between the input and hidden units determine when each hidden unit is active, and so by modifying these weights, a hidden unit can choose what it represents. We also distinguish single-layer and multi-layer architectures. The single-layer organisation, in which all units are connected to one another, constitutes the most general case and is of more potential computational power than hierarchically structured multi-layer organisations. In multi-layer networks, units are often numbered by layer, instead of following a global numbering. The perceptron figure 4. Units labelled A1, A2, Aj , Ap are called association units and their task is to extract specific, localised featured from the input images. Perceptrons mimic the basic idea behind the mammalian visual system. They were mainly used in pattern recognition even though their capabilities extended a lot more. The impact that the book had was tremendous and caused a lot of neural network researchers to loose their interest. The book was very well written and showed mathematically that single layer perceptrons could not do some basic pattern recognition operations like determining the parity of a shape or determining whether a shape is connected or not. The Learning Process The memorisation of patterns and the subsequent response of the network can be categorised into two general paradigms: The associative mapping can generally be broken down into two mechanisms: This is used to provide pattern completion, ie to produce a pattern whenever a portion of it or a distorted pattern is presented. In the second case, the network actually stores pairs of patterns building an association between two sets of patterns. Yet another paradigm, which is a variant associative mapping is classification, ie when there is a fixed set of categories into which the input patterns are to be classified. This type of learning mechanism is essential for feature discovery and knowledge representation. Every neural network posseses knowledge which is contained in the values of the connections weights. Modifying the knowledge stored in the network as a function of experience implies a learning rule for changing the values of the weights. Information is stored in the weight matrix W of a neural network. Learning is the determination of the weights. Following the way learning is performed, we can distinguish two major categories of neural networks: In such networks, the weights are fixed a priori according to the problem to solve. All learning methods used for adaptive neural networks can be classified into two major categories: Supervised learning which incorporates an external teacher, so that each output unit is told what its desired response to input signals ought to be. During the learning process global information may be required. Paradigms of supervised learning include error-correction learning, reinforcement learning and stochastic learning. An important issue conserning supervised learning is the problem of error convergence, ie the minimisation of error between the desired and computed unit values. The aim is to determine a set of weights which minimises the error. One well-known method, which is common to many learning paradigms is the least mean square LMS convergence. Unsupervised learning uses no external teacher and is based upon only local information. It is also referred to as self-organisation, in the sense that it self-organises data presented to the network and detects their emergent collective properties. Paradigms of unsupervised learning are Hebbian lerning and competitive learning. We

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say that a neural network learns off-line if the learning phase and the operation phase are distinct. A neural network learns on-line if it learns and operates at the same time. Usually, supervised learning is performed off-line, whereas unsupervised learning is performed on-line. The behaviour of an ANN Artificial Neural Network depends on both the weights and the input-output function transfer function that is specified for the units. This function typically falls into one of three categories: For threshold units, the output is set at one of two levels, depending on whether the total input is greater than or less than some threshold value. For sigmoid units, the output varies continuously but not linearly as the input changes. Sigmoid units bear a greater resemblance to real neurones than do linear or threshold units, but all three must be considered rough approximations. To make a neural network that performs some specific task, we must choose how the units are connected to one another see figure 4. The connections determine whether it is possible for one unit to influence another. The weights specify the strength of the influence. We can teach a three-layer network to perform a particular task by using the following procedure: We present the network with training examples, which consist of a pattern of activities for the input units together with the desired pattern of activities for the output units. We determine how closely the actual output of the network matches the desired output. We change the weight of each connection so that the network produces a better approximation of the desired output. Assume that we want a network to recognise hand-written digits. We might use an array of, say, sensors, each recording the presence or absence of ink in a small area of a single digit. The network would therefore need input units one for each sensor, 10 output units one for each kind of digit and a number of hidden units. For each kind of digit recorded by the sensors, the network should produce high activity in the appropriate output unit and low activity in the other output units. To train the network, we present an image of a digit and compare the actual activity of the 10 output units with the desired activity. We then calculate the error, which is defined as the square of the difference between the actual and the desired activities. Next we change the weight of each connection so as to reduce the error. We repeat this training process for many different images of each different images of each kind of digit until the network classifies every image correctly. To implement this procedure we need to calculate the error derivative for the weight  $EW$  in order to change the weight by an amount that is proportional to the rate at which the error changes as the weight is changed. One way to calculate the  $EW$  is to perturb a weight slightly and observe how the error changes. But that method is inefficient because it requires a separate perturbation for each of the many weights. Another way to calculate the  $EW$  is to use the Back-propagation algorithm which is described below, and has become nowadays one of the most important tools for training neural networks. This process requires that the neural network compute the error derivative of the weights  $EW$ . In other words, it must calculate how the error changes as each weight is increased or decreased slightly. The back propagation algorithm is the most widely used method for determining the  $EW$ . The back-propagation algorithm is easiest to understand if all the units in the network are linear. The algorithm computes each  $EW$  by first computing the  $EA$ , the rate at which the error changes as the activity level of a unit is changed. For output units, the  $EA$  is simply the difference between the actual and the desired output. To compute the  $EA$  for a hidden unit in the layer just before the output layer, we first identify all the weights between that hidden unit and the output units to which it is connected. We then multiply those weights by the  $EAs$  of those output units and add the products. This sum equals the  $EA$  for the chosen hidden unit. After calculating all the  $EAs$  in the hidden layer just before the output layer, we can compute in like fashion the  $EAs$  for other layers, moving from layer to layer in a direction opposite to the way activities propagate through the network. This is what gives back propagation its name. Once the  $EA$  has been computed for a unit, it is straight forward to compute the  $EW$  for each incoming connection of the unit. The  $EW$  is the product of the  $EA$  and the activity through the incoming connection. Note that for non-linear units, see Appendix C the back-propagation algorithm includes an extra step. Before back-propagating, the  $EA$  must be converted into the  $EI$ , the rate at which the error changes as the total input received by a unit is changed.

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## Chapter 3 : Brains Neurons | Compare Prices at Nextag

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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 neuronsâ€™ 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.

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## Chapter 4 : Chemistry for Biologists: Nerves and hormones

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This is an open access article under the terms of the Creative Commons Attribution License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited. This article has been corrected. This article has been cited by other articles in PMC. Abstract The suprachiasmatic nucleus SCN of the hypothalamus is the principal circadian pacemaker of the brain. It co-ordinates the daily rhythms of sleep and wakefulness, as well as physiology and behaviour, that set the tempo to our lives. Disturbance of this daily pattern, most acutely with jet-lag but more insidiously with rotational shift-work, can have severely deleterious effects for mental function and long-term health. The present review considers recent developments in our understanding of the properties of the SCN that make it a robust circadian time-keeper. Daily timing by these loops pivots around the negative regulation of the Period Per and Cryptochrome Cry genes by their protein products. The period of the circadian cycle is set by the relative stability of Per and Cry proteins, and this can be controlled by both genetic and pharmacological interventions. Circadian pacemaking in the SCN therefore provides an unrivalled context in which to understand how a complex, adaptive behaviour can be organised by the dynamic activity of a relatively few gene products, operating in a clearly defined neuronal circuit, with both cell-autonomous and emergent, circuit-level properties. VIP, DREADD, pharmacogenetic, paracrine, sleep Circadian rhythms are those daily cycles of behaviour and physiology that persist when an individual human subject, an experimental animal or a plant, is isolated in a time-free environment. Their persistence betrays the presence of an internal clock that is able, autonomously, to define periods of approximately circa- 1 day -dian. Under natural conditions, these circadian clocks are entrained to the cycle of light and darkness, so that they enable the physiology and behaviour of the organism to anticipate, and thereby adapt to, the solar day and night. In mammals, the principal pacemaker in the brain is the suprachiasmatic nucleus SCN 1 and this receives direct innervation from specialised ganglion cells of the retina that mediate entrainment of the clock by light 2. The role of the SCN is to generate a stable internal representation of solar time, and then to convey that via neural, behavioural and endocrine pathways to co-ordinate all aspects of daily functions across the brain and body. Importantly, the SCN can generate daily time autonomously; the retinal innervation serves solely to synchronise the circadian oscillator, not to sustain it. In this regard, the SCN is a remarkable piece of neurobiology: With the discovery of the molecular feedback loops that constitute the core circadian time-keeper see below , recognition of the medical relevance of circadian clocks has burgeoned. First, all major organs have local circadian clock mechanisms; the SCN is not the sole clock, although it is the orchestrator of innumerable clocks distributed across the body 3. Consequently, vital metabolic processes such as hepatic nitrogen metabolism, gluconeogenesis, cardiovascular function and renal de-toxification all follow precisely defined, interlocking cycles that optimise metabolic performance. The capabilities of brain and body therefore vary as a function of circadian time; thus, we should look upon ourselves as h machines. The consequential misalignment of local clocks with each other and with environmental time, as well as the temporal scramble that occurs during their progressive re-adjustment, is reflected in the various aspects of tiredness, mental confusion and general dysphoria. A far more insidious threat to public health arising from clock disruption comes from rotational shift-work, with epidemiological studies revealing significantly increased risks of cancer, cardiovascular disease and obesity, as well as diabetes 6. Furthermore, animal-based studies have revealed the mechanistic links behind these phenomena, with, for example, circadian disruption leading to insulin resistance 7 , 8. Put simply, if the liver, pancreas and skeletal muscle are not working in time and in tune, effective regulation of blood glucose and insulin is compromised. In addition, there has been a longstanding recognition that psychiatric conditions, especially major depressive disorders, are affected by and in turn affect circadian processes 9. Although mechanistic links remain elusive,

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animal models have again provided novel insights into how clocks, light, sleep and mood may interact. More immediate in terms of public health is the impact of the loss of tight circadian control of sleep on the care and life quality of patients with neurodegenerative diseases. It is the difficulties involved in trying to care for someone without a regular sleep cycle in a home setting that is the principal cause of institutionalisation, with its incumbent personal, social and economic costs. The hope, therefore, is that by determining the basic molecular and neurobiological mechanisms that govern circadian pacemaking, not only will an engaging piece of biology be decoded, but also new opportunities will be presented to address diseases characteristic of modern society. A molecular pacemaker built around feedback loops. At a molecular level, the core oscillatory mechanism of the SCN commences with trans-activation of *Per* and *Cry* genes by heterodimers of *Clock* and *Bmal1*, basic helix-loop-helix transcription factors that associate via so-called PAS dimerisation domains, and act via E-box enhancer elements in their target genes [12, 13]. Fig. Over the course of the circadian morning, the levels of *Per* and *Cry* mRNA accumulate in SCN neurones and, by the end of the circadian day, *Per* and *Cry* proteins appear, form complexes and start to enter the nucleus where they interfere with the actions of *Clock* and *Bmal1*, in part by recruiting transcriptional inhibitory complexes. This ultimately releases E-boxes from negative regulation and the cycle is ready to start anew with a new circadian day. The combined loss of these genes renders mice behaviourally arrhythmic, as does the loss of *Cry1* and *Cry2*, *Bmal1* alone, or *Per1* and *Per2* in combination [1, 2].

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## Chapter 5 : The neurones and supporting elements of the brain of a selachian - CORE

*The neurones and supporting elements of the brain of a selachian* By Gilbert Logan Houser Topics: Chondrichthyes, Nervous system.

The soma houses the cell nucleus and most of the genomic expression and synthetic machinery that elaborates the proteins, lipids, and sugars that constitute the neuronal cytoplasm and membranes. The membranous system bounds and defines different intracellular compartments and includes the outer cell membrane the plasmalemma that encompasses the global compartment defining cellularity itself. Avian optic lobe neuron showing axon emerging from dendrite Cajal. Spinal motor neuron labeled with antibodies to sodium channels at the axon initial segment green and neurofilaments red reproduced with permission. Polarization of a developing spinal motor neuron that accompanies the clustering of sodium channels near the axon hillock red. Early in the evolution of CNS, and during development, such cells were devoid of any plasmalemmal extensions. These bald neurons may be found in present day forms as receptors cells in, for instance, the carotid glomus, in gustatory system in vertebrate tongue or as photoreceptors in the retina. The point being that neurons can have many types of branching or, in fact, no branches at all. For the majority of neurons, however, the receiving or input pole generally consists of extensively branching tree-like extensions of the soma membrane known as dendrites coined in by William His from dendros Greek meaning tree which arise in vertebrate neurons directly from the cell body the body is also a receiving site in most neurons. With invertebrate neurons the dendrites arise most commonly from the axon see Figure 3. The axon conducts propagating electrochemical signals termed action potentials usually initiated at the axon hillock, green in Figure 2 B away from the soma. There are some dendrites that also serve as output systems see dendro dendritic synapses by G. There are, however, exceptions to these general rules of neuronal organization. In some neurons, e. A dye-filled antennal lobe projection neuron, one of the second-order neurons of the *Drosophila* olfactory system [http:](http://) Giant neuron in a cricket? C Confocal image of a leech ganglion from an embryo in which mechanosensory P cells were injected with a combination of the fluorescent dyes Alexa and neurobiotin visualized with Steptavidin-Cy3 and a local bending interneuron was injected with Alexa The electrically coupled cells appear in red. The interneuron appears in yellow due to colocalization of the dyes reproduced with permission. Two neurons in the auditory pathway of crickets named AN2 red and ON1 green. Examples of the great variety of neuronal form are shown in Figure 1 -Figure 6. Those in images Figure 2 -Figure 6 have been made using more recent staining techniques. This set of images represents the common variety of neurons as well as the extremes of morphology to illustrate the enormous variety neurons can generate morphologically. Neuronal function Their electrophysiological properties are as rich as their morphology, and endow neurons with a vast set of electrical properties and functional styles. The voltage and ligand dependent ionic conductances that generate and modulate such excitability can implement autorhythmic properties either as single cell oscillators or resonators that ultimately dictate network oscillatory properties. Early electrophysiological results from the study of motoneurons led to the view that neurons do precious little, other than integrate and fire. Intelligence is in the network was the initial credo in those early years. Many other newer parameters have entered electrophysiology over the last three decades. Plasticity mostly as Long Term Potentiation and Long Term Depression and intrinsic electrical properties have been particularly significant. Neurons are characterized by four main functional properties; a electrical excitability mostly across the plasmalemma , b secretion mostly vesicular and peptide extruding channel dependent , c molecular synthesis mostly proteins , and d growth and plasticity. This short review will deal mainly with electrical excitability. Secretion, mostly as the mechanism responsible for excitatory or inhibitory synaptic transmission, will be dealt in other entries. Electrical excitability When considering neuronal function, electrical excitability is indeed one of the main themes of concern. Passive electrical properties Passive properties refer to the capacitative and resistive aspects inherent in neuronal membranes, along with the resistivity inherent in the

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cytoplasm and the extracellular milieu. Together, these properties provide an electrical resemblance between neuronal processes axons and dendrites and conduction in electrical cables and hence are termed cable properties. While the basic assumption of most electrophysiologists is that the membrane potential may be initially considered as having a resting value the resting potential that is uniformly distributed along neuronal compartments, this is an oversimplification, as the ionic conductances and pumps which are responsible for setting the resting potential need not have a fixed density throughout the neuronal membrane. Even so, isopotentiality is inherent in most initial cable property assumptions. The value of the electrical field in mV is related to the driving force emf for each of the ionic species that can move across the membrane and the magnitude of the conductance for each ionic species  $i$ . While a more or less uniform permeability may be found for the leakage channels a constantly open channel permeable to potassium, voltage gated channels allowing sodium, potassium, calcium, and chloride ions across the plasmalemma also contribute to the membrane potential to the extent of the magnitude of their conductance at a given time. Note however that these latter conductances, because they are voltage-gated, deviate from the simple passive character of the leakage channel. Because of the complexity afforded by the branching pattern of the dendritic tree, the passive electrical properties of neurons were initially difficult to envision without a proper mathematical model. In the mid sixties of last century Wilfred Rall see Rall Model was the first to define the electrotonic cable properties of branching dendritic trees and to show the importance of such branching patterns on synaptic summation at different sites in the dendritic arbor. Neurons conduct waves of membrane potential passively electrotonically a short distance along their processes as the result of currents that flow intracellularly along the longitudinal resistance and simultaneously across the plasmalemmal membrane as resistive or capacitive current. When active properties are engaged these changes can travel the entire length of these processes. Active electrical properties By active electrical properties it is meant that the electrical potentials across the plasma membrane may be affected by the activation of voltage, ligand, or second messenger gated transmembrane ionic channels. The generation of action potentials is an example of electrical properties brought about by active, voltage-dependent means. Here the electric field across the membrane will act on the voltage sensors of transmembrane ionic channels channel sites with dipole moment properties that will trigger conformational changes, often allosteric, that will change channel ionic conductance. In the specific case of action potentials voltage-gated channels, the inflow of sodium or calcium ions depolarizes the plasma membrane. In turn, opening voltage-gated potassium channels and the resulting current flow repolarizes the plasma membrane. Although the conductance of most voltage-gated channels are increased by membrane depolarization, the conductance of some channels is increased when the membrane is hyperpolarized. Other examples of active electrical properties are those brought about by ligand-gated ionic conductances, where the binding of a neurotransmitter will gate ionic conductances allowing the generation of excitatory or inhibitory synaptic potentials. Yet another form of electrical activity is represented by intrinsic subthreshold oscillations, where the excitability of the cell is gated in such a fashion that the membrane potential is not uniform but rather in a state of continuous fluctuation, generating an oscillatory sinusoidal-like membrane profile—often with phase reset properties indicating chaotic, dynamic kinetics. Evolution of the neuron. Drawing of Purkinje cell Purkinje Drawings of dissected motor neurons Deiters Neuronal integration In an active neuron the superposition of passive and active electrical properties serves to allow the cell the possibility of summing the transmembrane potential either linearly or non-linearly and to reach depolarization levels sufficiently high to trigger action potentials. These can be conducted either along the length of the axon or dendritic tree, in an all-or-none continuous manner, in a saltatory fashion, or in a decremental mode. Axonal spike Neurons have but one axon, that is, a single process leaving the soma or a dendrite. However, axons branch either in the form of collaterals along the axonal length or at a terminal arborization known as the telodendron distal dendrite. These terminals are usually the site of presynaptic boutons that establish synaptic contacts with other neurons, muscles, or glands. Because they originate from a single initial segment axons send very similar spike sequences to all their branches. However, spike failure at branch points or changes in conduction

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velocity, secondary to changes in axonal diameter after branching, can reset conduction patterns and conduction times. A good example is the isochrony of spike conduction time found in the axons from the inferior olive that terminate in the cerebellum as what is called climbing fibers. Dendritic spikes In addition to action potentials in axons, dendrites may also generate regenerative events. For the most part these potentials decrement with distance but may reach the most distal dendritic branches See Figure 7. Such action potentials can be centripetal towards the soma or centrifugal back propagating away from the soma depending on the dendritic morphology and the distribution and density of voltage gated ionic channels over the dendritic tree. For more information, see Dendrites , Dendritic spikes , and Dendritic processing. Variety of neurons in which various markers were used to distinguish individual neurons and parts of neurons. Hippocampal neuron transfected micro-injected with GABA A receptor red and co-stained for neurofilaments green reproduced with permission. Triple stained neuron made by Wim Annaert. Three rabbit retinal ganglion cells injected with Alexa Fluor hydrazide top cell, red , Alexa Fluor hydrazide middle cell, green , or both the Alexa Fluor and the Alexa Fluor hydrazides bottom cell, yellow at colocalization. Intrinsic electrical properties and subthreshold oscillations Beyond action potentials and synaptic transmission there is the question of the electrical activity generated autonomously by neurons. In most cases the autonomous intrinsic activity results in a modulation of the resting potential and so the overall state of the neuron in the sense not only of synaptic modulation or the modulation produced by peptidergic, hormonal and metabolic activity, but also of other parameters such as pH and free radical NO and CO activity modulation. Beyond modulation, the parameter that is most relevant in defining intrinsic activity, other than resting potential, is the types and distributions of plasma membrane channels and second messenger activated modulation of channels. Subthreshold oscillatory activity was originally discovered in the inferior olive and then found in many other central nervous system neurons Llinas In addition to the olive such neurons have been found in: These oscillations are supported by persistent sodium currents and calcium current Cav3 and Cav1 subunits , in conjunction with voltage and calcium gated potassium currents. The frequency of these oscillations can vary from high gamma band 80 Hz , Mu 25 Hz , beta 15 Hz , alpha 10 Hz , theta 6 Hz, delta 3 Hz and slow sleep oscillation bellow 1Hz. These oscillations are viewed as supporting time-locked neuronal coherence and resonance. As such they are thought to be of significance in determining large functional brain states such as sleep, wakefulness, and dreaming and abnormal states such as epilepsy and thalamocortical dysrhythmia. Non-uniformity in channels density Finally, the electrical signature of neurons is defined by the passive integrative properties of the dendrites and soma and the non-linear electrical properties superimposed by the presence of voltage, ligand, second messenger, and metabotropic conductances supported by specialized ionic plasma membrane bound channels. These modulate excitability by their number, functional phenotype, and distribution over the dendritic, somatic and axonal neuronal segments. While certain characteristic properties can be assigned to given cellular phenotypes, the fact is that every neuron is unique both in its individually detailed shape and its connectivity. Perhaps it is the diversity of such parameters that allow the CNS to be as reliable as it actually is. Neurons marked and photographed in place. Pyramidal neurons of mouse cortex. Three Golden Columns - reproduced with permission. A pyramidal shaped neuron in the cortex and a second neuron in the underlying thalamus? Neurons in Drosophila, Andreas Prokop. Retinal cells Wallace B. Retroviral vector expressing GFP in adult mouse hippocampus. GFP expression in the dentate gyrus 4 weeks after virus injection express mature neuronal markers. Neuronal marker is NerN red, Cy3. Nature Vol , 28 Feb , pg Neurons within song control nucleus RA. Cortical neuron showing axon projection. The initial differentiation of these cells into true neurons or muscle cells precedes the further differentiation of nerve cells into sensory neurons in communication relation to the external world or motor neurons in direct communication with muscles or glands Figure 4 Ab. The final stage occurs with the development of interneurons establishing contacts between sensory and motor neurons Figure 4 Ac. The latter represent the vast majority of neurons in the brain and ganglia.

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## Chapter 6 : SELACHIAN - Definition and synonyms of selachian in the English dictionary

*The neurones and supporting elements of the brain of a selachian. By Gilbert Logan. Houser. Abstract. Mode of access: Internet.*

Another neurone, relay or sensory neurone To an organ e. Motor neurones are sometimes called effector neurones since they produce a physical response to stimulation. The general structure of nerve cells The neurone in general contains a cell body with a nucleus that controls the activity of the cell. The cytoplasm within the cell is extended to produce dendrons. Each dendron has a number of long fine structures called dendrites. These dendrites are stimulated by electrical impulses from other neurones. The information is then passed to the cell body. The axon is the long thin section of the neurone, which can be up to a metre long. This is formed by a single extension of the cell body cytoplasm. The axon always transmits impulses away from the cell body. Axons end in a series of synaptic knobs. These structures stimulate other nerves or a target organ, in which case a physical response happens e. Another important feature is Schwann cells. These cells are found along the length of the axon. Schwann cells wrap around the axon with small gaps between each cell. Neurones with Schwann cells are called myelinated neurones. These cells act as an electrical insulator and speed up transmission of impulses. There are neurones that are unmyelinated; they transmit impulses more slowly than myelinated neurones. Nerves and their impulses All living cells maintain an electrical potential difference across the cell membrane, i. This is called the membrane potential. Neurones have the ability to change their membrane potential. Under normal conditions no stimulation the membrane of a neurone has a negative charge -ve , compared to its surroundings. This is known as the resting potential. The resting potential depends on the concentration of four ions within the cell: When a membrane is in this condition it is said to be polarised. When a neurone is stimulated the electrical potential of its cell membrane is altered, it is depolarised. Depolarisation changes the permeability of the membrane towards sodium ions at the site of the stimulation causing a sudden influx of sodium ions into the axon. Now the overall charge inside the cell is more positive. This is known as the action potential. An animation showing the propagation of the action potential can be viewed on: This flow of potassium ions continues until the resting potential is achieved, that is the concentration of the ions, is restored in this region of the axon and the membrane is re-polarised. As the concentration is restored in the first section, the polarisation of an adjacent section of the membrane is depolarised. The ion transfer reaction is repeated. These reactions are localised, they start at the first stimulation point on the axon. The first reaction starts a wave of localised ion transfer reactions. These reactions propagate a series of action potentials followed by resting potentials repeated at regular intervals along. In this way electrical or nerve impulses are transported along the whole length of the axon by the movement of ions between the axon and its external environment. The Synapse Once the nerve impulse has passed to the end of the axon, the dendrites, it needs to be transferred to another neurone or tissue. At the end of each dendrite is a bulbous structure called a synaptic knob. The synaptic knob contains many structures common to living cells. In addition they have synaptic vesicles. These vesicles contain a chemical that assists the transfer of the impulse, a neurotransmitter called acetylcholine. The pre-synaptic membrane binds to the end of the adjacent neurone. Large protein molecules called receptor molecules are found on the surface of the postsynaptic membrane. There is a gap between the two structures about 20 nm wide known as the synaptic cleft. The nerve impulse is transported across the synaptic cleft by a similar method used to transport the impulse along the length of the axon, that is by the propagation of action potentials. Acetylcholine - click on image to open When the nerve impulse reaches the pre-synaptic membrane it depolarises the membrane. This causes changes in the electrical potential of the immediate environment, i. The empty vesicles return to the cytoplasm. The acetylcholine diffuses across the synaptic cleft and fuses with the receptor molecules at the surface of the post-synaptic membrane. As soon as acetylcholine depolarises the post-synaptic membrane, it must be removed from its surface to allow for the transmission of another impulse. This is achieved with

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assistance of water and a suitable biological enzyme. The acetylcholine molecule is hydrolysed by water. This reaction breaks the acetylcholine to make two products. Choline from acetylcholine These products are released by the receptor molecules. They diffuse across the cleft and back into the pre-synaptic neurone where they recombine to form acetylcholine. These molecules are stored in the synaptic vesicles for future use. A lot of energy is required for the recombination process that is provided by the many mitochondria present. Successive nerve impulse transmissions build up on the post-synaptic membrane until enough depolarisation has taken place and an action potential is generated. The impulse is then transported by the propagation of action potentials along the length of this neurone to another neurone or to a target organ. The effect of drugs on synaptic transmissions Discovering the chemical structure of neurotransmitters has given chemists an understanding of the action of drugs and poisons on the nervous system. There are many drugs that are known to influence the functioning of synaptic transmissions. An example is nicotine found in tobacco products. Nicotine is only one of different compounds found in tobacco smoke. Nicotine - click on image to open Nicotine is part of a group of nitrogen-containing chemicals called alkaloids. Alkaloids have hydrocarbon-based skeletons, i. Examples of other alkaloids are caffeine, morphine and cocaine. Nicotine mimics the action of neurotransmitter chemicals like acetylcholine. Both molecules are based on hydrocarbon skeletons but the important fact about these structures is that they contain a nitrogen atom with a positive charge. This makes the structures very reactive in the part of the molecule that has the charge. Acetylcholine receptors on post-synaptic membranes will accept nicotine because it has a similar arrangement of is atoms and similar charge on the nitrogen atom. When tobacco is burned, small droplets of tar containing nicotine are inhaled and find their way to the lungs and eventually to the alveoli or air sacs. Nicotine is a weak base pH 8. It is rapidly absorbed through the fine membrane of the air sac and the mouth into the bloodstream. From this point nicotine is distributed very quickly throughout the body, taking about eight seconds to reach the brain. In the brain it creates a burst of activity amongst the acetylcholine receptors to give a feeling of pleasure. The initial concentration of nicotine is high after one inhalation. It takes about 45 minutes for this concentration to be reduced by half. At low concentrations it acts as a stimulant at higher levels it acts as an inhibitor, i. When nicotine is bound to the postsynaptic receptor, it depolarises the membrane triggering the influx of sodium ions from surrounding tissues. This initiates a wave of action potentials as before. However nicotine is not removed by hydrolysis so the stimulation is maintained, i. However eventually nicotine is broken down mainly in the liver by oxidation, in a number of stages with the assistance of enzymes. This over-stimulation happens at all axons exposed to nicotine and it has an effect on all organs and functions. One adverse effect of the over-stimulation of nerve fibres is the constriction of blood vessels, at the same time stimulating the heart making it beat faster and increasing the blood pressure. As the level of nicotine falls the affected neurones have a chance to recover. However it is likely that long-term use of nicotine is likely to result will result in chronic illness or death as there will always be permanent tissue as well as nerve damage. Hormonal coordination Hormones do not belong to one particular chemical group. Some are amines, nitrogen-containing molecules, others are protein and polypeptide in origin. A few are steroids that are derived from fats and lipids. These chemicals messengers are passed in very small amounts, directly into the bloodstream by glands that collectively form the endocrine system. Once in the bloodstream the hormones are carried to all parts of the body. They bring about specific effects in the behaviour and development of animals. The basic similarities between the nervous and the endocrine systems are that they provide the body with methods to communicate with its internal and external environments in order to coordinate responses. They both employ chemicals to transmit messages and respond to stimulus caused by changes in their environments. However there are differences in: Hormones are transported all over the body via the blood, so response times will vary. Puberty is the stage in human development when children become adults. This transition takes years to complete and is controlled mainly by the hormones oestrogen, progesterone and testosterone that are steroids in origin.

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## Chapter 7 : Neuron - Scholarpedia

Buy *The Neurones and Supporting Elements of the Brain of a Selachian* () by Gilbert Logan Houser (ISBN: ) from Amazon's Book Store. Everyday low prices and free delivery on eligible orders.

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Nerves allow electrical impulses to propagate along their elongated cell extensions and facilitate the transfer of information throughout the body. Neural tissue is found within the central nervous system CNS and the peripheral nervous system PNS and the composition and constituent parts of neurones and their surrounding cells differ only slightly. The gray matter includes nerve cell bodies and some short branches from these cell bodies. The white matter is composed of the long extensions of the nerve, the nerve fibres. Within the central nervous system there are two major cell types; neurons which are the "functional" cells of the central nervous system and glial cells which play a supporting role within the CNS. Within the CNS there are also a number of other cells that play a supportive role to the neurone and these include astrocytes, oligodendrocytes, microglial cells, ependymal cells and choroid plexus epithelial cells.

**PNS Neurons** The PNS contains a series of paired nerves that extend from the spinal nerves originating from the spinal cord and cranial nerves that originate from the brain stem. Supportive cells that surround the nerves in the PNS are called Schwann cells rather than Glial cells. The main neurotransmitter found in the PNS is acetylcholine.

**Neurons** A typical neuron can be exemplified by a motor neuron in which the cell body of the nerve is located within the gray matter of the spinal cord and the nerve fibre, or axon, extends to the muscle. Nerve axons can be very long permitting electrical impulses to be sent over long distances throughout the body. The information below specifically regarding neurons is interchangeable between the CNS and PNS and therefore links have been provided where appropriate on this page to the PNS Structure.

**Basic Nerve Structure** The basic structure of a nerve is that of a cell body soma, which has a single long nerve fibre axon, attached at one end to the cell body and at the other end to another nerve cell body or to a structure requiring nerve impulses such as skeletal muscle. The interface between two nerves or a nerve and another structure is called the nerve synapse. The cell body of the nerve itself also contains numerous dendrites which increase its surface area enabling other nerve axons to connect with the cell body. The cell body usually has connections with many other axons from other nerve cells with many synapses on the cell body. The axon of the nerve cell is usually surrounded by some form of insulating protection, or myelination. This protective layer is called the myelin sheath.

**Soma Cell Body** The soma, or cell body, is the central part of the neuron and contains the nucleus of the cell, the rough and smooth endoplasmic reticulum, ribosomes and golgi apparatus. Similar processes that would be undertaken by any cell occur within the soma and due to the organelles it contains, the soma is where most protein synthesis occurs. Collectively, these branching structures of the dendrites are known as the "dendritic tree". The dendritic tree is the site where input to the neuron occurs via synapses with axons from other nerve cells. However, dendrites themselves are unable to propagate nerve impulses in the manner of axons as dendrites are unable to secrete neurotransmitters. Similarly axons do not possess the chemoreceptors that are found within the dendrites and are therefore unable to receive nerve impulses. Nerve impulses are therefore conducted in one direction only.

**Axon Nerve Fibre** The axon is a very fine projection that can measure up to thousands of times the diameter of the soma in length. The axon carries nerve signals away from the soma. The primary function of glial cell is to provide support to the neuronal cells. There are several different types of glial-style cell; in the PNS these are referred to as Schwann cells and whilst in the CNS they are referred to as Oligodendrocytes. Glial cells form a protective networked layer around the neuron within the CNS and also help to maintain the fluid content of the tissue surrounding the nerve. This protective layer is referred to as the myelin sheath and axons that are wrapped within this myelin sheath are able to conduct nerve impulses at higher speeds than those that are not wrapped. During foetal development the glial cells wrap around axons numerous times and as the glial cell matures it loses the majority of its cytoplasm.

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The remaining cellular structure consists of many layers of tightly packed lipid membranes around the axon. Periodically along the surface of the wrapped glial cell there are gaps approximately every mm. These gaps in the sheath are called the Nodes of Ranvier and are important in the conduction of impulses along the axon.

**Oligodendrocytes** Oligodendrocyte The term "oligodendrocyte" literally means a "cell with many branches". Oligodendrocyte precursors are found throughout the CNS and these precursors are post-mitotic, meaning oligodendrocytes can be replaced. Oligodendrocytes can myelinate up to 40 axons at a time. The myelin sheath formed by oligodendrocytes has numerous functions including; decreasing any ion leakage from the axon lowering the capacitance of the cell membrane and increasing impulse speed along the axon by allowing saltatory conduction of action potentials between the nodes of Ranvier.

**Nerve Impulse Propagation** Nerves are able to create, amplify and propagate electrical impulses that run along their axon. These nerve impulses are a form of action potential that is carried by ions. The nerve impulse is in effect an electrical difference between the inside and outside of the axon and is caused by ion movements across the membrane. Nerve impulses can occur from a number of sources including sensory cells, action potentials from other connected nerves or spontaneous depolarisation of the nerve cell membrane.

**Unmyelinated Axons** Conduction of an action potential along an unmyelinated axon, i. At a given moment when the action-potential threshold is reached voltage-gated sodium channels within the membrane of the axon will open resulting in an influx of ions following their electro-chemical gradient. The extracellular area of the axon therefore loses its positive charge, becoming more negative resulting in a current of positive charge that flows through the tissue towards the axon. The membrane of the axon is not a perfect insulator and at some regions on the axon, particularly within the area of the axon in front of the action potential, the voltage-dependant ion channels have not activated yet. This means that some of the positive charge is able to flow out of the axon membrane in these regions and this positive charge outflow is mainly via potassium. This outward leakage of potassium results in the current within the axon only being able to travel a short distance before the nerve impulse decays. However the effect of the current locally on the voltage-gated channels means that the nerve impulse is able to open voltage-gated channels within its immediate vicinity and this is enough for the signal to propagate along the nerve. The axon membrane at rest has a slightly negative charge and the local current of positive charge during an action potential reduces this negative charge. This process is called depolarisation and when this has progressed sufficiently, it results in the membrane potential reaching threshold level allowing the voltage-gated sodium channels to open. These channels only stay open for approximately 0. These voltage-gated channels are unable to open again for a short period post depolarisation. This is called the refractory period. The internal electrical resistance within the axon is the determining factor regarding how fast the local positive current impulse can pass through. The less the internal resistance, the higher the conduction velocity of the nerve impulses due to the locally positive current being able to depolarise the membrane to threshold level over a greater distance. This internal resistance decreases as a function of axon diameter and therefore thicker axons are able to conduct nerve impulses more rapidly.

**Myelinated Axons** The addition of the myelin sheath to nerve axons greatly enhances the speed with which they are able to conduct nerve impulses. For an unmyelinated nerve to conduct impulses at the same rate, it would have to have a diameter of approximately times that of the myelinated nerve. As with unmyelinated axons, an area of locally positive charge flows through the cytosol from the activated area within the axon. The major difference with myelinated nerves is that where the current effectively leaks away in unmyelinated nerves, here the current is only able to leak away through nodes of Ranvier see above. Therefore even a weak current is able to depolarise the axon membrane to the threshold level. There is a small loss of current but as these nodes are relatively small the local current is able to travel much further in myelinated axons resulting in a much higher conduction velocity. This type of impulse propagation is called saltatory conduction. Myelination of axons in mammals means that the nervous system can sustain a large number of high velocity axons within a relatively small space.

**Synapses** The synapses found at the end of axons are fundamental to the functioning of the nervous system as they facilitate communication between nerves and provide an interconnected network for

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many of the complex processes required by organisms. Synapses are required as the lipid bi-layer of the cell membrane has a relatively large electrical resistance making electrical impulse propagation directly between cells difficult. The most common form of nerve synapse is the chemical synapse which utilises neurotransmitters. When a nerve impulse reaches a synapse, neurotransmitters are released by the presynaptic terminal of the synapse and these transmitters diffuse to the membrane of the post-synaptic membrane where they bind to receptors. These receptors cause an inhibition or excitement in that nerve resulting in either blocking further electrical impulses or the further propagation of a signal.

**Neuromuscular Synapses** These chemical synapses provide connection between nerves and skeletal muscle cells. These synapses are most commonly found residing within groups of muscle cells where each neuron is in contact with several muscle cells but each muscle cell is only ever connected to one neuron. The nerve axon branches out prior to the muscle cells allowing multiple synapses with muscle cells from a single nerve axon. A synaptic cleft of approximately nm is found between the nerve synapse and the muscle cell. The nerve terminal membrane or pre-synaptic membrane contains numerous vesicles that contain the neurotransmitter, in this case most commonly acetylcholine ACh. Once released from the vesicles the ACh diffuses across the synaptic cleft and binds to receptors in the muscle cell membrane. The movement of potassium is relatively small due to there being only a small electrochemical gradient between the extra and intracellular environment. However there is a large influx of sodium into the cell and consequently this causes depolarisation in the muscle cell thus propagating the impulse into mechanical movement. Within the neuromuscular junction, the release of vesicles is facilitated by an influx of calcium into the pre-synaptic nerve just prior to exocytosis. There are several mechanisms that reduce the intracellular concentration of calcium once vesicles begin to be released to ensure that the neurotransmitter release is brief to prevent hyperpolarisation. The particular neurotransmitter ACh is heavily recycled within the synaptic cleft via endocytosis and over time the levels of endocytosis and exocytosis balance one-another resulting in a stable pre-synaptic membrane. Depolarisation within the muscle cell will last as long as the ACh is present in sufficient quantities within the synaptic cleft. In reality this only lasts a few milliseconds as the synaptic cleft also contains the enzyme acetylcholinesterase which hydrolyses the ACh into acetate and choline. Due to this mechanisms, the calcium restriction and the endocytosis, only one action potential is generated within the muscle fibre.

**Inter-neuron Synapses** The interaction between nerves within the synapse is fairly similar to that within the neuromuscular junction but there are several key differences. Firstly the receiving neuron will be receiving information from multiple other nerves rather than just one nerve as per a muscle cell. Secondly there are many more types of neurotransmitters utilised by inter-neuron synapses than just ACh. There are also a wider range of types of synapses between neurons that include both excitatory which will propagate a nerve impulse, but also inhibitory which will prevent another synapse on the same nerve propagating a nerve impulse. Excitatory synapses will lead to depolarisation of the target neuron and if this depolarisation reaches the threshold level, an impulse will be propagated. This depolarisation is referred to as excitatory post-synaptic potential EPSP as it brings the membrane potential of the target cell closer to the threshold for an action potential. If several excitatory synapses on the same nerve excite simultaneously, the target cell will receive the sum of all these individual synaptic potentials. Therefore multiple e active excitatory synapses are more likely to cause a threshold depolarisation. Inhibitory synapses between neurons also have many synapses from other nerves connecting to a single nerve providing a network of connections. However, the neurotransmitter molecules within an inhibitory synapse usually open ligand-gated channels for chloride-ions and potassium ions. This adjustment in membrane potential is called a inhibitory postsynaptic potential IPSP as it takes the membrane potential of the cell further from the threshold level required to generate an action potential.

**Astrocytes** Astrocytes, or astroglia, are star-shaped glial cells within the brain which have many processes that envelope synapses made between neurons. Astrocytes have several functions including the biochemical support of the endothelial cells in forming the blood-brain barrier, provision of nutrients to nervous tissues and to form scar tissue during repair of the brain. The fluorescent image of astrocytes on the right is possible because astrocytes express glial fibrillary acidic protein GFAP

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which facilitates their identification.

### Chapter 8 : Full text of "The neurones and supporting elements of the brain of a selachian .."

*Gilbert L. Houser, The neurones and supporting elements of the brain of a selachian, Journal of Comparative Neurology, 11, 2, (), (). Wiley Online Library.*

### Chapter 9 : Circadian Pacemaking in Cells and Circuits of the Suprachiasmatic Nucleus

*The translations of selachian from English to other languages presented in this section have been obtained through automatic statistical translation; where the essential translation unit is the word «selachian» in English.*