

Neuronal State Theory of Cognition, Learning, Sleep and Dreams

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Research Article

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Posted Date: July 19th, 2021

DOI: <https://doi.org/10.21203/rs.3.rs-732972/v1>

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Neuronal State Theory of Cognition, Learning, Sleep and Dreams

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Abstract

Understanding the workings of brain and nervous system; the cognitive processes they run has fascinated humanity for centuries. In an attempt to answer these questions, this paper presents a novel framework called ‘Neuronal State Theory’ rooted in the notion that a neuron has different neuronal states resulting from the concentrations of ionic species inside neurons. It is shown how all cognitive processes result from neuronal state theory. Furthermore, it is also shown all cognitive research fields (attention, memories, learning, imagination, sleep and dreams) emerge and are different aspects of the same presented framework.

Keywords:

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Introduction

How does cognitive processes in a human brain works? What are memories; where and how they are stored; how do we retrieve them? How do we learn and imagine? Why do we have a need to sleep, and when we do sleep, why do we dream? What are dreams? These questions, have fascinated humanity for centuries, as they are crucial in understanding human experience; our inherent ability to interact with the environment. If one attempts to answer these questions, the logical starting point is to study the workings of a human brain and the nervous system, and their resulting cognitive processes, which in totality is an intricate task. However, human race has made significant progress, not just in understanding the workings of nervous system, but also the cognition. The answers to the above questions have been diligently exposed in the literature, both in quantity and quality, individually and collaboratively. For example, there is a rich literature on memory models alone, along with the reviews of these models. In this article, conscious decision has been made, not to provide a survey of any literature, because it won't serve any purpose in giving context to the notions of this articles. The purpose of this article is to establish a novel framework/theory rooted in the universally accepted physiological aspects of neurons and it will be shown that how cognitive processes results from the presented framework. Secondly, it will be shown that all fields of cognitive research are emergent fields of the presented framework. In other words, all cognitive research fields are different aspects of a single framework presented in this article. The thesis presented in this article is divided into two parts. Part 1 develops necessary concepts of the framework and Part 2 shows how cognitive processes and cognitive research fields result from the concepts of the framework.

Neuron – a working unit of brain and nervous system has been studied extensively. It is well established that there are three general types of neurons, Receptor Neurons, Interneurons and Effector Neurons. These are the basic machinery of brain and nervous system and all the

processes of brain and nervous system results from the workings of these basic units; individually and in combination. The foundational notion of the presented framework is that neurons have neuronal states, which depends on the ionic species inside the neurons. To serve this purpose, the starting point of the discussion will be to establish the workings of a single neuron in terms of neuronal states. In section 1, all the necessary definitions, notions and notations of neuronal states are established. Furthermore, Action Potentials and Subthreshold Potentials for a single neuron in terms of its states are discussed. In section 2, communication between pre- and post-synaptic neurons based on their states will be discussed using a ‘3-Component Factory, Manager and Worker’ Model. Once the framework of communication between neurons is established, activations of neurons and factors affecting neuronal activation will be discussed in section 3. In section 4, concepts of state adaptabilities i.e. likelihood a neuron activates to a particular state will be presented. In section 5, output responses of a neuron, along with the neuronal behaviours will be discussed. Furthermore, notion of probabilities that a neuron generates a particular output response will be established. In section 6, set theory will be used to generalise the discussion to any arbitrary, X-Neuronal system, where X-neuronal system can be a human nervous system. Furthermore, a 4th component called ‘Directorial Board’ will be added to the previously used 3 Component Factory, Manager, Worker Model. It will be established that Directorial Board comprises of Local Directorial Boards LDB, which are crucial in cognitive processes. In depth workings and functionality of LDBs will be established.

In Part 2, it will be shown that cognitive processes result from the framework of neuronal state theory. Furthermore, types of cognitive processes, their architectures and capacity of X-Neuronal system to run cognitive processes will be presented. In the following sections 8-12, it will be shown that cognitive research fields like attention, memories, learning, imagination, sleep and dreams are emergent fields of neuronal state theory which emerge from a single

framework. All the necessary definitions, architecture and examples will be presented of these emergent fields. In conclusion, a novel neuronal state theory will be presented and this single theory will explain every architectural aspect of cognition, learning, sleep and dreams.

Part 1

In part 1 of this article, all the mandatory basic elements and concepts of neuronal state theory will be discussed.

1. Neuronal States

In this section, the workings of a single neuron in terms of neuronal states is presented along with the necessary basic definitions, notions and notations used in this article. Consider an arbitrary Receptor Neuron defined as R_{1x} , where $x = 0,1,2, \dots, y$ representing the state of neuron R_1 and y represents the total number of possible states R_1 can be in. It is well known that a neuron maintains a resting membrane potential of about -70 mV, by having low concentrations of Na^+ ions and high concentration of K^+ ions inside the cell. Other ionic species are also involved, but for simplicity, only Na^+ and K^+ ions will be discussed first and in later sections, the discussion will be generalised to all involved ionic species. For the purpose of this article, let the low concentration of Na^+ denoted by a value c_{Na} and high concentration of K^+ denoted by a value c_K inside the cell at the resting membrane potential be the rest state of a neuron, and this rest state is defined by $x = 0$. The arbitrary neuron R_1 in rest state is defined as R_{1_0} . The states of R_1 where $x > 0$ are the activated states of R_1 . Change in ionic concentrations of Na^+ and K^+ ions from the rest state concentrations c_{Na} and c_K gives rise to the activated states. It should be noted that R_1 in activated state can have membrane potential equal to the resting membrane potential, as the ionic concentrations of the involved ions inside the cell will be different than those of R_1 in rest state.

Now, consider R_{1_0} stimulated by an arbitrary stimulus s , depolarising R_1 by the movement of Na^+ ions inside the cell through voltage-gated Na^+ channels, increasing the membrane potential of R_1 and causing the transition of states of R_1 from R_{1_0} to R_{1_α} where $\alpha > 0$. i.e. $s \mapsto R_{1_0} \Rightarrow$

$R_{1\alpha}$. It is universally realised that depolarisation of neuron by influx of Na^+ ions has generally two outcomes; either the neuron triggers Action Potential (AP), if depolarisation causes the voltage to increase beyond a certain threshold T , or the neuron has subthreshold membrane potential if the depolarisation is below T . It is imperative that both cases be discussed in terms of states of R_1 for the purposes of this article.

1.1. Action Potential

If depolarising of R_1 by stimulus s is above threshold T , R_1 will undergo Action Potential (AP). A typical curve of membrane potential over time for R_1 is shown Figure 1. The focus here is to discuss the neuronal states of R_1 along the membrane potential curve. To serve this purpose, certain points (B, C, D, E and F) on the curve of Figure 1 are selected. It has been previously established that at Point B i.e. at the time of stimulation, R_1 is in rest state R_{1_0} . After stimulation, R_{1_0} starts transitioning to an activated state, with the influx of Na^+ ions. Let the activated state of R_1 at Points C, D and E be given by $R_{1\alpha}$, $R_{1\beta}$ and $R_{1\xi}$ respectively. It should be noted that along the path B to C, neuronal state transitions from one activated state to another, until it is updated to $R_{1\alpha}$ at Point C. Likewise, the state transitions to $R_{1\beta}$ at Point D after several state transitions along the path C to D. R_1 in state $R_{1\beta}$ has membrane potential equal to that of rest state R_{1_0} , nonetheless $R_{1\beta}$ is an activated state, since the ionic concentrations inside R_1 is different. At Point E, the state updates to $R_{1\xi}$ which is still an activated state. However, at Point F, R_1 returns back to its rest state R_{1_0} . If the neuron in question undergoes AP, neuronal states complete a cycle of state updates, starting from rest state and returning to rest state. For neuron R_1 undergoing AP, the cycle of state update is given by $R_{1_0} \Rightarrow R_{1\alpha} \Rightarrow R_{1\beta} \Rightarrow R_{1\xi} \Rightarrow R_{1_0}$. Neuron R_1 becomes ready to receive another stimulus in rest state R_{1_0} .

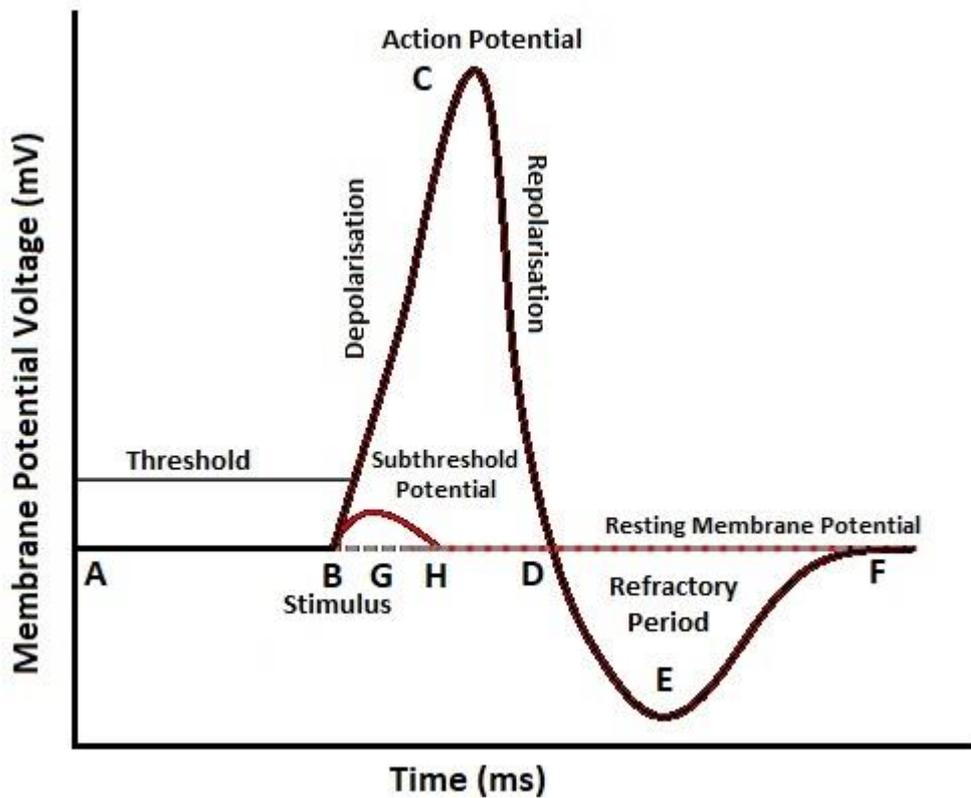


Figure 1. Typical Membrane Potential of neuron R_1 overtime. From time A to B, R_1 has resting membrane potential and is in rest state R_{1_0} with c_{Na} and c_K inside the cell. At B, R_1 is stimulated by s . From B to C, drastic influx of Na^+ ions by voltage-gated ion channels causing the voltage to increase. Since the voltage is beyond threshold voltage, AP occurs at C. Influx of Na^+ ions increases the osmolarity of R_1 , causing water molecules to move inside R_1 by osmosis, resulting in swelling of R_1 . At C, Na^+ gates close, stopping the influx of Na^+ ions, K^+ gates open. Na^+/K^+ -ATPase is activated, as a result of R_1 swelling. From C to D, K^+ ions move out of R_1 and Na^+/K^+ -ATPase pump moves 3 Na^+ ions out and 2 K^+ ions inside R_1 using 1 ATP molecule, causing the net repolarisation of R_1 . At time D, R_1 returns back to the resting membrane potential. Beyond D to F, is the refractory period of R_1 . Membrane potential falls below the resting membrane potential from D to E, likely due to the continuous flow of K^+ ions out of R_1 , and the workings of Na^+/K^+ -ATPase pump. From E to F, Na^+/K^+ -ATPase pump continues to work, until R_1 returns to resting membrane potential by having c_{Na} and c_K concentrations inside R_1 . Hence at F, R_1 returns to its rest state R_{1_0} . However, at B if stimulus s does not depolarise R_1 beyond threshold, membrane potential follow the path B to H. Na^+ ions flow into R_1 from B to G. At G, Na^+ gates close and K^+ gates are opened. From G to H, K^+ ions move out of R_1 repolarising it. It is likely, that small depolarisation from B to G is insensitive to the triggering of Na^+/K^+ -ATPase pump. Repolarisation from G to H is the out flow of K^+ ions. At H, R_1 reaches resting membrane potential, however it is still in some arbitrary activated state, because the ionic concentration is different from the ionic concentration at rest state R_{1_0} .

1.2. Subthreshold Potential

If depolarising of R_1 by stimulus s falls below threshold T , R_1 has subthreshold potential shown by Figure 1. In terms of neuronal states, points B, G and H are selected on the membrane potential curve of Figure 1. Let the activated states at Points G and H be given by $R_{1\sigma}$ and $R_{1\epsilon}$ respectively. Neuronal state of R_1 transitions from R_{1_0} at B to $R_{1\sigma}$ at G with the influx of sodium ions inside R_1 . Followed by the state transition to $R_{1\epsilon}$ at Point H which results from the outflow of K^+ ions. $R_{1\epsilon}$ at H has resting membrane potential, nonetheless R_1 is in activated state for the reasons of different ionic concentrations inside R_1 at point H. In subthreshold potentials, it is an incomplete cycle of state update given by $R_{1_0} \Rightarrow R_{1\sigma} \Rightarrow R_{1\epsilon}$ where the final state of R_1 is an activated state. Neuron R_1 in this case will receive another stimulus in activated state $R_{1\epsilon}$. Consider neuron R_1 in its current activated state $R_{1\epsilon}$ stimulated by an arbitrary stimulus s_1 . As discussed previously, the depolarisation of R_1 cause the transition of state from $R_{1\epsilon}$ to a new excited state. If the depolarisation still falls below the threshold T , R_1 will return to the resting membrane potential, but will be in the new unique excited state, with different ionic concentrations inside R_1 from state $R_{1\epsilon}$. A typical membrane potential curve overtime of R_1 under different stimuli representing Graded Potentials is shown in Figure 2. *Graded Potentials are changes in membrane potential which are always below threshold potential, but cause neuron to transition to a unique excited state on returning to the resting membrane potential after every potential change. Only after AP, the neuron returns to its rest state.*

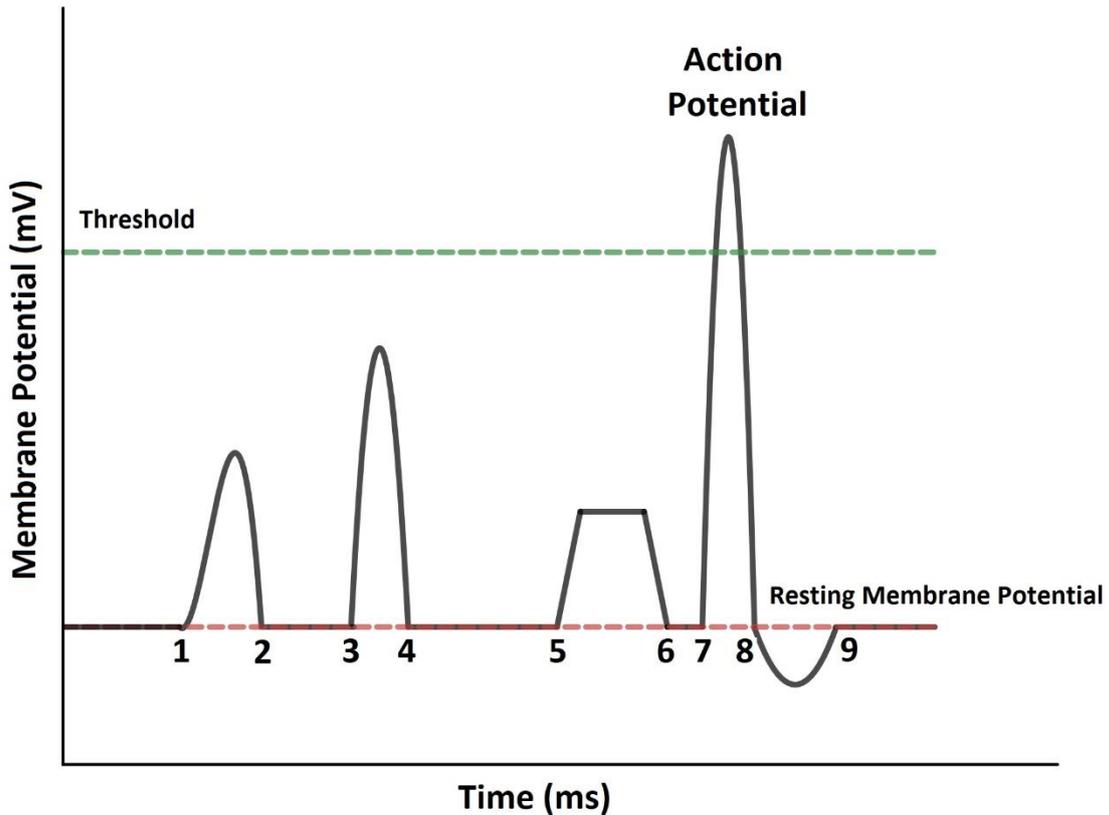


Figure 2. Typical Membrane Potential Curve overtime of Neutron R_1 . At Point 1, R_1 in excited state R_{1_e} at rest membrane potential is stimulated by an arbitrary stimulus s_1 . Depolarisation falls below threshold, so R_1 transitions to a new excited state at Point 2 from R_{1_e} , which is also at resting membrane potential. At 3, R_1 is stimulated again by a new stimulus s_2 , which causes depolarisation greater than stimulus s_1 , but still less than Threshold. R_1 transitions to yet another excited state at resting membrane potential at Point 4. At Point 5, R_1 is stimulated by a stimulus s_3 , which is longer in duration, yet small enough to cause depolarisation below threshold. R_1 transitions to a new excited state at Point 6. These changes in the membrane potential overtime represents Graded Potential, where potentials are always below threshold, but neuron R_1 is in unique excited state after returning to the resting membrane potential. However, at Point 7, R_1 is stimulated by a stimulus, which depolarises R_1 beyond Threshold causing AP. After AP, R_1 undergoes refractory period from Point 8 to Point 9. At Point 9, R_1 returns to its resting state R_{1_0} , completing the state update cycle.

2. Neuron to Neuron Communication

In this section, communication between two neurons is presented. It is well understood that synapses play a crucial role in neuronal communication. Two fundamentally different types of synapses (chemical and electrical) have been well established. First, chemical synapse will be discussed to establish necessary definitions and generalisations. A neuron after undergoing AP, release neurotransmitters into the synaptic cleft. These released neurotransmitters bind to the receptors, located on the plasma membrane of the postsynaptic neuron, which forms the basis of chemical signal transmission between two neurons. The response of postsynaptic neuron can be either excitatory (depolarisation) or inhibitory (hyperpolarisation) depending on the type of neurotransmitters. For the notions of this article, the mechanisms of neurotransmitter release and types of neurotransmitter are not important, rather the precedent will be given to the response of the postsynaptic neuron. In the previous section, the depolarisation and repolarisation and resulting states of the neurons were discussed only taking Na^+ and K^+ ion species into consideration. First of all, it is imperative that other ionic species be taken into consideration, which can potentially depolarise or repolarise neuron. To serve this purpose, the rest state of neuron R_{1_0} depends on the concentrations of ions denoted by c_i at resting membrane potential inside R_1 , where $i = 1,2,3 \dots j$; i represent the specific ionic specie and j are the total number of ionic species. Any change from these concentrations at rest state, regardless of the quantitative value of concentrations, transitions neuron to an activated state. As an example, suppose c_3 represents the concentration of Cl^- ions inside R_1 at rest state. Change in c_3 either by influx or outflux changes the rest state of R_1 to an activated state.

Now consider two neurons R_{1_x} and R_{2_x} , where $x = 0,1,2, \dots y$ representing the state of neuron R_1 and R_2 ; y represents the total number of possible states R_1 and R_2 can be in. Furthermore, consider R_1 to be a presynaptic neuron and R_2 to be a postsynaptic neuron and both neurons

are in rest state R_{1_0} and R_{2_0} respectively. At some arbitrary time (t), R_{1_0} is stimulated by a stimulus s depolarising R_{1_0} beyond threshold potential resulting in AP. Neurotransmitters are released into the synaptic cleft. These neurotransmitters bind to the receptors of R_{2_0} making it permeable to specific type of ionic species. The type of neurotransmitters determines which ionic species become more permeable, to move in or out of postsynaptic neuron R_2 . As an example, if the neurotransmitters from R_1 are excitatory, Na^+ voltage-gated channels open causing the influx of Na^+ ions in R_2 resulting in depolarisation and transition to an excited state. The amount of depolarisation then governs the behaviour of R_2 whether, it will undergo AP or subthreshold potential, as discussed for a single neuron in previous section. On the contrary, if the neurotransmitters from R_1 are inhibitors, Cl^- ions might become more permeable to move in R_2 causing hyperpolarisation and R_2 transition to some unique excited state.

2.1. Factory, Manager and Worker Model

For chemical synapse communication, neurotransmitters from the presynaptic neuron governs the response of postsynaptic neuron i.e. how it will behave. However, the process of generating that response or behaviour is carried out by the movement of ions in and out of postsynaptic neuron. At this point, '3- Component Factory, Manger and Worker Model' will be introduced to model the workings of neuron and the role of neurotransmitters and ions in it. The function of the factory is to generate a specific output, by the instructions given by the Managers and the intermediary processes to generate that output is carried out by the workers. For chemical synapse communication, neuron is modelled as factory, neurotransmitters as Managers and workers as ions. However, for receptor neurons which are stimulated directly by an external stimulus; external stimulus act as Manager. Furthermore, let's define ions moving inside the neuron for the purposes of depolarisation and/or hyperpolarisation by 'specialised workers'.

As an example, Na^+ ions and Cl^- ions moving inside the neuron for depolarisation or hyperpolarisation are specialised workers. In principle, ‘specialised workers’ are the subset of ‘workers’. Ions moving out of the neuron are defined as workers, but in later time if they move in any neuron, they act as specialised workers. In this article, focus will mostly be on the specialised workers. Diagrammatically, the components of ‘Factory, Manger and Worker Model’ are shown in Figure 3.

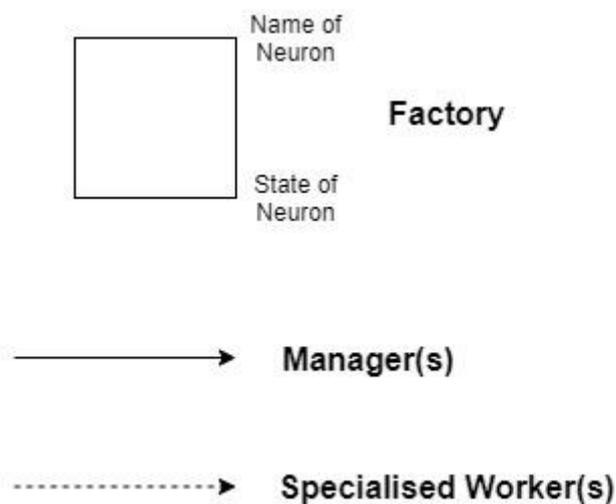


Figure 3. 3-Component Factory, Manager and Worker Model. Factory represents receptor neuron, with name and state of a neuron. For example, the receptor neuron $R_{2,0}$, 2 is the name of the neuron and 0 represents state, which is a rest state. Managers represent the neurotransmitters for chemical synapse communications and ions for electrical synapse communications. For receptor neurons directly stimulated by external stimulus; external stimulus represents the Manager. Specialised Workers are the ions moving into the neuron for depolarisation and/or hyperpolarisation.

2.2. Electrical Synapse Communication

In electrical synapses, the gap junction between two neurons allows the direct movement of ions between pre and postsynaptic neuron, without the release of neurotransmitters. This direct movement of ions into postsynaptic neuron causing depolarisation or hyperpolarisation and as a result state of postsynaptic neuron changes. To serve this purpose, from the notions of the

proposed 3-Component model, in electrical synapse communication, the ions moving inside the postsynaptic neuron through the gap junction, act both as Managers and specialised workers. These ions give instructions for output to postsynaptic neuron, as well as carry out the processes to generate that output.

2.3. Interneuron and Effector Neuron

The discussion so far has been focused on the receptor neurons. In this section, the other two types of neurons (interneuron and effector neurons) are defined. Consider arbitrary interneuron and effector neuron be defined as I_{1x} and E_{1x} , respectively; where $x = 0, 1, 2, \dots, y$ representing the state of interneuron I_1 and effector neuron E_1 . Furthermore, y represents the total number of possible states I_1 and E_1 can be in. Rest state for both these types of neurons is also given by $x = 0$ and $x > 0$ are the activated states. The workings of these neurons individually are similar to those of a single neuron discussed previously. However, unlike receptor neuron these type of neurons do not get stimulated by external stimulus, in normal circumstances. Stimulation of these neurons is caused by other neurons, either chemically or electrically.

2.4. 4-Neuronal System

Consider a 4-neuronal System ($R_{1_0} \rightarrow R_{2_0} \rightarrow I_{1_0} \rightarrow E_{1_0}$), where R_{1_0} is stimulated by a stimulus s . Chemical synaptic communication between these neurons, assuming all 4 neurons undergo AP is shown in Figure 4. The output response of stimulus s generated by this 4 neuronal system is given by the released neurotransmitters from effector neuron E_1 . These neurotransmitters can further bind to the receptors in the effector cells, which can be of any type, depending on the stimulus s . Now consider this same 4-neuronal system ($R_{1_0} \rightarrow R_{2_0} \rightarrow$

$I_{1_0} \rightarrow E_{1_0}$), where R_{1_0} is stimulated by a new arbitrary stimulus s_1 . This stimulus cause R_{1_0} and R_{2_0} to undergo AP, however, I_{1_0} depolarise below the AP threshold and remains in the subthreshold potential. Diagrammatically, this case is shown in Figure 5, using the proposed 3-component model. In this case, no output response for stimulus s_1 is generated. However, s_1 puts the interneuron I_1 to a unique excited state at resting membrane potential. As per the discussions in the previous section, the specialised workers (ions) which caused depolarisation of I_1 remains inside I_1 . As an example, if Na^+ ions were the specialised workers responsible for depolarisation, they will remain in I_1 . Some workers move out of I_1 to bring I_1 back to the resting membrane potential, however it will be in excited state because of the high concentration of specialised workers inside I_1 . Now consider this 4-neuronal system ($R_{1_0} \rightarrow R_{2_0} \rightarrow I_{1_0} \rightarrow E_{1_0}$), where R_{1_0} is stimulated by a new arbitrary stimulus s_2 . In this case, assume R_1 and R_2 communicated using electrical synapse and other neurons using chemical synapses and undergo AP. This combination of electrical and chemical synapse communication of 4-neuronal system is shown Figure 6. The response time for output in this case will be faster than the response time generated by the system with only chemical synaptic communication. Lastly, consider this 4-neuronal system ($R_{1_0} \rightarrow R_{2_0} \rightarrow I_{1_0} \rightarrow E_{1_0}$), where R_{1_0} is stimulated by a new arbitrary stimulus s_3 . In this case, assume R_1 and R_2 communicated using electrical synapse other neurons chemically. Furthermore, R_2 and I_1 undergo AP and managers from I_1 hyperpolarise E_1 with the workers moving out of E_1 . This combination of communication is shown in Figure 7. No response output for stimulus s_3 is generated and the only consequence of s_3 is the transition of E_1 to an activated state.

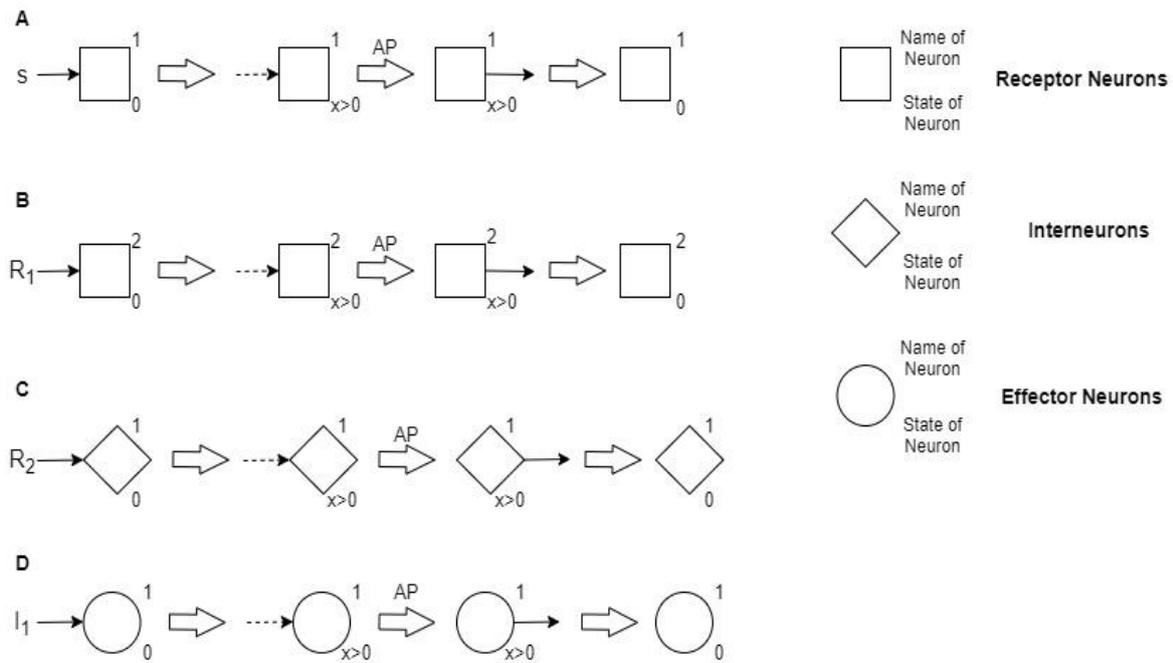


Figure 4: Chemical Synaptic Communication between 4-neuronal system ($R_{1_0} \rightarrow R_{2_0} \rightarrow I_{1_0} \rightarrow E_{1_0}$) undergoing AP.

- A) R_1 in rest state is stimulated by stimulus s , which acts as a manager. Specialised workers start moving in R_1 exciting and depolarisation R_1 to cause AP. Managers (neurotransmitters) are released into synaptic cleft of postsynaptic Neuron R_2 . R_1 returns to the rest state.
- B) Managers from R_1 binds to the receptors of R_2 which is in rest state. Specialised workers moving into R_2 causing depolarisation and AP as a result. Managers are released and R_2 returns to its rest state.
- C) Managers from R_2 binds to receptors of interneuron I_1 , resulting in the depolarisation beyond threshold, which in turns release managers to give instructions to the effector neuron E_1 . I_1 returns to its rest state.
- D) Managers from I_1 instruct depolarising workers to move in E_1 causing state change and depolarisation which leads to AP. Neurotransmitters are released. These neurotransmitters is the output response of the stimulus s for this 4 neuronal system. E_1 then returns to its rest state.

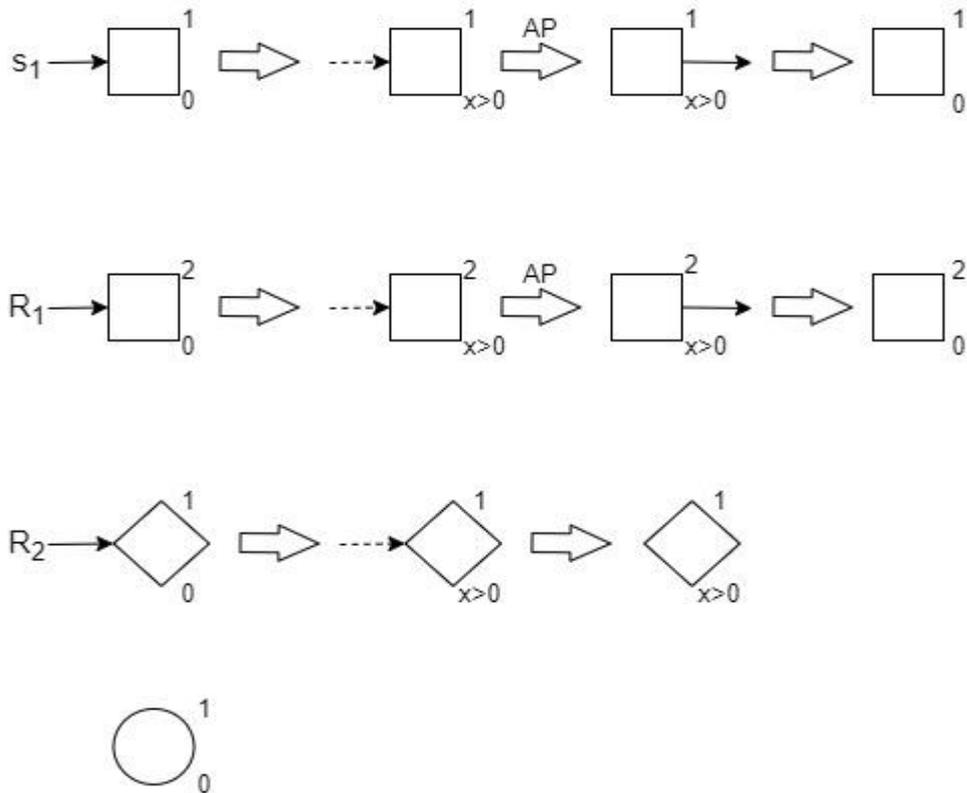


Figure 5. Chemical synaptic communication between 4-neuronal system ($R_{1_0} \rightarrow R_{2_0} \rightarrow I_{1_0} \rightarrow E_{1_0}$) where R_{1_0} is stimulated by stimulus s_1 . As a result both R_1 and R_2 undergo AP releasing managers, before returning back to their rest states. Managers from R_2 cause the specialised workers to move in I_1 resulting in depolarisation. However, depolarisation in this case is below the AP threshold potential. No neurotransmitters are released by I_1 and I_1 transitions to a unique excited state. E_1 does not receive managers from I_1 and remains in the rest state. As a result, no output response by this 4 neuronal system is generated for stimulus s_1 , but s_1 causes the interneuron I_1 to transition to a unique excited state.

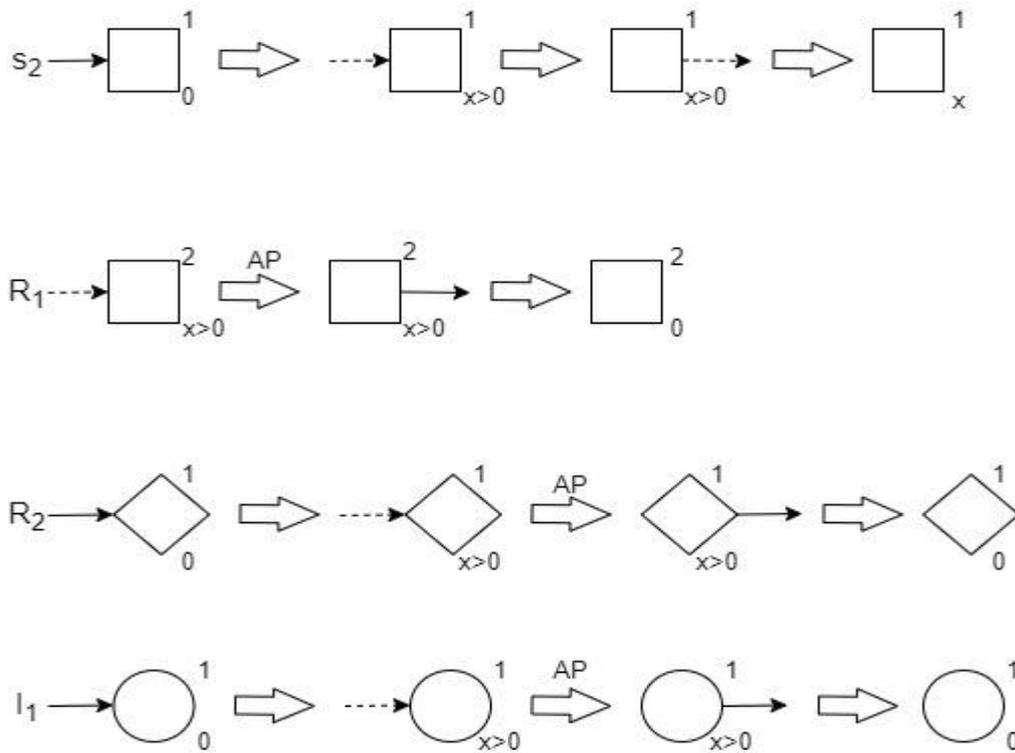


Figure 6. Combination of electrical and chemical synapse communication between a 4-neuronal system ($R_{1_0} \rightarrow R_{2_0} \rightarrow I_{1_0} \rightarrow E_{1_0}$) where R_{1_0} is stimulated by stimulus s_2 . R_1 and R_2 communicate electrically, where specialised workers move from R_1 to R_2 and act as both managers and specialised workers for R_2 . The state of R_1 after the movement of specialised workers out of it can be either rest state or activated state. If all the specialised workers which entered R_1 moves to R_2 and movement of workers is 0, R_1 returns to the rest state. However, if only a number of specialised workers from the total moves into R_2 or some workers inside R_1 moves as specialised workers into R_2 , R_1 will be in activated state. R_2 , I_1 and E_1 all undergo AP and to produce an outcome response to stimulus s_2 .

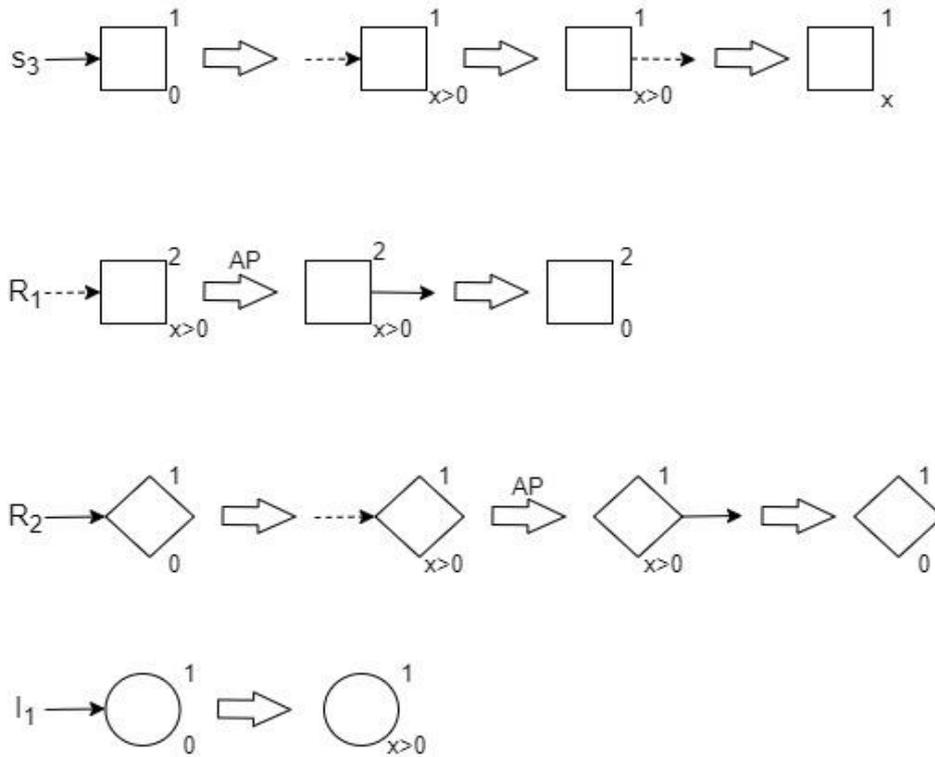


Figure 7. Combination of Electrical and chemical synapse communication between 4-neuronal system ($R_{1_0} \rightarrow R_{2_0} \rightarrow I_{1_0} \rightarrow E_{1_0}$) where R_{1_0} is stimulated by stimulus s_3 . R_1 and R_2 communicate electrically. R_2 and I_1 communicate chemically and both undergo AP. Managers from I_1 hyperpolarise E_1 and no specialised workers move into E_1 , but hyperpolarisation is achieved by the movement of workers out of E_1 . As a result, E_1 transitions to a unique excited state and no response output for stimulus s_3 is generated.

3. Neuronal Activation by Stimuli

The discussion so far has been on the postsynaptic neurons receiving stimuli for activation from a single presynaptic neuron. It is necessary that one discusses scenarios, where postsynaptic neurons receive stimuli from more than one presynaptic neuron at the same time. Consider an interneuron I_{1x} in the rest state and let the number of stimuli I_{1x} receives is given by $[S]_{I_1}$. The number of stimuli $[S]_{I_1}$ is equal to the number of presynaptic neurons for I_{1x} . Consider a case, where I_{1x} receives stimuli from two neurons R_{1x} and R_{2x} by chemical synaptic communication. For this case, $[S]_{I_1} = 2$. Furthermore, let the output generated by R_{1x} and R_{2x} which stimulates I_{1x} is given by $[O_1]_{R_1}$ and $[O_1]_{R_2}$, respectively. The focus will now be diverted to the contribution of each stimuli $[O_1]_{R_1}$ and $[O_1]_{R_2}$ in the activation of I_{1x} . To establish the necessary concepts, first the contribution of a single stimulus on I_{1x} will be discussed. To serve this purpose, consider the stimulus by R_{1x} i.e. $[O_1]_{R_1}$, which stimulates I_{1x} . Since, the neurons I_{1x} and R_{1x} communicate chemically, $[O_1]_{R_1}$ is in the form of neurotransmitters. Let the total number of neurotransmitters released by R_{1x} is defined by $[\varepsilon]_{R_1}$ and is given by equation 1.

$$[\varepsilon]_{R_1} = \sum_{\gamma=1}^{\tau} [\varepsilon_{\gamma}]_{R_1} \quad (1)$$

$[\varepsilon_{\gamma}]_{R_1}$ represents the total number of neurotransmitters of type γ , where $\gamma = 1, 2, 3, \dots, \tau$, representing individual type of neurotransmitters and τ represents the total types of neurotransmitters released by R_1 . Furthermore, $[\varepsilon_{\gamma}]_{R_1}$ is a set given by equation 2, where $\mathbf{g} = 1, 2, 3, \dots, \varepsilon_{\gamma}$. The element of set $[\varepsilon_{\gamma}]_{R_1}$ i.e. $[\varepsilon_{\gamma \mathbf{g}}]_{R_1}$ denotes the individual neurotransmitter of type γ , and \mathbf{g} labels each neurotransmitter.

$$[\varepsilon_{\gamma}]_{R_1} = \{[\varepsilon_{\gamma \mathbf{g}}]_{R_1}\} \quad (2)$$

The output $[O_1]_{R_1}$ of neuron R_{1_x} is given by equation 1. i.e. $[O_1]_{R_1} = \sum_{\gamma=1}^{\tau} [\epsilon_{\gamma}]_{R_1}$. These released neurotransmitters by R_{1_x} are ready to bind to the receptor sites on the plasma membrane of postsynaptic neuron I_{1_x} to start the activation process of I_{1_x} . Each individual released neurotransmitter by R_{1_x} has a certain affinity or likelihood to bind with receptor sites of I_{1_x} ; some having high affinity of binding while others have low affinity of binding. Based on the affinities, not all neurotransmitters will bind to the receptor sites of I_{1_x} . It is imperative that one discusses these affinities of binding and factors which affects these affinities.

3.1. Affinity of Neurotransmitter's binding

Let the affinity of binding of neurotransmitter g of type γ released by neuron R_{1_x} is defined by $[\mathcal{A}_{\gamma g}]_{R_1}$. The value of $[\mathcal{A}_{\gamma g}]_{R_1}$ varies between 0 and 1 i.e. $0 \leq [\mathcal{A}_{\gamma g}]_{R_1} \leq 1$. If $[\mathcal{A}_{\gamma g}]_{R_1} = 0$, the likelihood of neurotransmitter γg binding to the postsynaptic neuron I_{1_x} is 0, so γg won't bind to I_{1_x} . On the contrary, if $[\mathcal{A}_{\gamma g}]_{R_1} = 1$, γg will certainly bind to the receptor site of I_{1_x} .

The affinity of binding of neurotransmitters of type γ is the average value of the affinities of individual neurotransmitters of type γ given by equation 3.

$$[\mathcal{A}_{\gamma}]_{R_1} = \frac{\sum_{g=1}^{\epsilon_{\gamma}} [\mathcal{A}_{\gamma g}]_{R_1}}{\epsilon_{\gamma}} \quad (3)$$

Likewise, the affinity of binding of neurotransmitters, which are released by the presynaptic neuron R_{1_x} is the average of the affinities of neurotransmitters of type γ given by equation 4, where τ is the total types of neurotransmitters released.

$$[\mathcal{A}]_{R_1} = \frac{\sum_{\gamma=1}^{\tau} \left(\frac{\sum_{g=1}^{\epsilon_{\gamma}} [\mathcal{A}_{\gamma g}]_{R_1}}{\epsilon_{\gamma}} \right)}{\tau} \quad (4)$$

The closer the value of $[\mathcal{A}]_{R_1}$ to 1, the likelihood of more neurotransmitters binding to the postsynaptic neurons becomes high. It should be noted the concepts of affinities of neurotransmitter binding is applicable to all three types of neurons communicating chemically.

3.2. Factors affecting Affinities of Binding

This section discusses five factors that affect the affinities of neurotransmitters' binding to the postsynaptic neuron. Like previous section, R_{1_x} will be used as presynaptic neuron and I_{1_x} as a postsynaptic neuron. Furthermore, it will be assumed that the trajectories of neurotransmitters from presynaptic neuron ends on the location of binding sites on postsynaptic neuron. In other words, based on trajectories alone, the binding affinities of neurotransmitters is 1.

3.2.1. Number Density of Binding Sites

The first factor that affects the neurotransmitters' affinities of binding is the number density of binding sites on the plasma membrane of the postsynaptic neuron I_{1_x} . Consider that the total number of binding sites on the plasma membrane of I_{1_x} is defined by $[\mathcal{N}]_{I_1}$. Furthermore, assume that the surface of plasma membrane is given by an arbitrary 2-dimensional geometry with total surface area $[\mathcal{A}r]_{I_1}$. The number density of binding sites for I_{1_x} , defined by $[\rho]_{I_1}$ is given by equation 5. Number density of binding sites is the total number of binding sites over the total surface area of the plasma membrane. Likewise, the number density of binding sites of type γ is the total number of binding sites for type γ neurotransmitters over total surface area of plasma membrane, given by equation 6.

$$[\rho]_{I_1} = \frac{[\mathcal{N}]_{I_1}}{[\mathcal{A}r]_{I_1}} \quad (5)$$

$$[\rho_\gamma]_{I_1} = \frac{[\mathcal{N}_\gamma]_{I_1}}{[\mathcal{A}r]_{I_1}} \quad (6)$$

It should be noted that more than one type of neurotransmitters can share the same binding site.

To serve this purpose, the total number of binding sites $[\mathcal{N}]_{I_1}$ is given by equation 7, where

$[\mathcal{N}_{shared}]_{I_1}$ is the number of binding sites shared by different types of neurotransmitters.

$$[\mathcal{N}]_{I_1} = (\sum_{\gamma=1}^{\tau} [\mathcal{N}_{\gamma}]_{I_1}) - [\mathcal{N}_{shared}]_{I_1} \quad (7)$$

If $[\mathcal{N}_{shared}]_{I_1} = 0$, the number density $[\rho]_{I_1}$ is equal to the sum of the number densities of each

type of neurotransmitters, given by equation 8. If $[\mathcal{N}_{shared}]_{I_1} > 0$, $[\rho]_{I_1}$ is less than the sum

of number densities of each type of neurotransmitters, given by equation 9.

$$[\rho]_{I_1} = \sum_{\gamma=1}^{\tau} [\rho_{\gamma}]_{I_1} \quad (8)$$

$$[\rho]_{I_1} < \sum_{\gamma=1}^{\tau} [\rho_{\gamma}]_{I_1} \quad (9)$$

3.2.1.1. Availability of Binding sites to stimulus $[O_1]_{R_1}$

The neurotransmitters from R_1 has access to only a certain surface area of the plasma membrane $[\mathcal{A}r]_{I_1}$ of postsynaptic neuron I_1 , depending on the location and size of synapse

between R_1 and I_1 . The number of binding sites available to stimulus $[O_1]_{R_1}$ is located on the surface area of plasma membrane accessible to the stimulus $[O_1]_{R_1}$. Let this accessible area of

I_1 by stimulus $[O_1]_{R_1}$ is defined by a quantity named surface area coverage $[\delta_{R_1}]_{I_1}$. The number

density of binding sites for neurotransmitters of type γ for stimulus $[O_1]_{R_1}$ i.e. $[\rho_{\gamma R_1}]_{I_1}$ is given

by equation 10, where $[\mathcal{N}_{\gamma \delta_{R_1}}]_{I_1}$ are the number of binding sites of type γ on the surface area

coverage $[\delta_{R_1}]_{I_1}$

$$[\rho_{\gamma R_1}]_{I_1} = \frac{[\mathcal{N}_{\gamma \delta_{R_1}}]_{I_1}}{[\delta_{R_1}]_{I_1}} \quad 10$$

As the value of $[\rho_{\gamma R_1}]_{I_1}$ increases, the affinity $[\mathcal{A}_{\gamma g}]_{R_1}$ of an individual neurotransmitter g of type γ binding to postsynaptic neuron increases. Let the relationship between $[\mathcal{A}_{\gamma g}]_{R_1}$ and $[\rho_{\gamma R_1}]_{I_1}$ be given by equation 11, where $K_{\gamma g}$ is the constant of proportionality. Based on equation 11, $[\mathcal{A}_{\gamma g}]_{R_1}$ becomes asymptotic to 1, as values of $[\rho_{\gamma R_1}]_{I_1}$ becomes large, justifying equation 11 to be a reasonable relationship between $[\mathcal{A}_{\gamma g}]_{R_1}$ and $[\rho_{\gamma R_1}]_{I_1}$.

$$[\mathcal{A}_{\gamma g}]_{R_1} = \frac{K_{\gamma g} [\rho_{\gamma R_1}]_{I_1}}{K_{\gamma g} [\rho_{\gamma R_1}]_{I_1} + 1} \quad 11$$

Using equation 3 and 11, the affinity $[\mathcal{A}_{\gamma}]_{R_1}$ of type γ neurotransmitters binding to sites of I_1 based on number density is given by equation 12. Likewise, using equations 4 and 12, the affinity $[\mathcal{A}]_{R_1}$ of stimulus $[O_1]_{R_1}$ binding to the postsynaptic neuron I_1 in terms of number density is given by equation 13.

$$[\mathcal{A}_{\gamma}]_{R_1} = \frac{\sum_{g=1}^{\varepsilon_{\gamma}} \left(\frac{K_{\gamma g} [\rho_{\gamma R_1}]_{I_1}}{K_{\gamma g} [\rho_{\gamma R_1}]_{I_1} + 1} \right)}{\varepsilon_{\gamma}} \quad 12$$

$$[\mathcal{A}]_{R_1} = \frac{\sum_{\gamma=1}^{\tau} \left(\frac{K_{\gamma g} [\rho_{\gamma R_1}]_{I_1}}{K_{\gamma g} [\rho_{\gamma R_1}]_{I_1} + 1} \right)}{\tau} \quad 13$$

3.2.1.2. Example Cases of Number Density of Binding Sites

Consider the surface of plasma membrane of postsynaptic neuron I_1 in 2-dimensional Cartesian coordinates (x, y) is given by $y = f(x)$ where $f(x) = 10$ from $0 < x < 10$. The total surface area $[\mathcal{A}r]_{I_1}$ of plasma membrane is the integral of surface given by equation 14, where $a = 0$ and $b = 10$.

$$[\mathcal{A}r]_{I_1} = \int_a^b f(x)dx \quad 14$$

Based on equation 14, the surface of plasma membrane has a square geometry with surface area $[\mathcal{A}r]_{I_1} = 100$, shown in figure 8. Furthermore, let the surface area coverage $[\delta_{R_1}]_{I_1}$ by the stimulus $[O_1]_{R_1}$ of presynaptic neuron R_1 on the surface of plasma membrane of postsynaptic neuron is given by equation 15, where $f(x) = 2$ in the interval $2 < x < 5$. Based on equation 15, the surface area coverage of stimulus $[O_1]_{R_1}$ is equal to 6 showing rectangular geometry shown in figure 8.

$$[\delta_{R_1}]_{I_1} = \int_{a=2}^{b=5} f(x)dx \quad 15$$

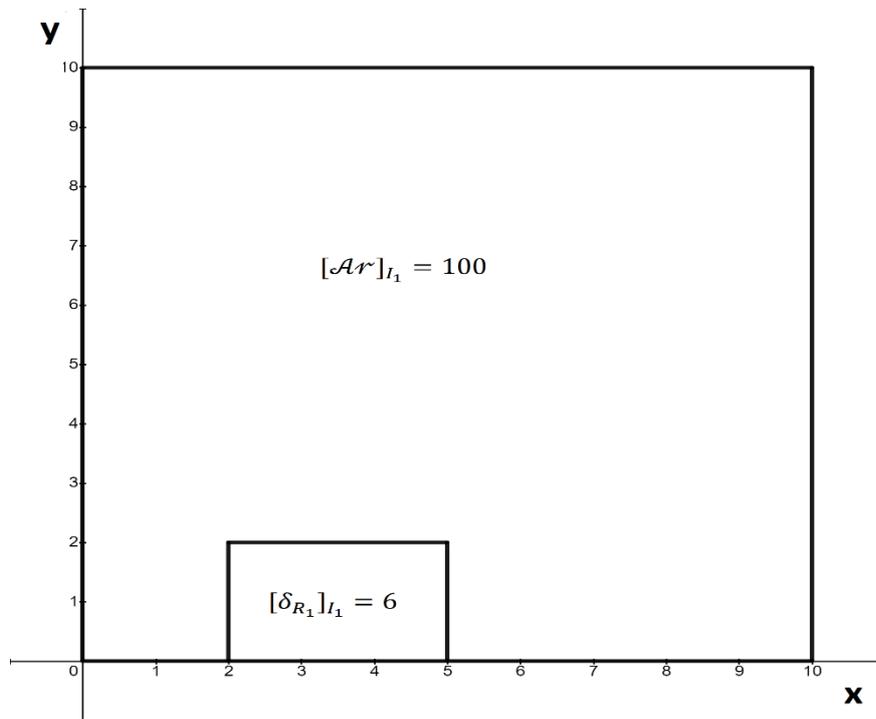


Figure 8: 2-Dimension Cartesian (x, y) Representation of Surface Plasma membrane of postsynaptic neuron I_1 and surface area coverage by stimulus of presynaptic neuron R_1 on the surface membrane of I_1 .

Example Case 1

In case 1, assume that total number of binding sites on the surface area $[\mathcal{A}r]_{I_1}$ is $[\mathcal{N}]_{I_1} = 10000000$, making $[\rho]_{I_1} = \frac{10000000}{100} = 100000 \text{ sites/unit}^2$. Furthermore, assume that number of binding sites on the surface area coverage $[\delta_{R_1}]_{I_1}$ by stimulus $[O_1]_{R_1}$ is $[\mathcal{N}_{\delta_{R_1}}]_{I_1} = 10000$, which corresponds to $[\rho_{R_1}]_{I_1} = \frac{10000}{6} = 1666.66 \text{ sites/unit}^2$. Let the stimulus $[O_1]_{R_1}$ of presynaptic neuron R_1 comprises one only one type of neurotransmitters i.e. $\gamma = 1$ with the total number of released neurotransmitters equal to $\epsilon_1 = 100$. Furthermore, let the number of binding sites on the surface area coverage of type $\gamma = 1$ neurotransmitters be $[\mathcal{N}_{1\delta_{R_1}}]_{I_1} = 240$, which corresponds to $[\rho_{1R_1}]_{I_1} = \frac{240}{6} = 40 \text{ sites/unit}^2$. Now, the affinity of binding for $g = 1$ neurotransmitter of type $\gamma = 1$ will be calculated using equation 11. For this case, assume that the constant of proportionality $K_{\gamma g}$ for all neurotransmitters of type $\gamma = 1$ is same and equal to 0.01 i.e. $K_{1g} = 0.01$. The affinity of binding of $g = 1$ neurotransmitter of type $\gamma = 1$ is $[\mathcal{A}_{11}]_{R_1} = \frac{0.01(40)}{[0.01(40)+1]} = 0.2857$. Since for this example case $K_{1g} = 0.01$ is same for all neurotransmitters and stimulus $[O_1]_{R_1}$ comprises of only type $\gamma = 1$ neurotransmitter, the affinity of type $\gamma = 1$ neurotransmitters is $[\mathcal{A}_1]_{R_1} = 0.2857$ (equation 12) and affinity of stimulus $[O_1]_{R_1}$ is $[\mathcal{A}]_{R_1} = 0.2857$ (equation 13).

Example Case 2

In this case, assume that the stimulus comprises $[O_1]_{R_1}$ of two types of neurotransmitters i.e. $\tau=2$ with $\gamma = 1,2$. Let the total number of released neurotransmitters be $[\epsilon]_{R_1} = 150$ (equation 1) with $[\epsilon_1]_{R_1} = 100$ and $[\epsilon_2]_{R_1} = 50$. Assume that both types of neurotransmitters do not share any binding sites on the surface area coverage. Furthermore, let the number of binding sites on the surface area coverage of type $\gamma = 2$ neurotransmitters be $[\mathcal{N}_{2\delta_{R_1}}]_{I_1} = 180$, which

corresponds to $[\rho_{2R_1}]_{I_1} = \frac{180}{6} = 30 \text{ sites/unit}^2$ and $[\rho_{1R_1}]_{I_1} = 40 \text{ sites/unit}^2$ as calculated in example case 1. The constant of proportionality $K_{\gamma g}$ for type $\gamma = 2$ neurotransmitters $K_{2g} = 0.02$ and is same for all neurotransmitters of type $\gamma = 2$. As in previous example case, $K_{1g} = 0.01$ for type $\gamma = 1$ neurotransmitters. Based on these values, the affinity of binding of $g = 1$ neurotransmitter of type $\gamma = 1,2$ is $[\mathcal{A}_{11}]_{R_1} = 0.2857$ and $[\mathcal{A}_{21}]_{R_1} = 0.375$ (equation 11). Since $K_{1g} = 0.01$ is same for all neurotransmitters of type $\gamma = 1$ and $K_{2g} = 0.02$ for type $\gamma = 2$ neurotransmitters, the affinities of type $\gamma = 1,2$ neurotransmitters are $[\mathcal{A}_1]_{R_1} = 0.2857$ and $[\mathcal{A}_2]_{R_1} = 0.375$ (equation 12). Based on equation 13, the affinity of stimulus $[O_1]_{R_1}$ for this example case is $[\mathcal{A}]_{R_1} = \frac{0.2857+0.375}{2} = 0.330$. The likelihood of neurotransmitters binding to the postsynaptic neuron I_1 is more in example case 2 than in example case 1.

3.2.2. Distribution of Binding Sites

The second factor under consideration for its effect on the binding affinities of neurotransmitters is the distribution of binding sites on the plasma membrane of postsynaptic neuron. As previously, R_1 and I_1 will be considered as pre and postsynaptic neurons, respectively. Let the distribution of binding sites on the plasma membrane of I_1 be defined by $[\mu]_{I_1}$. Consider the surface of plasma membrane of I_1 by an arbitrary 2-dimension geometry in Cartesian coordinates (x, y) with surface area $[\mathcal{A}\mathcal{r}]_{I_1}$ and total number of binding sites $[\mathcal{N}]_{I_1}$ shown in figure 9A. Let the location of each binding site is defined by a position vector \vec{V}_j , originating from origin. The total number of position vectors is equal to the total number of binding sites $[\mathcal{N}]_{I_1}$, therefore $j = 1,2,3 \dots [\mathcal{N}]_{I_1}$. The numbering of position vectors is done using the magnitude $|\vec{V}_j|$ (equation 16) and angle of vector with the x-axis θ_j (equation 17). The position vector with the lowest magnitude is taken by convention to be \vec{V}_1 i.e. $j = 1$.

The position vector with highest magnitude is $\overline{V}_{[\mathcal{N}]_{I_1}}$. In case, the position vectors have equal magnitude, the vector with small θ_j precedes in numbering.

$$|\overline{V}_j| = \sqrt{x_j^2 + y_j^2} \quad 16$$

$$\tan(\theta_j) = \frac{y_j}{x_j} \quad 17$$

3.2.2.1. Distribution Matrices of Binding Sites

The locations of $[\mathcal{N}]_{I_1}$ number of binding sites on the plasma membrane represents the distribution of binding sites. Consider a matrix $[a_{1j}] \in \mathbb{R}^{1 \times [\mathcal{N}]_{I_1}}$, where elements of $[a_{1j}]$ represents the location of binding sites i.e. $a_{1j} = \overline{V}_j$ with $j = 1, 2, \dots, [\mathcal{N}]_{I_1}$. The distribution of binding sites on the plasma membrane of I_1 is given by a matrix of dimensions $1 \times [\mathcal{N}]_{I_1}$ (equation 18).

$$[\mu]_{I_1} = [a_{1j}] \in \mathbb{R}^{1 \times [\mathcal{N}]_{I_1}} = [\overline{V}_1 \overline{V}_2 \dots \overline{V}_{[\mathcal{N}]_{I_1}}] \quad 18$$

As discussed previously, the stimulus $[O_1]_{R_1}$ by neuron R_1 has surface area coverage $[\delta_{R_1}]_{I_1}$ on the plasma membrane of I_1 . The total number of binding sites on $[\delta_{R_1}]_{I_1}$ is given by $[\mathcal{N}_{\delta_{R_1}}]_{I_1}$ shown in figure 9B. Let the distribution of binding sites on $[\delta_{R_1}]_{I_1}$ be defined by $[\mu_{R_1}]_{I_1}$. Furthermore, let the position vectors of binding sites on $[\delta_{R_1}]_{I_1}$ be defined by $\overline{V}_{R_{1j}}$ where $j = 1, 2, \dots, [\mathcal{N}_{\delta_{R_1}}]_{I_1}$. The numbering of position vectors on surface area coverage is done using magnitude $|\overline{V}_{R_{1j}}|$ and angle $\theta_{R_{1j}}$, with $\overline{V}_{R_{11}}$ having the lowest magnitude. Consider a matrix $[a_{R_{1j}}] \in \mathbb{R}^{1 \times [\mathcal{N}_{\delta_{R_1}}]_{I_1}}$ with $[a_{R_{1j}}] = \overline{V}_{R_{1j}}$ and $j = 1, 2, \dots, [\mathcal{N}_{\delta_{R_1}}]_{I_1}$. The distribution of binding sites on the surface area coverage $[\delta_{R_1}]_{I_1}$, in terms of location of binding sites is given by equation 19.

$$[\mu_{R_1}]_{I_1} = [a_{R_{11j}}] \in \mathbb{R}^{1 \times [\mathcal{N}_{\delta_{R_1}}]_{I_1}} = [\overrightarrow{V_{R_{11}}} \overrightarrow{V_{R_{12}}} \dots \overrightarrow{V_{[\mathcal{N}_{\delta_{R_1}}]_{I_1}}}] \quad 19$$

Likewise, the distribution of binding sites on the surface area coverage $[\delta_{R_1}]_{I_1}$ for neurotransmitters of type γ can be derived. Let the distribution of binding sites for type γ neurotransmitters on $[\delta_{R_1}]_{I_1}$ is defined by $[\mu_{\gamma R_1}]_{I_1}$. The total number of binding sites for type γ neurotransmitters is given by $[\mathcal{N}_{\gamma \delta_{R_1}}]_{I_1}$. Let the position vectors of type γ binding sites on $[\delta_{R_1}]_{I_1}$ be defined by $\overrightarrow{V_{\gamma R_{1j}}}$ where $j = 1, 2, \dots, [\mathcal{N}_{\gamma \delta_{R_1}}]_{I_1}$ with magnitudes $|\overrightarrow{V_{\gamma R_{1j}}}|$ and angle $\theta_{\gamma R_{1j}}$. The representation of type $\gamma = 1, 2$ neurotransmitters in terms of position vectors and angles is shown in Figure 9C. Consider a matrix $[a_{\gamma R_{11j}}] \in \mathbb{R}^{1 \times [\mathcal{N}_{\gamma \delta_{R_1}}]_{I_1}}$ with $[a_{\gamma R_{11j}}] = \overrightarrow{V_{\gamma R_{1j}}}$ and $j = 1, 2, \dots, [\mathcal{N}_{\gamma \delta_{R_1}}]_{I_1}$. The distribution of binding sites $[\mu_{\delta_{R_1}}]_{I_1}$ for type γ neurotransmitters on the surface area coverage $[\delta_{R_1}]_{I_1}$ is given by equation 20.

$$[\mu_{\gamma R_1}]_{I_1} = [a_{R_{11j}}] \in \mathbb{R}^{1 \times [\mathcal{N}_{\gamma \delta_{R_1}}]_{I_1}} = [\overrightarrow{V_{\gamma R_{11}}} \overrightarrow{V_{\gamma R_{12}}} \dots \overrightarrow{V_{[\mathcal{N}_{\gamma \delta_{R_1}}]_{I_1}}}] \quad 20$$

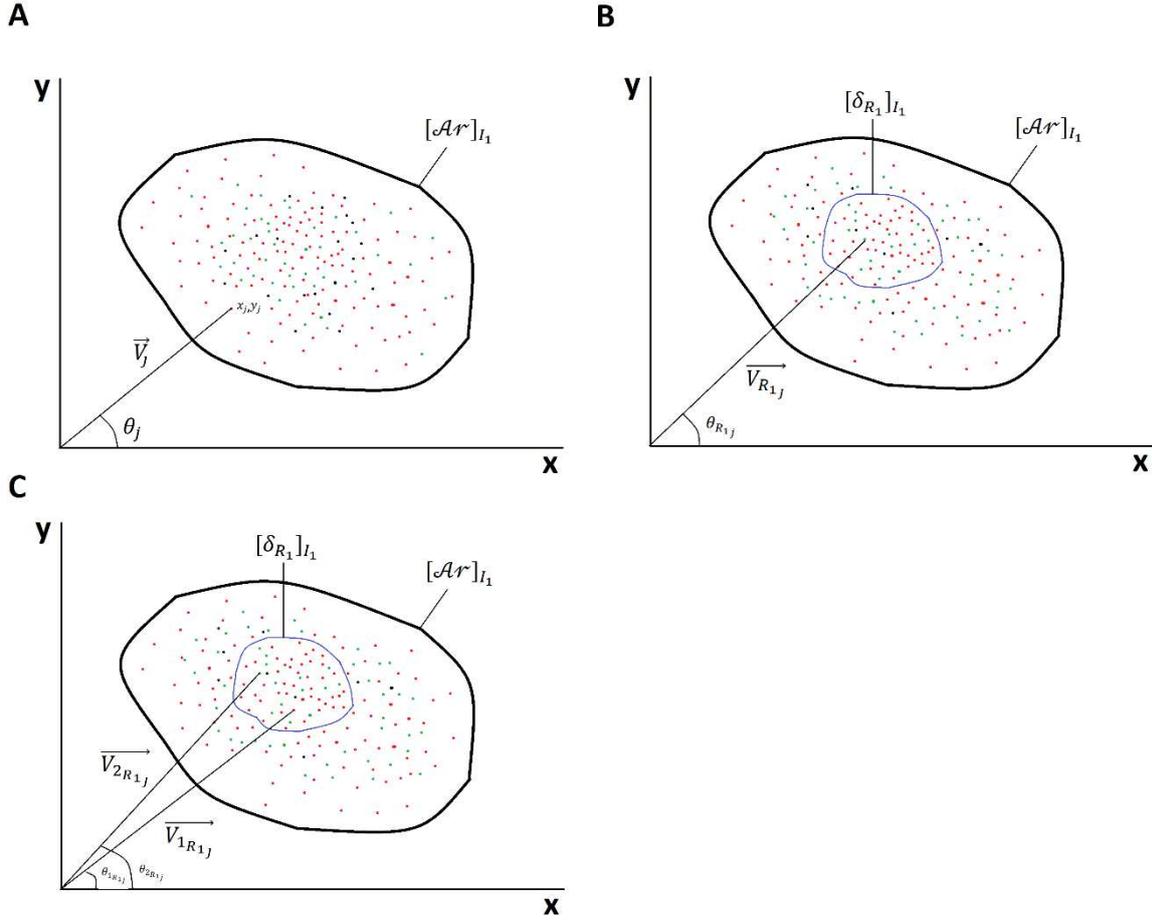


Figure 9: 2-Dimensional Geometry of Plasma Membrane of I_1 in Cartesian coordinates.

- A) Surface Area of plasma membrane $[Ar]_{I_1}$ with the number of binding sites represented as colored points. Each colored point represents a different type of neurotransmitter. The location of binding site is represented by \vec{V}_j position vector with magnitude $|\vec{V}_j|$ and angle θ_j .
- B) Surface area coverage $[\delta_{R_1}]_{I_1}$ by stimulus of R_1 on the plasma membrane of I_1 . The location of $[\mathcal{N}_{\delta_{R_1}}]_{I_1}$ number of binding sites is represented by \vec{V}_{R_1j} position vectors, where $j = 1, 2, \dots, [\mathcal{N}_{\delta_{R_1}}]_{I_1}$ with magnitudes $|\vec{V}_{R_1j}|$ and angles θ_{R_1j} .
- C) Representation of type $\gamma = 1, 2$ neurotransmitters on the surface area coverage $[\delta_{R_1}]_{I_1}$. Red points represent binding sites for neurotransmitters of type $\gamma = 1$ with position vectors \vec{V}_{1R_1j} and angles θ_{1R_1j} where $j = 1, 2, \dots, [\mathcal{N}_{1\delta_{R_1}}]_{I_1}$. Similarly, green points represent binding sites for type $\gamma = 2$ neurotransmitters with position vectors \vec{V}_{2R_1j} and angles θ_{2R_1j} where $j = 1, 2, \dots, [\mathcal{N}_{2\delta_{R_1}}]_{I_1}$.

3.2.2.2. Effect of Distribution on the Binding Affinity

The affinity of binding $[\mathcal{A}_{\gamma g}]_{R_1}$ for a g neurotransmitter of type γ released by presynaptic neuron R_1 depends on density $[\rho_{\gamma R_1}]_{I_1}$ and constant of proportionality $K_{\gamma g}$ (equation 11). Keeping the density $[\rho_{\gamma R_1}]_{I_1}$ of type γ binding sites fixed, the distribution $[\mu_{\gamma R_1}]_{I_1}$ affects the value of constant of proportionality $K_{\gamma g}$. Consider an arbitrary distribution $[\mu_{\gamma R_1}]_{I_1}$ of type γ binding sites given by the matrix of equation 20. Based on this distribution $[\mu_{\gamma R_1}]_{I_1}$, each neurotransmitter of type γ has a value of constant of proportionality $K_{\gamma g}$ where $g = 1, 2, \dots [\epsilon_{\gamma}]_{R_1}$. Consider a matrix $[b_{\gamma 1g}]_{R_1} \in \mathbb{R}^{1 \times [\epsilon_{\gamma}]_{R_1}}$ where elements of $[b_{\gamma 1g}]_{R_1}$ represent the values of $K_{\gamma g}$ of individual neurotransmitters of type γ i.e. $[b_{\gamma 1g}]_{R_1} = K_{\gamma g}$ with $g = 1, 2, \dots [\epsilon_{\gamma}]_{R_1}$. The values of $K_{\gamma g}$ for type γ is given by the matrix of equation 21.

$$[b_{\gamma 1g}]_{R_1} \in \mathbb{R}^{1 \times [\epsilon_{\gamma}]_{R_1}} = [K_{\gamma 1} \ K_{\gamma 2} \ \dots \ K_{\gamma [\epsilon_{\gamma}]_{R_1}}] \quad 21$$

The affinity of binding $[\mathcal{A}_{\gamma}]_{R_1}$ of neurotransmitter of type γ is the average of affinities of individual neurotransmitters calculated using $K_{\gamma g}$ (equation 12). A given arbitrary distribution of binding sites for type γ has a unique constant of proportionality matrix $[b_{\gamma 1g}]_{R_1}$, which affects the affinity of binding $[\mathcal{A}_{\gamma}]_{R_1}$. For example, consider two distinct distributions of binding sites (A and B) with of type γ and their corresponding constant of proportionality matrices given by equation 22 and 23. Furthermore, assume densities of binding sites for both distributions A and B is equal. Both distribution will have different affinities of binding and the distribution with high sum of constant of proportionalities i.e. $\sum_{g=1}^{[\epsilon_{\gamma}]_{R_1}} K_{\gamma g}$ will have high affinity of binding $[\mathcal{A}_{\gamma}]_{R_1}$. Likewise, for every neurotransmitter type with a given distribution of binding sites, there exist the constant of proportionality matrix. The affinity $[\mathcal{A}]_{R_1}$ of stimulus $[O_1]_{R_1}$ binding to the

postsynaptic neuron I_1 in terms of distributions of binding sites can be calculated using equation 13.

$$[\overrightarrow{V_{\gamma R_{11}}} \overrightarrow{V_{\gamma R_{12}}} \cdots \overrightarrow{V_{[\mathcal{N}_{\gamma \delta_{R_1}}]_{I_1}}}]_A \rightarrow [K_{\gamma_1} K_{\gamma_2} \cdots K_{\gamma_{[\varepsilon_{\gamma}]_{R_1}}}]_A \quad 23$$

$$[\overrightarrow{V_{\gamma R_{11}}} \overrightarrow{V_{\gamma R_{12}}} \cdots \overrightarrow{V_{[\mathcal{N}_{\gamma \delta_{R_1}}]_{I_1}}}]_B \rightarrow [K_{\gamma_1} K_{\gamma_2} \cdots K_{\gamma_{[\varepsilon_{\gamma}]_{R_1}}}]_B \quad 24$$

3.2.2.3. Example Case

Consider the surface area coverage $[\delta_{R_1}]_{I_1}$ by stimulus $[O_1]_{R_1}$ of R_1 on the plasma membrane of I_1 given by the surface $y = f(x)$ where $f(x) = 8$ from $0 < x < 10$. This corresponds to a square geometry (figure 10) with $[\delta_{R_1}]_{I_1} = 64 \text{ units}^2$. The stimulus $[O_1]_{R_1}$ comprises of two types of neurotransmitters i.e. $\tau=2$ with $\gamma = 1,2$. The total number of released neurotransmitters be $[\varepsilon]_{R_1} = 10$ (equation 1) with $[\varepsilon_1]_{R_1} = 6$ and $[\varepsilon_2]_{R_1} = 4$. The total number of binding sites for type $\gamma = 1,2$ on $[\delta_{R_1}]_{I_1}$ are $[\mathcal{N}_{1\delta_{R_1}}]_{I_1} = 10$ and $[\mathcal{N}_{2\delta_{R_1}}]_{I_1} = 8$. The corresponding densities of binding sites are $[\rho_{1R_1}]_{I_1} = \frac{10}{64} = 0.156 \text{ sites/unit}^2$ and $[\rho_{2R_1}]_{I_1} = \frac{8}{64} = 0.125 \text{ sites/unit}^2$. The location of binding sites are shown in figure 10. Based on the locations of binding sites, the distributions $[\mu_{1R_1}]_{I_1}$ and $[\mu_{2R_1}]_{I_1}$ of binding sites in terms of position vector matrices

$[a_{R_{11j}}] \in \mathbb{R}^{1 \times 10}$ and $[a_{R_{11j}}] \in \mathbb{R}^{1 \times 8}$ are given by equation 25 and 26, respectively.

$$[\mu_{1R_1}]_{I_1} = [(1.5,1)(2,2)(4,2)(2,4)(4,4)(5,3)(2,6)(4,5)(7,5)(6,7)] \quad 25$$

$$[\mu_{2R_1}]_{I_1} = [(1,3)(3,2)(6,2)(4.5,5)(7,1)(1,7.5)(7.5,3)(4,7.5)] \quad 26$$

Furthermore, assume the corresponding constant of proportionalities of neurotransmitters of type $\gamma = 1,2$ in the form of matrices $[b_{11g}]_{R_1} \in \mathbb{R}^{1 \times 6}$ and $[b_{21g}]_{R_1} \in \mathbb{R}^{1 \times 4}$ are given by equations 27 and 28 respectively.

$$[b_{11g}]_{R_1} \in \mathbb{R}^{1 \times 6} = [(0.1)(0.5)(5)(0.08)(10)(0.5)] \quad 27$$

$$[b_{21g}]_{R_1} \in \mathbb{R}^{1 \times 4} = [(2)(15)(0.7)(50)] \quad 28$$

Using the given data and equation 12, the affinity of binding of type $\gamma = 1,2$ is calculated to be $[\mathcal{A}_1]_{R_1} = 0.2033$ and $[\mathcal{A}_2]_{R_1} = 0.4486$. The affinity of binding for stimulus $[O_1]_{R_1}$ is calculated to be $[\mathcal{A}]_{R_1} = \frac{0.2033+0.4486}{2} = 0.32595$.

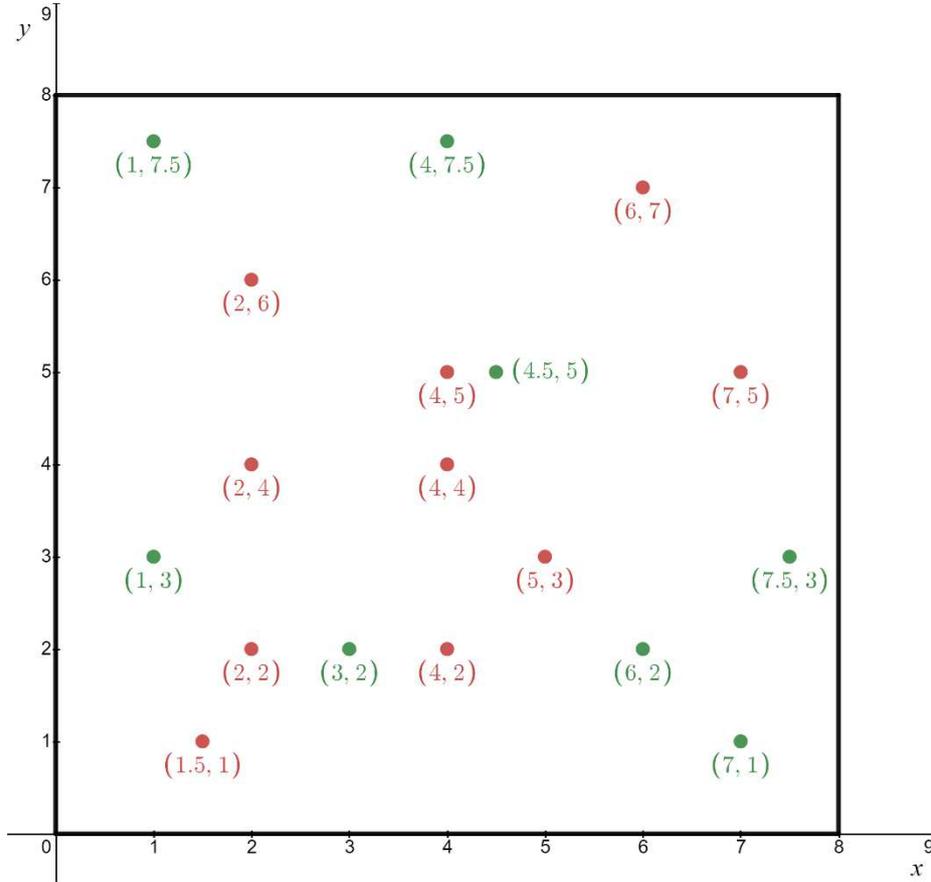


Figure 10. Representation of the locations of binding sites on the surface area coverage $[\delta_{R_1}]_{I_1}$ of plasma membrane I_1 . Surface area coverage $[\delta_{R_1}]_{I_1}$ has square geometry in 2-Dimensional Cartesian coordinates (x, y) . The types of neurotransmitter binding sites are represented using color points. Red points represent type $\gamma = 1$ binding sites with $[\mathcal{N}_{1\delta_{R_1}}]_{I_1} = 10$. Green points represent type $\gamma = 2$ binding sites with $[\mathcal{N}_{2\delta_{R_1}}]_{I_1} = 8$.

3.2.3. Number of Released Neurotransmitters

The third factor that affects affinity of binding is the number of released neurotransmitters. The stimulus $[O_1]_{R_1}$ of presynaptic neuron R_1 comprises of total number of released neurotransmitters given by $[\varepsilon]_{R_1}$ which is made of different types of neurotransmitters (equation 1). Consider the total number of neurotransmitters of type γ i.e. $[\varepsilon_\gamma]_{R_1}$. As discussed previously, these neurotransmitters bind to the binding sites of type γ on the surface area coverage of postsynaptic neuron I_1 . The total number of binding sites for type γ on the surface area coverage of postsynaptic neuron I_1 is given by $[\mathcal{N}_{\gamma\delta_{R_1}}]_{I_1}$. If $[\varepsilon_\gamma]_{R_1} > [\mathcal{N}_{\gamma\delta_{R_1}}]_{I_1}$, the neurotransmitters compete for binding sites, resulting in decrease affinity of binding $[\mathcal{A}_\gamma]_{R_1}$ for neurotransmitters of type γ . Likewise, if $[\varepsilon_\gamma]_{R_1} < [\mathcal{N}_{\gamma\delta_{R_1}}]_{I_1}$, likelihood of each neurotransmitter binding to the binding sites increases. In other words, if the ratio $\frac{[\varepsilon_\gamma]_{R_1}}{[\mathcal{N}_{\gamma\delta_{R_1}}]_{I_1}} < 1$, $[\mathcal{A}_\gamma]_{R_1}$ is less and if $\frac{[\varepsilon_\gamma]_{R_1}}{[\mathcal{N}_{\gamma\delta_{R_1}}]_{I_1}} > 1$, $[\mathcal{A}_\gamma]_{R_1}$ is more. Now the focus will be to find the relationship between the ratio $\frac{[\varepsilon_\gamma]_{R_1}}{[\mathcal{N}_{\gamma\delta_{R_1}}]_{I_1}}$ and the affinity of binding $[\mathcal{A}_{\gamma g}]_{R_1}$ of the g neurotransmitter of type γ . Based on equation 11, the ratio $\frac{[\varepsilon_\gamma]_{R_1}}{[\mathcal{N}_{\gamma\delta_{R_1}}]_{I_1}}$ governs the value of constant of proportionality $K_{\gamma g}$, since the number density of binding sites is fixed. To establish the relationship between $\frac{[\varepsilon_\gamma]_{R_1}}{[\mathcal{N}_{\gamma\delta_{R_1}}]_{I_1}}$ and $K_{\gamma g}$, two set of equations will be used, given by equations 29 and 30, $[K_{\gamma g}]_{max}$ is the maximum value of constant of proportionality. Furthermore, χ_γ is the rate parameter for neurotransmitter of type γ , which is the rate of decrease in $K_{\gamma g}$ with increase in ratio $\frac{[\varepsilon_\gamma]_{R_1}}{[\mathcal{N}_{\gamma\delta_{R_1}}]_{I_1}}$ from 1.

$$K_{\gamma g} = [K_{\gamma g}]_{max} \quad \text{if} \quad \frac{[\varepsilon_\gamma]_{R_1}}{[\mathcal{N}_{\gamma\delta_{R_1}}]_{I_1}} \leq 1 \quad 29$$

$$K_{\gamma_g} = [K_{\gamma_g}]_{max} \cdot e^{-\chi_\gamma \left[\frac{[\epsilon_\gamma]_{R_1}}{[\mathcal{N}_{\gamma\delta_{R_1}}]_{I_1}} - 1 \right]} \quad \text{if } \frac{[\epsilon_\gamma]_{R_1}}{[\mathcal{N}_{\gamma\delta_{R_1}}]_{I_1}} \geq 1 \quad 30$$

Once the value of K_{γ_g} is calculated, the affinity of binding $[\mathcal{A}_{\gamma_g}]_{R_1}$ can be calculated using equation 11. It should be noted that each neurotransmitter of type γ will have the same constant of proportionality K_{γ_g} given by equation 29 or 30, depending on the ratio $\frac{[\epsilon_\gamma]_{R_1}}{[\mathcal{N}_{\gamma\delta_{R_1}}]_{I_1}}$ and therefore $[\mathcal{A}_\gamma]_{R_1} = [\mathcal{A}_{\gamma_g}]_{R_1}$. The affinity of binding of stimulus $[O_1]_{R_1}$ can be calculated using equation 13.

3.2.4. Kinetic Energy of Neurotransmitters

The fourth factor that affects affinity of binding is the kinetic energy of neurotransmitters. Let the kinetic energy of g neurotransmitter of type γ released by presynaptic neuron R_1 is defined by $[\kappa_{\gamma g}]_{R_1}$ in appropriate units of energy. The affinity of binding $[\mathcal{A}_{\gamma g}]_{R_1}$ depends on the value of the kinetic energy $[\kappa_{\gamma g}]_{R_1}$ and not all kinetic energy values result in binding of neurotransmitters to binding site, which points to $[\mathcal{A}_{\gamma g}]_{R_1}$ values 0 or closer to 0. From equation 11, the binding affinity depends on constant of proportionality $K_{\gamma g}$ and density of binding sites $[\rho_{\gamma R_1}]_{I_1}$. The density of binding sites is fixed, so the kinetic energy $[\kappa_{\gamma g}]_{R_1}$ governs the value of constant of proportionality $K_{\gamma g}$ which in turns affect the affinity of binding $[\mathcal{A}_{\gamma g}]_{R_1}$. In other words, if constant of proportionality $K_{\gamma g}$ resulting from kinetic energy is high, $[\mathcal{A}_{\gamma g}]_{R_1}$ is high.

The next step is to establish a relationship between kinetic energy $[\kappa_{\gamma g}]_{R_1}$ of a neurotransmitter and it's constant of proportionality $K_{\gamma g}$. To establish this, a function with shape parameter ℓ_γ and scale parameter Λ_γ is used, given by equation 31. Both parameters ℓ_γ and Λ_γ are defined for values greater than 0 i.e. $\ell_\gamma > 0$ and $\Lambda_\gamma > 0$. Furthermore, each neurotransmitter type γ has its own values of shape and scale parameter. For instance, for neurotransmitter of type $\gamma = 1$, shape parameter $\ell_1 = 5$ and scale parameter $\Lambda_1 = 0.5$. It should be noted that different types of neurotransmitters can have one of both values of ℓ_γ and Λ_γ same. Once the parameters of specific type of neurotransmitter are well defined, the functions of equation 31, gives the relationship between kinetic energy $[\kappa_{\gamma g}]_{R_1}$ and constant of proportionality $K_{\gamma g}$.

$$K_{\gamma g} = \left\{ \frac{\ell_\gamma}{\Lambda_\gamma} \left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_\gamma} \right)^{\ell_\gamma} e^{-\left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_\gamma} \right)^{\ell_\gamma}} \right\} \text{ For } [\kappa_{\gamma g}]_{R_1} \geq 0 \quad 31$$

Based on equation 31, the kinetic energy $[\kappa_{\gamma g}]_{R_1}$ which corresponds to highest value of constant of proportionality $K_{\gamma g}$ is given by $\frac{dK_{\gamma g}}{d[\kappa_{\gamma g}]_{R_1}} = 0$. The derivative of equation 31 is shown in equation 32, with its derivation given in Appendix A.

$$\frac{dK_{\gamma g}}{d[\kappa_{\gamma g}]_{R_1}} = \frac{\ell_{\gamma}^2}{\Lambda_{\gamma} \cdot (\Lambda_{\gamma})^{\ell_{\gamma}}} ([\kappa_{\gamma g}]_{R_1})^{\ell_{\gamma}-1} \left(e^{-\left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_{\gamma}}\right)^{\ell_{\gamma}}} \right) \left[1 - \left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_{\gamma}}\right)^{\ell_{\gamma}} \right] \quad 32$$

After equating equation 32 with 0 and simplification, the kinetic energy $[\kappa_{\gamma g}]_{R_1}$ with highest $K_{\gamma g}$ is given by equation 33. Based on equation 31, the highest constant of proportionality $K_{\gamma g}$ will always be at kinetic energy equal $[\kappa_{\gamma g}]_{R_1}$ to scale parameter Λ_{γ} .

$$[\kappa_{\gamma g}]_{R_1} = \Lambda_{\gamma} \quad 33$$

Given by $[\kappa_{\gamma g}]_{R_1}$, the affinity of binding $[\mathcal{A}_{\gamma g}]_{R_1}$ for g neurotransmitter of type γ can be calculated using equations 31 and equations 11. The focus now will be diverted to the affinity of binding $[\mathcal{A}_{\gamma}]_{R_1}$ of type γ neurotransmitters. The total number of type γ neurotransmitters released by presynaptic neuron is given by $[\mathcal{E}_{\gamma}]_{R_1}$ with each neurotransmitter g of type γ having its own kinetic energy $[\kappa_{\gamma g}]_{R_1}$ and its own constant of proportionality $K_{\gamma g}$. If the value of $[\mathcal{E}_{\gamma}]_{R_1}$ is large, it is more convenient to incorporate statistical distributions to estimate average kinetic energy of type γ neurotransmitters, rather than having kinetic energy of individual neurotransmitter. Regardless of the method the practitioner employs, constant of proportionality is calculated using equation 31, with appropriate parameter and subsequently, the binding affinity $[\mathcal{A}_{\gamma}]_{R_1}$ is calculated using equation 12. Likewise, the binding affinity $[\mathcal{A}]_{R_1}$ of stimulus $[O_1]_{R_1}$ is calculated using equation 13.

3.2.4.1. Example Case

Consider stimulus $[O_1]_{R_1}$ comprises of two types of neurotransmitters i.e. $\tau=2$ with $\gamma = 1,2$. The total number of released neurotransmitters be $[\varepsilon]_{R_1} = 8$ (equation 1) with $[\varepsilon_1]_{R_1} = 5$ and $[\varepsilon_2]_{R_1} = 3$. Furthermore, consider the kinetic energies of released neurotransmitters in appropriate units for type $\gamma = 1,2$ are given by equations 34 and 35, respectively.

$$[\kappa_{1g}]_{R_1} \{g = 1,2,.. [\varepsilon_1]_{R_1}\} = \{0.2, 0.4, 0.5, 0.5, 0.8\} \quad 34$$

$$[\kappa_{2g}]_{R_1} \{g = 1,2,.. [\varepsilon_2]_{R_1}\} = \{0.3, 0.35, 0.45\} \quad 35$$

The number densities of binding sites on the postsynaptic neuron I_1 for type $\gamma = 1,2$ neurotransmitters are $[\rho_{1R_1}]_{I_1} = 0.15 \text{ sites/unit}^2$ and $[\rho_{2R_1}]_{I_1} = 0.23 \text{ sites/unit}^2$. In this example, the effect of distribution of binding sites on binding affinities is neglected. Furthermore, the relationship between kinetic energy $[\kappa_{\gamma g}]_{R_1}$ and constant of proportionality $K_{\gamma g}$ is given by equation 31, where $\ell_1 = 5$, $\Lambda_1 = 0.5$ for type $\gamma = 1$ neurotransmitters and $\ell_2 = 2.5$, $\Lambda_2 = 0.35$ for type $\gamma = 2$ neurotransmitters. Based on these parametric values, the relationship between kinetic energy and constant of proportionality is shown in figure 11. The calculated values of constant of proportionalities for neurotransmitters for type $\gamma = 1,2$ are given in equations 36 and 37, respectively. The binding affinities of neurotransmitters $[\mathcal{A}_{1g}]_{R_1}$ and $[\mathcal{A}_{2g}]_{R_1}$ using equation 11 are given in equations 38 and 39, respectively.

$$K_{1g} \{g = 1,2,.. [\varepsilon_1]_{R_1}\} = \{0.10136, 2.36124, 3.679, 3.679, 0.00293\} \quad 36$$

$$K_{2g} \{g = 1,2,.. [\varepsilon_2]_{R_1}\} = \{2.461, 2.628, 2.0544\} \quad 37$$

$$[\mathcal{A}_{1g}]_{R_1} \{g = 1,2,.. [\varepsilon_1]_{R_1}\} = \{0.01497, 0.26154, 0.3556, 0.3556, 0.000439\} \quad 38$$

$$[\mathcal{A}_{2g}]_{R_1} \{g = 1, 2, \dots [\epsilon_2]_{R_1}\} = \{0.361, 0.377, 0.321\}$$

39

Using equation 12, the binding affinities of type $\gamma = 1, 2$ are calculated to be $[\mathcal{A}_1]_{R_1} = 0.1976$ and $[\mathcal{A}_2]_{R_1} = 0.353$. The binding affinity of stimulus $[O_1]_{R_1}$ is calculated to be $[\mathcal{A}]_{R_1} = 0.2753$ (equation 13).

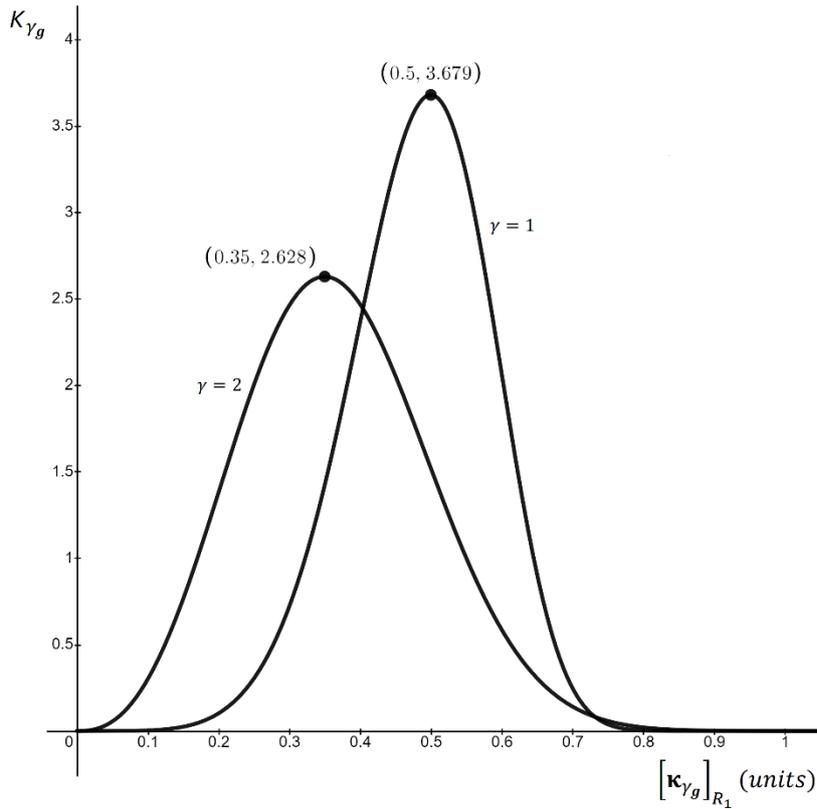


Figure 11. Relationship between kinetic energy $[\kappa_{\gamma g}]_{R_1}$ and constant of proportionality $K_{\gamma g}$ for type $\gamma = 1, 2$ neurotransmitters. The maximum constant of proportionality $K_{1g} = 3.679$ at $[\kappa_{1g}]_{R_1} = \Lambda_1 = 0.5$ for type $\gamma = 1$ neurotransmitter. The maximum constant of proportionality $K_{2g} = 2.628$ at $[\kappa_{2g}]_{R_1} = \Lambda_2 = 0.35$ for type $\gamma = 2$ neurotransmitter.

3.2.5. Orientation of Neurotransmitters

The fifth factor that's affects affinity of neurotransmitter binding is the orientation of neurotransmitter. Consider an arbitrary neurotransmitter \mathbf{g} of type γ released by presynaptic neuron R_1 . Let the geometry of neurotransmitter \mathbf{g} be given by an arbitrary 3-dimensional geometry in Cartesian coordinates (x, y, z) , with origin of coordinates at the centroid of geometry, as shown in figure 12. Furthermore, let the orientation of neurotransmitter \mathbf{g} of type γ is defined by $[\Theta_{\gamma g}]_{R_1}$. The orientation $[\Theta_{\gamma g}]_{R_1}$ is represented in 3-dimensional spherical coordinates (r, θ, ϕ) . Consider an arbitrary point I on the surface of geometry of neurotransmitter \mathbf{g} , shown in figure 12. Orientation $[\Theta_{\gamma g}]_{R_1}$ of neurotransmitter \mathbf{g} of type γ is given by $[\Theta_{\gamma g}]_{R_1} = [[\mathbf{r}_{\gamma g}]_{R_1}, [\theta_{\gamma g}]_{R_1}, [\phi_{\gamma g}]_{R_1}]$, where point I act as the reference point for orientation of neurotransmitter. As the coordinates of point I change, the orientation of neurotransmitter changes. It should be noted that the radial distance $[\mathbf{r}_{\gamma g}]_{R_1}$ will always remain fixed in every orientation. So change in orientation $[\Theta_{\gamma g}]_{R_1}$ depends on the change in polar angle $[\theta_{\gamma g}]_{R_1}$ and azimuthal angle $[\phi_{\gamma g}]_{R_1}$.

Now that the convention of orientation is established, the effect of orientation $[\Theta_{\gamma g}]_{R_1}$ on the affinity of binding $[\mathcal{A}_{\gamma g}]_{R_1}$ for \mathbf{g} neurotransmitter of type γ will be discussed. Based on equation 11, orientation $[\Theta_{\gamma g}]_{R_1}$ governs the constant of proportionality $K_{\gamma g}$, since the number density of binding sites is fixed. To understand relationship between $[\Theta_{\gamma g}]_{R_1}$ and $K_{\gamma g}$, consider that there exists an orientation $[\Theta_{\gamma g}]_{R_1}$ of neurotransmitter \mathbf{g} where the value of constant of proportionality $K_{\gamma g}$ is maximum. Let this orientation be called optimum orientation defined by

$[[\Theta_{\gamma g}]_{R_1}]_{opt}$ and is given by $[[\Theta_{\gamma g}]_{R_1}]_{opt} = [[\mathbf{r}_{\gamma g}]_{R_1}, [[\theta_{\gamma g}]_{R_1}]_{opt}, [[\phi_{\gamma g}]_{R_1}]_{opt}]$. The optimum orientation tells that the values of polar angle and azimuthal angle for point I should

be $[\theta_{\gamma g}]_{R_1} = [[\theta_{\gamma g}]_{R_1}]_{opt}$ and $[\phi_{\gamma g}]_{R_1} = [[\phi_{\gamma g}]_{R_1}]_{opt}$, respectively, and at these angles for point I, the value of constant of proportionality will be maximum i.e. $[K_{\gamma g}]_{max}$. Any changes of polar angle from $[[\theta_{\gamma g}]_{R_1}]_{opt}$ to $[\theta_{\gamma g}]_{R_1}$ and azimuthal angle from $[[\phi_{\gamma g}]_{R_1}]_{opt}$ to $[\phi_{\gamma g}]_{R_1}$ will decrease the value of constant of proportionality from $[K_{\gamma g}]_{max}$. Let the changes in polar angles and azimuthal angle be defined by $\Delta [\theta_{\gamma g}]_{R_1}$ and $\Delta [\phi_{\gamma g}]_{R_1}$ respectively and are given by equations 40 and 41, respectively. It should be noted the only the positive absolute values of change in both polar angle and azimuthal angle will be used in this article, for simplicity.

$$\Delta [\theta_{\gamma g}]_{R_1} = \left| [\theta_{\gamma g}]_{R_1} - [[\theta_{\gamma g}]_{R_1}]_{opt} \right| \quad 40$$

$$\Delta [\phi_{\gamma g}]_{R_1} = \left| [\phi_{\gamma g}]_{R_1} - [[\phi_{\gamma g}]_{R_1}]_{opt} \right| \quad 41$$

The change in orientation from optimum orientation $[[\theta_{\gamma g}]_{R_1}]_{opt}$ defined by $\Delta [\theta_{\gamma g}]_{R_1}$ is just the sum of change in polar angle and azimuthal angle given by equation 42.

$$\Delta [\theta_{\gamma g}]_{R_1} = \Delta [\theta_{\gamma g}]_{R_1} + \Delta [\phi_{\gamma g}]_{R_1} \quad 42.$$

To establish the relationship between changes in orientation $\Delta [\theta_{\gamma g}]_{R_1}$ and constant of proportionality $K_{\gamma g}$, exponential functions will be used. Let the relationship between change in orientation $\Delta [\theta_{\gamma g}]_{R_1}$ and constant of proportionality $K_{\gamma g}$ is given by equation 43, where χ_{γ} is the rate parameter for neurotransmitter of type γ , and governs the rate of decrease in constant of proportionality with increase in change in orientation. Furthermore, the changes in polar and azimuthal angle are given in radians, as a convention for equation 43.

$$K_{\gamma_g} = [K_{\gamma_g}]_{max} \cdot e^{-\chi_{\gamma} \cdot \Delta[\theta_{\gamma_g}]_{R_1}}$$

43

Once the value of K_{γ_g} is obtained, the affinity of binding $[\mathcal{A}_{\gamma_g}]_{R_1}$ can be calculated using equation 11. Likewise, the affinities $[\mathcal{A}_{\gamma}]_{R_1}$ and $[\mathcal{A}]_{R_1}$ can be calculated using equations 12 and 13, respectively.

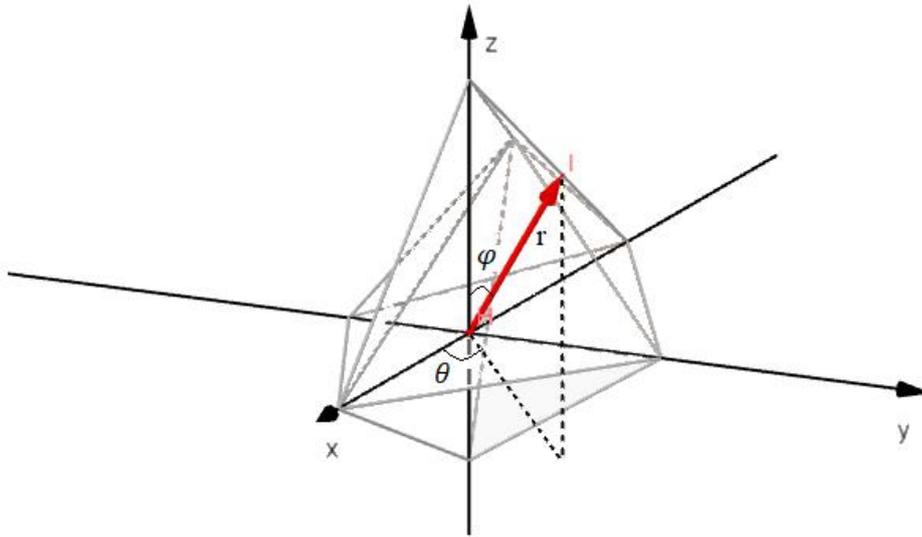


Figure 12. Representation of an arbitrary geometry of type γ neurotransmitter in 3-dimensional Cartesian coordinates. The origin of Cartesian coordinates represent the centroid of the geometry of the neurotransmitter. The orientation of the neurotransmitter is represented in 3-dimensional spherical coordinates, with point I as the reference point of orientation. The orientation of neurotransmitter is given by (r, θ, φ) , where r is the radial distance, θ is the polar angle and φ is the azimuthal angle. Any change in orientation of neurotransmitter results from the change in polar and/or azimuthal angle.

3.2.5.1. Example Case

Consider stimulus $[O_1]_{R_1}$ comprises of two types of neurotransmitters i.e. $\tau=2$ with $\gamma = 1,2$. The total number of released neurotransmitters be $[\varepsilon]_{R_1} = 5$ (equation 1) with $[\varepsilon_1]_{R_1} = 3$ and $[\varepsilon_2]_{R_1} = 2$. Furthermore, consider the optimum orientations of type $\gamma = 1,2$ are given by equations 44 and 45, respectively. Both polar and azimuthal angles are given in radians.

$$[[\boldsymbol{\Theta}_{1g}]_{R_1}]_{opt} = \left[[\mathbf{r}_{1g}]_{R_1}, [[\boldsymbol{\theta}_{1g}]_{R_1}]_{opt}, [[\boldsymbol{\phi}_{1g}]_{R_1}]_{opt} \right] = [2,1,1] \quad 44$$

$$[[\boldsymbol{\Theta}_{2g}]_{R_1}]_{opt} = \left[[\mathbf{r}_{2g}]_{R_1}, [[\boldsymbol{\theta}_{2g}]_{R_1}]_{opt}, [[\boldsymbol{\phi}_{2g}]_{R_1}]_{opt} \right] = [1,0,0] \quad 45$$

The orientations of type $\gamma = 1$ neurotransmitters is given by $[\boldsymbol{\Theta}_{11}]_{R_1} = [2,1.5,0.78]$, $[\boldsymbol{\Theta}_{12}]_{R_1} = [2,1,0.9]$ and $[\boldsymbol{\Theta}_{13}]_{R_1} = [2,1.2,1]$. Similarly, the orientation of type $\gamma = 2$ neurotransmitters is given by $[\boldsymbol{\Theta}_{21}]_{R_1} = [1,0.1,1.2]$ and $[\boldsymbol{\Theta}_{22}]_{R_1} = [1,0,0]$. The number densities of binding sites on the postsynaptic neuron I_1 for type $\gamma = 1,2$ neurotransmitters are $[\rho_{1R_1}]_{I_1} = 0.15 \text{ sites/unit}^2$ and $[\rho_{2R_1}]_{I_1} = 0.23 \text{ sites/unit}^2$. In this example, the effect of distribution of binding sites and kinetic energy on binding affinities is neglected. Furthermore, maximum constant of proportionality and rate parameter for type $\gamma = 1$ is given by $[K_{1g}]_{max} = 3$ and $\chi_1 = 4$. Likewise, $[K_{2g}]_{max} = 2$ and $\chi_2 = 6$, for type $\gamma = 2$ neurotransmitters. The relationship between $\Delta[\boldsymbol{\Theta}_{\gamma g}]_{R_1}$ and $K_{\gamma g}$ for type $\gamma = 1,2$ neurotransmitters is shown in figure 13. Using equations 40, 41, 42 and 43, the constant of proportionalities for type $\gamma = 1,2$ are calculated and are shown in equation 46 and 47, respectively.

$$[K_{11}, K_{12}, K_{13}] = [0.1684, 2.01096, 1.3480] \quad 46$$

$$[K_{21}, K_{22}] = [0.000819, 2] \quad 47$$

Using equation 11, the affinity of binding $[\mathcal{A}_{1g}]_{R_1}$ and $[\mathcal{A}_{2g}]_{R_1}$ are calculated to be $[[\mathcal{A}_{11}]_{R_1}, [\mathcal{A}_{12}]_{R_1}, [\mathcal{A}_{13}]_{R_1}] = [0.0246, 0.23174, 0.16819]$ and $[[\mathcal{A}_{21}]_{R_1}, [\mathcal{A}_{22}]_{R_1}] = [0.0001883, 0.3151]$. The affinities of binding for type $\gamma = 1,2$ are calculated to be $[\mathcal{A}_1]_{R_1} = 0.14151$ and $[\mathcal{A}_2]_{R_1} = 0.15764$ (equation 12). The binding affinity of stimulus $[O_1]_{R_1}$ is calculated to be $[\mathcal{A}]_{R_1} = 0.1496$ (equation 13).

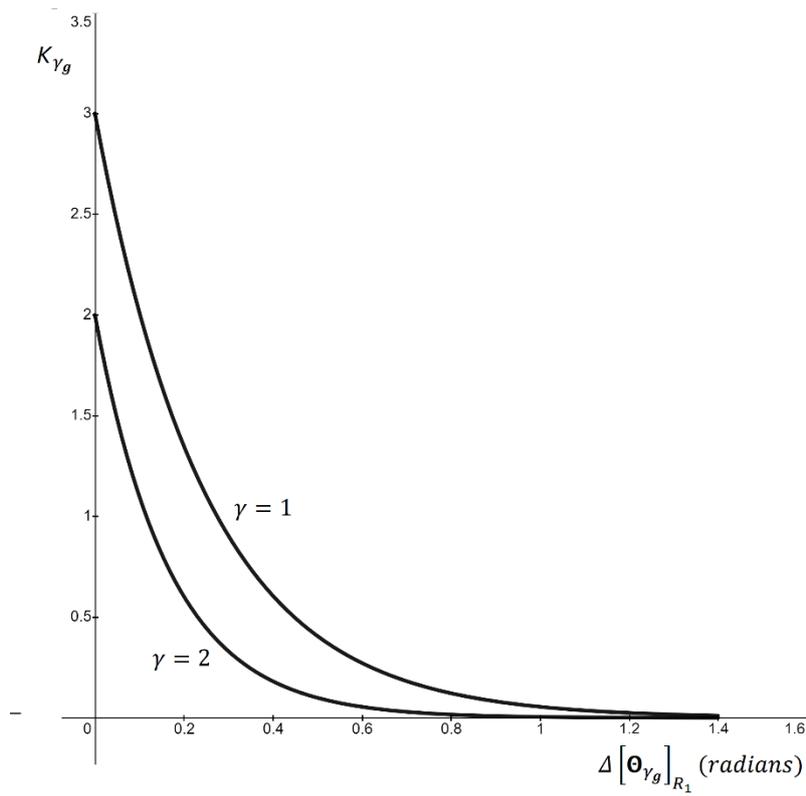


Figure 13. Relationship between changes in orientation and constant of proportionality for type $\gamma = 1,2$ neurotransmitters. The maximum constant of proportionalities are $[K_{1g}]_{max} = 3$ and $[K_{2g}]_{max} = 2$, when $\Delta[\Theta_{\gamma g}]_{R_1} = 0$, representing optimum orientations of each type of neurotransmitters. As $\Delta[\Theta_{\gamma g}]_{R_1}$ increases, constant of proportionality decreases with rates $\chi_1 = 4$, $\chi_2 = 6$ for $\gamma = 1,2$ neurotransmitters.

3.3. Constant of Proportionality K_{γ_g}

The constant of proportionality K_{γ_g} of equation 11 is governed by four factors; distribution of binding site $[\mu_{\gamma_{R_1}}]_{I_1}$, ratio $\frac{[\varepsilon_{\gamma}]_{R_1}}{[N_{\gamma\delta R_1}]_{I_1}}$, kinetic energy $[\kappa_{\gamma_g}]_{R_1}$ and orientation $[\Theta_{\gamma_g}]_{R_1}$. So far in example cases, only one factor at a time is used to calculate the value of K_{γ_g} , neglecting others. However, if incorporates all four factors in calculating K_{γ_g} , the constant of proportionality is given by equation 48 i.e. the product of contribution of each factor on the value of constant of proportionality K_{γ_g} . Based on equation 48, the affinity of binding $[\mathcal{A}_{\gamma_g}]_{R_1}$ is given by equation 49, incorporating all five discussed factors.

$$K_{\gamma_g} = [K_{\gamma_g}]_{\mu} \cdot [K_{\gamma_g}]_{\frac{\varepsilon_{\gamma}}{N_{\gamma\delta}}} \cdot [K_{\gamma_g}]_{\kappa} \cdot [K_{\gamma_g}]_{\Theta} \quad 48$$

$$[\mathcal{A}_{\gamma_g}]_{R_1} = \frac{[K_{\gamma_g}]_{\mu} \cdot [K_{\gamma_g}]_{\frac{\varepsilon_{\gamma}}{N_{\gamma\delta}}} \cdot [K_{\gamma_g}]_{\kappa} \cdot [K_{\gamma_g}]_{\Theta} [\rho_{\gamma_{R_1}}]_{I_1}}{[K_{\gamma_g}]_{\mu} \cdot [K_{\gamma_g}]_{\frac{\varepsilon_{\gamma}}{N_{\gamma\delta}}} \cdot [K_{\gamma_g}]_{\kappa} \cdot [K_{\gamma_g}]_{\Theta} [\rho_{\gamma_{R_1}}]_{I_1} + 1} \quad 49$$

3.4. Contribution of Stimuli in Neuronal Activation

The contribution of stimulus $[O_1]_{R_1}$ in activation of postsynaptic neuron I_1 is correlated with the affinity of binding $[\mathcal{A}]_{R_1}$ of stimulus $[O_1]_{R_1}$. Higher the $[\mathcal{A}]_{R_1}$, higher the likelihood of released neurotransmitters of binding to the receptor binding sites of I_1 , so higher the contribution of stimulus $[O_1]_{R_1}$ in activation of I_1 . In other words, not all released neurotransmitters of presynaptic neuron contributes to the activation of postsynaptic neuron and only the neurotransmitters than bind to the receptor binding sites contributes in activation of postsynaptic neuron. Based on the contribution or number of R_1 neurotransmitters binding to I_1 , the postsynaptic neuron becomes permeable to specific ionic species and the movement

of these ionic species results in activation of I_1 to a specific activated state. Now, consider postsynaptic neuron I_1 receiving stimuli from two presynaptic neurons R_1 and R_2 , simultaneously i.e. $[S]_{I_1} = 2$. Furthermore, the output generated by R_1 and R_2 which stimulates I_1 is given by $[O_1]_{R_1}$ and $[O_1]_{R_2}$, respectively. Both $[O_1]_{R_1}$ and $[O_1]_{R_2}$ have affinities of binding $[\mathcal{A}]_{R_1}$ and $[\mathcal{A}]_{R_2}$, which is correlated to the contributions of stimuli of presynaptic neurons. Based on these contributions, the postsynaptic neuron I_1 attains a unique activated state. The activation of postsynaptic neuron I_1 results from the sum of the contributions of stimuli from presynaptic neurons R_1 and R_2 , which is correlated with the affinities $[\mathcal{A}]_{R_1}$ and $[\mathcal{A}]_{R_2}$. Likewise, the postsynaptic neuron I_1 can be stimulated by any number of presynaptic neurons, both simultaneously or at time intervals. The activation of I_1 results from the sum of contributions of each stimulus, and is correlated with the affinity of binding of each stimulus.

3.5. Fate of Neurotransmitters

Depending on the affinity of binding of stimulus, not all released neurotransmitters bind to the postsynaptic neuron. The question now is that what happens to the neurotransmitters that do not bind to the postsynaptic neuron. To address this question, consider R_1 and I_1 as pre and postsynaptic neurons. Furthermore, consider the time at which neurotransmitters are released by R_1 is denoted by time $t = 0$. At time $t = 0$, each individual released neurotransmitter \mathbf{g} of type γ has a position, momentum, kinetic energy $[\kappa_{\gamma\mathbf{g}}]_{R_1}$ and orientation $[\Theta_{\gamma\mathbf{g}}]_{R_1}$. Let the position of neurotransmitter \mathbf{g} of type γ is given by a 3-dimensional Vector $\left[\begin{array}{c} \longrightarrow \\ L_{\gamma\mathbf{g}} \end{array} \right]_{R_1}$ in Cartesian

coordinates (x, y, z) , shown in Figure 14. Furthermore, let the momentum of neurotransmitter g

neurotransmitter of type γ is given by $\begin{bmatrix} \rightarrow \\ M_{\gamma g} \end{bmatrix}_{R_1}$.

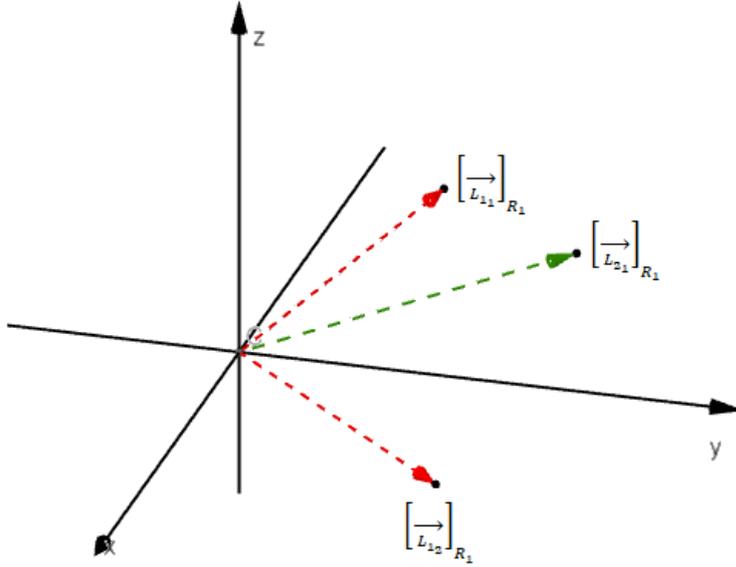


Figure 14. Representation of Position Vector of neurotransmitters in Cartesian coordinates. Two types of neurotransmitters $\gamma = 1, 2$ are presented, with red vectors for type $\gamma = 1$ and green vector for type $\gamma = 2$. A global reference frame is selected for all types of neurotransmitters. Position vector $\begin{bmatrix} \rightarrow \\ L_{\gamma g} \end{bmatrix}_{R_1}$ points from the origin of global reference frame to the centroid of the geometry of the neurotransmitter.

With increase in time i.e. $t > 0$, the position $\begin{bmatrix} \rightarrow \\ L_{\gamma g} \end{bmatrix}_{R_1}$ and momentum $\begin{bmatrix} \rightarrow \\ M_{\gamma g} \end{bmatrix}_{R_1}$ evolves with

respect to time. Let the position and momentum at time $t = 0$ be called initial

position $\left[\begin{bmatrix} \rightarrow \\ L_{\gamma g} \end{bmatrix}_{R_1} \right]_{ini}$ and initial momentum $\left[\begin{bmatrix} \rightarrow \\ M_{\gamma g} \end{bmatrix}_{R_1} \right]_{ini}$ of neurotransmitter. With evolution

of time $t > 0$, the values of position and momentum change given by $\Delta \begin{bmatrix} \rightarrow \\ L_{\gamma g} \end{bmatrix}_{R_1}$ and $\Delta \begin{bmatrix} \rightarrow \\ M_{\gamma g} \end{bmatrix}_{R_1}$,

resulting in neurotransmitter following a certain trajectory. Let the trajectory of

neurotransmitter g of type γ be given by $\left[\begin{array}{c} \longrightarrow \\ T_{\gamma g} \end{array} \right]_{R_1}$. Along the trajectory $\left[\begin{array}{c} \longrightarrow \\ T_{\gamma g} \end{array} \right]_{R_1}$, the neurotransmitter may collide with other neurotransmitters resulting in the altering of trajectory $\left[\begin{array}{c} \longrightarrow \\ T_{\gamma g} \end{array} \right]_{R_1}$. Furthermore, during collisions, the momentum $\left[\begin{array}{c} \longrightarrow \\ M_{\gamma g} \end{array} \right]_{R_1}$ and kinetic energy $[\kappa_{\gamma g}]_{R_1}$ is exchanged between neurotransmitters and during collisions, the orientation $[\Theta_{\gamma g}]_{R_1}$ of neurotransmitters changes. From time $t = 0$, the trajectory $\left[\begin{array}{c} \longrightarrow \\ T_{\gamma g} \end{array} \right]_{R_1}$ of neurotransmitters keeps evolving with respect to time. The fate of the neurotransmitter depends on the trajectory $\left[\begin{array}{c} \longrightarrow \\ T_{\gamma g} \end{array} \right]_{R_1}$. For instance, if at any time $t > 0$, $\left[\begin{array}{c} \longrightarrow \\ T_{\gamma g} \end{array} \right]_{R_1}$ leads the neurotransmitter to the surface area coverage on the postsynaptic neuron, the neurotransmitter has a certain affinity of binding to the receptor binding sites, depending on the factors discussed previously. Furthermore, if the trajectory $\left[\begin{array}{c} \longrightarrow \\ T_{\gamma g} \end{array} \right]_{R_1}$ at any instance of time leads the neurotransmitter back to the presynaptic neuron, reuptake of neurotransmitter for metabolism can happen. At this point, it is highly plausible that the $\left[\begin{array}{c} \longrightarrow \\ T_{\gamma g} \end{array} \right]_{R_1}$ can take the neurotransmitter beyond the synaptic cleft between pre and postsynaptic neuron. It is imperative that one discusses the fate of neurotransmitter whose trajectory $\left[\begin{array}{c} \longrightarrow \\ T_{\gamma g} \end{array} \right]_{R_1}$ leads it beyond synaptic cleft.

3.5.1. Trajectories beyond Synaptic Cleft

Let the neurotransmitters whose trajectories lead them beyond synaptic cleft be called ‘drifting neurotransmitters’. In this section, two cases for drifting neurotransmitters will be discussed.

Case 1

Consider a drifting neurotransmitter g of type γ whose trajectory $\left[\begin{array}{c} \longrightarrow \\ T_{\gamma g} \end{array} \right]_{R_1}$ leads it to the binding site of type γ beyond the surface area coverage $[\delta_{R_1}]_{I_1}$ on postsynaptic neuron I_1 . As the drifting neurotransmitter reaches the receptor binding site, it has a certain affinity of binding $[\mathcal{A}_{\gamma g}]_{R_1}$ to the binding site. For this drifting neurotransmitter, the affinity of binding $[\mathcal{A}_{\gamma g}]_{R_1}$ depends on the kinetic energy $[\kappa_{\gamma g}]_{R_1}$ and orientation $[\Theta_{\gamma g}]_{R_1}$ at the time of arrival, given by equation 50. If the drifting neurotransmitter binds to the binding site, it stimulates postsynaptic neuron and has the contribution in the activation of postsynaptic neuron I_1 .

$$[\mathcal{A}_{\gamma g}]_{R_1} = \frac{[K_{\gamma g}]_{\kappa} \cdot [K_{\gamma g}]_{\theta}}{[K_{\gamma g}]_{\kappa} \cdot [K_{\gamma g}]_{\theta+1}} \quad 50$$

Case 2

Consider a drifting neurotransmitter g of type γ whose trajectory $\left[\begin{array}{c} \longrightarrow \\ T_{\gamma g} \end{array} \right]_{R_1}$ leads it to the binding site of type γ on a nearby neuron. Let this nearby neuron be I_{2x} which is in rest state i.e. $x = 0$, at the time of arrival of drifting neurotransmitter. This drifting neurotransmitter has a certain affinity of binding $[\mathcal{A}_{\gamma g}]_{R_1}$ to the binding site of I_2 , given by equation 50. If the drifting neurotransmitter binds to the receptor binding site, it stimulates I_2 and contributes in the

activation of I_2 to a certain activated state $x > 0$. Now, consider a scenario where I_2 is stimulated by a presynaptic neuron R_3 at a certain time t . Coincidentally, a drifting neurotransmitter g of type γ binds to I_2 at the same time t . The activation of I_2 depends on both the contributions of presynaptic neuron R_3 and the drifting neurotransmitter.

At this point, it is necessary that one differentiate between the stimulation of neuron by presynaptic neuron and stimulation by drifting neurotransmitter. To establish this difference, let the stimulation by presynaptic neuron be called 'Direct Stimulation' and stimulation by drifting neurotransmitter be called 'Indirect Stimulation'. Based on this premise, the stimulation of any arbitrary neuron at any instance of time is given by the sum of direct and indirect stimulation i.e. *Stimulation = Direct Stimulation + Indirect Stimulation*. Likewise, the activation of any arbitrary neuron at any instance of time depends on the sum of the contributions of each direct stimulation and each indirect stimulation.

4. State Adaptabilities of a Neuron

In this section, the concept of state adaptabilities of a neuron will be introduced. Consider a receptor neuron R_{1x} with the total number of possible states equal to y . State adaptability $[\Omega_x]_{R_1}$ is the probability that the neuron R_{1x} will attain an activated state x , at a given instance of time t . The value of $[\Omega_x]_{R_1}$ varies from 0 to 1 i.e. $0 \leq [\Omega_x]_{R_1} \leq 1$. In other words, state adaptabilities are the likelihoods that the neuron attains a certain state. Furthermore, since the total number of possible activated states of neuron R_{1x} is $y - 1$ as $x = 0$ is the rest state, the total number of state adaptabilities are $y - 1$ and the sum of state adaptabilities for neuron R_{1x} at any instance of time t is given by equation 51.

$$\sum_{x=1}^y [\Omega_x]_{R_1} (0 < t \leq \infty) = 1 \quad (51)$$

Consider an arbitrary state adaptability $[\Omega_1]_{R_1}$ and assume at time $t = 0$, all possible activated states of R_{1x} are equally probable i.e. $[\Omega_x]_{R_1}(t = 0) = \frac{1}{y-1}$ and sum of all $y - 1$ state adaptabilities satisfies equation 51. Ω_1 of R_1 at $t = 0$ is given by equation 52.

$$[\Omega_1]_{R_1}(t = 0) = \frac{1}{y-1} \quad (52)$$

With the evolution of time i.e. $t > 0$, the state adaptability $[\Omega_1]_{R_1}$ changes depending on the state adaptability factors $[\eta_1]_{R_1}$, which can be $[\Omega_1]_{R_1} - \text{increasing}$, $[\Omega_1]_{R_1} - \text{decreasing}$ or $[\Omega_1]_{R_1} - \text{constant}$. State adaptability factors $[\eta_x]_{R_1}$ aid neuron to adapt itself functionally, e.g. optimizing the number and distribution of binding sites on its plasma membrane, preparing it to attain a certain state at any instance of time. As an example, hypothetically if the state adaptability of any arbitrary state at an instance of time reaches 1, from that instance, the neuron is fully adapted functionally to attain that arbitrary state at that instant and later instances. However, this state adaptability can fall below 1, if some other state adaptability factors

increase the state adaptabilities of other states. In a sense, state adaptability factors $[\eta_1]_{R_1}$ governs the rate of state adaptabilities overtime. Let the rate of state adaptability is given by $\frac{d[\Omega_1]_{R_1}}{dt} = [\dot{\Omega}_x]_{R_1}$. For $[\Omega_1]_{R_1}$, the rate of state adaptability is given by $[\dot{\Omega}_1]_{R_1}$. So, $[\Omega_1]_{R_1}$ – *increasing* adaptability factors governs positive rate i.e. $+\dot{[\Omega_1]}_{R_1}$, likewise $[\Omega_1]_{R_1}$ – *decreasing* governs negative rate i.e. $-\dot{[\Omega_1]}_{R_1}$ and for $[\Omega_1]_{R_1}$ – *constant*, the rate is equal to zero i.e. $[\dot{\Omega}_1]_{R_1} = 0$. The concepts of state adaptabilities and state adaptability factors applies to all three types of neurons.

4.1. Example Case of State Adaptability with time

Consider an effector neuron E_{1_x} with total number of possible states $y = 101$ and assume at time $t = 0$ all the possible activated states are equally probable. Therefore, at time $t = 0$, the state adaptability of state $x = 1$ i.e. $[\Omega_1]_{E_1}$ is given by equation 53. At any instance of time, sum of state probabilities is given by equation 51 with subscript of E_1 instead of R_1 .

$$[\Omega_1]_{E_1}(t = 0) = \frac{1}{100} \quad (53)$$

Consider from time interval $0 < t \leq 5$, neuron E_{1_x} only attains state $x = 1$, ten times. Let the number of unique attained states in an interval is defined by $[\Phi]_{E_1}$ and the number of times that unique state is attained in that interval is defined by $[\lambda_x]_{E_1}$. In time interval $0 < t \leq 5$, $[\Phi]_{E_1} = 1$ and $[\lambda_1]_{E_1} = 10$. Both $[\Phi]_{E_1}$ and $[\lambda_1]_{E_1}$ in the time interval $0 < t \leq 5$ are two of the state adaptability factors $[\eta_1]_{E_1}$ which governs the rate of state adaptability $[\dot{\Omega}_1]_{E_1}$. Let $[\dot{\Omega}_1]_{E_1}$ governed by $[\Phi]_{E_1}$ and $[\lambda_1]_{E_1}$ in the time interval $0 < t \leq 5$ is given by equation 54.

$$[\dot{\Omega}_1]_{E_1}(0 < t \leq 5) = 0.06t \quad (54)$$

Based on equation 54, both $[\Phi]_{E_1} = 1$ and $[\lambda_1]_{E_1} = 10$ are $[\Omega_1]_{E_1}$ – *increasing* state adaptability factors. This is a reasonable assumption as in the given interval, neuron E_1 only attains $x = 1$ state, so the neuron will adapt itself functionally to increase the probability of attaining $x = 1$ beyond the given interval. Likewise, with the increase in the number of times neuron attains $x = 1$ in the interval, the neuron will adapt itself faster, increasing the state adaptability of state $x = 1$. The state adaptability in the time interval $0 < t \leq 5$ is the integral of equation 54 is given by equation 55 with $[\Omega_1]_{E_1}$ at time $t = 0$ given by equation 53.

$$[\dot{\Omega}_1]_{E_1} (0 < t \leq 5) = 0.03t^2 + 0.01 \quad (55)$$

Consider in time interval $5 < t \leq 10$, neuron E_{1x} attains 10 unique states i.e. $[\Phi]_{E_1} = 10$ and each unique state is attained once i.e. $[\lambda_x]_{E_1} = 1$. Let the rate of state adaptability, governed by $[\Phi]_{E_1} = 10$ and $[\lambda_x]_{E_1} = 1$ in the time interval $5 < t \leq 10$ is given by equation 56.

$$[\dot{\Omega}_1]_{E_1} (5 < t \leq 10) = -0.1 \quad (56)$$

Based on equation 56, both $[\Phi]_{E_1}$ and $[\lambda_x]_{E_1}$ are $[\Omega_1]_{E_1}$ – *decreasing* state adaptability factors, which is reasonable as each unique attained state governs the neuron to adapt accordingly, decreasing the probability of attaining $x = 1$ state. The state adaptability of state $x = 1$ in the interval $5 < t \leq 10$ is given by equation 57.

$$[\Omega_1]_{E_1} (5 < t \leq 10) = -0.1t + 1.26 \quad (57)$$

Now, consider in time interval $10 < t \leq 20$, neuron E_{1x} only attains state $x = 1$, twice i.e. $[\Phi]_{E_1} = 1$ and $[\lambda_1]_{E_1} = 2$. Based on these $[\eta_1]_{E_1}$, let the rate of state adaptability in the time interval $10 < t \leq 20$ be given by equation 58; the corresponding state adaptability is given by equation 59.

$$[\dot{\Omega}_1]_{E_1} (10 < t \leq 20) = 0.026 \quad (58)$$

$$[\Omega_1]_{E_1}(10 < t \leq 20) = 0.026t \quad (59)$$

Lastly, consider in the time interval $20 < t \leq 35$, both $[\Phi]_{E_1} = 0$ and $[\lambda_x]_{E_1} = 0$, i.e. the neuron E_{1_x} doesn't attain any state. The rate of state adaptability in this interval will be 0 and the state adaptability of state $x = 1$ will remain constant. i.e. $[\Omega_1]_{E_1}(20 < t \leq 35) = 0.52$. The state adaptability of state $x = 1$ in the time interval $0 \leq t \leq 35$ is shown in figure 15.

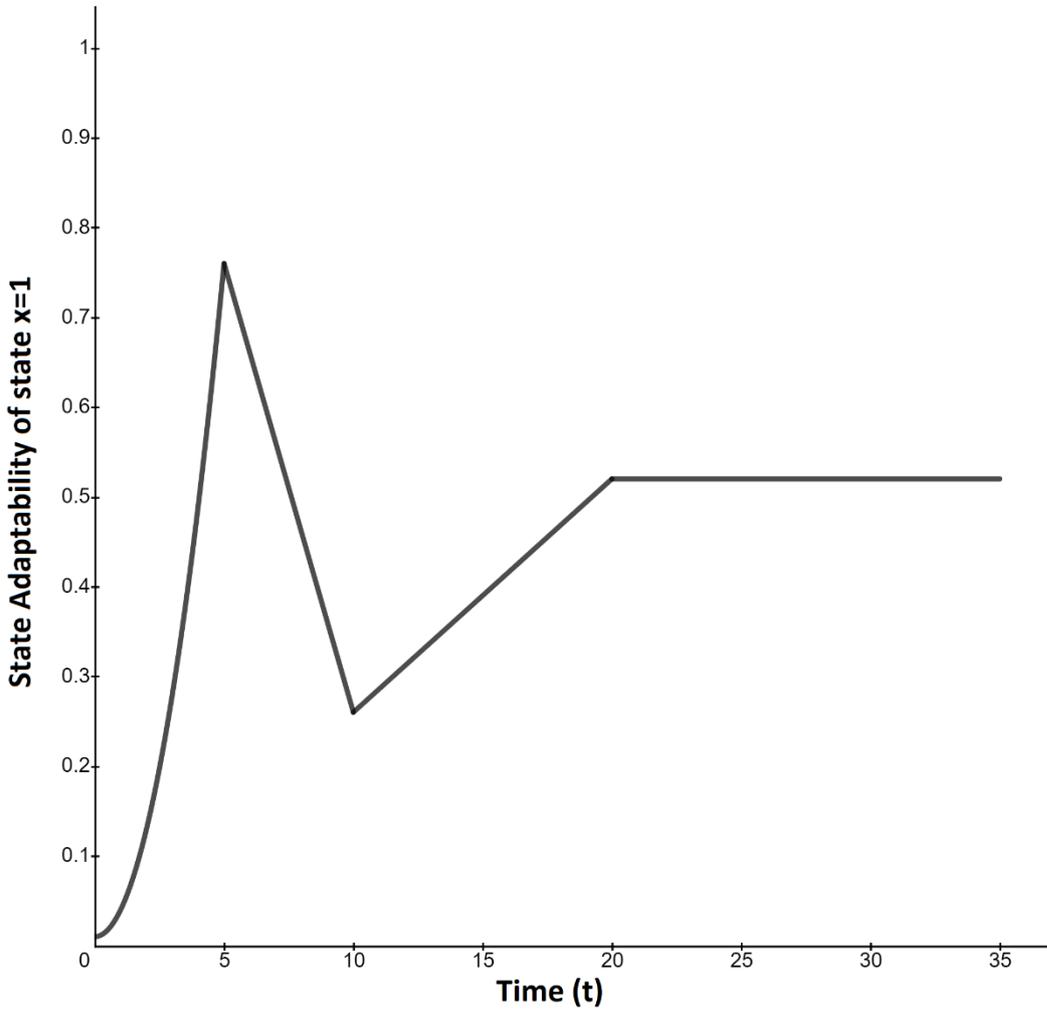


Figure 15. State Adaptability evolution of state $x = 1$ in the time interval $0 \leq t \leq 35$. At $t = 0$, $[\Omega_1]_{E_1} = 0.01$. In interval $0 < t \leq 5$, $[\Omega_1]_{E_1}$ increases from $[\Omega_1]_{E_1} = 0.01$ reaching the maximum value of $[\Omega_1]_{E_1} = 0.76$ at $t = 5$. From $5 < t \leq 10$, $[\Omega_1]_{E_1}$ decreases till time $t = 10$, reaching the value of 0.26. In the next interval $10 < t \leq 20$, $[\Omega_1]_{E_1}$ again increases reaching $[\Omega_1]_{E_1} = 0.52$ at $t = 20$. From $t = 20$ till $t = 35$, $[\Omega_1]_{E_1}$ remains constant at 0.52.

5. Output Responses of a Neuron

In this section, output responses of a neuron resulting from neuronal attained states will be exposed. Consider a neuron R_{1x} and let the output responses of R_{1x} be given by $[O_a]_{R_1}$, where $a = 1,2,3, \dots b$; a represents a unique output response and b are the total number of possible output responses neuron R_{1x} can generate. The output responses of a neuron are generally of two types depending on the type of communication; release of neurotransmitters results from chemical synaptic communication and movement of ions through gap junction from pre to post-synaptic neuron in electrical synaptic communication. To cater these two types of output responses, consider a set $[O]_{R_1}$, which is defined by equation 60, where $[O_C]_{R_1}$ represents a set of responses resulting from the chemical synaptic communication and $[O_F]_{R_1}$ represents a set of output responses by electrical synaptic communication. $[O_C]_{R_1}$ and $[O_F]_{R_1}$ are defined by equation 61 and 62, respectively.

$$[O]_{R_1} = \{[O_C]_{R_1} \cup [O_F]_{R_1}\} \quad (60)$$

$$[O_C]_{R_1} = \{[O_{C_U}]_{R_1}\} \quad (61)$$

$$[O_F]_{R_1} = \{[O_{F_Y}]_{R_1}\} \quad (62)$$

$[O_{C_U}]_{R_1}$ represents a unique output response resulting from chemical synaptic communication and $U = 1,2,3, \dots V$ and V are the total number of unique output responses of neuron R_1 resulting from chemical synaptic communication. Likewise, $[O_{F_Y}]_{R_1}$ represents unique neuronal output from electrical synaptic communication and $Y = 1,2,3, \dots Z$ and Z the total number of unique neuronal outputs from electrical synaptic communication are. The total number of output responses b of neuron R_1 are given by equation 63.

$$[b]_{R_1} = [V]_{R_1} + [Z]_{R_1} \quad (63)$$

It has been previously established that the total number of possible states of neuron R_{1x} is given by y . Let a set $[y]_{R_1}$ represents all these possible states and $[y]_{R_1}$ comprises of a rest state $[x_0]_{R_1}$ and three sets of states: states corresponding to subthreshold potentials, states corresponding to AP and states corresponding to electrical synaptic communication. Let the subthreshold potential states be defined by a set $[x_{STP}]_{R_1}$, AP states by a set $[x_{AP}]_{R_1}$ and electrical synaptic communication states by a set $[x_{ESC}]_{R_1}$. The elements of set $[y]_{R_1}$ are given by equation 64. The elements of sets $[x_{STP}]_{R_1}$, $[x_{AP}]_{R_1}$ and $[x_{ESC}]_{R_1}$ are given by equations 65, 66 and 67 respectively, where $J = 1,2,3, \dots, K$, $L = 1,2,3, \dots, M$ and $P = 1,2,3, \dots, Q$. J , L and P represent an individual subthreshold potential state, AP state and electrical synaptic communication state, respectively and K , M and Q are the total number of possible subthreshold potential states, AP states and electrical synaptic communication states, respectively. The total number of possible states of neuron R_{1x} therefore are given by equation 68.

$$[y]_{R_1} = \{ [x_0]_{R_1} \cup [x_{STP}]_{R_1} \cup [x_{AP}]_{R_1} \cup [x_{ESC}]_{R_1} \} \quad (64)$$

$$[x_{STP}]_{R_1} = \{ [x_{STP_J}]_{R_1} \} \quad (65)$$

$$[x_{AP}]_{R_1} = \{ [x_{AP_L}]_{R_1} \} \quad (66)$$

$$[x_{ESC}]_{R_1} = \{ [x_{ESC_P}]_{R_1} \} \quad (67)$$

$$[y]_{R_1} = 1 + [K]_{R_1} + [M]_{R_1} + [Q]_{R_1} \quad (68)$$

It has been previously discussed for chemical synaptic communication only the AP states produces an output response, so for neuron R_{1x} undergoing chemical synaptic communication, the elements of set $[x_{AP}]_{R_1}$ will produce output responses which are the subset of set $[O_C]_{R_1}$. Similarly, for electrical synaptic communication, the elements of set $[x_{ESC}]_{R_1}$ will produce

output responses from the set $[O_F]_{R_1}$. The output responses $[O_a]_{I_1}$ and $[O_a]_{E_1}$ of interneuron I_1 and effector neuron E_1 are given similarly to that of receptor neuron R_1 . Equation 60-68 holds for all three types of neurons with the appropriate neuron labelling.

5.1. Neuronal Behaviour

In this section, two types of neuronal behaviours (ideal neuronal behaviour and real neuronal behaviour) will be discussed. Consider a receptor neuron R_{1x} undergoing chemical and electrical synaptic communication overtime. Let $[\alpha]_{R_1}$ be the number of unique AP and ESC states attained by the neuron R_{1x} in a given time interval. Furthermore, let $[\beta]_{R_1}$ be the number of unique output responses produced as a result of $[\alpha]_{R_1}$ in that time interval. If in the given time interval, $[\alpha]_{R_1}$ is equal to $[\beta]_{R_1}$ i.e. number of unique attained states is equal to the number of unique output responses, neuron R_{1x} exhibits ideal neuronal behaviour, given by equation 69. Each AP state and each ESC state from sets $[x_{AP}]_{R_1}$ and $[x_{ESC}]_{R_1}$ respectively will produce a unique output response from sets $[O_C]_{R_1}$ and $[O_F]_{R_1}$ respectively. In other words, neuron's behaviour is classified as ideal if the total number of unique attained states is equal to the total number of output responses produced in a given time interval. On the contrary, if $[\alpha]_{R_1} > [\beta]_{R_1}$ i.e. the number of unique attained states is greater than the number of unique output responses in a time interval, neuron R_{1x} exhibits real neuronal behaviour, given by equation 70. One or more than one AP and/or ESC state can produce the same output response. In real neuronal behaviour, not all AP and ESC states have the potential to produce a unique output response. Interneurons and effector neurons exhibits similar types of behaviour.

$$\left[\frac{\beta}{\alpha}\right]_{R_1} = 1 \quad (69)$$

$$\left[\frac{\beta}{\alpha}\right]_{R_1} < 1 \quad (70)$$

5.1.1. Example Case of Neuronal Behaviour

Consider an interneuron I_{1x} in a rest state i.e. $x = 0$ at time $t = 0$. In the time interval $0 < t \leq 5$, neuron I_{1x} attains 3 AP states ($[x_{AP1}]_{I_1}, [x_{AP2}]_{I_1}, [x_{AP3}]_{I_1}$) and 2 ESC states ($[x_{ESC1}]_{I_1}, [x_{ESC2}]_{I_1}$). As a consequence of these attained states, consider neuron I_{1x} produces output responses ($[O_{C1}]_{I_1}, [O_{C2}]_{I_1}, [O_{C3}]_{I_1}$) by AP states and ($[O_{F1}]_{I_1}, [O_{F2}]_{I_1}$) by ESC states. In time interval $0 < t \leq 5$, both $[\alpha]_{I_1}$ and $[\beta]_{I_1}$ are equal to 5 and $\left[\frac{\beta}{\alpha}\right]_{I_1} = 1$, so I_{1x} exhibits ideal behaviour. In the time interval $5 < t \leq 15$, I_{1x} attains 3 AP states ($[x_{AP4}]_{I_1}, [x_{AP5}]_{I_1}, [x_{AP6}]_{I_1}$) and 4 ESC states ($[x_{ESC3}]_{I_1}, [x_{ESC4}]_{I_1}, [x_{ESC5}]_{I_1}, [x_{ESC6}]_{I_1}$). The resulting output responses are ($[O_{C4}]_{I_1}, [O_{C4}]_{I_1}, [O_{C5}]_{I_1}$) by AP states and ($[O_{F3}]_{I_1}, [O_{F3}]_{I_1}, [O_{F3}]_{I_1}, [O_{F4}]_{I_1}$) by ESC states. In this time interval, $[\alpha]_{I_1} = 7$ and $[\beta]_{I_1} = 4$ and $\left[\frac{\beta}{\alpha}\right]_{I_1} = \frac{4}{7}$ which is less than 1, so neuron I_{1x} exhibits real behaviour. In the time interval $15 < t \leq 35$, neuron I_{1x} attains 2 AP states ($[x_{AP1}]_{I_1}, [x_{AP3}]_{I_1}$) and 1 ESC state ($[x_{ESC5}]_{I_1}$). The resulting output responses in this interval are ($[O_{C1}]_{I_1}, [O_{C3}]_{I_1}$) by AP states and ($[O_{F3}]_{I_1}$) by ESC state. In time interval $15 < t \leq 35$, both $[\alpha]_{I_1}$ and $[\beta]_{I_1}$ are equal to 3 and $\left[\frac{\beta}{\alpha}\right]_{I_1} = 1$, so I_{1x} exhibits ideal behaviour. It should be noted that in this time interval, neuron I_{1x} attained states which were attained in previous time intervals and each attained state produced the same output as in previous intervals i.e. $[x_{AP1}]_{I_1} \rightarrow [O_{C1}]_{I_1}$, $[x_{AP3}]_{I_1} \rightarrow [O_{C3}]_{I_1}$ and $[x_{ESC5}]_{I_1} \rightarrow [O_{F3}]_{I_1}$. The point to ponder is that once a state produced an output response, it will always produce the same output response. Furthermore, if one considers time interval $0 < t \leq 35$, neuron I_{1x} exhibits real behaviour since in this interval $[\alpha]_{I_1} = 12$, $[\beta]_{I_1} = 9$ and $\left[\frac{\beta}{\alpha}\right]_{I_1} = 0.75$. At time $t > 5$, neuron I_{1x} starts exhibiting real behaviour transitioning from

ideal behaviour, and will always exhibit real behaviour in the interval $0 < t \leq \infty$ starting from time $t > 5$. In other words, the instant two attained states produce the same output response in the interval $0 < t \leq \infty$, neuron's behaviour transitions to real from ideal.

5.2. Probabilities of Output Responses of a Neuron

Consider a receptor neuron R_{1x} and let the probabilities of output responses $[O_a]_{R_1}$ generated by R_{1x} is given by $[\psi_{O_a}]_{R_1}$. The total number of probabilities of output responses is equal to the total number of possible unique output responses of a neuron i.e. b . Furthermore, the sum of total number of probabilities of output responses is given by equation 71.

$$\sum_{a=1}^b [\psi_{O_a}]_{R_1} (0 < t \leq \infty) = 1 \quad (71)$$

Consider an output response $[O_1]_{R_1}$ with the probability $[\psi_{O_1}]_{R_1}$. The probability $[\psi_{O_1}]_{R_1}$ is equal to the sum of state adaptabilities of states which produce output response $[O_1]_{R_1}$, given by equation 72. For instance, if only one state (AP or ESC) produce the output response $[O_1]_{R_1}$, the probability that the neuron will produce $[O_1]_{R_1}$ is equal to state adaptability of that state. The value of $[\psi_{O_1}]_{R_1}$ varies from $0 \leq [\psi_{O_1}]_{R_1} \leq 1$. The probabilities of output responses applies to all three types of neurons

$$[\psi_{O_1}]_{R_1} = \sum_{x \rightarrow [O_1]_{R_1}} [\Omega_x]_{R_1} \quad (72)$$

5.2.1. Example Case of Output Response Probability

Consider an output response probability $[\psi_{O_2}]_{E_1}$ of an effector neuron E_{1x} . The total number of possible states and unique output responses of E_{1x} are given by $[y]_{E_1} = 101$ and $[b]_{E_1} = 50$ respectively. Assume 3 states ($x = 2,3,4$) of neuron E_{1x} produce the output response $[O_2]_{E_1}$ i.e. $x = 2,3,4 \rightarrow [O_2]_{E_1}$. Furthermore, assume at time $t = 0$, all activated states of E_{1x} are equally probable with state adaptabilities $[\Omega_x]_{E_1}(t = 0) = \frac{1}{100}$. Using equation 72 for neuron E_{1x} , the probability of output response $[O_2]_{E_1}$ at time $t = 0$ is given by $[\psi_{O_2}]_{E_1}(t = 0) = 0.03$. Consider in the time interval $0 < t \leq 5$, the state adaptabilities of states $x = 2,3,4$ are given by equation 73, 74 and 75 respectively.

$$[\Omega_2]_{E_1}(0 < t \leq 5) = 0.02t^2 + 0.01 \quad (73)$$

$$[\Omega_3]_{E_1}(0 < t \leq 5) = 0.01t^2 + 0.01 \quad (74)$$

$$[\Omega_4]_{E_1}(0 < t \leq 5) = 0.002t^2 + 0.01 \quad (75)$$

Based on equations 73, 74 and 75, all three state adaptabilities are increasing, so they are governed by $[\Omega_{2,3,4}]_{E_1}$ – *increasing* state adaptability factors. As a consequence, $[\psi_{O_2}]_{E_1}$ in the time interval $0 < t \leq 5$ will increase, given by equation 76.

$$[\psi_{O_2}]_{E_1}(0 < t \leq 5) = 0.032t^2 + 0.03 \quad (76)$$

Consider in the time interval $5 < t \leq 10$, the state adaptabilities of states $x = 2,3,4$ are given by equations 77, 78 and 79 respectively.

$$[\Omega_2]_{E_1}(5 < t \leq 10) = -0.003t^2 + 0.585 \quad (77)$$

$$[\Omega_3]_{E_1}(5 < t \leq 10) = 0.001t + 0.255 \quad (78)$$

$$[\Omega_4]_{E_1}(5 < t \leq 10) = -0.0008t^2 + 0.08 \quad (79)$$

In this time interval, $[\Omega_2]_{E_1}$ and $[\Omega_4]_{E_1}$ are governed by $[\Omega_{2,4}]_{E_1} - \text{decreasing}$ adaptability factors as both state adaptabilities are decreasing. On the contrary, $[\Omega_3]_{E_1}$ is increasing. As a consequence, in the time interval $5 < t \leq 10$, $[\psi_{O_2}]_{E_1}$ decreases, given by equation 80.

$$[\psi_{O_2}]_{E_1} (5 < t \leq 10) = -0.0038t^2 + 0.001t + 0.92 \quad (80)$$

Consider in the time interval $10 < t \leq 25$, the rates of state adaptabilities of states $x = 2,3,4$ are 0 i.e. $[\dot{\Omega}_2, \dot{\Omega}_3, \dot{\Omega}_4]_{E_1} = 0$. In this time interval the state adaptabilities remain constant ($[\Omega_2]_{E_1} = 0.285$, $[\Omega_3]_{E_1} = 0.265$, $[\Omega_4]_{E_1} = 0$). As a result, $[\psi_{O_2}]_{E_1} = 0.55$ remains constant in interval $10 < t \leq 25$. The evolutions of state adaptabilities and $[\psi_{O_2}]_{E_1}$ in the time interval $0 < t \leq 25$ are shown in figures 16 and 17, respectively.

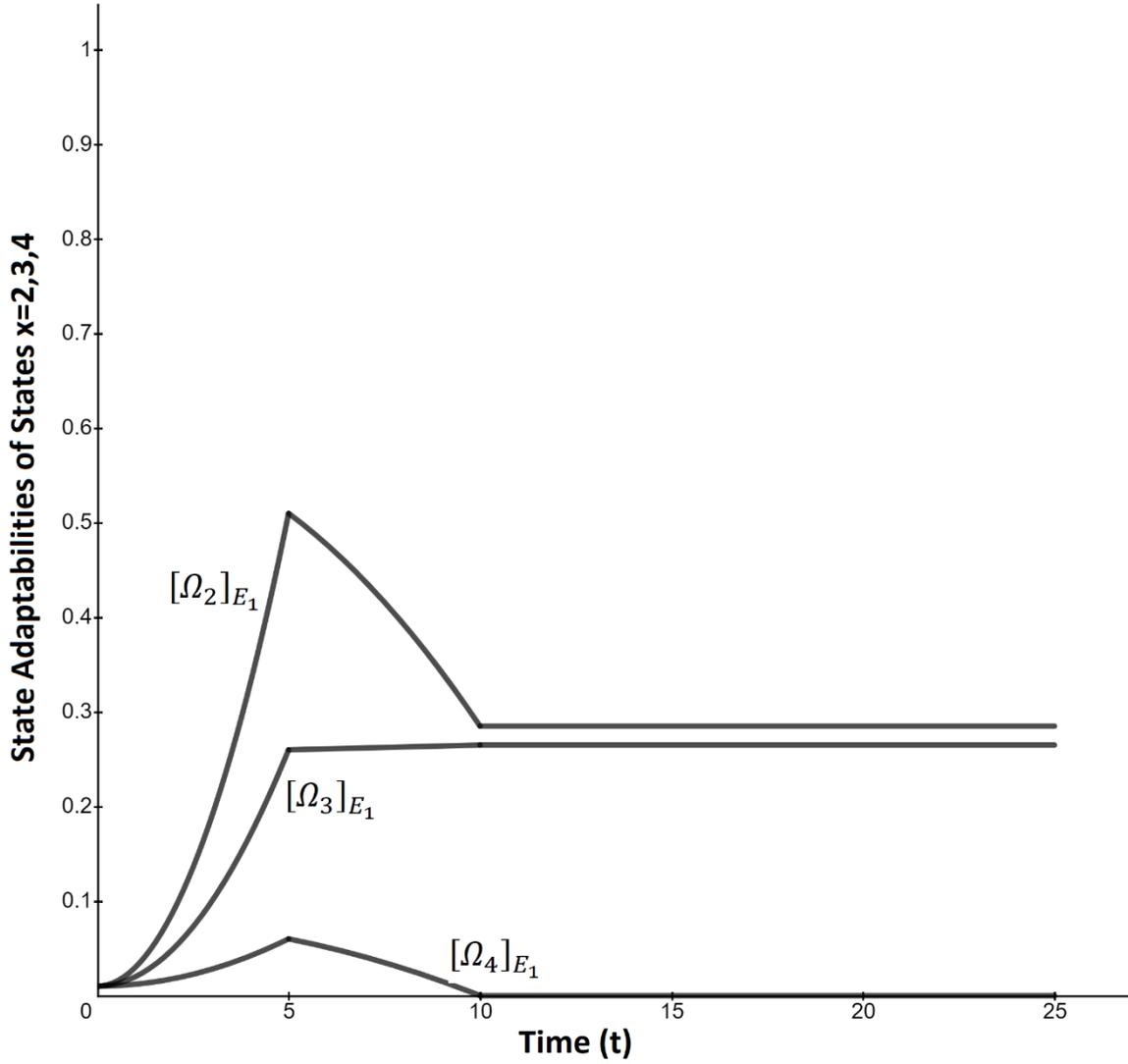


Figure 16. Evolution of state adaptabilities $[\Omega_2, \Omega_3, \Omega_4]_{E_1}$ in time interval $0 < t \leq 25$. At time $t = 5$, two state adaptabilities reach maximum values ($[\Omega_2 = 0.56, \Omega_4 = 0.06]_{E_1}$) from 0.03 with rates $[\dot{\Omega}_2]_{E_1} = 0.04t$ and $[\dot{\Omega}_4]_{E_1} = 0.004t$. $[\Omega_3]_{E_1}$ reaches the value of 0.26 at $t = 5$ with rate $[\dot{\Omega}_3]_{E_1} = 0.02t$. In time interval $5 < t \leq 10$ both $[\Omega_2]_{E_1}$ and $[\Omega_4]_{E_1}$ decrease with rates $[\dot{\Omega}_2]_{E_1} = -0.006t$ and $[\dot{\Omega}_4]_{E_1} = -0.0016t$, while $[\Omega_3]_{E_1}$ increase reaching the maximum value of 0.265 at $t = 10$ with rate $[\dot{\Omega}_3]_{E_1} = 0.001$. At $t = 10$, $[\Omega_2]_{E_1}$ reaches 0.285, while $[\Omega_4]_{E_1}$ reaches 0. In the time interval $10 < t \leq 25$, all three state adaptabilities remain constant $[\Omega_2]_{E_1} = 0.285$, $[\Omega_3]_{E_1} = 0.265$, $[\Omega_4]_{E_1} = 0$ since rates are 0.

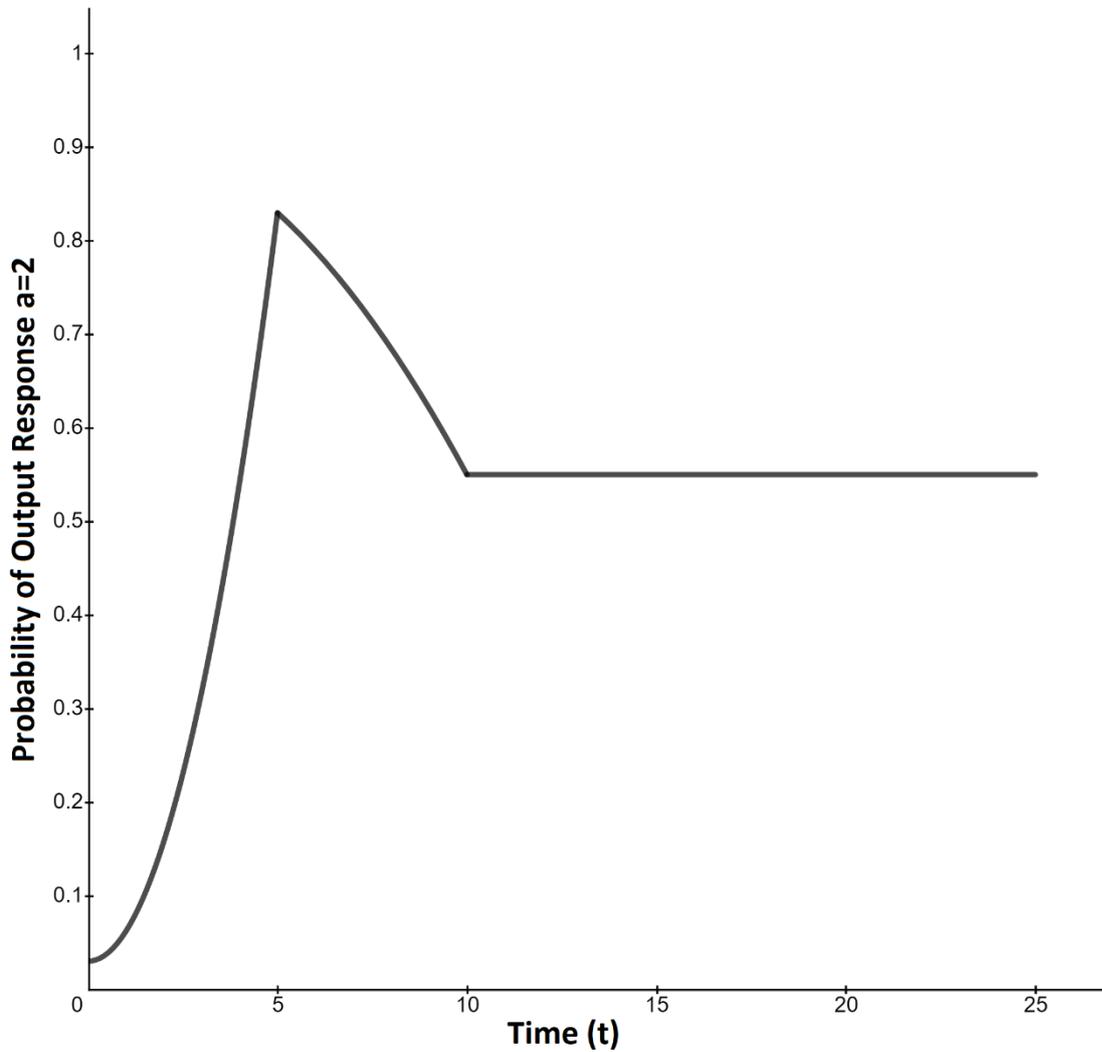


Figure 17. Evolution of Probability of Output Response $[\psi_{o_2}]_{E_1}$ in the time interval $0 < t \leq 25$. At time $t = 5$, $[\psi_{o_2}]_{E_1}$ reaches maximum value of 0.83 from 0.03 at time $t = 0$ with rate $[\dot{\psi}_{o_2}]_{E_1} = 0.064t$. In the time interval $5 < t \leq 10$, $[\psi_{o_2}]_{E_1}$ decreases with the rate $[\dot{\psi}_{o_2}]_{E_1} = -0.0076t + 0.001$ reaching $[\psi_{o_2}]_{E_1} = 0.55$ at $t = 10$. In the time interval $10 < t \leq 25$, $[\psi_{o_2}]_{E_1} = 0.55$ constant as the rate $[\dot{\psi}_{o_2}]_{E_1} = 0$.

6. X-Neuronal System

In this section, concepts of set theory are used to define a system with X neurons. Consider a set N which is given by equation 81.

$$N = \{R, I, E\} \quad (81)$$

The elements of N are three sets, R, I and E , where R represents Receptor neurons set, I interneurons sets and E effector neurons set. Furthermore R, I and E are defined by equation 82, 83 and 84, where $j = 1, 2, 3, \dots p$; $k = 1, 2, 3, \dots q$; $l = 1, 2, 3, \dots r$ and $x = 0, 1, 2, \dots y$.

$$R = \{R_{j_x}\} \quad (82)$$

$$I = \{I_{k_x}\} \quad (83)$$

$$E = \{E_{l_x}\} \quad (84)$$

j represents an individual receptor neuron and p are the total number of receptor neurons. Similarly, k represents an individual interneuron and q are the total number of interneurons. Lastly, l represents an individual effector neuron and r are the total number of effector neurons. Furthermore, x represents the state of an individual neuron, with $x = 0$ representing the rest state and $x > 0$ representing the activated states. The total number of unique states an individual neuron can be in is given by y . The total number of neurons X in the system is given by equation 85. Diagrammatically, the set N representing the X -Neuronal system is shown in Figure 18. Using equations 68 and 63, the total number of possible states and total unique output responses of X -neuronal system are given by equations 86 and 87, respectively.

$$X = p + q + r \quad (85)$$

$$[y]_X = \sum_{j=1}^p [y]_{R_j} + \sum_{k=1}^q [y]_{I_k} + \sum_{l=1}^r [y]_{E_l} \quad (86)$$

$$[b]_X = \sum_{j=1}^p [b]_{R_j} + \sum_{k=1}^q [b]_{I_k} + \sum_{l=1}^r [b]_{E_l} \quad (87)$$

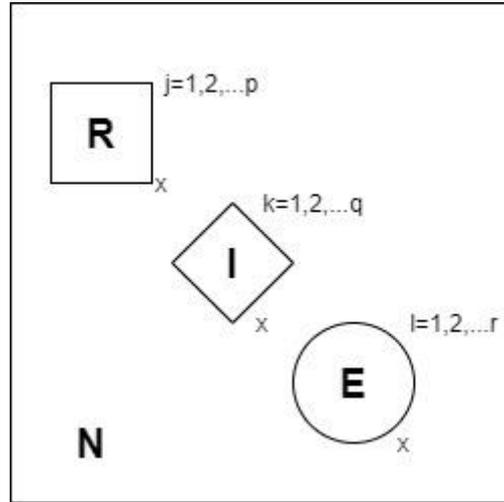


Figure 18. Set N comprising of three sets R, I and E , representing X -Neuronal System with $X = p + q + r$.

6.1. Directorial Board

In this section, a 4th component called ‘Directorial Board’ will be added to the previously proposed 3-component model. Let the Directorial board be defined by a set D , which is the subset of set N i.e. $D \subseteq \{N\}$. Directorial board represents a set of unique neurons from sets R, I and E . Furthermore, Directorial Board is further comprised of ‘Local Directorial Boards’ given by equation 88, where $h = 1, 2, 3, \dots, w$, representing an individual Local Directorial Board (LDB) and w are the total number of LDBs. Furthermore, $u = 0, 1, 2, \dots, v$ are the possible states of an individual LDB, where $u = 0$ represents the rest state and $u > 0$ represents the activated states; v are the total number of possible states D_h can be in.

$$D = \{D_{h_u}\} \quad (88)$$

Local Directorial Boards are the subset of set D i.e. $D_{h_u} \subseteq \{D\}$ and therefore also represents a set of neurons from sets R, I and E . The state of set D_h depends on the states of the neurons which D_h represents. As an example, consider D_1 which is defined by equation 89.

$$\mathbf{D}_{1_u} = \{R_{100_x}, I_{100_x}, E_{100_x}\} \quad (89)$$

Furthermore, assume the total number of possible states of each neuron is 100. i.e. $y = 100$. The state of \mathbf{D}_1 depends on the states of neurons R_{100} , I_{100} and E_{100} . If R_{100} , I_{100} and E_{100} are in the rest state i.e. $x = 0$, then \mathbf{D}_1 will be in the rest state i.e. $u = 0$. On the contrary, if any of the neurons R_{100} , I_{100} and E_{100} is in the activated state, \mathbf{D}_1 will be in the activated state with $u > 0$. The total number of possible states v of \mathbf{D}_h are given by the products of the total number of possible states of each neurons. The total number of possible activated states of \mathbf{D}_h are given by $v - 1$. For \mathbf{D}_1 , v is given by equation 90 and the total number of possible activated states of \mathbf{D}_1 is equal to 99999.

$$[v]_{\mathbf{D}_1} = 100 \cdot 100 \cdot 100 = 10^6 \quad (90)$$

6.1.1. Relationship between Local Directorial Boards

As mentioned previously, there are w number of total LDBs and each LDB is a subset of Directorial board i.e. $\mathbf{D}_{h_u} \subseteq \{\mathbf{D}\}$. The relationship between LDBs is rather more intimate and complex, such that one LDB can be a subset of another LDB. In addition, some LDBs can have neurons unique to them and others can have neurons which are common to both. In order to describe relationship between LDBs, consider 4 arbitrary LDBs (\mathbf{D}_{1_u} , \mathbf{D}_{2_u} , \mathbf{D}_{3_u} , \mathbf{D}_{4_u}) out of w , which are defined by equations 89, 91, 92 and 93, respectively.

$$\mathbf{D}_{2_u} = \{I_{100_x}, I_{101_x}, I_{102_x}, E_{100_x}, E_{101_x}\} \quad (91)$$

$$\mathbf{D}_{3_u} = \{R_{100_x}, I_{100_x}, I_{101_x}, I_{102_x}, I_{103_x}, E_{100_x}, E_{101_x}\} \quad (92)$$

$$\mathbf{D}_{4_u} = \{R_{200_x}, I_{200_x}, E_{200_x}\} \quad (93)$$

Neurons I_{100_x} and E_{100_x} belongs to two LDBs D_{1_u} and D_{2_u} i.e. $\{I_{100_x}, E_{100_x}\} = D_{1_u} \cap D_{2_u}$. Furthermore, $D_{1_u}, D_{2_u} \subseteq D_{3_u}$ so D_{3_u} contains all the neurons of D_{1_u} and D_{2_u} and neuron I_{103_x} i.e. $D_{3_u} = \{D_{1_u} \cup D_{2_u}, I_{103_x}\}$. Neurons R_{200_x}, I_{200_x} and E_{200_x} are unique to the LDB D_{4_u} . Representation of these 4 LDBs is shown in Figure 19.

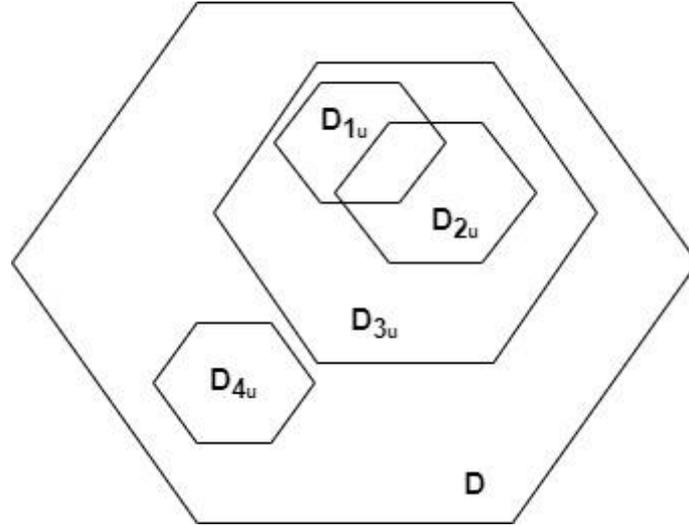


Figure 19. Representation of 4- arbitrary LDBs ($D_{1_u}, D_{2_u}, D_{3_u}, D_{4_u}$) which are subsets of Directorial Board D . $D_{1_u} \cap D_{2_u}$ represents neurons shared by both LDBs. $D_{1_u}, D_{2_u} \subseteq D_{3_u}$ and D_{4_u} is independent of the other three LDBs.

6.1.2. Communication between LDBs and Neuron

Consider an LDB D_{1_u} defined by equation 89 and an interneuron I_{1_x} , both in rest states i.e. $u = x = 0$. Furthermore, let D_{1_u} and I_{1_x} communicate by chemical synaptic communication and all neurons involved in communication achieve AP states. Communication between neurons of D_{1_u} and I_{1_x} using 3-component model is presented in figure 20. It should be noted that neurons of D_{1_u} are stimulated by arbitrary neuron/neurons, which are subset of set N . Let two neurons, I_{1000_x} and I_{1001_x} acts as presynaptic neurons for the neurons of D_{1_u} , and communicate chemically and electrically, as shown in figure 21. The two neurons I_{1000_x} and I_{1001_x} are further stimulated by some arbitrary neuron/neurons of set N and so on. However, for neuron I_{1_x} only the three neurons

of \mathbf{D}_{1_u} are the direct stimulators, so only \mathbf{D}_{1_u} will act as its LDB. It is mandatory for the neurons of LDB to have direct communication with a neuron or system of neurons to be called the system's LDB. To further elaborate this concept, consider \mathbf{D}_{1_u} again and three neurons I_{2_x} , I_{3_x} and E_{1_x} all in rest state i.e. $u = x = 0$. Furthermore, assume the neurons communicate chemically and electrically as shown in figure 22. As figure 22 shows, neurons I_{2_x} and I_{3_x} receives managers from R_{100_x} and I_{100_x} of \mathbf{D}_{1_u} , so for both I_{2_x} and I_{3_x} , the neurons R_{100_x} and I_{100_x} act as their LDB. It can be defined by a new set using equation 68. Likewise, for neurons E_{1_x} only E_{100_x} of \mathbf{D}_{1_u} directly stimulates it, so only neuron E_{100_x} acts as an LDB for E_{1_x} . Let LDB for neurons I_{2_x} and I_{3_x} be defined by \mathbf{D}_{100_u} and LDB for neuron E_{1_x} be defined by \mathbf{D}_{101_u} and are given by equation 94 and 95, respectively. For the three neuronal system (I_{2_x} , I_{3_x} and E_{1_x}), \mathbf{D}_{1_u} is the union of sets \mathbf{D}_{100_u} and \mathbf{D}_{101_u} i.e. $\mathbf{D}_{1_u} = \mathbf{D}_{100_u} \cup \mathbf{D}_{101_u}$. Individually each neuron in a system can have its own LDB, but for the whole system, the LDB is the union of LDBs of individual neurons.

$$\mathbf{D}_{100_u} = \{R_{100_x}, I_{100_x}\} \quad (94)$$

$$\mathbf{D}_{101_u} = \{E_{100_x}\} \quad (95)$$

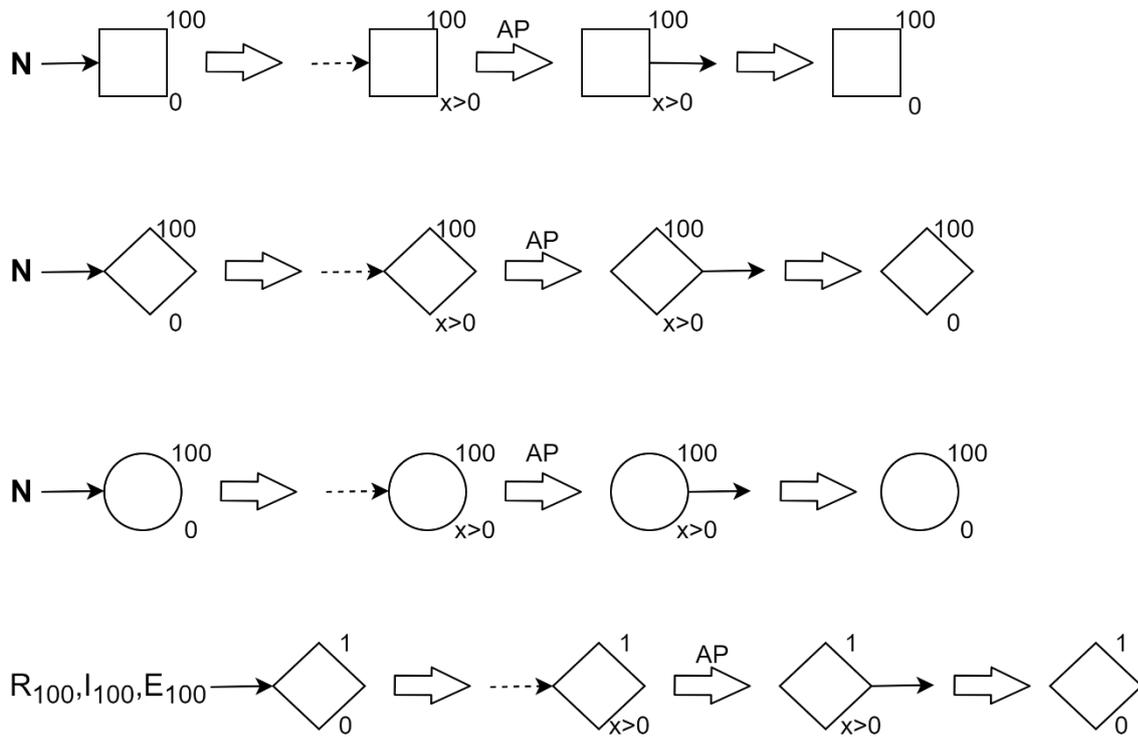


Figure 20. Communication between LDB \mathbf{D}_{1_u} and an interneuron I_{1_x} . LDB \mathbf{D}_{1_u} comprises of 3 neurons $R_{100_x}, I_{100_x}, E_{100_x}$ initially all in rest state. Neurons of \mathbf{D}_{1_u} receives managers from some arbitrary neuron or neurons, which are the subset of set \mathbf{N} (equation 81). As a result, specialised workers start flowing in the neurons, causing APs and managers are released by them as an output response. Neurons of \mathbf{D}_{1_u} returns back to their rest states $u = 0$. The managers from neurons $R_{100_x}, I_{100_x}, E_{100_x}$ of \mathbf{D}_{1_u} are received by neuron I_{1_x} , resulting in the movement of specialised workers inside I_{1_x} . Neuro I_{1_x} achieves an AP state and release managers as an output response and then returns to its rest state $x = 0$.

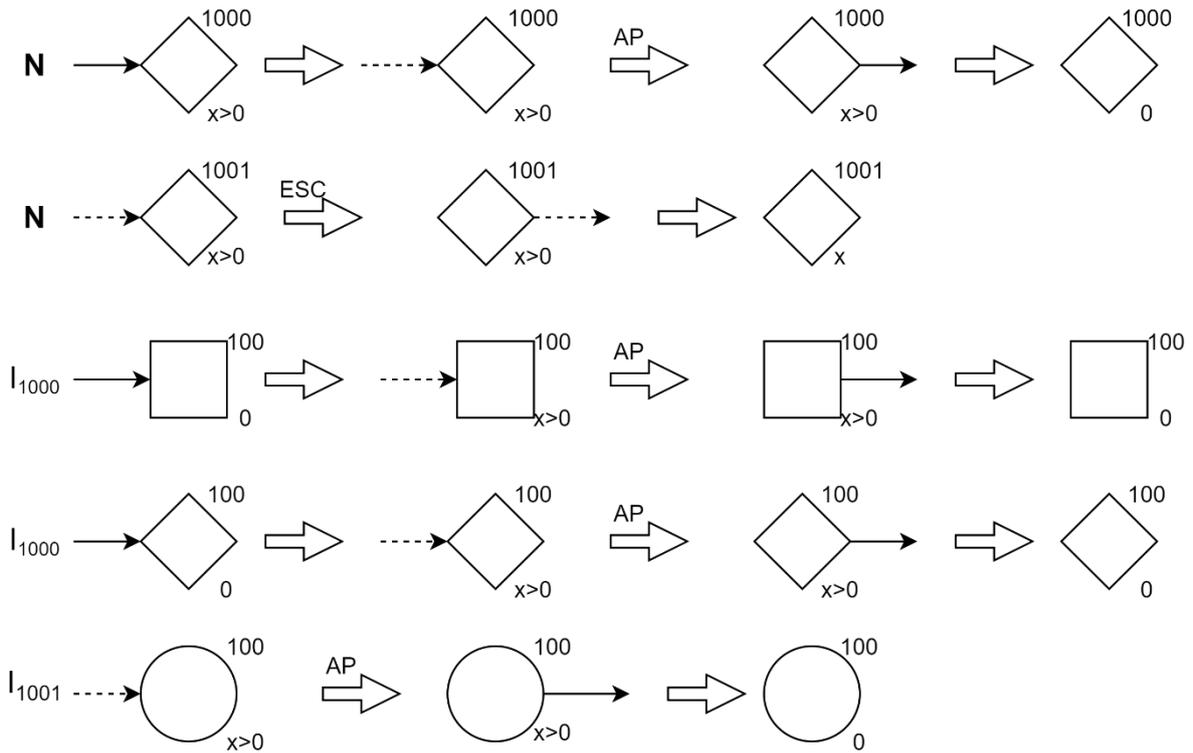


Figure 21. Communication between neurons I_{1000x} and I_{1001x} and D_{1u} , using 3-component model. They are stimulated by some arbitrary neuron/neurons of set N . Initially, neuron I_{1000x} is in activated subthreshold state and communicate chemically with arbitrary neuron/neurons of set N . Neuron I_{1000x} undergo AP to release managers which are received by neurons R_{100x}, I_{100x} . Both neurons R_{100x}, I_{100x} undergo AP releasing managers which are received by the interneuron I_{1x} shown in figure 20. Neuron I_{1001x} communicates electrically with arbitrary neurons of set N , thus release managers in the form of ions or workers, which are received by E_{100x} of D_{1u} . The specialised workers by I_{1001x} cause E_{100x} to undergo AP releasing managers which are received by interneuron I_{1x} (figure 20).

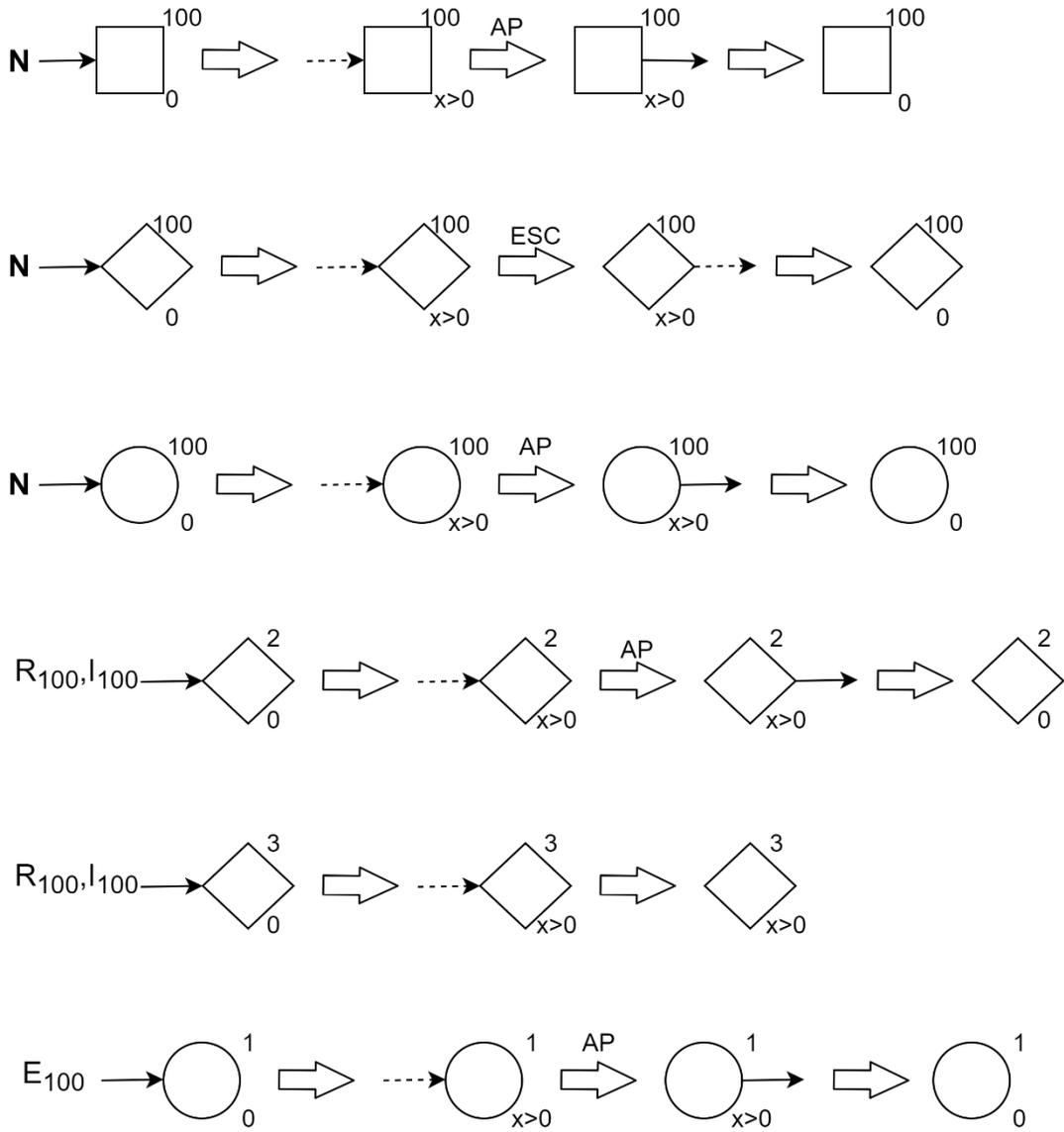


Figure 22. Communication between D_{1u} and neurons I_{2x} , I_{3x} and E_{1x} using 3-component model. Initially all neurons of D_{1u} are in rest state and are stimulated by neurons of set N . As a result, neurons R_{100x} , E_{100x} undergo AP release managers in the form of neurotransmitters and neuron I_{100x} release managers in the form of workers or ions. Neurons I_{2x} and I_{3x} receives managers from R_{100x} and I_{100x} of D_{1u} , while neuron E_{1x} only receives managers from E_{100x} of D_{1u} . Both I_{2x} and I_{3x} communicate chemically with R_{100x} and electrically with I_{100x} . This communication causes I_{2x} to undergo AP and release managers, while neuron I_{3x} only attains a subthreshold potential state and remains in that state, until stimulated further. Neurons E_{100x} and E_{1x} communicate chemically with E_{1x} undergoing AP to release managers and returning to rest state.

6.1.3. Function of Local Directorial Board

Now that the workings of LDBs has been well established, the focus will now be diverted to their functions. To establish the functionality of LDBs, 3 cases will be discussed. Consider two neuronal system (R_{1_x} and I_{1_x}) communicating chemically and let R_{1_x} be the presynaptic neuron for I_{1_x} . Furthermore, let the neuron R_1 is stimulating by a stimulus $[S]_{R_1}$. For this discussion, indirect stimuli by drifting neurotransmitters is assumed to be 0.

Case 1

In case 1, R_1 attains an AP state $[x_{AP_1}]_{R_1}$ by stimulus $[S]_{R_1}$ and consequently an output response $[O_{C_1}]_{R_1}$ i.e. $[S]_{R_1} \rightarrow [x_{AP_1}]_{R_1} \rightarrow [O_{C_1}]_{R_1}$. The output response $[O_{C_1}]_{R_1}$ of R_1 is the stimulus for postsynaptic neuron I_1 . Based on the contribution of stimulus $[O_{C_1}]_{R_1}$, neuron I_1 attains an activated state. Let this activated state be an AP state given by $[x_{AP_1}]_{I_1}$ and consequently I_1 generates an output response. Let the output response generated by I_1 is given by $[O_{C_1}]_{I_1}$. The communication of this two-neuronal system can be modelled using 3-Component Model. For this two-neuronal system, the output response generated by the stimulus $[S]_{R_1}$ is given by $[O_{C_1}]_{I_1}$ i.e. $[S]_{R_1} \rightarrow [x_{AP_1}]_{R_1} \rightarrow [O_{C_1}]_{R_1} \rightarrow [x_{AP_1}]_{I_1} \rightarrow [O_{C_1}]_{I_1}$.

Now consider LDB D_{1_u} which communicates with neuron I_{1_x} , as shown in figure 20. Let the stimulation of I_1 by D_{1_u} causes I_1 to attain an AP state $[x_{AP_2}]_{I_1}$ and consequently an output response $[O_{C_2}]_{I_1}$. If one consider two-neuronal system along with the LDB D_{1_u} , where stimuli of R_1 and D_{1_u} reaches I_{1_x} simultaneously. The activation of neuron I_{1_x} will depend on the contributions of direct stimuli R_1 and D_{1_u} , since indirect stimuli is assumed to be 0. As equation 89 shows, D_{1_u} comprises of 3 neurons ($R_{100_x}, I_{100_x}, E_{100_x}$). Let the output responses of these neurons which stimulate I_{1_x} be given by $([O_{C_1}]_{R_{100}}, [O_{C_1}]_{I_{100}}, [O_{C_1}]_{E_{100}})$. The stimuli for neuron I_1 is given by $[S]_{I_1} = 4$, which $[S]_{I_1}$ is shown in equation 96.

$$[S]_{I_1} = \{[O_{C_1}]_{R_1}, [O_{C_1}]_{R_{100}}, [O_{C_1}]_{I_{100}}, [O_{C_1}]_{E_{100}}\} \quad 96$$

The activation of neuron I_1 depends on the sum of the contributions of each stimulus of $[S]_{I_1}$. Let the activated state of I_1 by stimuli $[S]_{I_1}$ be an AP state $[x_{AP_3}]_{I_1}$ resulting in an output response $[O_{C_3}]_{I_1}$. If one considers both R_1 and D_{1u} , the output response for stimulus $[S]_{R_1}$ is $[O_{C_3}]_{I_1}$ instead of $[O_{C_1}]_{I_1}$. The function of LDB D_{1u} in this case is to provide additional stimuli to neuron I_1 which in turn contributes in the activation of neuron I_1 and as a result altering the output response of stimulus $[S]_{R_1}$.

Case 2

In case 2, the output response of neuron I_1 by presynaptic neuron R_1 alone is same as in case 1 i.e. $[S]_{R_1} \rightarrow [x_{AP_1}]_{R_1} \rightarrow [O_{C_1}]_{R_1} \rightarrow [x_{AP_1}]_{I_1} \rightarrow [O_{C_1}]_{I_1}$. The contributions of stimuli of D_{1u} cause I_1 to attain a subthreshold potential state given by $[x_{STP_1}]_{I_1}$. Consider that neuron I_1 is stimulated by the stimuli of both R_1 and D_{1u} . Based on the contributions of each stimulus, it is possible that neuron I_1 attains a subthreshold potential state. Assume this STP state by contributions of R_1 and D_{1u} stimuli is given by $[x_{STP_1}]_{I_1}$. If the eventual state of I_1 is $[x_{STP_1}]_{I_1}$, neuron I_1 will not generate an output response. In this case, no output response of stimulus $[S]_{R_1}$ will be generated and neuron I_1 will remain in an activated state $[x_{STP_1}]_{I_1}$, until it receives further stimuli. So, in this case, the function of LDB D_{1u} was to repolarise the membrane potential of I_1 , causing it to remain in subthreshold values. A stimulus $[S]_{R_1}$ which was supposed to produce an output response $[O_{C_1}]_{I_1}$ will not produce any response, because of the inference of the local directorial board D_{1u} .

Case 3

In this case, the contribution of stimulus of presynaptic neuron R_1 causes I_1 to attain a subthreshold potential state $[x_{STP_3}]_{I_1}$. Furthermore, the contribution of stimuli of D_{1u} causes I_1 to attain an AP state $[x_{AP_3}]_{I_1}$ resulting in an output response $[O_{C_3}]_{I_1}$ as in case 1. Consider the contributions of R_1 and D_{1u} stimuli in activation of I_1 , simultaneously. Neuron I_1 attains an AP state given by $[x_{AP_4}]_{I_1}$ resulting in an output response $[O_{C_4}]_{I_1}$. In this case, stimulus $[S]_{R_1}$, which was only supposed to activate neuron I_1 into STP state $[x_{STP_3}]_{I_1}$ will produce an output response $[O_{C_4}]_{I_1}$, because of the interference of LDB D_{1u} .

6.1.4. Timing of Stimuli of LDB

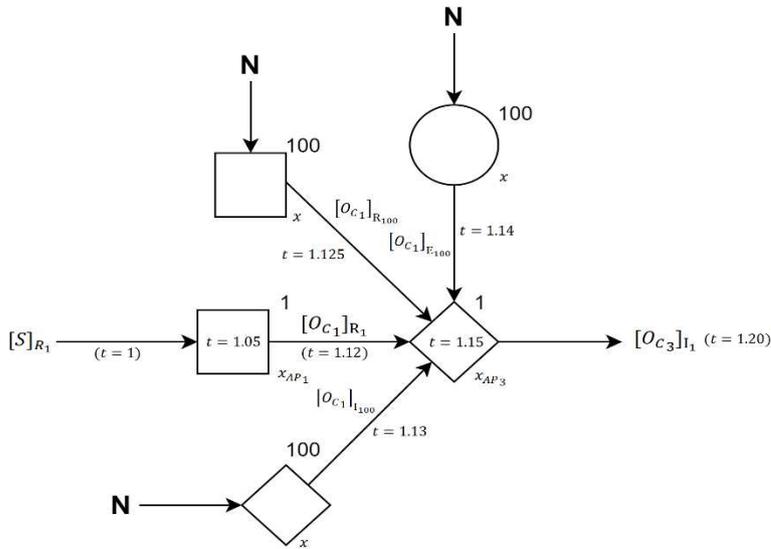
It should be noted that the timings of stimuli by LDBs is of utmost importance. To elaborate this point further, consider neuron I_1 which receives stimulus $[O_{C_1}]_{R_1}$ from presynaptic neuron R_1 at time $t = 0$ and attains an AP state at time $t = 0.05$. Let the time interval from time neuron I_1 receives stimulus $[O_{C_1}]_{R_1}$ to the time it attains an AP state be denoted by $[T]_{I_1}$. For neuron I_1 , $[T]_{I_1}$ is given by $0 \leq [T]_{I_1} \leq 0.05$. If the stimuli of LDB D_{1u} arrives at neuron I_1 in time interval $[T]_{I_1}$, D_{1u} will contribute in the eventual activation state of I_1 . However, if stimuli of D_{1u} arrives at I_1 beyond time interval $[T]_{I_1}$, D_{1u} will not contribute in the activation of I_1 and only the contribution of stimulus $[O_{C_1}]_{R_1}$ will govern the activation of I_1 .

6.2. Example Case of Two-Neuronal System and LDB

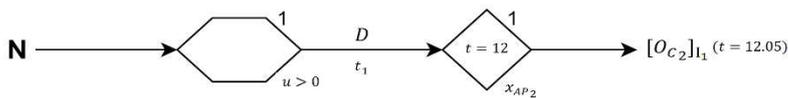
In this section, an example case of two-neuronal system (R_{1_x} and I_{1_x}) and LDB \mathbf{D}_{1_u} (equation 89) of I_{1_x} with respect to time will be presented. Consider at time ($t = 0$), all neurons are in rest state i.e. $x = u = 0$. Furthermore, in this example, all involved neurons communicate chemically. At time $t = 1$, presynaptic neuron R_{1_0} is stimulated by a stimulus $[S]_{R_1}$ resulting in R_1 attaining an AP state $[x_{AP_1}]_{R_1}$ at time $t = 1.05$. As a consequence, R_1 produce an output response $[O_{C_1}]_{R_1}$ at $t = 1.10$ i.e. $[S]_{R_1}(t = 1) \rightarrow [x_{AP_1}]_{R_1}(t = 1.05) \rightarrow [O_{C_1}]_{R_1}(t = 1.10)$. The output response $[O_{C_1}]_{R_1}$ stimulates postsynaptic neuron I_{1_0} at time $t = 1.12$. Likewise, the stimuli from neurons ($R_{100}, I_{100}, E_{100}$) of LDB \mathbf{D}_{1_u} given by $([O_{C_1}]_{R_{100}}, [O_{C_1}]_{I_{100}}, [O_{C_1}]_{E_{100}})$ reach I_1 at times ($t = 1.125, 1.13, 1.14$), respectively. Based on the contributions of stimuli of R_{1_x} and \mathbf{D}_{1_u} , neuron I_1 attains an AP state $[x_{AP_3}]_{I_1}$ at time $t = 1.15$, which results in an output response $[O_{C_3}]_{I_1}$ at $t = 1.2$. Neuron I_1 returns to its rest state at time $t = 1.23$. The time for stimulus $[S]_{R_1}$ to produce an output response is given by $t = 0.2$. The generation of output response is shown in Figure 23A. Now, consider at time $t = 4$ and $t = 7$, neuron R_{1_0} is stimulated by a stimulus $[S]_{R_1}$, resulting in neuron I_1 attaining an AP state $[x_{AP_3}]_{I_1}$, at time $t = 4.15$ and $t = 7.15$, from contributions of stimuli of R_{1_x} and \mathbf{D}_{1_u} . As a consequence, I_1 produce an output response $[O_{C_3}]_{I_1}$ at time $t = 4.20$ and $t = 7.20$ and returning to rest state at time $t = 4.23$ and $t = 7.23$. In the time intervals, $1.23 \leq t \leq 4.12$ and $4.23 \leq t \leq 7.12$, neuron I_1 will remain in the rest state. Furthermore, beyond time $t \geq 7.23$, will remain in rest state, until it is stimulated further. So far in the time interval $0 \leq t \leq 7.20$, neuron I_1 has produced output response $[O_{C_3}]_{I_1}$ three times by the stimulus $[S]_{R_1}$. In other words, the generation of output response $[O_{C_3}]_{I_1}$ shown in figure 23A is repeated 3 times.

Now consider only LDB \mathbf{D}_{1u} stimulates the neuron I_{10} . The output responses ($[O_{C1}]_{R_{100}}, [O_{C1}]_{I_{100}}, [O_{C1}]_{E_{100}}$) of \mathbf{D}_{1u} neurons reach I_1 at times ($t = 11.975, 11.98, 11.99$) resulting in I_1 attaining an AP state $[x_{AP2}]_{I_1}$ at time $t = 12$. As a consequence, I_1 produce an output response $[O_{C2}]_{I_1}$ at time $t = 12.05$ and returning to rest state at time $t = 12.08$. The generation of output response $[O_{C2}]_{I_1}$ by the LDB \mathbf{D}_{1u} alone is shown in figure 23B. Furthermore, the generation of output response $[O_{C2}]_{I_1}$ by the LDB \mathbf{D}_{1u} shown in figure 23B is repeated 3 times, where I_1 attains AP state $[x_{AP2}]_{I_1}$ at time $t = (14, 16, 18)$, produce output response $[O_{C2}]_{I_1}$ at time $t = (14.05, 16.05, 18.05)$ and return to rest state at time $t = (14.08, 16.08, 18.08)$. In the time interval, $0 \leq t \leq 18.05$, neuron I_1 has behaved ideally (equation 69) with $\left[\frac{\beta}{\alpha}\right]_{I_1} = \frac{2}{2} = 1$. Furthermore, I_1 generated 7 output responses; 3 $[O_{C3}]_{I_1}$ responses and 4 $[O_{C2}]_{I_1}$ responses.

A



B



$$D = ([O_{C_1}]_{R_{100}}, [O_{C_1}]_{I_{100}}, [O_{C_1}]_{E_{100}})$$

$$t_1 = (t = 11.975, t = 11.98, t = 11.99)$$

Figure 23. Generation of output responses of neuron I_1 .

A) Generation of output response $[O_{C_3}]_{I_1}$ at time $t = 1.20$ by the stimulus $[S]_{R_1}$.

B) Generation of the output response $[O_{C_2}]_{I_1}$ at time $t = 12.05$ by LDB D_{1_u} alone. Local

Directorial board D_{1_u} is represented by a Hexagon, with appropriate name and state.

Moving forward, the LDBs will be represented by the hexagon.

6.2.1. Evolution of $[\Omega_x]_{I_1}$ and $[\psi_{O_a}]_{I_1}$ with respect to time

Now that the working of two neuron system and LDB has been established in the time interval $0 \leq t \leq 18.05$, the focus will now be diverted to the state adaptabilities $[\Omega_x]_{I_1}$ and probabilities of output responses $[\psi_{O_a}]_{I_1}$ of I_1 in time interval $0 \leq t \leq 18.05$. To establish the significances of $[\Omega_x]_{I_1}$ and $[\psi_{O_a}]_{I_1}$ in the broader scheme of things, neuron I_1 will be treated as a ‘Model Neuron’ with the following properties.

- $[y]_{I_1} = 6$ (Equation 68); With $[K]_{I_1} = 2$, $[M]_{I_1} = 3$, $[Q]_{I_1} = 0$.
- $[b]_{I_1} = 3$ (Equation 63); With $[V]_{I_1} = 3$, $[Z]_{I_1} = 0$.
- I_1 is an ideal neuron in any time interval i.e. $\left[\frac{\beta}{\alpha}\right]_{I_1} = 1$

Assume that in the time interval $0 \leq t \leq 1.15$, state adaptabilities of all activated states of I_1 are equally probable i.e. $[\Omega_x]_{I_1} = \frac{1}{[K]_{I_1} + [M]_{I_1}} = 0.2$. The two state adaptabilities of importance at this point are the state adaptabilities of AP states $[x_{AP_3}]_{I_1}$ and $[x_{AP_2}]_{I_1}$. Assume that every time, neuron I_1 attains AP state $[x_{AP_3}]_{I_1}$, the state adaptability $[\Omega_{x_{AP_3}}]_{I_1}$ increase 0.15 and simultaneously $[\Omega_{x_{AP_2}}]_{I_1}$ decrease 0.05. Furthermore, every time neuron I_1 attains AP state $[x_{AP_2}]_{I_1}$, the state adaptability $[\Omega_{x_{AP_2}}]_{I_1}$ increase 0.2 and simultaneously $[\Omega_{x_{AP_3}}]_{I_1}$ decrease 0.16. Based on these assumptions, the evolution of state adaptabilities $[\Omega_{x_{AP_3}}]_{I_1}$ and $[\Omega_{x_{AP_2}}]_{I_1}$ in the time interval, $0 \leq t \leq 18.05$ is shown in Figure 24. Furthermore, since neuron I_1 is assumed to be an ideal neuron, the probabilities of output responses $[\psi_{O_{C_3}}]_{I_1}$ and $[\psi_{O_{C_2}}]_{I_1}$ are equal to the state adaptabilities $[\Omega_{x_{AP_3}}]_{I_1}$ and $[\Omega_{x_{AP_2}}]_{I_1}$ respectively (equation 72).

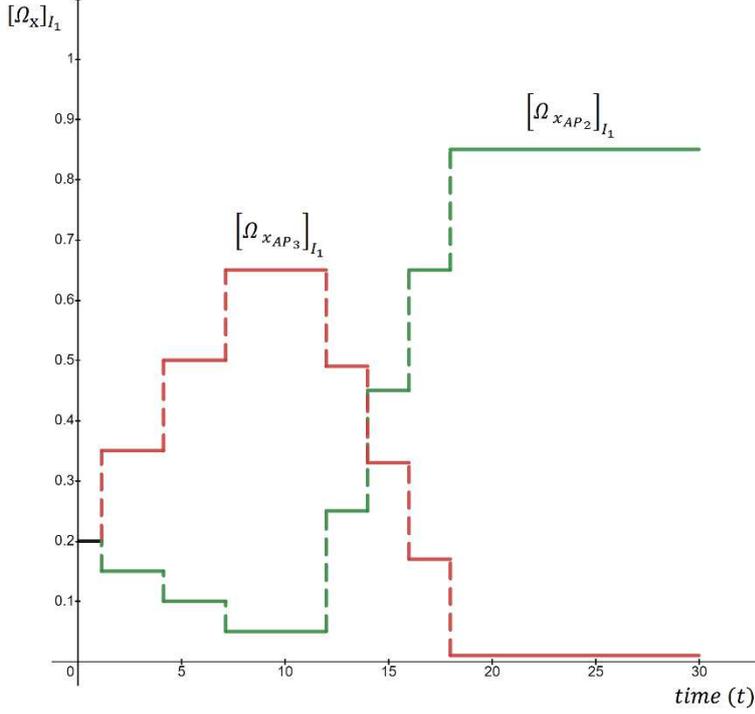


Figure 24. Evolution of state adaptabilities $[\Omega_{x_{AP_3}}]_{I_1}$ and $[\Omega_{x_{AP_2}}]_{I_1}$ of neuron I_1 with respect to time. Before $t = 1.15$, both $[\Omega_{x_{AP_3}}]_{I_1} = 0.2$ and $[\Omega_{x_{AP_2}}]_{I_1} = 0.2$. At times $t = (1.15, 4.15, 7.15)$, neuron I_1 attains $[x_{AP_3}]_{I_1}$ causing $[\Omega_{x_{AP_3}}]_{I_1}$ to increase to $[\Omega_{x_{AP_3}}]_{I_1} = (0.35, 0.5, 0.65)$ and simultaneously, causing $[\Omega_{x_{AP_2}}]_{I_1}$ to decrease to $[\Omega_{x_{AP_2}}]_{I_1} = (0.15, 0.1, 0.05)$, respectively. Similarly, at times $t = (12, 14, 16, 18)$, neuron I_1 attains $[x_{AP_2}]_{I_1}$ causing $[\Omega_{x_{AP_2}}]_{I_1}$ to increase to $[\Omega_{x_{AP_2}}]_{I_1} = (0.25, 0.45, 0.65, 0.85)$ and simultaneously, causing $[\Omega_{x_{AP_3}}]_{I_1}$ to decrease to $[\Omega_{x_{AP_3}}]_{I_1} = (0.49, 0.33, 0.17, 0.01)$, respectively.

6.2.2. Significance of $[\Omega_x]_{I_1}$ and $[\psi_{o_a}]_{I_1}$

In the time interval $1.15 \leq t \leq 7.20$, the state adaptability $[\Omega_{x_{AP_3}}]_{I_1}$ and probability of output response $[\psi_{O_{C_3}}]_{I_1}$ increases reaching at 0.65, as neuron I_1 keeps attaining AP state $[x_{AP_3}]_{I_1}$ resulting in output response $[O_{C_3}]_{I_1}$. This signifies that neuron I_1 starts adapting itself as soon as it attains the state $[x_{AP_3}]_{I_1}$ at time $t = 1.15$, and keeps adapting itself functionally as it keeps attaining $[x_{AP_3}]_{I_1}$, increasing the likelihood of attaining $[x_{AP_3}]_{I_1}$ and producing $[O_{C_3}]_{I_1}$ beyond the time interval $1.15 \leq t \leq 7.20$. However, for neuron to keep adapting itself to attain

state $[x_{AP_3}]_{I_1}$, it has to keep receiving stimuli ($[O_{C_1}]_{R_1}, [O_{C_1}]_{R_{100}}, [O_{C_1}]_{I_{100}}, [O_{C_1}]_{E_{100}}$) from neurons ($R_1, R_{100}, I_{100}, E_{100}$), respectively. This in turn means that neurons ($R_1, R_{100}, I_{100}, E_{100}$) have to keep producing outputs ($[O_{C_1}]_{R_{100}}, [O_{C_1}]_{I_{100}}, [O_{C_1}]_{E_{100}}$) by attaining specific AP states. In order for these neurons to keep attaining specific AP states for output responses ($[O_{C_1}]_{R_{100}}, [O_{C_1}]_{I_{100}}, [O_{C_1}]_{E_{100}}$), they need to adapt themselves to increase the state adaptabilities of those AP states and as a consequence, the probabilities of output responses ($[\psi_{O_{C_1}}]_{R_1}, [\psi_{O_{C_1}}]_{R_{100}}, [\psi_{O_{C_1}}]_{I_{100}}, [\psi_{O_{C_1}}]_{E_{100}}$).

However, as the likelihood of state $[x_{AP_3}]_{I_1}$ increased to 0.65, neuron I_1 received stimuli from LDB \mathbf{D}_{1_u} alone, resulting in attaining $[x_{AP_2}]_{I_1}$ state instead of $[x_{AP_3}]_{I_1}$ at time $t = 12$. This halts the increase in $[\Omega_{x_{AP_3}}]_{I_1}$, and neuron I_1 starts adapting itself to attain state $[x_{AP_2}]_{I_1}$ and produce output response $[O_{C_2}]_{I_1}$. As neuron I_1 keeps attaining $[x_{AP_2}]_{I_1}$ in the time interval $12 \leq t \leq 18.05$ by stimulation from \mathbf{D}_{1_u} alone, the state adaptability $[\Omega_{x_{AP_2}}]_{I_1}$ keeps increasing and reaching 0.85. Neurons of LDB \mathbf{D}_{1_u} , keep adapting themselves to provide the desire environment needed by neuron I_1 to increase the likelihood of $[x_{AP_2}]_{I_1}$ state and $[O_{C_2}]_{I_1}$ response. As discussed previously, the process of neurons adapting themselves depend on the rate of state adaptability which is governed by state adaptability factors $[\eta_x]_{I_1}$. Depending on these state adaptability factors, some neurons adapt faster while others adapt slower.

6.3. Workings of Two-Neuronal System (I_{1x} and E_{5x})

Consider a Model Effector Neuron E_{5x} with the following properties.

- $[y]_{E_5} = 3$ (Equation 68); With $[K]_{E_5} = 0$, $[M]_{E_5} = 2$, $[Q]_{E_5} = 0$.
- $[b]_{E_5} = 1$ (Equation 63); With $[V]_{E_5} = 1$, $[Z]_{E_5} = 0$.
- E_5 is a real neuron in the time interval $0 \leq t \leq \infty$ i.e. $\left[\frac{\beta}{\alpha}\right]_{E_5} = 0.5$.

Now consider that the output response $[O_{C_3}]_{I_1}$ generated by neuron I_1 at time $t = 1.20$ reaches neuron E_5 at $t = 1.23$, causing E_5 to attain an AP state $[x_{AP_1}]_{E_5}$ at $t = 1.25$. As a result, E_5 produces an output response $[O_{C_1}]_{E_5}$ at $t = 1.30$ i.e. $[O_{C_3}]_{I_1}(t = 1.23) \rightarrow [x_{AP_1}]_{E_5}(t = 1.25) \rightarrow [O_{C_1}]_{E_5}(t = 1.30)$. Similarly, neuron E_5 also generate output response $[O_{C_1}]_{E_5}$ at $t = 4.30$ and $t = 7.30$ after receiving stimulus $[O_{C_3}]_{I_1}$ from presynaptic neuron I_1 . At time $t = 12.08$, E_5 receives stimulus $[O_{C_2}]_{I_1}$ from I_1 , causing E_5 to attain an AP state $[x_{AP_2}]_{E_5}$ at $t = 12.10$. As a result, E_5 produces an output response $[O_{C_1}]_{E_5}$ at $t = 12.15$ i.e. $[O_{C_2}]_{I_1}(t = 12.08) \rightarrow [x_{AP_2}]_{E_5}(t = 12.10) \rightarrow [O_{C_1}]_{E_5}(t = 12.15)$. It should be noted that both AP states $[x_{AP_1}]_{E_5}$ and $[x_{AP_2}]_{E_5}$ produce the same output response $[O_{C_1}]_{E_5}$, since $[b]_{E_5} = 1$ for real model neuron E_5 . Furthermore, E_5 will again produce output response $[O_{C_1}]_{E_5}$ at $(t = 14.15, 16.15, 18.15)$. So, in the time interval $0 \leq t \leq 18.15$, E_5 will produce output response $[O_{C_1}]_{E_5}$ seven times.

Now consider the 3-neuronal system (R_{1x} , I_{1x} and E_{5x}) and LDB D_{1u} of I_{1x} . The generation of output response by the stimulus $[S]_{R_1}$ is $[O_{C_1}]_{E_5}$ produced by effector neuron E_5 as shown in Figure 25A.

If the neuron I_1 is stimulated by D_{1u} alone, the output response produced by E_5 will still be $[O_{C_1}]_{E_5}$ as it was for the stimulus $[S]_{R_1}$. The generation of output response when neuron I_1

is stimulated by D_{1u} alone is shown in Figure 25B. The generation of same output response, as shown in Figure 25 is of utmost importance. It signifies that once a stimulus produces an output response at any instance of time, using a set of neurons. It is possible to produce the same output response at a later instance of time, using another set of neurons, without the need of stimulus. For this example case, stimulus $[S]_{R_1}$ used set $\{R_{1x}, I_{1x}, E_{5x}, D_{1u}\}$ to produce output response $[O_{C_1}]_{E_5}$ at times $(t = 1.30, 4.30, 7.30)$. However, the same output response $[O_{C_1}]_{E_5}$ was produced at times $(t = 12.15, 14.15, 16.15, 18.15)$ using set $\{I_{1x}, E_{5x}, D_{1u}\}$, without the need of $[S]_{R_1}$ and R_{1x} . This concept will be immensely crucial in the discussions of later sections.

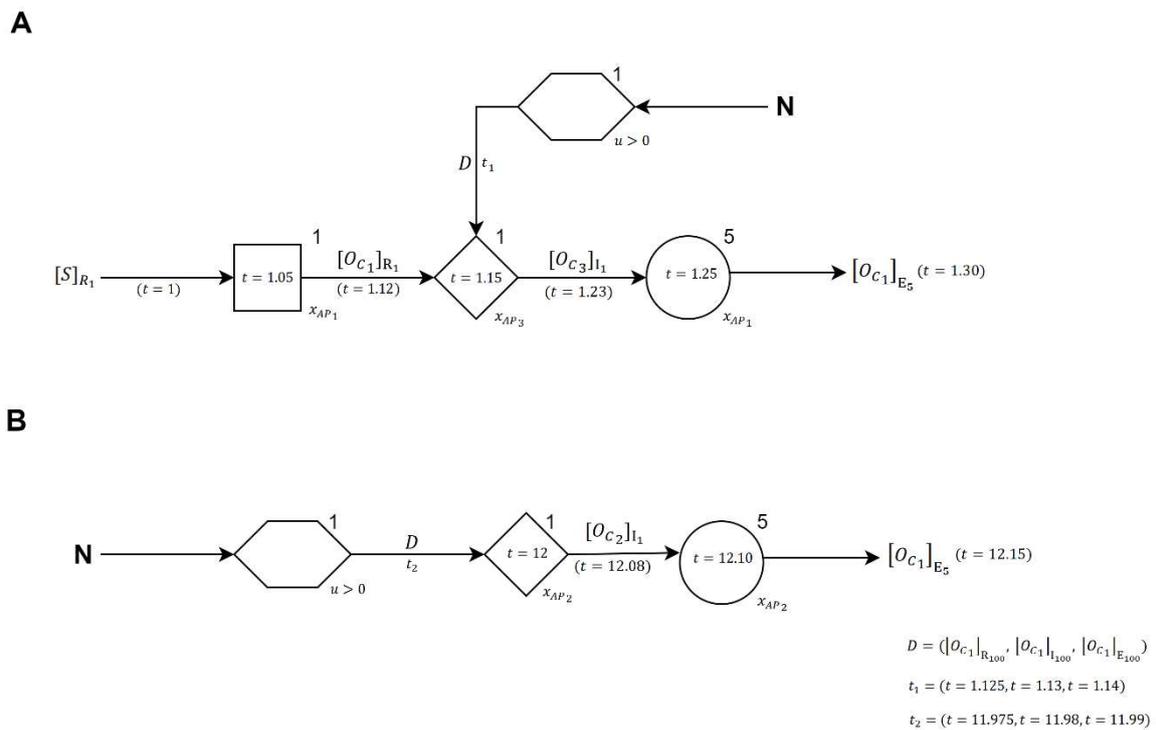


Figure 25. Generation of output response $[O_{C_1}]_{E_5}$.

- A) Generation of $[O_{C_1}]_{E_5}$ by the stimulus $[S]_{R_1}$ at time $t = 1.30$, using neurons $\{R_{1x}, I_{1x}, E_{5x}, D_{1u}\}$.
- B) Generation of $[O_{C_1}]_{E_5}$ at time $t = 12.15$ by neurons $\{I_{1x}, E_{5x}, D_{1u}\}$, where neuron I_1 was stimulated by LDB D_{1u} .

PART 2

Now that all the mandatory elements required to study cognitive processes have been well established. In part 2, those elements will be used to understand the architecture of cognitive processes and in later sections, it will be shown how every field of cognitive research (Attention, Memory, Learning, Imagination, Sleep and Dreams) emerges from the elements discussed in part 1.

7. Architecture of Cognition

First and foremost, the architecture of cognition of X -Neuronal system will be discussed, using certain aspects of set theory. At this point, it becomes a necessity that one properly define stimuli and classify different categories of stimuli for an X -Neuronal system. To serve this purpose, consider stimulus S_c , where $c = 1, 2, 3, \dots, d$; c represents a unique stimulus and d are the total number of possible stimuli an X -Neuronal system can receive. For the purposes of this article, the stimuli will be classified into two categories; *External Stimuli* and *Internal Stimuli*. External Stimuli are stimuli from the environment outside the organism of X -Neuronal system, e.g. light, sound, heat. On the contrary, internal stimuli are stimuli from within the environment of organism of X -Neuronal system. To cater both these categories of stimuli, consider a Stimuli Set \mathbf{S} defined by equation 97.

$$\mathbf{S} = \{\mathbf{S}_{external} \cup \mathbf{S}_{internal}\} \quad 97$$

Stimuli Set \mathbf{S} is the union of external stimuli set $\mathbf{S}_{external}$ and internal stimuli set $\mathbf{S}_{internal}$. Sets $\mathbf{S}_{external}$ and $\mathbf{S}_{internal}$ are further defined by equations 98 and 99, respectively, where G represents a unique external stimulus and H are the total number of possible external stimuli. Likewise, A represents a unique internal stimulus and B are the total number of possible

internal stimuli. The total number of possible stimuli d for X -Neuronal system are given by equation 100.

$$\mathbf{S}_{external} = \{S_{ext_G}\}; G = 1,2,3, \dots H \quad 98$$

$$\mathbf{S}_{internal} = \{S_{int_A}\}; A = 1,2,3, \dots B \quad 99$$

$$d = H + B \quad 100$$

Now that the stimuli are defined and categorized, consider an arbitrary stimulus S_c which stimulates X -Neuronal system at an arbitrary time t , causing the system to produce a stimulus response. Let the stimulus response produced by X -Neuronal system as a result of stimulus S_c is defined by $[\mathcal{R}]_{S_c}$. X -Neuronal system is defined by a set \mathbf{N} (equation 81). In general, X -Neuronal system will only use some of the elements of set \mathbf{N} to produce a stimulus response $[\mathcal{R}]_{S_c}$. Let the elements of set \mathbf{N} used by X -Neuronal System to produce $[\mathcal{R}]_{S_c}$ be defined by a set $[\mathbf{N}]_{S_c}$, where $[\mathbf{N}]_{S_c}$ is a subset of set \mathbf{N} i.e. $[\mathbf{N}]_{S_c} \subseteq \mathbf{N}$. As an example, consider the example case shown in figure 25A. Set $[\mathbf{N}]_{S_c}$ i.e. neurons used by X -Neuronal System while responding to stimulus $[S]_{R_1}$ is given by $[\mathbf{N}]_{[S]_{R_1}} = \{R_{1_x}, I_{1_x}, E_{5_x}\}$. It is imperative that one properly define Stimulus Response $[\mathcal{R}]_{S_c}$ to keep everything in perspective. For this article, $[\mathcal{R}]_{S_c}$ is defined as follows:

$[\mathcal{R}]_{S_c}$ is a set; the elements of $[\mathcal{R}]_{S_c}$ comprises of output response of neurons from set $[\mathbf{N}]_{S_c}$ that do not stimulate other neurons of X -Neuronal system while responding to stimulus S_c .

As an example, Stimulus response $[\mathcal{R}]_{S_c}$ for stimulus $[S]_{R_1}$ (figure 25A) is given by $[\mathcal{R}]_{[S]_{R_1}} = \{[O_{C_1}]_{E_5}\}$. It should be absolutely clear before moving forward in discussion that output responses of set $[\mathcal{R}]_{S_c}$ can stimulate effector cells, e.g. glands cells, muscle cells and organ cells. So far, three sets (\mathbf{S} , $[\mathbf{N}]_{S_c}$, $[\mathcal{R}]_{S_c}$) are defined.

The next set required to explain the architecture of cognitive process is the local directorial board \mathbf{D}_{h_u} (equation 88). It should be noted that like set $[\mathbf{N}]_{S_c}$, set \mathbf{D}_{h_u} is a subset of set \mathbf{N} i.e. $\mathbf{D}_{h_u} \subseteq \mathbf{N}$. Although the elements of \mathbf{D}_{h_u} are used by X -Neuronal system to produce $[\mathcal{R}]_{S_c}$, they are not part of set $[\mathbf{N}]_{S_c}$. The elements of \mathbf{D}_{h_u} sort of direct the behavioural responses of elements of set $[\mathbf{N}]_{S_c}$ by providing additional stimuli, hence they are represented by a separate set and not part of $[\mathbf{N}]_{S_c}$. The elements of set \mathbf{D}_{h_u} communicate with the elements of set $[\mathbf{N}]_{S_c}$ using 3-component model. For example case shown in figure 25A, LDB is \mathbf{D}_{1_u} and the elements of \mathbf{D}_{1_u} communicate with the element I_{1_x} of $[\mathbf{N}]_{[S]_{R_1}}$, as shown in figure 20. Lastly, one more set is required to fully explain the architecture of cognitive process. Consider a set $\boldsymbol{\gamma}$; the elements of set $\boldsymbol{\gamma}$ comprises of drifting neurotransmitters which indirectly stimulates the elements of $[\mathbf{N}]_{S_c}$, while elements of $[\mathbf{N}]_{S_c}$ are responding to the stimulus S_c .

To fully explain the architecture of an arbitrary cognitive process, one needs a stimulus S_c from set \mathbf{S} and 4 set ($[\mathbf{N}]_{S_c}$, $[\mathcal{R}]_{S_c}$, \mathbf{D}_{h_u} , $\boldsymbol{\gamma}$). Diagrammatically, the architecture of an arbitrary cognitive process is shown in Figure 26.

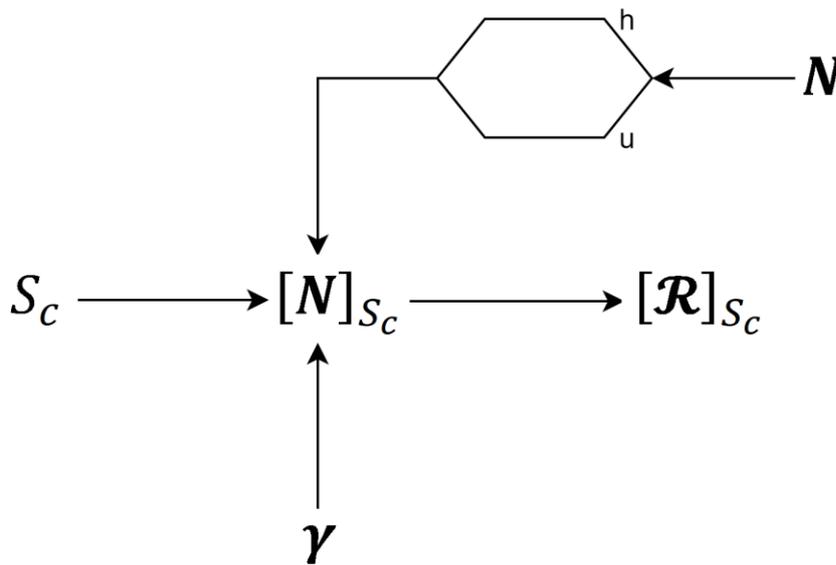


Figure 26. Architecture of an Arbitrary Cognitive Process. Stimulus S_c which is a subset of set \mathbf{S} stimulates X -Neuronal system to produce a Stimulus Response $[\mathcal{R}]_{S_c}$, by using a set of

neurons $[N]_{S_c}$. During the generation of stimulus response $[\mathcal{R}]_{S_c}$, neurons can be stimulated directly by an LDB D_{h_u} and/or stimulated indirectly by drifting neurotransmitters represented by set γ . All the involved neurons communicate using 3-component ‘Factory, Managers and Workers’ Model.

7.1. Example Case of an Arbitrary Cognitive Process

In this section, an example case of an arbitrary cognitive process will be presented to better understand the architecture and salient features of cognitive processes. Consider an X -Neuronal system, where $X = 300$, with $p, q, r = 100$ (equation 85). Directorial Board D for this X -Neuronal system represents 200 neurons. The neurons in this example case will only communicate chemically and will be treated as ‘Model Neurons’ with the following properties.

- $[y] = 10$ (Equation 68); With $[K] = 3, [M] = 6, [Q] = 0$.
- $[b] = 6$ (Equation 63); With $[V] = 6, [Z] = 0$.
- All the neurons in the time interval $0 \leq t \leq \infty$ behave ideally i.e. $\left[\frac{\beta}{\alpha}\right] = 1$.
- Initially, all neurons are in rest state i.e. $x = 0$.

Consider an external stimulus S_{ext_1} , which is subset of set S (equation 97) stimulating X -Neuronal system at an arbitrary time t , resulting in X -Neuronal system producing a stimulus response $[\mathcal{R}]_{S_{ext_1}}$. The neurons used by X -Neuronal system to produce $[\mathcal{R}]_{S_{ext_1}}$ are represented by $[N]_{S_{ext_1}}$ and are given by equation 101.

$$[N]_{S_{ext_1}} = \{ R_{10_x}, R_{11_x}, R_{12_x}, R_{50_x}, R_{51_x}, R_{52_x}, R_{53_x}, R_{54_x}, R_{55_x}, I_{10_x}, I_{11_x}, I_{12_x}, \\ I_{13_x}, I_{14_x}, I_{15_x}, I_{16_x}, E_{10_x}, E_{11_x}, E_{12_x}, E_{13_x}, E_{50_x}, E_{51_x}, E_{52_x} \} \quad 101$$

Furthermore, during this cognitive process, an LDB D_{3_u} defined by equation 92 governs the behavioural responses of certain elements of set $[N]_{S_{ext_1}}$ by providing addition stimuli. In addition to that, 3 drifting neurotransmitters; 2 of type $\gamma = 1$ and 1 of type $\gamma = 2$, bind to the

receptor binding sites of the 3 elements of set $[N]_{S_{ext_1}}$. For this example case, set γ is given by equation 102, where 1 and 2 represents the types of neurotransmitters. Based on the external stimulus S_{ext_1} and sets $[N]_{S_{ext_1}}$, D_{3u} and γ , X-Neuronal system produces a stimulus response $[R]_{S_{ext_1}}$. The architecture of this cognitive process, along with the states and output response of elements of $[N]_{S_{ext_1}}$ are shown in Figure 27. Based on figure 27, $[R]_{S_{ext_1}}$ is given by equation 103.

$$\gamma = \{1, 1, 2\} \quad 102$$

$$[R]_{S_{ext_1}} = \{ [O_{c_6}]_{E_{50}}, [O_{c_5}]_{E_{51}}, [O_{c_1}]_{E_{52}} \} \quad 103$$

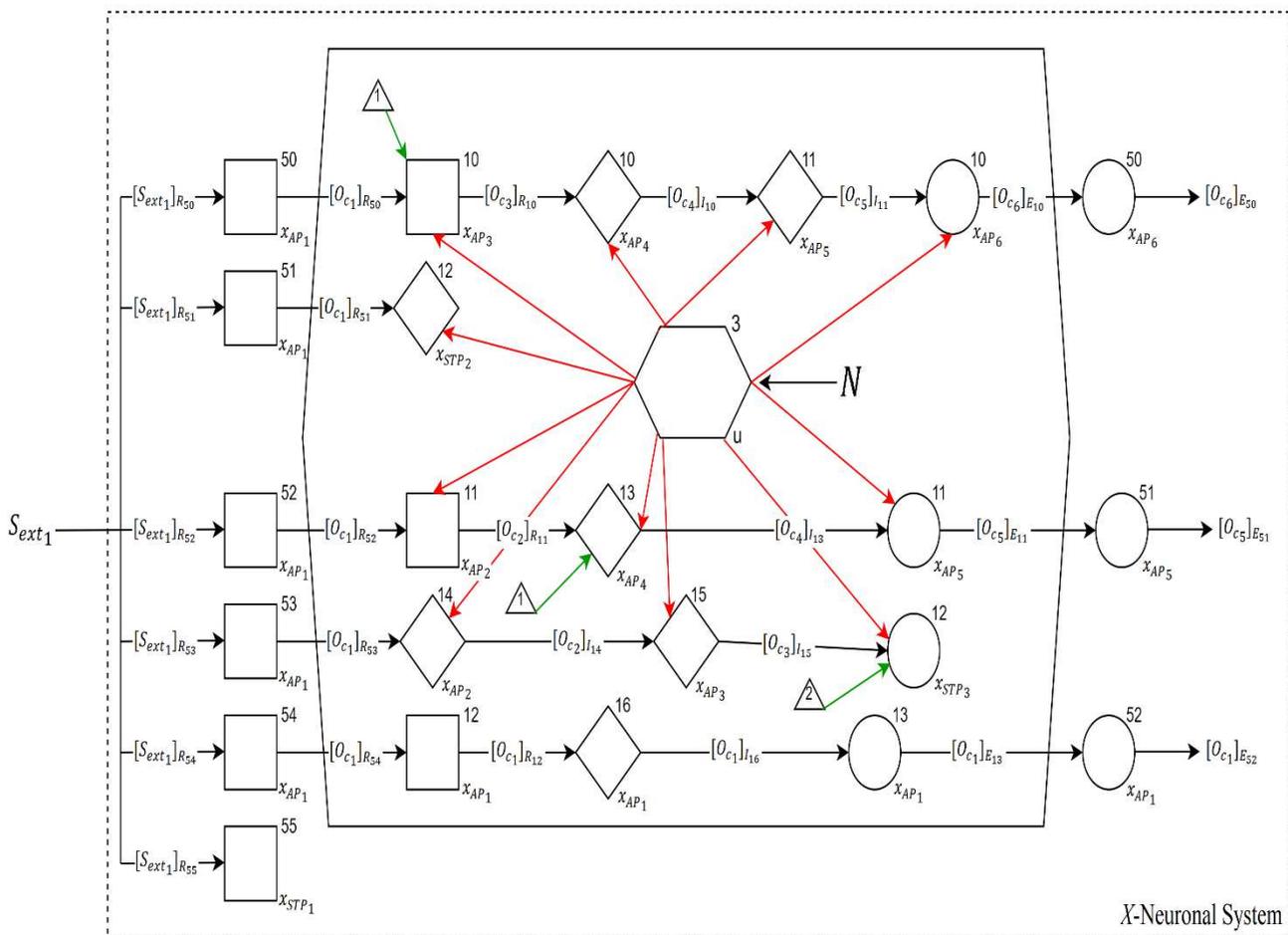


Figure 27. Architecture of an Arbitrary Cognitive process. X-Neuronal system which represented by set N is shown by a dotted quadrilateral. Directorial board D is represented by a hexagon. Individual drifting neurotransmitters are represented by a triangle with 1 and 2 represent the type of neurotransmitters. Neurons of LDB D_{3u} communicate with 11 neurons of

set $[N]_{S_{ext_1}}$. For simplicity output responses of D_{3_u} neurons are not shown and attention is given to the neurons of set $[N]_{S_{ext_1}}$. Neurotransmitters of set γ (equation 102) bind to the receptor sites of neurons R_{10_x} , I_{13_x} , E_{12_x} . The stimulus response $[R]_{S_{ext_1}}$ for stimulus S_{ext_1} is given by the output responses of neurons E_{50_x} , E_{52_x} , E_{53_x} .

7.2. Salient Features of an Arbitrary Cognitive Process

Based on Figure 27, it can be seen that external stimulus S_{ext_1} provide neurotransmitters/managers to 6 receptor neurons, 5 of which attained AP state x_{AP_1} and one neuron R_{55_x} attained an STP state x_{STP_1} . Based on the discussions of neuronal states and 3-component model, R_{55_x} has an incomplete AP cycle, so it will remain in activated state, holding specialised workers/ions inside it. If R_{55_x} is stimulated at a later time during another cognitive process, it will initially be in activated state x_{STP_1} while receiving stimulus. Likewise, neurons I_{12_x} and E_{12_x} remained in activated states x_{STP_2} and x_{STP_3} after the arbitrary cognitive process was complete. For neuron I_{12_x} , direct stimuli from R_{51_x} and D_{3_u} governed the eventual activated state x_{STP_2} . It is possible that without the additional stimuli from LDB D_{3_u} , the eventual state of I_{12_x} could have been an AP state, by stimulation from R_{51_x} alone, which could have resulted in different stimulus response $[R]_{S_{ext_1}}$ for stimulus S_{ext_1} . The fate of neuron E_{12_x} to attain activated state x_{STP_3} was governed by direct stimuli from I_{15_x} and D_{3_u} and indirect stimulus from a drifting neurotransmitter of type $\gamma = 2$. In a nutshell, *it is a feature of an arbitrary cognitive process that some of the neurons of X-Neuronal system can remain in activated state holding specialised workers inside them, even after the cognitive process is finished.*

The second feature that would be discussed is that it is possible for some neurons of set $[N]_{S_{ext_1}}$ to not receive additional direct stimuli from LDB D_{3_u} and/or indirect stimuli from drifting neurotransmitters. For example, neurons R_{12_x} , I_{16_x} , E_{13_x} which are subset of Directorial Board D did not receiver stimuli from D_{3_u} and/or drifting neurotransmitters of set γ , for this

cognitive process. This signifies that output response $[O_{C_1}]_{E_{52}}$ of set $[\mathcal{R}]_{S_{ext_1}}$ was only governed by $[S_{ext_1}]_{R_{54}}$, with intermediary neurons R_{12_x} , I_{16_x} , E_{13_x} in between. So, it is not unreasonable to say, that the generation of output response $[O_{C_1}]_{E_{52}}$ in itself is a cognitive process, without the stimulation (both direct and/or indirect) from LDB D_{3_u} and drifting neurotransmitters, given by equation 104. Cognitive process of equation 104 was initiated by X-Neuronal system when neuron R_{54_x} received stimulus $[S_{ext_1}]_{R_{54}}$.

$$\begin{aligned}
 [S_{ext_1}]_{R_{54}} &\rightarrow [x_{AP_1}]_{R_{54}} \rightarrow [O_{C_1}]_{R_{54}} \rightarrow [x_{AP_1}]_{I_{16}} \rightarrow [O_{C_1}]_{I_{16}} \rightarrow [x_{AP_1}]_{E_{13}} \rightarrow [O_{C_1}]_{E_{13}} \rightarrow \\
 [x_{AP_1}]_{E_{52}} &\rightarrow [O_{C_1}]_{E_{52}}
 \end{aligned}
 \tag{104}$$

The architecture of this cognitive process is given by $[S_{ext_1}]_{R_{54}} \rightarrow [N]_{[S_{ext_1}]_{R_{54}}} \rightarrow [\mathcal{R}]_{[S_{ext_1}]_{R_{54}}}$, where $[N]_{[S_{ext_1}]_{R_{54}}} = \{R_{54_x}, R_{12_x}, I_{16_x}, E_{13_x}, E_{52_x}\}$ and $[\mathcal{R}]_{[S_{ext_1}]_{R_{54}}} = \{[O_{C_1}]_{E_{52}}\}$.

In a nutshell, *a cognitive process of generating a stimulus response $[\mathcal{R}]_{S_c}$ for stimulus S_c can be a combination of many cognitive process running in series and parallel.*

Based on the cognitive process of equation 104, it can be confidently said that every time a neuron of X-Neuronal system is stimulated, a new unique cognitive process is initiated. For example, consider neuron E_{10_x} (figure 27), which was stimulated by stimulus $[O_{C_5}]_{I_{11}}$. The time neuron E_{10_x} got stimulated marked the initiation of a unique cognitive process and this cognitive process produced a stimulus response $[\mathcal{R}]_{[O_{C_5}]_{I_{11}}} = \{[O_{C_6}]_{E_{50}}\}$. Furthermore, this cognitive process is a part of the cognitive process of figure 27. Now consider neuron R_{55_x} which was stimulated by stimulus $[S_{ext_1}]_{R_{55}}$, marking the initiation of a unique cognitive process by X-Neuronal system. The fate of this cognitive process is rather unique, because neuron R_{55_x} didn't

produced a stimulus response i.e. $[\mathcal{R}]_{[S_{ext1}]_{R_{55}}}$ is a null set. This cognitive process only stimulated neuron R_{55_x} to an activated subthreshold potential state x_{STP_1} . This signifies a new type of cognitive process where no stimulus response is generated and this process only stimulates neuron to an STP activated state. Furthermore, this type of cognitive processes prepare neurons in a certain activated states for other unique cognitive processes at later times. For example, neuron R_{55_x} will be in STP state x_{STP_1} for later cognitive processes. The state x_{STP_1} of R_{55_x} holds the essence of cognitive process initiated with stimulus $[S_{ext1}]_{R_{55}}$. Likewise, cognitive processes initiated by stimuli $[S_{ext1}]_{R_{51}}$ and $[S_{ext1}]_{R_{53}}$ also represent cognitive process of this type, where neurons I_{12_x} and E_{12_x} remained in activated states STP x_{STP_2} and x_{STP_3} , respectively. In a nutshell, *some cognitive processes only stimulate neurons of an X-Neuronal system to an activated subthreshold states and these processes do not produce a stimulus response.*

At this point, it has become an absolute necessity to formally define different types of cognitive processes, to keep everything in perspective for moving forward in discussion.

7.3. Definitions of Cognitive Processes

In this section, two types of cognitive processes (type 1 and type 2) will be formally defined. Type 1 cognitive processes are processes which generate a stimulus response $[\mathcal{R}]_{S_c}$ for stimulus S_c . Type 2 cognitive processes are processes which only stimulates neurons of X-Neuronal system to an activated STP state, without generating a stimulus response.

Let an arbitrary cognitive process run by an X-Neuronal system be given by CP_i where $i = 1, 2, 3, \dots, j$; i represents a unique cognitive process and j are the total number of possible

cognitive process an X -Neuronal system can run. To cater both types of cognitive processes, consider a cognitive processes set $[CP]$ for an X -Neuronal system, defined by equation 105.

$$[CP] = \{ [CP]_{type\ 1} \cup [CP]_{type\ 2} \} \quad 105$$

Cognitive processes set $[CP]$ is the union of type 1 cognitive processes $[CP]_{type\ 1}$ set and type 2 cognitive processes $[CP]_{type\ 2}$. Sets $[CP]_{type\ 1}$ and $[CP]_{type\ 2}$ are further defined by equations 106 and 107, respectively, where $p = 1,2,3, \dots, q$ and $u = 1,2,3, \dots, v$.

$$[CP]_{type\ 1} = \{ [CP_p]_{type\ 1} \} \quad 106$$

$$[CP]_{type\ 2} = \{ [CP_u]_{type\ 2} \} \quad 107$$

p represents a unique type 1 cognitive process and q are the total number of possible type 1 cognitive processes. Likewise, u represents a unique type 2 cognitive process and v are the total number of possible type 2 cognitive processes. The total number of possible cognitive processes j an X -Neuronal system can run are given by equation 108.

$$j = q + v \quad 108$$

Based on these conventions, the definitions of cognitive processes $[CP_p]_{type\ 1}$ and $[CP_u]_{type\ 2}$ are given as follows:

A single arbitrary type 1 cognitive process $[CP_p]_{type\ 1}$ is the generation of a stimulus response $[R]_{S_c}$ for stimulus S_c by the elements of set $[N]_{S_c} \subseteq N$ with or without additional direct stimuli from elements of set D_{h_u} and/or indirect stimuli from elements of set γ . Furthermore, $[CP_p]_{type\ 1}$ can be a combination of other cognitive processes of type 1 and type

2.

A single arbitrary type 2 cognitive process $[CP_u]_{type\ 2}$ is the activation of elements of set $[N]_{S_c} \subseteq N$ to Subthreshold Potential states by stimulus S_c , with or without additional direct stimuli from elements of set D_{h_u} and/or indirect stimuli from elements of set γ . Stimulus response $[R]_{S_c}$ is an empty set for $[CP_u]_{type\ 2}$. Furthermore, $[CP_u]_{type\ 2}$ can be a combination of other type 2 cognitive processes.

Now that the definitions of cognitive processes of both types are formally defined, the focus will now be diverted to the time of cognitive process completion. Consider an arbitrary cognitive process CP_i , which is initiated at the time a neuron of X -Neuronal system is stimulated by the stimulus S_c . Let the initiation time of CP_i be defined by $[t_{initial}]_{CP_i}$ and completion time of CP_i be defined by $[t_{final}]_{CP_i}$. Cognitive process CP_i can be of two types. If CP_i is of type 1, the cognitive process is completed when all the neurons whose output responses are the elements of set $[R]_{S_c}$ have generated an output response. If the set $[R]_{S_c}$ has more than one elements, then $[t_{final}]_{CP_i}$ is the time of generation of output response of neuron which produced output response last. On the contrary, if CP_i is of type 2, then cognitive process is completed when element/elements of set $[N]_{S_c}$ have attained STP state/states. If more than one element of $[N]_{S_c}$ attained activated STP states, then $[t_{final}]_{CP_i}$ is the time of activation of neuron of set $[N]_{S_c}$ which attained STP state last. Based on the initiation and final completion time, the time of cognitive process completion defined by $[t]_{CP_i}$ is given by equation 109.

$$[t]_{CP_i} = [t_{final}]_{CP_i} - [t_{initial}]_{CP_i} \quad 109$$

At any given time, an X -Neuronal system is running a certain number of cognitive processes from set $[CP]$. Let the number of cognitive processes run by X -Neuronal system at any given time t be given by NCP . Furthermore, consider that these cognitive process are represented by a set $[CP]_t$, where elements of $[CP]_t$ represent the cognitive processes from set $[CP]$ run by X -

Neuronal system at time t . So set $[CP]_t$ is a subset of set $[CP]$ i.e. $[CP]_t \subseteq [CP]$. The question now becomes that which factors govern NCP and $[CP]_t$ at any given time. In principle, there can be significant number of governing factors which govern the number of cognitive processes represented by set $[CP]_t$, at any given time t . However, for the purposes of this article, one most fundamental governing factor will be discussed, which is given by the following statement.

Given an X-Neuronal system, the number of cognitive process NCP represented by set $[CP]_t$, the system can run at any given time t , depends on the availability of ions/workers. The higher the availability of ions/workers at any given time t , higher the NCP , X-Neuronal system can run.

From the perspective of 3-Component ‘Factory, Managers, Workers’ model, the above statement is self-explanatory, if less workers are available, less work will be done. Now the focus will be diverted to derive a relationship between NCP and availability of ions. To serve this purpose, let the number of available ions at any given time t be given by $[\dot{w}]_t$. It should be noted that these ions can be of different ionic species. For the purposes of this article, let the relationship between NCP and $[\dot{w}]_t$ be a linear relationship, given by equation 110, where $Const.$ is a constant. Based on equation 110, as the availability of ions increase, the number of cognitive processes an X-Neuronal system can run increase. In other words, the capacity of X-Neuronal system to run increase with the increase in the availability of ions.

$$NCP = Const. [\dot{w}]_t \quad 110$$

Now consider that the maximum number of cognitive processes an X-Neuronal system can run at any given time t be defined by $[NCP]_{max}$. Furthermore, in order to run $[NCP]_{max}$, X-Neuronal system requires a certain number of ions, which can be of different ionic species, depending on the cognitive processes. Let the number of ions required by X-Neuronal system

to run $[NCP]_{max}$ be defined by \dot{W} . If X -Neuronal system is running $[NCP]_{max}$, it is working at full 100% capacity. Let the capacity of X -Neuronal system at any given time t be defined by $[C]_t$. The capacity $[C]_t$ of X -Neuronal system is the ratio of number of cognitive processes at given time t over the maximum number of cognitive process.

$$[C]_t = \frac{NCP}{[NCP]_{max}} \times 100 \quad 111$$

As an example, if at time t , X -Neuronal system is running $NCP = 0.2 \cdot [NCP]_{max}$, it is running at 20% of its full capacity i.e. $[C]_t = 20\%$. Likewise, $[C]_t$ can also be given in terms of availability of ions, given by equation 112.

$$[C]_t = \frac{[\dot{w}]_t}{\dot{w}} \times 100 \quad 112$$

This is one of the significant features of the Neuronal State Theory presented in this article. *As the number of available ions $[\dot{w}]_t$ decrease, the capacity $[C]_t$ of X -Neuronal system to run cognitive process decrease.*

It has been previously established that type 2 cognitive processes only activate neurons of X -Neuronal system to STP states and these neurons hold specialised workers/ions inside them, even after cognitive process is complete. These held ions by neurons in STP activated states are not available to X -Neuronal system to run cognitive processes. The task of these ions is to keep neurons in STP states for later cognitive processes. So, it is reasonable to say that, *as the number of type 2 cognitive processes increase with time, the capacity of X -Neuronal system decrease, as more ions are held in neurons to maintain their STP states.*

This is a significant feature of Neuronal State Theory and it will be of utmost importance in the later sections of Sleep and Architecture of Dreams.

Now that the architecture and important definitions of cognitive processes have been reasonably well established, the next sections will discuss how different fields of cognitive research emerge from these architectures and framework developed in Part 1.

8. Attention

In this section, the architecture of attention will be discussed using the framework developed for cognitive processes. A cognitive process will be used as a test case to discuss the architecture of attention and later attention will be generalised using the salient features of this test case. Consider an X -Neuronal system, where $X = 300$, with $p, q, r = 100$. The neurons in this example case will only communicate chemically and will be treated as ‘Model Neurons’ with the following properties.

- $[y] = 10$ (Equation 68); With $[K] = 3, [M] = 6, [Q] = 0$.
- $[b] = 6$ (Equation 63); With $[V] = 6, [Z] = 0$.
- All the neurons in the time interval $0 \leq t \leq \infty$ behave ideally i.e. $\left[\frac{\beta}{\alpha}\right] = 1$.
- Initially, all neurons are in rest state i.e. $x = 0$.

Consider that at time t , X -Neuronal system is stimulated by an external stimulus S_{ext_2} (equation 98). As a consequence, X -Neuronal system starts a cognitive process of type 1. Let this type 1 cognitive process be denoted by $[CP_1]_{type 1}$. Since, the cognitive process is of type 1, X -Neuronal system will produce a stimulus response $[\mathcal{R}]_{S_{ext_2}}$, using a set $[N]_{S_{ext_2}}$. Let $[N]_{S_{ext_2}}$ for this cognitive process is given by equation 113.

$$[N]_{S_{ext_2}} = \{ R_{25_x}, R_{26_x}, R_{27_x}, R_{28_x}, R_{29_x}, R_{30_x}, R_{31_x}, R_{32_x}, R_{33_x}, I_{50_x}, I_{51_x}, I_{52_x}, \\ I_{53_x}, I_{54_x}, I_{55_x}, E_{25_x}, E_{26_x}, E_{27_x}, E_{28_x}, E_{29_x} \} \quad 113$$

Furthermore, for $[CP_1]_{type 1}$, neurons of set $[N]_{S_{ext_2}}$ do not receive direct and/or indirect stimuli from LDB or drifting neurotransmitters i.e. sets D_{hu} and γ are empty sets for $[CP_1]_{type 1}$. The architecture of $[CP_1]_{type 1}$ is shown in Figure 28. Based on Figure 28, the stimulus response $[R]_{S_{ext_2}}$ for stimulus S_{ext_2} , without additional stimuli from LDB and drifting neurotransmitter is given by equation 114. Furthermore, after completion of $[CP_1]_{type 1}$, neurons $\{R_{28_x}, R_{30_x}, R_{32_x}, R_{33_x}, I_{52_x}, E_{27_x}\}$ remain in x_{STP_1} states, holding ions inside them, as an essence of stimulus S_{ext_2} for future cognitive processes run by X-Neuronal system.

$$[R]_{S_{ext_2}} = \{ [O_{C_1}]_{E_{25}}, [O_{C_1}]_{E_{26}}, [O_{C_1}]_{E_{29}} \} \quad 114.$$

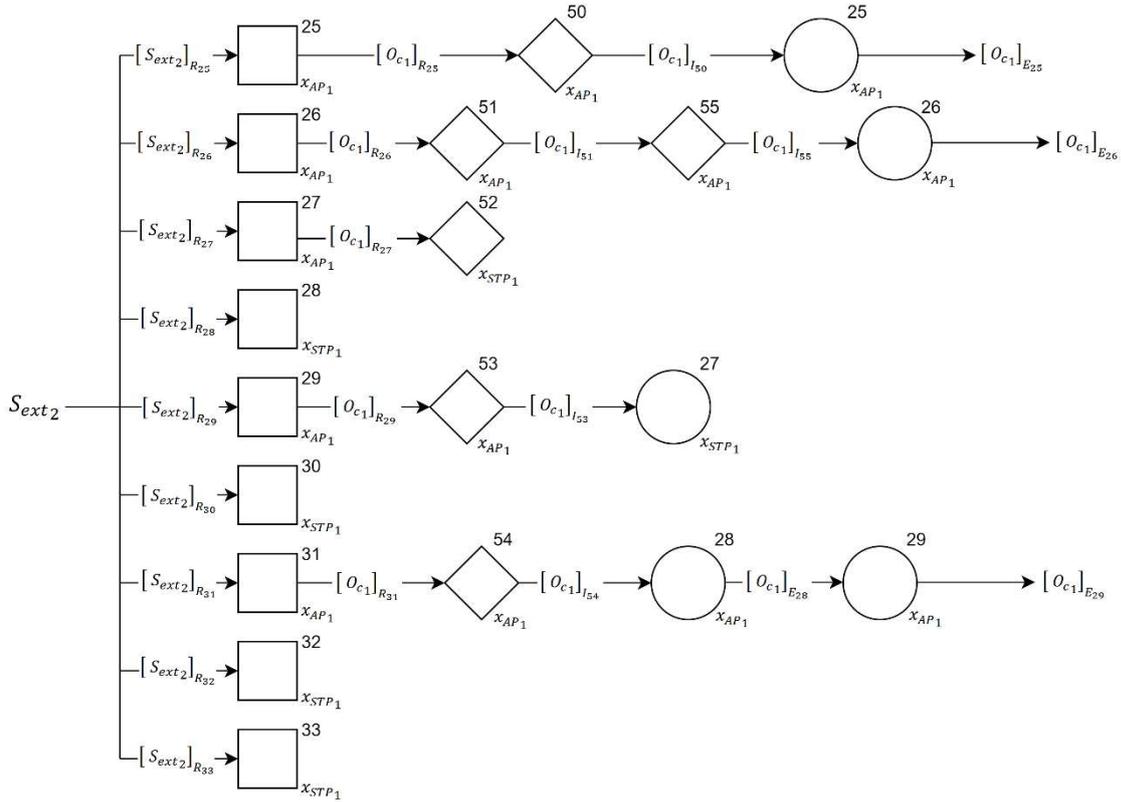


Figure 28. Architecture of type 1 cognitive process $[CP_1]_{type 1}$ run by X-Neuronal system using set $[N]_{S_{ext_2}} \subseteq N$ for an external stimulus S_{ext_2} .

Now the stimulus response $[\mathcal{R}]_{S_{ext_2}}$ for test cognitive process $[CP_1]_{type\ 1}$ will be abstracted with colored dices to make visualization of output responses of set $[\mathcal{R}]_{S_{ext_2}}$, easier for the reader. To serve this purpose, consider an arbitrary picture with considerable large number of colored dices. Furthermore, assume that the output responses of set $[\mathcal{R}]_{S_{ext_2}}$ represents these colored dices with the following rules.

- Each output response of set $[\mathcal{R}]_{S_{ext_2}}$ represents a unique dice on the arbitrary picture. So, set $[\mathcal{R}]_{S_{ext_2}}$ represents 3 unique dices on the arbitrary picture.
- Each possible output response of an element of set $[\mathcal{R}]_{S_{ext_2}}$ represents a unique color of the dice. As an example, output responses $[O_{C_1}]_{E_{25}}$ and $[O_{C_2}]_{E_{25}}$ represent unique colors of the same dice.

Since the total number of possible output responses of neurons in this example case is 6 i.e. $[b] = 6$. The possible colors a dice can have is 6. Let the possible output responses O_{CU} where $U = 1,2,3,4,5,6$ of elements of set $[\mathcal{R}]_{S_{ext_2}}$ be represented by colors {Red, Orange, Yellow, White, Green, Blue}, respectively. Based on these abstraction rules, the stimulus response $[\mathcal{R}]_{S_{ext_2}}$ for stimulus S_{ext_2} without direct/indirect additional stimuli is given by 3 unique dices of red color.

Now the question is, what would have happened to the test cognitive process $[CP_1]_{type\ 1}$ if it were to be stimulated by additional stimuli from LDB and/or drifting neurotransmitters. To address this question, effects of additional stimuli on the stimulus response $[\mathcal{R}]_{S_{ext_2}}$ of process $[CP_1]_{type\ 1}$ will be discussed.

Consider all neurons of X -Neuronal system are in rest state initially and at time t , system is stimulated by two stimuli (S_{ext_2}, S_{int_1}), simultaneously. As a consequence, X -Neuronal

system starts two cognitive processes; one for external stimulus S_{ext_2} and second for internal stimulus S_{int_1} . For external stimulus S_{ext_2} , the cognitive process is already denoted by $[CP_1]_{type_1}$. Let the cognitive process started by X -Neuronal system for internal stimulus S_{int_1} be denoted by $[CP_2]_{type_1}$. Set $[N]_{S_{int_1}}$ for cognitive process $[CP_2]_{type_1}$ is given by equation 115.

$$[N]_{S_{int_1}} = \{I_{20_x}, I_{21_x}, I_{22_x}, I_{23_x}, I_{50_x}, I_{55_x}, E_{25_x}, E_{27_x}, E_{29_x}, E_{30_x}, E_{60_x}, E_{61_x}, E_{62_x}\} \quad 115$$

Based on equations 113 and 115, it can be seen that both cognitive process $[CP_1]_{type_1}$ and $[CP_2]_{type_1}$ share common elements given by equation 116.

$$[N]_{S_{ext_2}} \cap [N]_{S_{int_1}} = \{I_{50_x}, I_{55_x}, E_{25_x}, E_{27_x}, E_{29_x}\} \quad 116$$

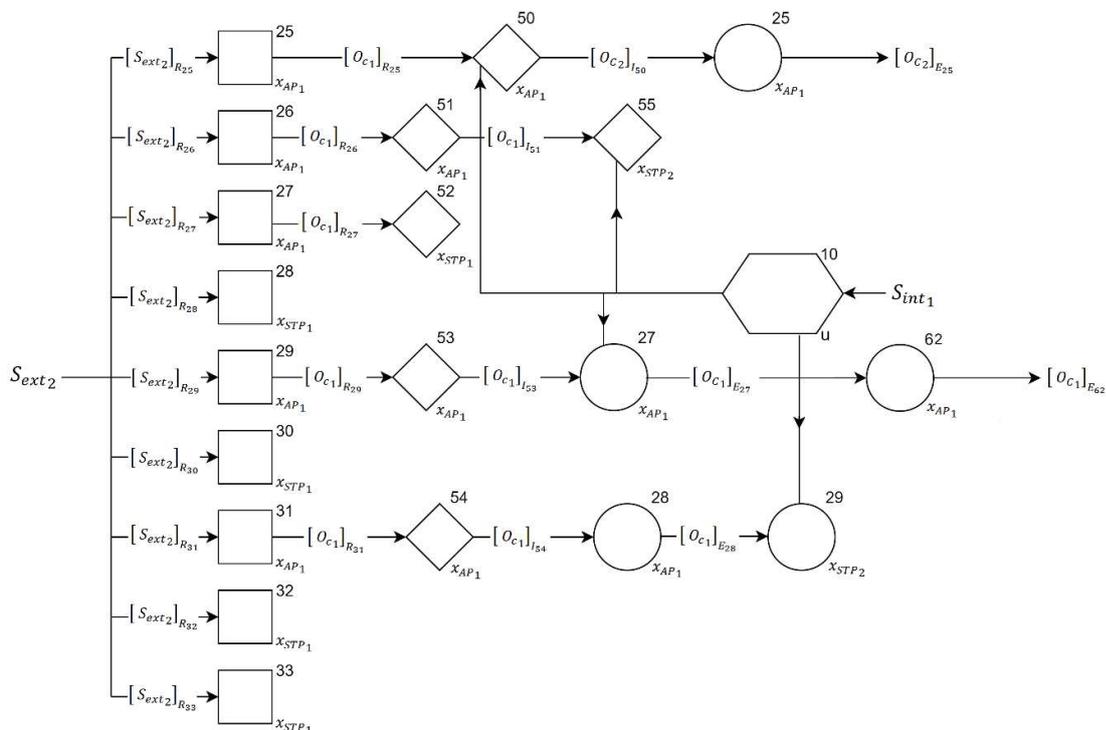
Since both cognitive processes share common elements and are started simultaneously by X -Neuronal system, each cognitive process can have effect on the other process depending on the times the stimuli reach neurons. At this point, the cognitive process $[CP_1]_{type_1}$ will be considered as a test process and the effect of $[CP_2]_{type_1}$ on $[CP_1]_{type_1}$ will be discussed. Based on the framework discussed previously, any additional direct stimuli elements of set $[N]_{S_c}$ receive are from LDB D_{h_u} . So depending on the timings of stimuli, the elements of set $[N]_{S_{int_1}}$ can act LDB for the elements of set $[N]_{S_{ext_2}}$. For the purposes of discussion, consider that stimuli from some elements of set $[N]_{S_{int_1}}$ indeed reach in time to affect the activated states of some elements of set $[N]_{S_{ext_2}}$, acting as its LDB. Let the LDB for cognitive process $[CP_1]_{type_1}$ be denoted by D_{10_u} and is comprised of elements of set $[N]_{S_{int_1}}$, given by equation 117.

$$D_{10_u} = \{I_{21_x}, I_{22_x}, I_{23_x}, E_{60_x}, E_{61_x}\} \quad 117$$

Based on the additional stimuli from \mathbf{D}_{10_u} , the architecture of cognitive process $[CP_1]_{type\ 1}$ is shown in Figure 29. Based on Figure 29, the stimulus response $[\mathcal{R}]_{S_{ext_2}}$ is given by equation 118. Based on these abstraction rules, the stimulus response $[\mathcal{R}]_{S_{ext_2}}$ for stimulus S_{ext_2} with direct additional stimuli from LDB \mathbf{D}_{10_u} is given by 2 unique dices of with red and orange color. It should be noted that because of additional stimuli from \mathbf{D}_{10_u} , the set $[\mathbf{N}]_{S_{ext_2}}$ also changes. Instead of equation 113, $[\mathbf{N}]_{S_{ext_2}}$ for cognitive process $[CP_1]_{type\ 1}$ is given by equation 119.

$$[\mathcal{R}]_{S_{ext_2}} = \{ [O_{C_2}]_{E_{25}}, [O_{C_1}]_{E_{62}} \} \quad 118$$

$$[\mathbf{N}]_{S_{ext_2}} = \{ R_{25_x}, R_{26_x}, R_{27_x}, R_{28_x}, R_{29_x}, R_{30_x}, R_{31_x}, R_{32_x}, R_{33_x}, I_{50_x}, I_{51_x}, I_{52_x}, I_{53_x}, I_{54_x}, I_{55_x}, E_{25_x}, E_{27_x}, E_{28_x}, E_{29_x}, E_{62_x} \} \quad 119$$



Architecture of type 1 cognitive process $[CP_1]_{type\ 1}$ run by X -Neuronal system using set $[\mathbf{N}]_{S_{ext_2}} \subseteq \mathbf{N}$ for an external stimulus S_{ext_2} , with additional direct stimuli from LDB \mathbf{D}_{10_u} .

8.1. Architecture of Attention

In this section, the architecture of attention will be discussed using the salient features of cognitive process $[CP_1]_{type\ 1}$ with and without addition stimuli. Based on this architecture, attention will be formally defined using the framework of neuronal state theory.

To discuss the architecture of attention, the concepts of data and information are immensely useful. To serve this purpose, it is imperative that one defines data and information in the context of this article. For the purposes of this article, the definitions of data and information are given as follows:

Data is unorganised raw facts that X-Neuronal system receives as stimulus S_c .

Information is the processed, organised and structure data given as the outputs of stimulus Response $[\mathcal{R}]_{S_c}$.

Now that the definitions of data and information are established. The focus will be diverted to the salient features of cognitive process $[CP_1]_{type\ 1}$, which pertains to the architecture of attention. Based on figure 29, it can be seen that with addition direct stimuli from D_{10_u} , the stimulus response $[\mathcal{R}]_{S_{ext_2}}$ (equation 118) is different than the stimulus response of same cognitive process without addition stimuli (equation 114). Using abstraction rules, 2 unique dices of red and orange color with addition stimuli as compare to 3 unique red dices without addition stimuli. This signifies that, for an arbitrary stimulus S_c , an X-Neuronal system can produce different stimulus responses $[\mathcal{R}]_{S_c}$. This point represents a very crucial feature of the architecture of cognition. In the context of data and information, *this point signifies that an X-Neuronal system can process the same data into different information, depending on the addition of other data during processing.* For instance, for cognitive process $[CP_1]_{type\ 1}$, X-Neuronal system processed the data received as stimulus S_{ext_2} into 2 different information.

Firstly, information as 3 unique red dices when no additional data was added to the data S_{ext_2} during processing. Secondly, information as 2 unique dices of red and orange color, when additional data was added to S_{ext_2} during processing. The additional added data comes from another cognitive process $[CP_2]_{type_1}$ and represents a portion of data S_{int_1} . At this point, it is very reasonable to say that X -Neuronal system was supposed to process data S_{ext_2} into information $[\mathcal{R}]_{S_{ext_2}}$ given by equation 114, however because of the addition of additional data from S_{int_1} , it processed S_{ext_2} into information given by equation 118. To generalise this feature, let the stimuli response $[\mathcal{R}]_{S_c}$ for a given stimulus S_c without any additional stimuli be given by $[\mathcal{R}_{ref}]_{S_c}$. In other words, $[\mathcal{R}_{ref}]_{S_c}$ is the information, an X -Neuronal system process for a given data S_c , without any additional data addition during the processing. This represents a significant feature of attention. *In the context of neuronal state theory, one of the features of architecture of attention is the processing of a given amount of data S_c by X -Neuronal system into information $[\mathcal{R}]_{S_c}$ which is different from $[\mathcal{R}_{ref}]_{S_c}$.*

Consider again that all neurons of X -Neuronal system are in rest state initially, and at time t , system is stimulated by a stimulus S_{ext_2} . If no additional stimuli are added during processing of data S_{ext_2} , X -Neuronal system is supposed to produce a stimulus response $[\mathcal{R}_{ref}]_{S_{ext_2}}$ given by equation 114. However, consider that during processing of S_{ext_2} , neurons of set $[\mathcal{N}]_{S_{ext_2}}$ are stimulated by additional direct stimuli from arbitrary set \mathbf{D}_{h_u} and indirect stimuli from drifting neurotransmitters, given by an arbitrary set $\boldsymbol{\gamma}$. Based on addition stimuli, X -Neuronal system did not produce any stimulus response $[\mathcal{R}]_{S_{ext_2}}$ for stimulus S_{ext_2} i.e. $[\mathcal{R}]_{S_{ext_2}}$ is an empty set. This time around, X -Neuronal system ran cognitive process started by stimulus S_{ext_2} as a type 2 cognitive process, where only elements of set $[\mathcal{N}]_{S_{ext_2}}$ remained in subthreshold states, without producing any response. In other words, X -Neuronal system did process data of

stimulus of stimulus S_{ext2} into any information. *This is one of an important features of architecture of attention, which signifies that it is possible that an X-Neuronal system doesn't process a given amount of data S_c into any information i.e. $[\mathcal{R}]_{S_c}$ is an empty set.*

The question now becomes that what is the fate of data S_{ext2} , if X-Neuronal system ran it as a type 2 cognitive process. To address this question, consider cognitive process $[CP_1]_{type\ 1}$ with and without additional stimuli as shown in figures 28 and 29, respectively. In both cases, $[CP_1]_{type\ 1}$ is the combination of many cognitive processes of types 1 and 2. To establish the fate of data, if X-Neuronal system ran it as a type 2 cognitive process, consider neuron R_{33_x} from figure 28. X-Neuronal system ran type 2 cognitive process, when R_{33_x} , initially at rest state received data $[S_{ext2}]_{R_{33}}$. Data $[S_{ext2}]_{R_{33}}$ represents small portion of data S_{ext2} . Upon completion of type 2 cognitive process, neuron R_{33_x} attained an activated x_{STP_1} state. Even though the data $[S_{ext2}]_{R_{33}}$ was not processes into any information, it is held or stored in neuron R_{33_x} as an x_{STP_1} activated state. Since $[S_{ext2}]_{R_{33}}$ represents a portion of data S_{ext2} , a portion of data S_{ext2} is stored in neuron R_{33_x} as an x_{STP_1} activated state. Likewise, consider neuron I_{55_x} from figure 29. X-Neuronal system started a cognitive process when I_{55_x} was stimulated by the data $[O_{C_1}]_{I_{51}}$. Data $[O_{C_1}]_{I_{51}}$ in itself is a unique stimulus, which caused X-Neuronal system to start a new cognitive process. However, this cognitive process is a small part of cognitive process $[CP_1]_{type\ 1}$. From the perspective of $[CP_1]_{type\ 1}$, data $[O_{C_1}]_{I_{51}}$ is a transformed form of the data $[S_{ext2}]_{R_{26}}$, where transformation of data is given by equation 120.

$$[S_{ext2}]_{R_{26}} \rightarrow [O_{C_1}]_{R_{26}} \rightarrow [O_{C_1}]_{I_{51}} \quad 120$$

So data $[O_{C_1}]_{I_{51}}$ which neuron I_{55_x} received, represents a transformed form of the portion of data S_{ext_2} . Furthermore, neuron I_{55_x} received addition stimuli during process of data $[O_{C_1}]_{I_{51}}$, from LDB D_{10_u} , which in turn represents transformed form of the portion of data S_{int_1} . Upon completion of process, I_{55_x} attained an activated state χ_{STP_2} . Transformed forms of the portions of data S_{ext_2} and S_{int_1} gets stored in the activated state χ_{STP_2} of neuron I_{55_x} . Similarly, if the whole cognitive process started by S_{ext_2} is a type 2 process, the portions of data S_{ext_2} is stored in the subthreshold states of neurons of set $[N]_{S_{ext_2}}$ in the same form or transformed form. This discussion is generalised for an arbitrary stimuli S_c by the following statements:

The portions of arbitrary data S_c , which are not processed into information $[\mathcal{R}]_{S_c}$ is never lost, rather it is stored in the neurons of set $[N]_{S_c}$ as activated subthreshold states. This stored data can be in same form as data S_c or in the transformed form of data S_c .

For a type 1 cognitive process, an X-Neuronal system can process portions of arbitrary data S_c into information $[\mathcal{R}]_{S_c}$, while storing other portions of data S_c into neurons of set $[N]_{S_c}$ as activated subthreshold states, in the same form or transformed form of data S_c .

For a type 2 cognitive process, an X-Neuronal system will store all the arbitrary data S_c (same or transformed form) into neurons of set $[N]_{S_c}$ as activated subthreshold states. The subthreshold states are given by the concentrations of ionic species inside the neurons.

It has been previously established, neurons in subthreshold activated states will receive stimuli in activated state for later cognitive process. Similarly, neurons of set $[N]_{S_{ext_2}}$ who remained in activated states will receive stimuli from later cognitive processes in those activated states. If these later cognitive processes produces stimuli responses, using activated neurons of set $[N]_{S_{ext_2}}$, then those stimuli responses will have essence of stimulus S_{ext_2} . In other words,

portions of data S_{ext_2} , which was stored in the activated subthreshold states of neurons of set $[N]_{S_{ext_2}}$, will be processed into information for later cognitive processes. This is one of the most significant features of the neuronal state theory, which states:

Stimuli responses of cognitive processes can be affected by the stimuli previously received by the X-Neuronal system.

In the context of architecture of attention, the processing of arbitrary data S_c into information $[\mathcal{R}_{ref}]_{S_c}$ can be affected by the previously stored data in neurons of X-Neuronal system as subthreshold states, which can result in data S_c processed into information $[\mathcal{R}]_{S_c}$

instead of $[\mathcal{R}_{ref}]_{S_c}$.

Now consider the cognitive process $[CP_2]_{type\ 1}$ of figure 28, which was started by X-Neuronal system for stimulus S_{ext_2} , at time t . At this point, it is necessary that one discusses the possibility that receiving of stimulus S_{ext_2} can be dependent on the previously ran cognitive process or processes by X-Neuronal system. For instance, consider an arbitrary type 1 cognitive process $[CP_\phi]_{type\ 1}$, which produced a stimulus response $[\mathcal{R}]_{S_c}$ for stimulus S_c . The stimulus response $[\mathcal{R}]_{S_c}$ for cognitive process $[CP_\phi]_{type\ 1}$ can cause the X-Neuronal system to receive stimulus S_{ext_2} at time t . It is very possible that X-Neuronal system would have received stimulus other than S_{ext_2} , if it were not for the cognitive process $[CP_\phi]_{type\ 1}$. Likewise, the receiving of a particular stimulus can be dependent on more than one previously ran cognitive processes. In the context of architecture of attention, it signifies, *data an X-Neuronal system receives at a given time t receives can be affected by the information of previously processed data.*

8.2. Definition of Attention

Now that the necessary features of architecture of attention are established, attention can be formally defined in the context of neuronal state theory, by the following statement:

For a given amount of data S_c (independent of or dependent on information of previously processes data), an X-Neuronal system receives at time t . Attention is the processing of data S_c (portions of S_c or complete S_c) into information $[\mathcal{R}]_{S_c}$ instead of $[\mathcal{R}_{ref}]_{S_c}$. Information $[\mathcal{R}]_{S_c}$ is affected by previously stored data in the neurons of set $[\mathbf{N}]_{S_c}$, as subthreshold states and the addition of additional data during processing of S_c from sets \mathbf{D}_{h_u} and $\boldsymbol{\gamma}$. Furthermore, $[\mathcal{R}]_{S_c}$ can be a null set, signifying storage of complete data S_c (same form or transformed form) in the neurons of set $[\mathbf{N}]_{S_c}$, which can affect the processing of data into information of later cognitive processes.

9. Memories

In this section, the architecture of memories will be discussed using the framework of neuronal state theory. Firstly, a single memory in the context of this article will be discussed and later an example case will be presented. Using the example case, salient features of architecture of memories will be discussed.

Consider an X -Neuronal system comprising of Model neurons communicating chemically, with the following properties.

- $[y] = 10$ (Equation 68); With $[K] = 3, [M] = 6, [Q] = 0$.
- $[b] = 6$ (Equation 63); With $[V] = 6, [Z] = 0$.
- All the neurons in the time interval $0 \leq t \leq \infty$ behave ideally i.e. $\left[\frac{\beta}{\alpha}\right] = 1$.
- Initially, all neurons are in rest state i.e. $x = 0$.

Consider a type 1 cognitive process $[CP_7]_{type 1}$ ran by X -Neuronal system, given by equation 121.

$$\begin{array}{ccc}
 S_1(t_1) \rightarrow [N]_{S_1} \rightarrow [\mathcal{R}]_{S_1}(t_2) & & \\
 \uparrow & & 121 \\
 \mathbf{D}_{h_u} & &
 \end{array}$$

Based on equation 121, X -Neuronal system received stimulus S_1 at time t_1 and produced a stimulus response $[\mathcal{R}]_{S_1}$ at time t_2 , using set $[N]_{S_1}$ with addition direct stimuli from an arbitrary LDB \mathbf{D}_{h_u} . Using abstraction rules of colored dices (discussed in attention section), the stimulus response $[\mathcal{R}]_{S_1}$ at time t_2 represents a unique number of colored dices. For the sake of the argument, assume $[\mathcal{R}]_{S_1}$ represents 6 unique dices of red color. Now, consider another type 1 cognitive process $[CP_8]_{type 1}$ ran by X -Neuronal system, given by equation 122.

$$S_2(t_3) \rightarrow [N]_{S_2} \rightarrow [\mathcal{R}]_{S_2}(t_4) \quad 122$$

Based on equation 122, X -Neuronal system received stimulus S_2 at time t_3 and produced a stimulus response $[\mathcal{R}]_{S_2}$ at time t_4 , using set $[\mathcal{N}]_{S_2}$ with no addition stimuli. Using abstraction rules of colored dices, stimulus response $[\mathcal{R}]_{S_2}$ at time t_4 represents unique number of colored dices. The question now becomes, what if stimulus response $[\mathcal{R}]_{S_2}$ at time t_4 represents the same colored dices as stimulus response $[\mathcal{R}]_{S_1}$ at time t_2 . In other words, elements of set $[\mathcal{R}]_{S_2}$ are exactly the same as elements of set $[\mathcal{R}]_{S_1}$. If both sets ($[\mathcal{R}]_{S_2}$ and $[\mathcal{R}]_{S_1}$) are same, X -Neuronal system has produced the same stimulus response for two different stimuli. In terms of data and information, X -Neuronal system has processed data S_1 at time t_1 and data S_2 at time t_3 into the same information. The ability of X -Neuronal system to process different data at different times into same information is the foundation of memory, in the neuronal state theory. Using the above discussion as foundation, generalised definition for a single memory in the context of neuronal state theory is given by the following statement:

Given an X -Neuronal system produced a stimulus response $[\mathcal{R}]_{S_c}$ for an arbitrary type 1 cognitive process $[CP_p]_{type\ 1}$ at time $t = t_0$ using neuronal set $[\mathcal{N}]_{S_c}$, a single memory of $[CP_p]_{type\ 1}$ is the generation of stimulus response $[\mathcal{R}]_{S_c}$ by the same X -Neuronal system at time $t > t_0$, using neuronal set $[\mathcal{N}]_{S_c}$ or other neuronal sets.

This is one of the most important features of neuronal state theory. Contrary to popular convention where memory research is carried out with storage and retrieval models, neuronal state theory states, memory has nothing to do with storage and retrieval, rather it is the ability of X -Neuronal system (which can be a human brain) to produce same information depending on the conditions at that instance of time, which was produced at earlier times by the same system. Memories in its essence are instantaneous and dynamic processes ran by the X -Neuronal system and are very much dependent on the state of X -Neuronal system at any given

time. This and other aspect of memories will be further elaborated by the example case in the next section.

9.1. Example Case

Consider an X -Neuronal system, where $X = 3 = 1200$, with $p, q, r = 400$. The neurons in this example case will only communicate chemically and will be treated as ‘Model Neurons’ with properties given in previous section.

Now consider a type 1 cognitive process $[CP_5]_{type\ 1}$ started by X -Neuronal system at time $t = t_1$ after receiving an external stimulus S_{ext_3} . At time $t = t_2$, X -Neuronal system produced a stimulus response $[\mathcal{R}]_{S_{ext_3}}$. The architecture of cognitive process $[CP_5]_{type\ 1}$ is shown in Figure 30. Based on figure 30, the neuronal set used by X -Neuronal system for $[CP_5]_{type\ 1}$ is given by equation 123.

$$[\mathbf{N}]_{S_{ext_3}} = \{ R_{300_x}, R_{301_x}, R_{302_x}, R_{303_x}, R_{304_x}, R_{305_x}, R_{306_x}, R_{307_x}, R_{308_x}, R_{309_x}, I_{300_x}, \\ I_{301_x}, I_{302_x}, I_{303_x}, I_{304_x}, I_{305_x}, I_{306_x}, E_{300_x}, E_{301_x}, E_{302_x}, E_{303_x}, E_{304_x}, E_{305_x} \} \quad 123$$

Some of the elements of set $[\mathbf{N}]_{S_{ext_3}}$ received additional direct stimuli from LDB \mathbf{D}_{h_u} . For the purposes of this discussion, details of \mathbf{D}_{h_u} are not important, hence they will not be discussed further. For cognitive process $[CP_5]_{type\ 1}$ a stimulus response was generated by X -Neuronal system, given by equation 124.

$$[\mathcal{R}]_{S_{ext_3}} = \{ [O_{C_1}]_{E_{300}}, [O_{C_2}]_{E_{301}}, [O_{C_3}]_{E_{302}}, [O_{C_4}]_{E_{303}}, [O_{C_5}]_{E_{304}}, [O_{C_6}]_{E_{305}} \} \quad 124$$

Using the abstraction rules of colored dices, $[\mathcal{R}]_{S_{ext_3}}$ represents 6 dices with colors {Red, Orange, Yellow, White, Green, Blue} respectively.

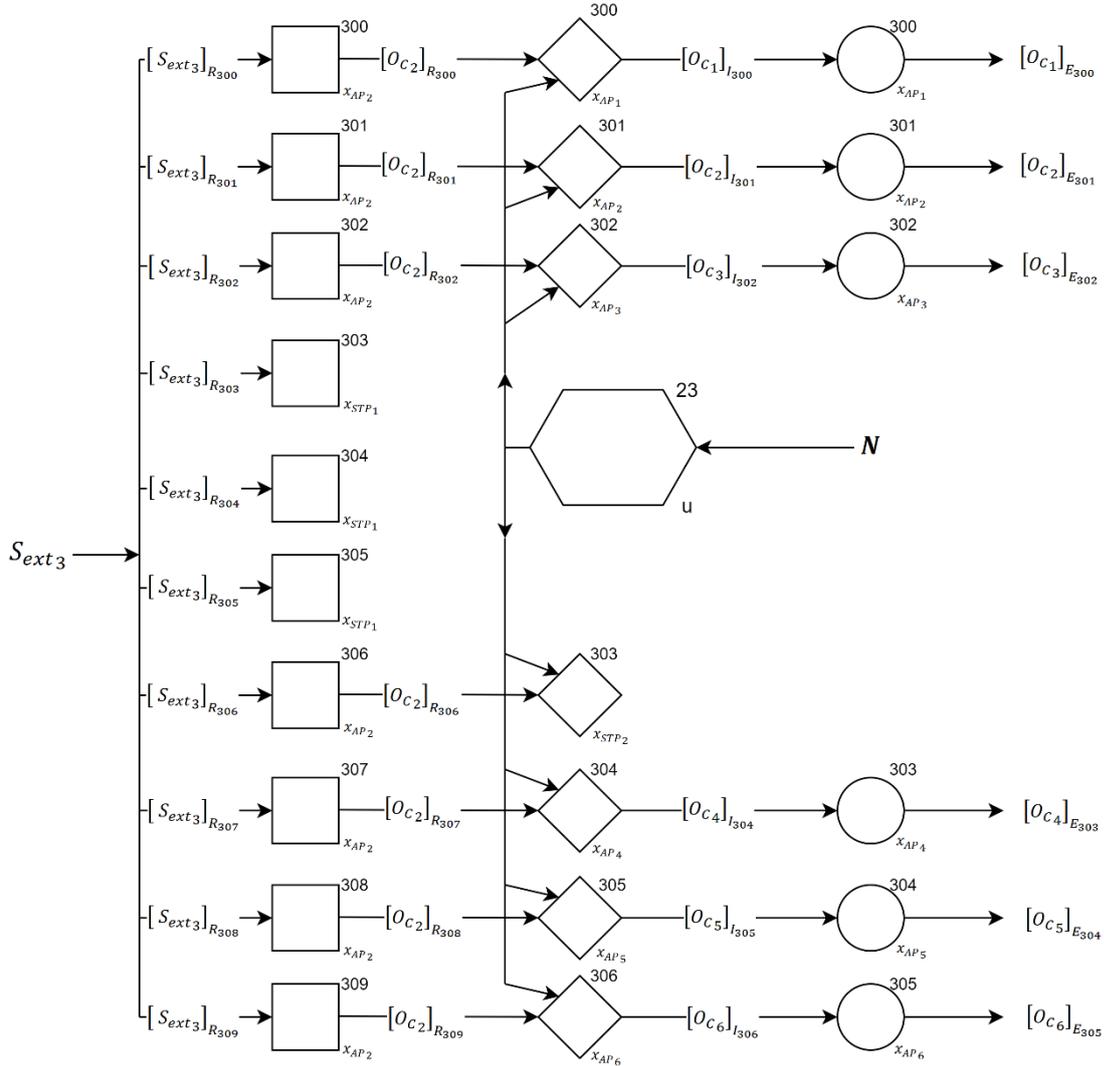


Figure 30. Architecture of type 1 cognitive process $[CP_5]_{type 1}$ ran by X-Neuronal system, where $X = 1200$, with $p, q, r = 400$.

Now, consider another type 1 cognitive process $[CP_6]_{type 1}$ started by same X-Neuronal system at time $t = t_3$ after receiving an internal stimulus S_{int_2} . At time $t = t_4$, X-Neuronal system produced a stimulus response $[\mathcal{R}]_{S_{int_2}}$. The architecture of cognitive process $[CP_6]_{type 1}$ is shown in Figure 31. Based on figure 31, sets $[N]_{S_{int_2}}$ and $[\mathcal{R}]_{S_{int_2}}$ are given by equations 125 and 126 respectively.

$$[\mathbf{N}]_{S_{int_2}} = \{I_{300_x}, I_{301_x}, I_{302_x}, I_{304_x}, I_{305_x}, I_{306_x}, E_{300_x}, E_{301_x}, E_{302_x}, E_{303_x}, E_{304_x}, E_{305_x}\} \quad 125$$

$$[\mathcal{R}]_{S_{int_2}} = \{[O_{C_1}]_{E_{300}}, [O_{C_2}]_{E_{301}}, [O_{C_3}]_{E_{302}}, [O_{C_4}]_{E_{303}}, [O_{C_5}]_{E_{304}}, [O_{C_6}]_{E_{305}}\} \quad 126$$

Based on equations 124 and 126, it can be seen that both cognitive processes $[CP_5]_{type\ 1}$ and $[CP_6]_{type\ 1}$ produced the same stimulus response i.e. $[\mathcal{R}]_{S_{ext_3}} = [\mathcal{R}]_{S_{int_2}}$. In terms of memory discussion, X -Neuronal system has produced a memory of cognitive process $[CP_5]_{type\ 1}$ at time $t = t_4$, using the internal stimulus S_{int_2} and neuronal set $[\mathbf{N}]_{S_{int_2}}$ (equation 125).

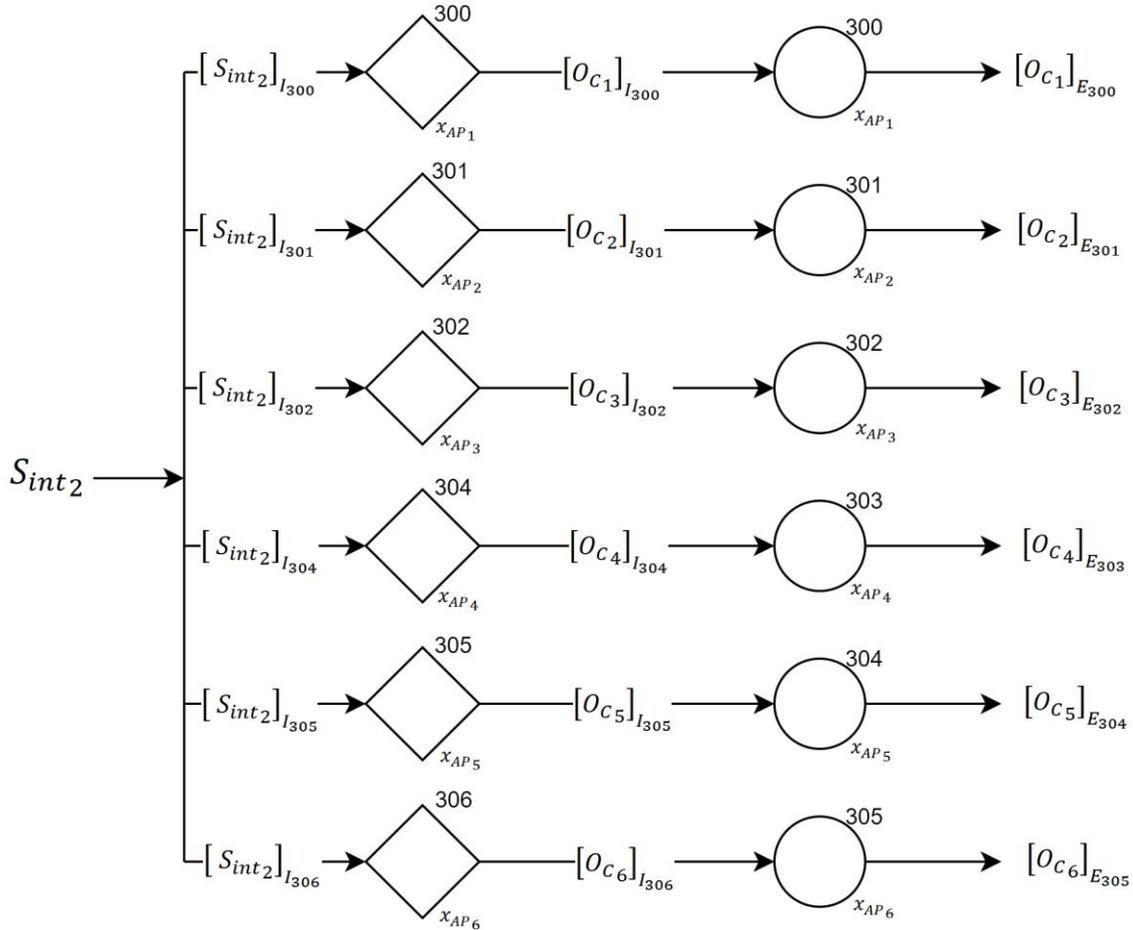


Figure 31. Architecture of type 1 cognitive process $[CP_6]_{type\ 1}$ ran by X -Neuronal system, where $X = 1200$, with $p, q, r = 400$.

9.2. Salient Features of Memories

In this section, salient features of memories will be discussed using the example case mentioned above. First of all, it can be seen that X -Neuronal system generated the memory of cognitive process $[CP_5]_{type\ 1}$ using neuronal set $[N]_{S_{int_2}}$ (equation 125) which is a subset of neuronal set $[N]_{S_{ext_3}}$ (equation 123) i.e. $[N]_{S_{int_2}} \subseteq [N]_{S_{ext_3}}$. This is a very significant feature of memories, which signifies that X -Neuronal system does not have to use all the neurons of set $[N]_{S_{ext_3}}$ to produce memory of cognitive process $[CP_5]_{type\ 1}$. In principle, X -Neuronal system can produce memory of $[CP_5]_{type\ 1}$ using different neuronal sets for different stimuli. Let an arbitrary neuronal set which can produce memory of cognitive process $[CP_5]_{type\ 1}$ be given by $[\mathcal{M}_m]_{[\mathcal{R}]_{S_{ext_3}}}$, where $m = 0, 1, 2, \dots, n$. Set $[\mathcal{M}_m]_{[\mathcal{R}]_{S_{ext_3}}}$ represents a unique neuronal set which can generate stimulus response same $[\mathcal{R}]_{S_{ext_3}}$ of cognitive process $[CP_5]_{type\ 1}$ at time $t > t_2$ and n represents the total number of possible neuronal sets which can generate stimulus response same as $[\mathcal{R}]_{S_{ext_3}}$. Set $[\mathcal{M}_m]_{[\mathcal{R}]_{S_{ext_3}}}$ is a subset of set N . As a convention, $[\mathcal{M}_0]_{[\mathcal{R}]_{S_{ext_3}}}$ will always represent neuronal set $[N]_{S_{ext_3}}$ i.e. the set which produced $[\mathcal{R}]_{S_{ext_3}}$ for the first time at $t = t_2$, and set $[N]_{S_{ext_3}}$ can produce $[\mathcal{R}]_{S_{ext_3}}$ at time $t > t_2$. Similarly, set $[N]_{S_{int_2}}$ also represents a unique $[\mathcal{M}_m]_{[\mathcal{R}]_{S_{ext_3}}}$ set. As an example, assume $[N]_{S_{int_2}}$ is represented by $[\mathcal{M}_1]_{[\mathcal{R}]_{S_{ext_3}}}$. The elements of set $[\mathcal{M}_m]_{[\mathcal{R}]_{S_{ext_3}}}$ can be less than, greater than or equal to set $[\mathcal{M}_0]_{[\mathcal{R}]_{S_{ext_3}}}$. For instance, $[\mathcal{M}_1]_{[\mathcal{R}]_{S_{ext_3}}}$ has less elements than $[\mathcal{M}_0]_{[\mathcal{R}]_{S_{ext_3}}}$. However, for memory of cognitive process $[CP_5]_{type\ 1}$ all n neuronal sets will have neurons whose output responses are the elements of set $[\mathcal{R}]_{S_{ext_3}}$. So for memory of $[CP_5]_{type\ 1}$, all n neuronal sets will have

neurons $\{E_{300_x}, E_{301_x}, E_{302_x}, E_{303_x}, E_{304_x}, E_{305_x}\}$. Now this feature of memories will be generalised for any X -Neuronal system, where X can be any natural number.

Given a stimulus response $[\mathcal{R}]_{S_c}$ of an arbitrary type 1 cognitive process $[CP_p]_{type\ 1}$ generated by an X -Neuronal system for stimulus S_c using neuronal set $[N]_{S_c}$ at time $t = t_0$. The memory of cognitive process $[CP_p]_{type\ 1}$ can be generated by X -Neuronal system at time $t > t_0$ using n number of neuronal sets, with a unique neuronal set represented as $[\mathcal{M}_m]_{[\mathcal{R}]_{S_c}}$. Furthermore, for memory generation of $[CP_p]_{type\ 1}$, all n neuronal sets will have neurons whose output response are the elements of set $[\mathcal{R}]_{S_c}$.

9.3. Impaired Memories

In this section, the concept of impaired memories in the context of neuronal state theory will be discussed. Consider $[\mathcal{R}]_{S_{ext_3}}$ (equation 124) which by abstraction rules of colored dices, $[\mathcal{R}]_{S_{ext_3}}$ represents 6 dices with colors {Red, Orange, Yellow, White, Green, Blue} respectively. In the example case, memory of $[CP_5]_{type\ 1}$ i.e. generation of $[\mathcal{R}]_{S_{ext_3}}$ was generated by cognitive process $[CP_6]_{type\ 1}$ at time $t = t_4$. Now consider at completion of cognitive process $[CP_6]_{type\ 1}$ at time $t = t_4$, X -Neuronal system generated $[\mathcal{R}]_{S_{int_2}}$ given by equation 127 instead of $[\mathcal{R}]_{S_{int_2}}$ (equation 126).

$$[\mathcal{R}]_{S_{int_2}} = \{ [O_{C_1}]_{E_{300}}, [O_{C_1}]_{E_{301}}, [O_{C_3}]_{E_{302}}, [O_{C_4}]_{E_{303}}, [O_{C_5}]_{E_{304}}, [O_{C_6}]_{E_{305}} \} \quad 127$$

Using abstraction of colored dices, $[\mathcal{R}]_{S_{int_2}}$ (equation 127) represents same 6 unique dices as $[\mathcal{R}]_{S_{ext_3}}$, but the color of one dice is red instead of orange i.e. neuron E_{301_x} generated output response $[O_{C_1}]_{E_{301}}$ instead of $[O_{C_2}]_{E_{301}}$. This is the foundational concept of impaired memory

in the context of neuronal state theory, where same neurons $\{E_{300_x}, E_{301_x}, E_{302_x}, E_{303_x}, E_{304_x}, E_{305_x}\}$ produce output responses at $t = t_4$ for cognitive process $[CP_6]_{type\ 1}$, which produced output responses at $t = t_2$ for cognitive process $[CP_5]_{type\ 1}$. However, one neuron E_{301_x} generated a different output response i.e. $[O_{C_1}]_{E_{301}}$ instead of $[O_{C_2}]_{E_{301}}$. In other words, X -Neuronal system generated an impaired memory of cognitive process $[CP_5]_{type\ 1}$ at time $t = t_4$, where one of the elements of set $[\mathcal{R}]_{S_{int_2}}$ (equation 127) produced by the same neuron is different from the element of set $[\mathcal{R}]_{S_{ext_3}}$. Since $[\mathcal{R}]_{S_{ext_3}}$ and $[\mathcal{R}]_{S_{int_2}}$ comprises of 6 elements, the memory of $[CP_5]_{type\ 1}$ generated by X -Neuronal system at time $t = t_4$ is $\frac{1}{6} \cdot 100 = 16.67\%$ impaired. Generalisation of impaired memories is given by the following statement:

Given a stimulus response $[\mathcal{R}]_{S_c}$ of an arbitrary type 1 cognitive process $[CP_p]_{type\ 1}$ generated by an X -Neuronal system for stimulus S_c using neuronal set $[N]_{S_c}$ at time $t = t_0$. Impaired memory of $[CP_p]_{type\ 1}$ is the generation of the output responses at time $t > t_0$ by same neurons which produced output responses of set $[\mathcal{R}]_{S_c}$ at time $t = t_0$. However, one or more generated output responses at time $t > t_0$ are different from set $[\mathcal{R}]_{S_c}$.

Let the percentage of memory impairment be represented by $[\%JM]_{[\mathcal{R}]_{S_c}}$ and is defined by equation 128.

$$[\%JM]_{[\mathcal{R}]_{S_c}} = \frac{\text{no.of generated output responses different from } [\mathcal{R}]_{S_c}}{\text{Total number of elements of } [\mathcal{R}]_{S_c}} \cdot 100 \quad 128$$

9.4. Likelihood of Memory Generation

In this section, the likelihood of memories generation by X -Neuronal system will be discussed. The concepts of state adaptabilities and probabilities of output responses of neurons, discussed in part 1 of this article are the foundations to discuss the likelihood of memory generation by X -Neuronal system.

From example case, consider memory of cognitive process $[CP_5]_{type\ 1}$ generated by X -Neuronal system at time $t = t_4$, using neuronal set $[\mathcal{M}_1]_{[\mathcal{R}]_{S_{ext_3}}}$. As discussed previously, n number of neuronal sets can generate memory of $[CP_5]_{type\ 1}$ and all of these n neuronal sets have neurons $\{E_{300_x}, E_{301_x}, E_{302_x}, E_{303_x}, E_{304_x}, E_{305_x}\}$, which produce output responses of set $[\mathcal{R}]_{S_{ext_3}}$. So, the likelihood of memory generation of $[CP_5]_{type\ 1}$ depends on the neurons $\{E_{300_x}, E_{301_x}, E_{302_x}, E_{303_x}, E_{304_x}, E_{305_x}\}$, regardless of which neuronal set $[\mathcal{M}_m]_{[\mathcal{R}]_{S_{ext_3}}}$ they belong to. In order to generate memory of $[CP_5]_{type\ 1}$, these neurons have to produce specific outputs. For instance, neuron E_{300_x} has to generate output response $[O_{C_1}]_{E_{300}}$. Neuron E_{300_x} has a certain probability of generating output response $[O_{C_1}]_{E_{300}}$ at any given time t defined by $[\psi_{O_{C_1}}]_{E_{300}}$. Since neuron E_{300_x} is treated as ‘Model Neuron’ which behaves ideally $\left[\frac{\beta}{\alpha}\right] = 1$ in time interval $0 \leq t \leq \infty$, the probability of output response $[\psi_{O_{C_1}}]_{E_{300}}$ will always be equal to the state adaptability of state which corresponding to output response $[O_{C_1}]_{E_{300}}$. So, $[\psi_{O_{C_1}}]_{E_{300}}$ is equal to the state adaptability $[\Omega_{x_{AP_1}}]_{E_{300}}$ of state x_{AP_1} i.e. $[\psi_{O_{C_1}}]_{E_{300}} = [\Omega_{x_{AP_1}}]_{E_{300}}$. The value of $[\Omega_{x_{AP_1}}]_{E_{300}}$ at any given time t is governed by state adaptability factors $[\eta_{x_{AP_1}}]_{E_{300}}$. If $[\Omega_{x_{AP_1}}]_{E_{300}}$ at any given time t is high, $[\psi_{O_{C_1}}]_{E_{300}}$ is also high. Similarly, other neurons $\{E_{301_x}, E_{302_x}, E_{303_x}, E_{304_x}, E_{305_x}\}$ also have probabilities of output responses which comprises of set $[\mathcal{R}]_{S_{ext_3}}$ at any given time t equal to state adaptabilities of states which

corresponds to required output responses for memory generation of $[CP_5]_{type\ 1}$. Let the likelihood of memory generation of $[CP_5]_{type\ 1}$ be represented by $[\mathcal{LM}]_{[\mathcal{R}]_{S_{ext3}}}$. The likelihood of memory generation of $[CP_5]_{type\ 1}$ by X -Neuronal system at any given time $t > t_2$ is given by equation 129.

$$[\mathcal{LM}]_{[\mathcal{R}]_{S_{ext3}}} = [\psi_{OC_1}]_{E_{300}} \cdot [\psi_{OC_2}]_{E_{301}} \cdot [\psi_{OC_3}]_{E_{302}} \cdot [\psi_{OC_4}]_{E_{303}} \cdot [\psi_{OC_5}]_{E_{304}} \cdot [\psi_{OC_6}]_{E_{305}}$$

129

If the probabilities of output responses which comprises of set $[\mathcal{R}]_{S_{ext3}}$ at any given time $t > t_2$ are high, the likelihood that X -Neuronal system will generate memory of $[CP_5]_{type\ 1}$ will be high at that instance of time and vice versa. The generalisation of likelihood of memory generation by X -Neuronal system is given by the following statement:

Given a stimulus response $[\mathcal{R}]_{S_c}$ of an arbitrary type 1 cognitive process $[CP_p]_{type\ 1}$ generated by an X -Neuronal system for stimulus S_c using neuronal set $[\mathcal{N}]_{S_c}$ at time $t = t_0$. Furthermore, $[\mathcal{R}]_{S_c}$ comprises of $\#$ number of elements, where each element is labelled by h and $h = 1, 2, 3, \dots, \#$. The likelihood of memory generation of $[CP_p]_{type\ 1}$ represented as $[\mathcal{LM}]_{[\mathcal{R}]_{S_c}}$ by X -Neuronal system at a given time $t > t_0$ is the product of probabilities of output responses which comprise set $[\mathcal{R}]_{S_c}$, given by equation 130.

$$[\mathcal{LM}]_{[\mathcal{R}]_{S_c}} = \prod_{h=1}^{\#} (\psi_h) \quad 130$$

9.5. Short-Term and Long-Term Memories

In this section, short-term and long-term memories in the context of neuronal state theory will be discussed. Consider stimulus response $[\mathcal{R}]_{S_{ext_3}}$ (equation 124) of cognitive process $[CP_5]_{type\ 1}$ generated by X -Neuronal system at time $t = t_2$. It has been previously established that memory generation of cognitive process $[CP_5]_{type\ 1}$ depends on neurons $\{E_{300_x}, E_{301_x}, E_{302_x}, E_{303_x}, E_{304_x}, E_{305_x}\}$ generating output responses of set $[\mathcal{R}]_{S_{ext_3}}$ at time $t > t_2$. Memory of cognitive process $[CP_5]_{type\ 1}$ being categorised short-term or long-term depends on the ability of the neurons $\{E_{300_x}, E_{301_x}, E_{302_x}, E_{303_x}, E_{304_x}, E_{305_x}\}$ to generate output responses of set $[\mathcal{R}]_{S_{ext_3}}$. If neurons can generate output responses of set $[\mathcal{R}]_{S_{ext_3}}$ for a long period of time beyond $t > t_2$, memory of $[CP_5]_{type\ 1}$ will be categorised as long-term memory. On the contrary, if neurons can generate output responses of set $[\mathcal{R}]_{S_{ext_3}}$ for only a short period of time beyond $t > t_2$, memory of $[CP_5]_{type\ 1}$ will be categorised as short-term memory. To define a proper distinction between short-term and long-term memory, consider a time interval $t_2 < t \leq t_{short}$. If neurons $\{E_{300_x}, E_{301_x}, E_{302_x}, E_{303_x}, E_{304_x}, E_{305_x}\}$ can only produce output responses of set $[\mathcal{R}]_{S_{ext_3}}$ in the time interval $t_2 < t \leq t_{short}$, memory of $[CP_5]_{type\ 1}$ will be categorised as short-term memory. However, if neurons $\{E_{300_x}, E_{301_x}, E_{302_x}, E_{303_x}, E_{304_x}, E_{305_x}\}$ can produce output responses of set $[\mathcal{R}]_{S_{ext_3}}$ beyond time interval $t_2 < t \leq t_{short}$, memory of $[CP_5]_{type\ 1}$ will be categorised as long-term memory. The generalisation of short-term and long-term memories is given by following statement:

Given a stimulus response $[\mathcal{R}]_{S_c}$ of an arbitrary type 1 cognitive process $[CP_p]_{type\ 1}$ generated by an X -Neuronal system for stimulus S_c using neuronal set $[N]_{S_c}$ at time $t = t_0$. Memory of $[CP_p]_{type\ 1}$ is categorised as short-term, if neurons whose output responses comprises of

set $[\mathcal{R}]_{S_c}$ can only produce output responses of set $[\mathcal{R}]_{S_c}$ in the time interval $t_0 < t \leq t_{short}$.
Memory of $[CP_p]_{type\ 1}$ is categorised as long-term, if neurons whose output responses comprises of set $[\mathcal{R}]_{S_c}$ can produce output responses of set $[\mathcal{R}]_{S_c}$ beyond the time interval $t_0 < t \leq t_{short}$.

10. Learning

In this section, the concepts of learning in the context of neuronal state theory will be discussed. Firstly, a single neuron of an X -Neuronal system will be used to establish foundational concepts of learning and later these concepts will be generalised for the whole X -Neuronal system.

Consider an arbitrary receptor neuron R_{j_x} in rest state at time $t = 0$. Assume that at time $t = 0$, R_{j_x} is a new-born neuron, which is yet to attain any activated state and generate any output response. Assume at time $t = t_1$, neuron R_{j_x} attained an AP state from set $[\mathbf{x}_{AP}]_{R_j}$ (equation 66) after receiving an arbitrary stimulus S_c and at time $t = t_2$ generated its first ever output response from set $[\mathbf{O}_C]_{R_j}$ (equation 61) out of the possible $[b]_{R_j}$ (equation 63) output responses. With time, neuron R_{j_x} continues to attain AP and ESC states from sets $[\mathbf{x}_{AP}]_{R_j}$ and $[\mathbf{x}_{ESC}]_{R_j}$ (equation 67) after receiving stimuli and continues to generate output responses from sets $[\mathbf{O}_C]_{R_j}$ and $[\mathbf{O}_F]_{R_j}$ (equation 62). The generated output responses can be unique or they can be output responses which were previously generated. For the foundations of learning, only the unique output responses generated by neuron R_{j_x} are important. The total number of possible output responses $[b]_{R_j}$ are also the number of unique output responses neuron R_{j_x} can generate. After neuron's R_{j_x} birth at time $t = 0$, let the number of unique output responses R_{j_x} has generated so far at a given time t be defined by $[\vartheta]_{R_j}$. As an example, assume for neuron R_{j_x} , $[b]_{R_j} = 100$ and at time $t = 5$, R_{j_x} has generated 10 unique output responses so far out of the total unique outputs $[b]_{R_j} = 100$. Therefore, at time $t = 5$, $[\vartheta]_{R_j} = 10$. Generation of unique output responses by neuron R_{j_x} is the foundation of learning of neuron R_{j_x} . In the context of neuronal state theory, learning of neuron R_{j_x} is defined by the following statement:

Learning of neuron R_{j_x} is the generation of unique output responses by R_{j_x} with time t .

In other words, neuron R_{j_x} learns when it generates a unique output response and as it continues to generate unique output responses, it continues to learn. While neuron R_{j_x} continues to learn with time, the value of $[\vartheta]_{R_j}$ approaches to $[b]_{R_j}$. The total number of unique output responses $[b]_{R_j}$ is the learning limit of neuron R_{j_x} . For instance, assume neuron R_{j_x} has generated all possible unique outputs $[b]_{R_j}$ at time $t = 1000$. So at time $t = 1000$, $[\vartheta]_{R_j} = [b]_{R_j}$, neuron R_{j_x} has reached its learning limit. Beyond time $t > 1000$, neuron R_{j_x} will continue to generate repeated output responses out of $[b]_{R_j}$. *Learning is the generation of output response out of $[b]_{R_j}$ for the first time.* In the context of neuronal state theory, learning is a concept rooted at individual neuronal level. Similarly other types of neurons (I_{k_x}, E_{l_x}) learn in a similar way.

10.1. Relationship between Learning and Time

Now that the concept of learning is established, a relationship of learning with respect to time will be discussed. Neuron R_{j_x} and concepts of ideal and real neuronal behaviour will be used for the discussion of learning and time relationship.

As discussed previously, R_{j_x} generated its first output response from set $[\mathbf{O}_C]_{R_j}$ at time $t = t_2$, resulting from attaining an AP state from set $[\mathbf{x}_{AP}]_{R_j}$, at time $t = t_1$. In the time interval $0 < t \leq t_2$, R_{j_x} exhibited ideal behaviour i.e. $\left[\frac{\beta}{\alpha}\right]_{R_j} = 1$ (equation 69). With time, R_{j_x} keeps attaining activated states from sets $[\mathbf{x}_{AP}]_{R_j}$ and $[\mathbf{x}_{ESC}]_{R_j}$ and keeps generating output responses from sets $[\mathbf{O}_C]_{R_j}$ and $[\mathbf{O}_F]_{R_j}$. It is a reasonable hypothesis that generally for any neuron, number of output producing activated states will be greater than the number of possible outputs and therefore for neuron R_{j_x} it is a reasonable assumption that $[M]_{R_j} + [Q]_{R_j} > [V]_{R_j} + [Z]_{R_j}$. So, at a certain time, R_{j_x} will transition to exhibiting real behaviour i.e. $\left[\frac{\beta}{\alpha}\right]_{R_j} < 1$. In the context of learning, the ability of a neuron R_{j_x} to produce unique output

responses will decrease with time, as more than one output producing states will start producing the same output response. It is very reasonable to hypothesize that, after the birth of neuron R_{j_x} , the ability of R_{j_x} to produce more unique output responses will be high and R_{j_x} will learn faster. With time, the ability of R_{j_x} to produce unique output responses decreases, hence learning slows down with time. A relationship between learning and time should have the essence of this hypothesis. Keeping this hypothesis in mind, the relationship between learning and time for neuron R_{j_x} is given by equation 131, where $[\dot{\mathcal{L}}]_{R_j}$ represents learning of neuron R_{j_x} and $[\mathcal{C}]_{R_j}$ represents learning constant of neuron R_{j_x} . The value of learning constant $[\mathcal{C}]_{R_j}$ depends on the ability of the neuron on average in time interval $0 < t < \infty$ to produce unique output responses. If the average ability of neuron is higher, the value of learning constant is higher $[\mathcal{C}]_{R_j}$ and vice versa. For simplicity, the detailed relationship between $[\mathcal{C}]_{R_j}$ and average ability of neuron to produce unique output responses will not be discussed.

$$[\dot{\mathcal{L}}]_{R_j} = [b]_{R_j} \cdot \left(\frac{[\mathcal{C}]_{R_j} \cdot t}{[\mathcal{C}]_{R_j} \cdot t + 1} \right) \quad 131$$

The rate of learning of neuron R_{j_x} defined by $[\dot{\mathcal{L}}]_{R_j}$ is the derivative of learning $[\mathcal{L}]_{R_j}$ with respect to time t , given by equation 132.

$$[\dot{\mathcal{L}}]_{R_j} = \frac{[b]_{R_j} \cdot [\mathcal{C}]_{R_j}}{([\mathcal{C}]_{R_j} \cdot t + 1)^2} \quad 132$$

Now the concept of learning potential of a neuron R_{j_x} will be discussed. A new-born neuron R_{j_x} at time $t = 0$, has the learning potential $[\mathcal{L}P]_{R_j}$ of 1, since it is yet to produce any unique output response or learn. With time, as neuron starts learning, the learning potential starts decreasing. If neuron R_{j_x} has produced all unique output responses $[b]_{R_j}$, the learning potential of R_{j_x} will be 0. So, for neuron R_{j_x} , the value of $[\mathcal{L}P]_{R_j}$ is between 1 and 0 i.e. $1 > [\mathcal{L}P]_{R_j} > 0$.

Furthermore, $[L]_{R_j}$ is inversely proportional to learning $[L]_{R_j}$. The relationship between learning potential $[LP]_{R_j}$ and time t is given by equation 133.

$$[LP]_{R_j} = 1 - \left(\frac{[\mathcal{L}]_{R_j} \cdot t}{[\mathcal{L}]_{R_j} \cdot t + 1} \right) \quad 133$$

Equations 131, 132 and 133 are applicable to all three types of neurons with the right labelling of neurons.

10.2. Learning of an X-neuronal system

Now that the learning concepts of a single neuron has been established, these concepts will be used to discuss learning an arbitrary X-neuronal system.

Consider an X-neuronal system which comprises of all three types of neurons (equation 85). The total number of unique output responses of an X-neuronal system are given by $[b]_X$ (equation 87) representing the sum of all unique output responses of neurons comprising X-neuronal system. Since learning happens at neuron level, learning of an X-neuronal system depends on the learning of neurons, which makes up the system. As neurons of X-neuronal system learn with time, X-neuronal system as a whole learns i.e. generation of unique output responses out of total $[b]_X$. *The learning limit of any arbitrary X-neuronal system is equal to the total number of unique output responses $[b]_X$.* If all the neurons of X-neuronal system have reached their individual learning limit, the system has reached its learning limit. Using equation 131, the learning of an X-neuronal system $[L]_X$ with time t is given by 134.

$$[L]_X = \sum_{j=1}^p [b]_{R_j} \left(\frac{[\mathcal{L}]_{R_j} \cdot t}{[\mathcal{L}]_{R_j} \cdot t + 1} \right) + \sum_{k=1}^q [b]_{I_k} \left(\frac{[\mathcal{L}]_{I_k} \cdot t}{[\mathcal{L}]_{I_k} \cdot t + 1} \right) + \sum_{l=1}^r [b]_{E_l} \left(\frac{[\mathcal{L}]_{E_l} \cdot t}{[\mathcal{L}]_{E_l} \cdot t + 1} \right) \quad 134$$

Likewise, the rate of learning $[\dot{L}]_X$ and learning potential $[LP]_X$ of X -neuronal system with time t is given by equations 135 and 136, respectively.

$$[\dot{L}]_X = \sum_{j=1}^p \frac{[b]_{R_j} \cdot [\mathcal{L}]_{R_j}}{([\mathcal{L}]_{R_j} \cdot t + 1)^2} + \sum_{k=1}^q \frac{[b]_{I_k} \cdot [\mathcal{L}]_{I_k}}{([\mathcal{L}]_{I_k} \cdot t + 1)^2} + \sum_{l=1}^r \frac{[b]_{E_l} \cdot [\mathcal{L}]_{E_l}}{([\mathcal{L}]_{E_l} \cdot t + 1)^2} \quad 135$$

$$[LP]_X = \sum_{j=1}^p 1 - \left(\frac{[\mathcal{L}]_{R_j} \cdot t}{[\mathcal{L}]_{R_j} \cdot t + 1} \right) + \sum_{k=1}^q 1 - \left(\frac{[\mathcal{L}]_{I_k} \cdot t}{[\mathcal{L}]_{I_k} \cdot t + 1} \right) + \sum_{l=1}^r 1 - \left(\frac{[\mathcal{L}]_{E_l} \cdot t}{[\mathcal{L}]_{E_l} \cdot t + 1} \right) \quad 136$$

A typical relationship between learning $[L]_X$ of an arbitrary X -neuronal system with time t is shown in Figure 32. The relationship curve shown in Figure 32 is particularly insightful, which signifies that X -neuronal system learns faster, initially and with time learning slows down. If one consider X -neuronal system to be the nervous system of a human with billions of neurons, the learning curve of figure 32 represents the learning of a human nervous system with time. Learning curve signifies that at early stage as a baby, rate of learning of neurons of nervous system and system as a whole is faster. With time, representing human aging, the rate of learning slows down. *This is a very significant and insightful feature of neuronal state theory which signifies that at early stages of life, humans learn faster and with time learning slows down.*

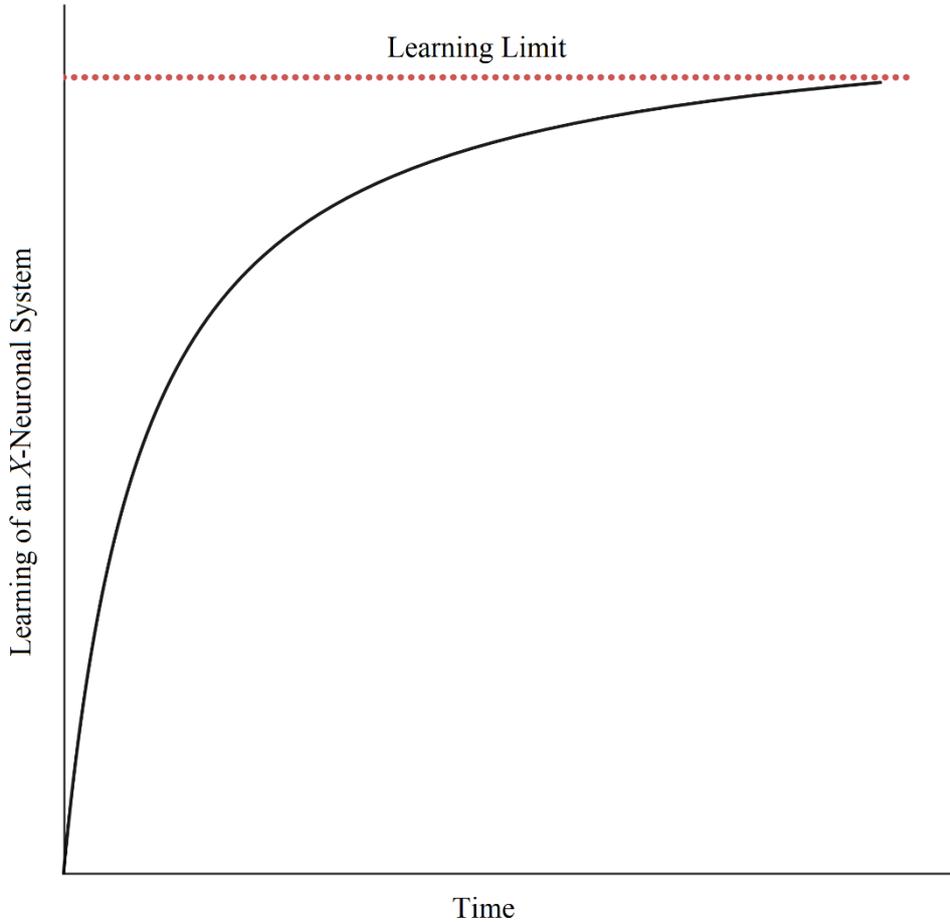


Figure 32. Typical learning curve $[L]_X$ of an arbitrary X -neuronal system with respect to time t . As time increases, learning curve starts becoming asymptotic to the learning limit $[b]_X$ of X -neuronal system.

10.3. Imagination

In this section, the concept of imagination will be discussed using the context of neuronal state theory. Imagination in its essence is one of the forms of learning. As discussed previously, learning of X -neuronal system is the generation of unique output responses of neurons of X -neuronal system with time. For learning in general, X -neuronal system can receive both external and internal stimuli from sets $\mathbf{S}_{external}$ (equation 98) and $\mathbf{S}_{internal}$ (equation 99), respectively, which in turn will cause the neurons to attain AP and ESC states and potentially generate unique output responses. However, in imagination - a form of learning, neurons of X -

neuronal system generate unique output responses only after receiving internal stimuli from set $\mathbf{S}_{internal}$. In general, imagination is the generation of unique output responses triggered by internal stimuli. Furthermore, not all neurons of X -neuronal system are capable of imagination. As an example, receptor neurons in skin are not capable of imagination. To serve this purpose, consider a set \mathbf{Im} which represents neurons of X -neuronal system capable of imagination. Set \mathbf{Im} is a subset of set \mathbf{N} i.e. $\mathbf{Im} \subseteq \mathbf{N}$. The generalised definition of imagination for an arbitrary X -neuronal system is given by the following statement:

Imagination - a form of learning is the generation of unique output responses with time t by neurons of set $\mathbf{Im} \subseteq \mathbf{N}$ triggered by the internal stimuli from set $\mathbf{S}_{internal}$.

10.4. Talent

In this section, notion of talent will be discussed using the framework of neuronal state theory. Firstly, a single neuron will be used to discuss necessary concepts of talent and later these concepts will be generalised for any arbitrary X -neuronal system.

Consider a receptor neuron R_{j_x} with total number of unique output responses equal to $[b]_{R_j}$. Neuron R_{j_x} is only capable of generating $[b]_{R_j}$ number of possible output responses. In other words, the numerical value of $[b]_{R_j}$ is the output response producing capability of neuron R_{j_x} , which R_{j_x} receives from the genetics of R_{j_x} containing organism. To elaborate this notion further, consider two organisms A and B who are stimulated by same external stimuli of light of increasing intensities at the same intervals of time. Furthermore, assume that the function of neuron R_{j_x} is to receive light stimuli and produce an output response, which will act as stimulus for other some other arbitrary neuron. Both organisms A and B will use their version of neuron R_{j_x} to receive light stimuli. Furthermore, assume organism A version of R_{j_x} has $[b]_{R_j} =$

10 and organism B version of R_{j_x} has $[b]_{R_j} = 5$, which depends on the genetics of the organism A and B, respectively. After receiving light stimuli, both versions of R_{j_x} will produce output responses. However, Organism A version of R_{j_x} can produce more unique output responses as compare to organism B. since $[b]_{R_j} \text{ of } A > [b]_{R_j} \text{ of } B$. In other words, organism A version of R_{j_x} is more capable of producing unique output responses as compare to organism B version, for same stimuli. It is very reasonable to say organism A version of R_{j_x} is more talented than organism B version of R_{j_x} for receiving light stimuli. Talent in the context of neuronal state theory is a neuron level feature and for neuron R_{j_x} is defined by the following statement:

Talent of neuron R_{j_x} is the unique output producing capability of R_{j_x} to produce unique output responses for light stimuli. The higher the output response producing capability of R_{j_x} , the higher the talent, neuron R_{j_x} has to receive light stimuli.

The above discussion is valid for all three types of neurons. Using the above discussion, talent of X-neuronal system will be discussed. Consider an arbitrary X-neuronal system which has to carry out a function \mathcal{F} . X-neuronal system uses a set of neurons defined by $[\mathbf{N}]_{\mathcal{F}}$ to carry out \mathcal{F} . The talent of X-neuronal system for function \mathcal{F} is defined by the following statement:

Talent of X-neuronal system for function \mathcal{F} is the capabilities of neurons of set $[\mathbf{N}]_{\mathcal{F}}$ to produce unique output responses. The higher the capabilities of neurons of set $[\mathbf{N}]_{\mathcal{F}}$, higher the talent X-neuronal system has for function \mathcal{F} .

11. Sleep

In this section, notion of sleep in the context of neuronal state theory will be discussed. The concept of capacity $[\mathbb{C}]_t$ of X -neuronal system to run cognitive process at a given time is the foundational framework to discuss sleep. It has been previously established that capacity $[\mathbb{C}]_t$ depends on the number of cognitive process NCP ran by X -neuronal system at a given time, out of $[NCP]_{max}$ (equation 111). Alternatively, $[\mathbb{C}]_t$ depends on the availability of ions $[\dot{w}]_t$ at any given time, out of \dot{W} (equation 112). To dwell further, an X -neuronal system requires the availability of certain specific ionic species to run certain cognitive processes. As an example, if the neurotransmitters involved in a certain cognitive process makes Na^+ ions more permeable, then X -neuronal system requires Na^+ ions to run that cognitive process. To discuss the notion of sleep, at first only Na^+ ionic species will be used and later the notion will be discussed for all responsible ionic species.

As discussed previously, a neuron maintains a rest state by having low concentration of Na^+ ions inside it. To attain activated state, Na^+ ions move inside the neuron. From 3-Component model, movement of ions inside the neuron are labelled as specialised workers. Therefore, for neuron to attain activated states, Na^+ ions act as specialised workers. Now consider an X -neuronal system with all of its neurons in rest state at time $t = 0$. Based on Na^+ ionic specie alone, all the neurons of X -neuronal system have low concentration of Na^+ ions inside them. Furthermore, consider that X -neuronal system is a subsystem of an organism. In other words, X -neuronal system is a part of an organism. At time $t = 0$, concentration of Na^+ ions outside the neurons of X -neuronal system is high. Let the organism be given by a System, which has two subsystem 1 and 2, as shown in Figure 33. Subsystem 1 represents X -neuronal system and subsystem 2 represents the entire organism without the X -neuronal system.

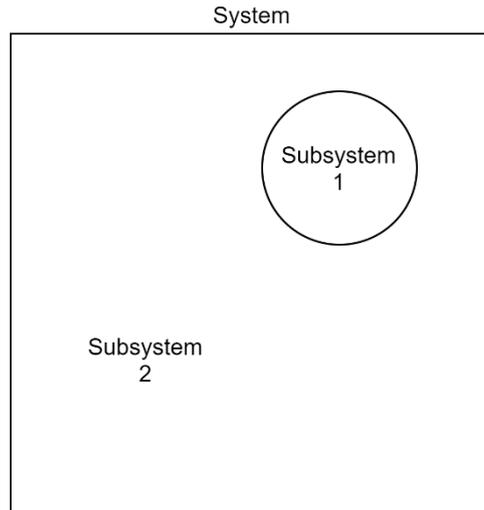


Figure 33. Representation of Organism as a system with subsystem 1 and subsystem 2. Subsystem 1 represents X -neuronal system and subsystem 2 represents the entire organism without the X -neuronal system.

Let the total number of Na^+ ions in system at a given time t be defined by $[\text{Na}^+]_{total}$. Furthermore, let the total number of Na^+ ions in subsystem 1 and 2 at a given time t be given by $[\text{Na}^+]_1$ and $[\text{Na}^+]_2$, respectively. The total number of Na^+ ions in system at a given time t is given by equation 137.

$$[\text{Na}^+]_{total} = [\text{Na}^+]_1 + [\text{Na}^+]_2 \quad 137$$

So at time $t = 0$, $[\text{Na}^+]_2 \gg [\text{Na}^+]_1$ since neurons of subsystem 1 are in rest state having low concentration/ number of Na^+ ions inside them. Furthermore, the number of Na^+ ions in the subsystem 2 are the ions available to X -neuronal system to run cognitive processes.

With time i.e. $t > 0$, subsystem 1 or X -neuronal system starts to run cognitive processes of type 1 and 2, after receiving stimuli, which cause the influx of Na^+ ions in subsystem 1 from subsystem 2. Let the rate of influx of Na^+ ions in subsystem 1 be given by $[\dot{\text{Na}}^+]_1$. When type 1 cognitive processes are complete, some of the involved neurons in those processes return to their rest state, which causes outflux of Na^+ ions from subsystem 1 to subsystem 2. Let the rate

outflux of Na^+ ions from subsystem 1 be given by $[\dot{N}a^+]_2$. Furthermore, some of the involved neurons in the processes remain in subthreshold states, holding Na^+ ions inside them. So at any given time t , there is an influx and outflux of Na^+ ions to and from subsystem 1. Let the rate of influx and outflux of Na^+ ions to and from subsystem 1 at any given time t be defined by net flux $[\dot{N}a^+]_{net}$ given by equation 138.

$$[\dot{N}a^+]_{net} = [\dot{N}a^+]_1 - [\dot{N}a^+]_2 \quad 138$$

The net flux $[\dot{N}a^+]_{net}$ depends on the number of cognitive processes run by subsystem 1 at a given time from receiving stimuli i.e. $[\dot{N}a^+]_{net} \propto NCP$. As subsystem 1 receive more stimuli, more cognitive processes are started, which in turn cause influx of Na^+ ions in the neurons of subsystem 1 with higher influx rate, resulting in the increase in $[\dot{N}a^+]_{net}$. It should be noted that at the same time, there is outflux of Na^+ ions as well, but influx rate is greater than outflux rate, so on average net flux increases. The net flux $[\dot{N}a^+]_{net}$ then affects the number of Na^+ ions in subsystem 1 and 2. With high $[\dot{N}a^+]_{net}$, the value of $[Na^+]_1$ increases and the value of $[Na^+]_2$ decreases. In addition to that, subsystem 1 also run type 2 cognitive processes, where involved neurons hold Na^+ ions inside them as subthreshold states. So, not all Na^+ ions return to subsystem 2, even when type 2 cognitive processes are complete. With time, as subsystem 1 continues to receive stimuli, the availability of Na^+ ions to ran cognitive processes decrease, because the number of Na^+ ions in subsystem 2 decreases. Based on Na^+ ions alone, the capacity $[C]_t$ of subsystem 1 to run cognitive processes decreases with time. The decrease in the capacity $[C]_t$ of subsystem 1 to run cognitive processes, because of the lack of availability of ions is the foundation for the notion of sleep.

11.1. Awake State

Now the concept of awake state in the context of neuronal state theory will be defined. Awake State is the ability of organism to receive external stimuli from the environment. Following the convention of current discussion, the stimuli comes from outside the system (figure 33) during awake state.

Consider at time $t = 0$, system shown in figure 33 is in awake state. With time $t > 0$, system receives external stimuli and subsystem 1 starts to run cognitive processes of type 1 and 2. This causes the influx and outflux of Na^+ ions to and from subsystem 1. However, during awake state, influx rate of Na^+ ions is higher than outflux rate, resulting in positive net flux i.e. $+\dot{[Na^+]_{net}}$. During awake state, the value of net flux keeps fluctuating with time, depending on the start and completion of cognitive processes, but will always be positive. This results in the increase in $[Na^+]_1$ and decrease in $[Na^+]_2$ during awake state.

11.2. Somnolence State

With time, $[Na^+]_1$ continue to increase and $[Na^+]_2$ continue to decrease and at one point of time, $[Na^+]_1$ becomes considerably larger than $[Na^+]_2$ i.e. $[Na^+]_1 \gg [Na^+]_2$. The availability of Na^+ ions in subsystem 2, required by subsystem 1 to start new cognitive process becomes scarce. In other words, $[\mathbb{C}]_t$ of subsystem 1 is significantly reduced. Once the capacity $[\mathbb{C}]_t$ of subsystem 1 is reduced below a certain threshold value defined by $[\mathbb{C}_{threshold}]_t$, subsystem 1 starts signalling organism/system to decrease receiving external stimuli. System goes into Somnolence state which is defined by the following state:

Somnolence state is a response of the system triggered when the capacity $[\mathbb{C}]_t$ of subsystem 1 falls below $[\mathbb{C}_{threshold}]_t$ because of the lack of availability of ions in subsystem 2 to start and run cognitive processes. In somnolence state, system reduce receiving external stimuli.

In somnolence state, the net flux $[\dot{Na}^+]_{net}$ although continue to fluctuate depending on the starting and completion of cognitive processes, but on average continue to decrease since the rate of influx $[\dot{Na}^+]_1$ decreases. The capacity of subsystem 1 still remains below $[C_{threshold}]_t$ and $[Na^+]_1$ continue to increase at a slower rate.

11.3. Sleep State

When the capacity $[C]_t$ of subsystem 1 falls further below $[C_{threshold}]_t$ to a certain value defined by $[C_{sleep}]_t$, subsystem 1 triggers system/organism to practically stop receiving external stimuli, because subsystem 1 is practically unable to start any cognitive processes, due to the scarcity of Na^+ ions in subsystem 2. As a result, system enters the sleep state. However, system in sleep state can still receive some external stimuli of high intensity. As an example, if one considers system as a human organism, then system can receive external stimuli like loud sound, high intensity lights, high temperatures. These high intensity stimuli can trigger system into somnolence or awake state. So in sleep state, a system can still receive some external stimuli. Let the external stimuli a system can receive even in sleep state be defined by a set $[S_{external}]_{SS}$. Set $[S_{external}]_{SS}$ is a subset of set $S_{external}$ (equation 98). In the context of neuronal state theory, sleep state of a system is defined by the following statement:

Sleep state is a response of the system triggered when the capacity $[C]_t$ of subsystem 1 falls below $[C_{sleep}]_t$ because of the lack of availability of ions in subsystem 2 to start and run cognitive processes. In sleep state, system practically stops receiving external stimuli, except external stimuli of set $[S_{external}]_{SS}$.

In sleep state, subsystem 1 practically stops the starting of new cognitive processes because of external stimuli. This decrease the influx of Na^+ ions in subsystem 1 and hence net flux on

average decreases further. In sleep state, function of subsystem 1 is rather unique and insightful, which will be the foundation of dreams discussed in the later section. In sleep state, subsystem 1 frees up Na^+ ions held in the neurons of subsystem 1. This is achieved by subsystem 1 by completing previously running cognitive processes. Furthermore, subsystem 1 start new cognitive processes, where neurons in subthreshold states holding Na^+ ions are stimulated by internal stimuli. When these cognitive processes are completed, neurons start returning to rest state, freeing up held Na^+ ions and these ions start moving to subsystem 2 from 1. So, in sleep state, rate of outflux is greater than rate of influx i.e. $[\dot{N}a^+]_2 \gg [\dot{N}a^+]_1$. As a result, the net flux, which although fluctuating in value because of influx and outflux rates is on average negative i.e. $-[\dot{N}a^+]_{net}$. Furthermore, in sleep state, the capacity $[\text{C}]_t$ of subsystem 1 increases, as $[\text{Na}^+]_2$ increases.

As capacity $[\text{C}]_t$ of subsystem 1 continue to increase during sleep state and reach a certain value defined by $[\text{C}_{awake}]_t$. Beyond $[\text{C}_{awake}]_t$, subsystem 1 becomes fully functional to start new cognitive processes for external stimuli. To serve this purpose, system is triggered to start receiving external stimuli again and hence system again enters awake state. At this point, system completes one awake state-sleep state (AS-SS) cycle and enters next (AS-SS) cycle.

In a nutshell, the availability of responsible ionic species for subsystem 1 to run cognitive processes triggers the awake, somnolence and sleep states of the system. If the availability is high, system remains in awake state and if the availability is scares, the system enters sleep state to free up ionic species held in subsystem 1, resulting in the increase in availability.

11.4. Availability of Ionic Species

So far the notions of different states of system have been discussed using only Na^+ ions. In this sections, all ionic species which are responsible to run cognitive processes will be used to discuss the ideas of previous section.

Consider a system which comprises of two subsystem 1 and 2, as shown in Figure 33, where subsystem 1 represents X -neuronal system and subsystem 2 represents the entire organism without the X -neuronal system. Let the total number of ions of all ionic species responsible to run cognitive processes in a system is defined by ω_{total} and is given by equation 139. In equation 139, $\mathfrak{X}_{\mathbb{P}}$ represents an individual ionic specie and \mathbb{Q} represents the total number of ionic species responsible to run cognitive processes. Furthermore, $\mathfrak{U}_{\mathbb{P}}$ represents the total number of ions of ionic specie \mathbb{P} .

$$\omega_{total} = \sum_{\mathbb{P}=1}^{\mathbb{Q}} \mathfrak{U}_{\mathbb{P}} \cdot \mathfrak{X}_{\mathbb{P}} \quad 139$$

Let the total number of ions of all \mathbb{Q} ionic species in subsystem 1 and 2 at any given time be defined by ω_1 and ω_2 , respectively. The value of ω_{total} at any given time is the sum of ω_1 and ω_2 , given by equation 140.

$$\omega_{total} = \omega_1 + \omega_2 \quad 140$$

As subsystem 1 is running cognitive processes, the number $\mathfrak{U}_{\mathbb{P}}$ of ionic species keep changing in subsystem 1 and subsystem 2, because of influx and outflux of ions, depending on the cognitive processes. Each individual ionic specie $\mathfrak{X}_{\mathbb{P}}$ will have its own influx and outflux rate. Let the influx and outflux rate of an individual ionic specie $\mathfrak{X}_{\mathbb{P}}$ be given by $[\dot{\mathfrak{X}}_{\mathbb{P}}]_1$ and $[\dot{\mathfrak{X}}_{\mathbb{P}}]_2$, respectively. Rate of influx of all \mathbb{Q} ionic species from subsystem 2 to subsystem 1 is the average of influx rates of individual ionic species given by equation 141. Likewise, rate of

outflux of all \mathbb{Q} ionic species from subsystem 1 to subsystem 2 is the average of outflux rates of individual ionic species given by equation 142. Net flux $\dot{\omega}_{net}$ is given by equation 143.

$$\dot{\omega}_1 = \frac{\sum_{\mathbb{P}=1}^{\mathbb{Q}} [\dot{x}_{\mathbb{P}}]_1}{\mathbb{Q}} \quad 141$$

$$\dot{\omega}_2 = \frac{\sum_{\mathbb{P}=1}^{\mathbb{Q}} [\dot{x}_{\mathbb{P}}]_2}{\mathbb{Q}} \quad 142$$

$$\dot{\omega}_{net} = \dot{\omega}_1 - \dot{\omega}_2 \quad 143$$

Net flux $\dot{\omega}_{net}$ depends on the number and type of cognitive processes run by the subsystem 1 at any given time. So, $\dot{\omega}_{net}$ keeps fluctuating depending on the start and completion of cognitive processes, which results in the movement of ionic species in and out of subsystem 1. If one considers system as human organism and subsystem 1 as human nervous system, the fluctuations in net flux $\dot{\omega}_{net}$ represents the electric activity as measured on the electroencephalogram (EEG). In other words, *electrical activity of human brain is the movement of ionic species in and out of neurons, depending on the cognitive processes run by the human nervous system at any given time t .*

As discussed in previous section, availability of responsible ionic species to run cognitive processes triggers different states of the system. So, in awake state, availability of ionic species is high i.e. ω_2 is high. With time t , ω_2 decreases and ω_1 increases in awake state, resulting in the decrease in the capacity $[\mathbb{C}]_t$ of subsystem 1. Somnolence state of the system is triggered when capacity $[\mathbb{C}]_t$ of subsystem at a given time t is below $[\mathbb{C}_{threshold}]_t$. System reduces the receiving of external stimuli in somnolence state, however, ω_1 continue to increase, but at a slower rate. Likewise, sleep state of the system is triggered when $[\mathbb{C}]_t$ of subsystem 1 is below $[\mathbb{C}_{sleep}]_t$. System practically stops receiving external stimuli. In sleep state, subsystem 1 starts freeing up held ions in the neurons by completing previously running cognitive

processes and starting new cognitive processes by stimulating neurons in subthreshold states using internal stimuli. In sleep state, ω_2 increases and ω_1 decreases, resulting in the increase in capacity $[C]_t$ of subsystem 1. When capacity $[C]_t$ increases to $[C_{awake}]_t$, awake state of the system is triggered. At this point, system completes one AS-SS cycle and start the next AS-SS cycle. Two typical AS-SS cycles using linear approximations of ω_1 and ω_2 with time t are shown in figure 34.

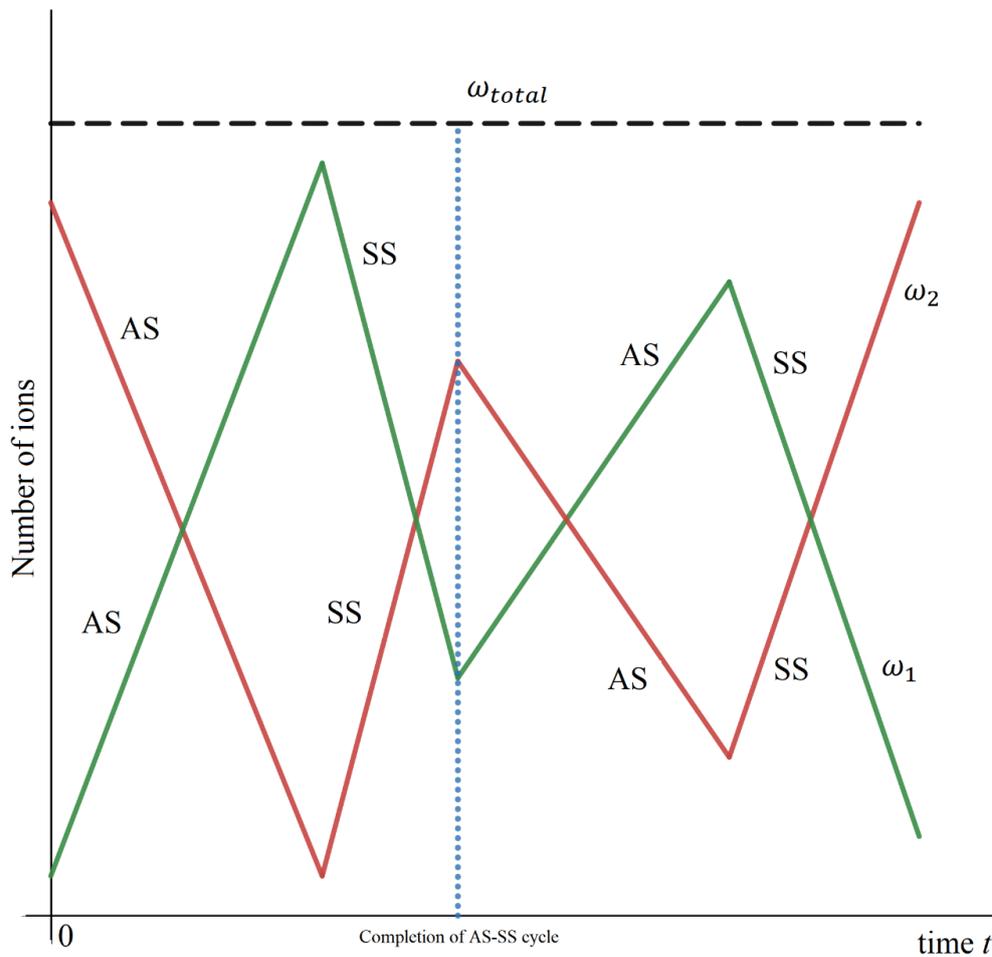


Figure 34. Linear approximations of number of ions in subsystem 1 and 2 with respect to time t , during awake state (AS) and sleep state (SS). Dashed black line represents total number of ions in a system. Red and green lines represents number of ions in subsystem 2 and 1, respectively. In AS, ω_2 decreases and ω_1 increases. In SS, ω_2 increases and ω_1 decreases. Dotted blue line represents the completion of one AS-SS cycle.

12. Dreams

In this section, the concept and architecture of dreams, in the context of neuronal state theory will be discussed. It has been previously established that in sleep state, X-neuronal system frees up ions held in the neurons by completing previously running cognitive processes. Furthermore, X-neuronal system start new cognitive process of type 1 and 2 by stimulating neurons in subthreshold states by internal stimuli. Upon completion of these new type 1 cognitive processes, ions move out of neurons, resulting in neurons attaining rest state. These notions of sleep state serve as the foundational basis to discuss dreams.

Consider an arbitrary type 1 cognitive process $[CP_{\varphi}]_{type\ 1}$ an X-neuronal system is running in the sleep state of an organism. Furthermore, consider that $[CP_{\varphi}]_{type\ 1}$ was started by an arbitrary stimulus S_c when organism was in awake state. X-neuronal system completes $[CP_{\varphi}]_{type\ 1}$ in sleep state by generating a stimulus response $[\mathcal{R}]_{S_c}$ using neuronal set $[N]_{S_c}$. Cognitive process $[CP_{\varphi}]_{type\ 1}$ is given by $S_c \rightarrow [N]_{S_c} \rightarrow [\mathcal{R}]_{S_c}$. The significance of $[CP_{\varphi}]_{type\ 1}$ lies in the fact that it was started in the awake state and completed in the sleep state. Completion of $[CP_{\varphi}]_{type\ 1}$ in sleep state is the essence of dreams. In the context of neuronal state theory, *a single dream is the generation of stimulus response $[\mathcal{R}]_{S_c}$ for stimulus S_c using neuronal set $[N]_{S_c}$ in the sleep state of an organism.*

Cognitive process $[CP_{\varphi}]_{type\ 1}$ was started in awake state. However, for generation of dream, cognitive processes can be started in the sleep state of an organism. So dreams are the generation of stimuli responses in the sleep state of an organism, regardless of the state an organism was in when it received stimuli.

12.1. Salient Features of Dreams

It has been established that during sleep state, X -neuronal system start new cognitive process of type 1 and 2 by stimulating neurons in subthreshold states. Consider an arbitrary effector neuron E_{l_x} in an arbitrary subthreshold state $[x_{STP_J}]_{E_l}$ during sleep state of an organism. Furthermore, consider that STP state $[x_{STP_J}]_{E_l}$ resulted from neuron E_{l_x} receiving stimuli for 2 type 1 cognitive processes $[CP_{50}]_{type\ 1}$ and $[CP_{51}]_{type\ 1}$, which were started for stimuli S_{50} and S_{51} respectively, in the awake state of the organism. In other words, neuron E_{l_x} was an element of neuronal sets $[N]_{S_{50}}$ and $[N]_{S_{51}}$. However, both cognitive processes were completed without E_{l_x} attaining an AP state, so it remained in STP state $[x_{STP_J}]_{E_l}$ after completion of $[CP_{50}]_{type\ 1}$ and $[CP_{51}]_{type\ 1}$. Neuron E_{l_x} holds parts of the stimuli S_{50} and S_{51} in same form or transformed form and if E_{l_x} generated an output response, that output response will have essence of stimuli S_{50} and S_{51} . Now considers that in sleep state, X -neuronal system starts a type 1 cognitive process by stimulating E_{l_x} by an arbitrary internal stimuli S_{int_A} . Upon completion of the process, neuron E_{l_x} produced an arbitrary output response $[O_{C_U}]_{E_l}$. Since $[O_{C_U}]_{E_l}$ was generated in the sleep state, it is a dream. Furthermore, generated output response $[O_{C_U}]_{E_l}$ has been effected by the stimuli S_{int_A} , S_{50} and S_{51} . *This is a very significant features of dreams signifying that dreams can be effected by the stimuli received in the awake state of an organism.*

Likewise in sleep state, more than one neuron in subthreshold states can be a part of neuronal set of a single cognitive process started by an internal stimulus. The resulting generation of stimulus response, will be affected by the stimuli which caused the said neurons to attain subthreshold states. In a nutshell, Dreams are the generation of stimuli responses of type 1 cognitive processes in the sleep state of an organisms. The cognitive processes can be started

in awake, somnolence or sleep state. Furthermore, dreams can be affected by previously received stimuli, depending on the states of the neurons. If neurons are in subthreshold states, when receiving stimuli, the resulting dreams will be affected by previously received stimuli which caused neurons to attain subthreshold states. Furthermore, *it should be noted that neurons of X-neuronal system can learn while generating dreams in sleep state. If neurons produce unique output responses while generating dreams, they are learning.*

12.2. Remembrance of Dreams

Now that the features of dreams are well established, in this section, the remembrance of dreams in the context of neuronal state theory will be defined.

Given an X-Neuronal system generated a dream $[\mathcal{R}]_{S_c}$ in sleep state of an organism, for an arbitrary type 1 cognitive process $[CP_p]_{type\ 1}$ at time $t = t_0$ using neuronal set $[N]_{S_c}$.

Remembrance of dream is the generation of stimulus response $[\mathcal{R}]_{S_c}$ by the same X-Neuronal system at time $t > t_0$, in the awake state of the organism using neuronal set $[N]_{S_c}$ or other neuronal sets.

In other words, Remembrance of dreams is merely the generation of the dream in the awake state of the organism. Furthermore, X-neuronal system can generate impaired dreams in the awake state. Impaired dreams in the context of neuronal state theory is defined by the following statement:

Given a generation of dream $[\mathcal{R}]_{S_c}$ of an arbitrary type 1 cognitive process $[CP_p]_{type\ 1}$ generated by an X-Neuronal system in sleep state of an organism, for stimulus S_c using neuronal set $[N]_{S_c}$ at time $t = t_0$. Impaired dream is the generation of the output responses at time $t > t_0$ in awake state of an organism, by same neurons which produced output responses

of set $[\mathcal{R}]_{S_c}$ at time $t = t_0$. However, one or more generated output responses at time $t > t_0$ are different from set $[\mathcal{R}]_{S_c}$.

Conclusion

In conclusion, this article presents a framework called ‘Neuronal State Theory’, which explains the architecture of the workings of human nervous system in running cognitive processes. This article also shows that every cognitive research field (Attention, Memories, Learning, Imagination, Sleep and Dreams) emerges from this single framework. Neuronal State Theory serves as a foundational framework for cognitive research and gives several useful insights in the workings of cognition; which is not limited to just human species. Furthermore, this framework can be potentially useful in the architecture of neuromorphic machines and human-like intelligence.

Appendix A

Derivation of derivative of $K_{\gamma g}$ with respect to $[\kappa_{\gamma g}]_{R_1}$.

$$\frac{dK_{\gamma g}}{d[\kappa_{\gamma g}]_{R_1}} = \frac{d}{d[\kappa_{\gamma g}]_{R_1}} \left\{ \frac{\ell_{\gamma}}{\Lambda_{\gamma}} \left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_{\gamma}} \right)^{\ell_{\gamma}} e^{-\left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_{\gamma}} \right)^{\ell_{\gamma}}} \right\} \quad A1$$

Taking the constant values out equation A1 can be written as A2

$$\frac{dK_{\gamma g}}{d[\kappa_{\gamma g}]_{R_1}} = \frac{\ell_{\gamma}}{\Lambda_{\gamma}} \left\{ \frac{d}{d[\kappa_{\gamma g}]_{R_1}} \left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_{\gamma}} \right)^{\ell_{\gamma}} e^{-\left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_{\gamma}} \right)^{\ell_{\gamma}}} \right\} \quad A2$$

Let $\left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_{\gamma}} \right)^{\ell_{\gamma}} = f$ and $e^{-\left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_{\gamma}} \right)^{\ell_{\gamma}}} = g$ and applying product rule on equation A2, given by equation A3, where \dot{f} and \dot{g} are given by A4 and A5, respectively.

$$\frac{dK_{\gamma g}}{d[\kappa_{\gamma g}]_{R_1}} = \frac{\ell_{\gamma}}{\Lambda_{\gamma}} \{ \dot{f}g + \dot{g}f \} \quad A3$$

$$\dot{f} = \frac{d}{d[\kappa_{\gamma g}]_{R_1}} \left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_{\gamma}} \right)^{\ell_{\gamma}} = \frac{\ell_{\gamma}}{\Lambda_{\gamma}} ([\kappa_{\gamma g}]_{R_1})^{\ell_{\gamma}-1} \quad A4$$

$$\dot{g} = \frac{d}{d[\kappa_{\gamma g}]_{R_1}} e^{-\left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_{\gamma}} \right)^{\ell_{\gamma}}} = -\frac{\ell_{\gamma} e^{-\left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_{\gamma}} \right)^{\ell_{\gamma}}} ([\kappa_{\gamma g}]_{R_1})^{\ell_{\gamma}-1}}{\Lambda_{\gamma}} \quad A5$$

Plugging equations A4 and A5 in equation A3 and simplifying the derivative of $K_{\gamma g}$ with respect to $[\kappa_{\gamma g}]_{R_1}$ is given by equation A6.

$$\frac{dK_{\gamma g}}{d[\kappa_{\gamma g}]_{R_1}} = \frac{\ell_{\gamma}^2}{\Lambda_{\gamma} (\Lambda_{\gamma})^{\ell_{\gamma}}} ([\kappa_{\gamma g}]_{R_1})^{\ell_{\gamma}-1} \left(e^{-\left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_{\gamma}} \right)^{\ell_{\gamma}}} \right) \left[1 - \left(\frac{[\kappa_{\gamma g}]_{R_1}}{\Lambda_{\gamma}} \right)^{\ell_{\gamma}} \right] \quad A6$$

Competing Interests:

The author declares no competing interest.