

## Mechanism of Molecular Neuroscience in Brain

Takashe Hirokava\*

Department of Neuroscience, Doshisha University, Kyoto, Japan

### Editorial

**Received:** 05-Jan-2022, Manuscript

No. neuroscience-22- 52138;

**Editor assigned:** 07-Jan-2022, Pre

QC No. neuroscience -22-

52138(PQ); **Reviewed:** 21-Jan-

2022, QC No. neuroscience -22-

52138; **Accepted:** 24-Jan-2022,

Manuscript No. neuroscience-22-

52138 (A); **Published:** 31-Jan-2022,

DOI:10.4172/neuroscience.6.1.005

**\*For Correspondence:**

Takashe Hirokava, Department of

Neuroscience, Doshisha University,

Kyoto, Japan

**E-mail:** takshirokava32@ac.jp

### EDITORIAL NOTE

Different kinds of reasoning demonstrate the importance of a molecular knowledge of the brain. First, let's look at some of the more practical arguments. It is simple to map the patterns of neurons and their connections when attempting to understand the brain only through the activity of neurons in pathways. Given the flexibility of synaptic connections and the qualities of synapses, however, knowledge of the brain could only be reached if all synaptic connections and neurons could be monitored at the same time, which is clearly impossible. <sup>[1-2]</sup> Only by being able to predict the dynamics and qualities of these neuron's synaptic connections, as well as the patterns of neurons in response to synaptic activity, can this problem be solved practically. Because the brain is more than a collection of circuits, comprehending them involves not just the capacity to anticipate the behaviour of neurons and synapses in circuits, but

## Research & Reviews: Neuroscience

also knowledge of how the brain operates at a higher level than neural circuits .<sup>[3-4]</sup> Different areas and cells of the brain connect with one another *via* more than synapses. Glia, in addition to neurons is a part of the brain's main information-processing engine. Understanding this mechanism will necessitate a molecular approach, or knowledge of the molecular structure. <sup>[5-7]</sup> Another counter argument is that molecular processes are too complicated to be tractable, and that this molecular logic will be unmanageable. This might be applicable to systems neuroscience as well, fails to recognise the distinction between 'complexity' and 'details.' In molecular neuroscience, the same is true. Details are crucial until principles are discovered; after principles are established. Translational research is an even more compelling practical reason for why molecular neuroscience is critical for understanding the brain. Diseases are caused by molecular deficiencies, despite the fact that they often present as system dysfunctions.<sup>[8-9]</sup> To cure a disease, we must first comprehend the disease process; which requires comparing it to a healthy normal condition. One might suggest that pathways will ultimately be more significant in neuropsychiatric disorders, and that understanding autism, for example, will require understanding the especially human pathways for language and empathy, because the condition presents as a dysfunction of these pathways. It is based on a basic misunderstanding of disease processes; just because a disease manifests as a disorder of certain human abilities, and presumably of their underlying circuits, does not mean that the disease process operates in this manner. The majority of genes connected to neuropsychiatric disorders are widely expressed, implying that they are involved in a diverse range of systems. <sup>[10-11]</sup>The presentations of neuropsychiatric disorders do not always imply that the underlying connections are the cause of the disease; rather, they imply that the pathways underlying these manifestations are a result of the disease. The central nervous system interacts with the peripheral nervous system, immunological system, endocrine system, and, most likely, the gut and skin microbiota in order to function properly. These interactions are only now being researched. Because these interactions are believed to be crucially engaged in neurodegenerative illness and other disorders, molecular and cellular biology approaches are needed to investigate them. However, only preliminary efforts toward understanding their processes have been taken to date.

The vast majority of human neurons do not regenerate and remain active throughout one's life; What keeps neurons alive for a long period, how do they age, and how do they die? These questions are critical for comprehending both normal brain function and neurodegenerative illnesses. Aging is an unavoidable reality in our life.

### REFERENCES

1. Skandsen T, et al. The epidemiology of mild traumatic brain injury: the Trondheim MTBI follow-up study. *Scand J Trauma Resusc Emerg Med* . 2018; 26:34.

## Research & Reviews: Neuroscience

- 2.Fazel S, et al. Suicide, fatal injuries and other cause of premature mortality in patients with traumatic brain injury; a 41-year Swedish population study. *JAMA Psychiatry*. 2014;71:326–333.
- 3.Olesen J, et al.The economic cost of brain disorders in Europe. *Eur J Neurol*.2012;19:55–62.
- 4.Sharp DJ, et al. Network dysfunction after traumatic brain injury. *Nat Rev Neurol*. 2014;10:156–66.
- 5.Kashluba S, et al. A longitudinal, controlled study of patient complaints following treated mild traumatic brain injury. *Arch Clin Neuropsychol*.2004;19:805–16.
- 6.Hou R, et al. When a minor head injury results in enduring symptoms: a prospective investigation of risk factors for postconcussional syndrome after mild traumatic brain injury. *J Neurol Neurosurg Psychiatry*. 2012;83:217–23.
- 7.Maas AIR, et al. Traumatic brain injury: integrated approaches to improve prevention, clinical care, and research. *Lancet Neurology* 2017;16: 987–1048.
- 8.Sinha S, et al. Workforce planning in neurosurgery. *Br J Neurosurg*. 2020;34:3–8.
- 9.Powell J,et al. Community based rehabilitation after severe traumatic brain injury: a randomised controlled trial. *J Neurol Neurosurg Psychiatry* .2002;72:193–202.
- 10.Adkins-Regan E. Neuroendocrinology of social behavior. *ILAR J*. 2009; 50: 5–14.
- 11.Adolphs R, et al. Fear and the human amygdala. *J Neurosci* .1995;15: 5879–5891.