Opinion

Neuronal migration and its importance in the human being

Abstract

Background: Neuronal migration is a topic of special relevance to the study of neuroscience, since the movement, distance and connection of individual neurons determines embryonic brain development. The alterations that can occur during this process are known as neuronal migration disorders (NMD).

Objective: To describe the reasons why neuronal migration disorders occur, as well as the processes involved in it.

Methodology: Reflective type, based on the search of information published in several databases, using as key terms neuronal migration and neuronal migration disorders.

Results: Alteration within the proliferative layers of the neural tube can lead to NMD and the reasons for its occurrence are due to genetic situations, altered neuronal connections, abnormal migration of convolutions and problems with autism spectrum disorders, as well as epilepsies.

Conclusion: All these results suggest that NMDs can cause neurological disorders and difficulties and syndromes when regions are affected.

Volume 12 Issue 6 - 2022

Rohnal José Rada Luna
Corporación Universitaria del Caribe CECAR, Psicologia, Colombia

Correspondence: Rohnal José Rada Luna, Corporación Universitaria del Caribe CECAR, Psicologia, Colombia , Email rohnal.rada@cecar.edu.co

Received: September 5, 2022 | **Published:** November 23, 2022

Opinion

Neuronal migration "is an essential process in the central nervous system (CNS) and plays a fundamental role in brain development during the embryonic period".[1] This is a phenomenon paramount to normal development in humans, as it links cells in appropriate spatial relationships with other cells.[2] During embryonic development, neurons are formed within proliferative layers of the neural tube and originate according to the radial migratory pathway depending on the area of the nervous system.[3,4]

Factors arising from essential neuronal migration processes lead to severe brain malformations, which can cause complex and heterogeneous neuronal development and migration disorders.[5] The lack of regulation in neuronal migration is known as neuronal migration disorders (NMD). Different studies have revealed that NMD have been associated with several intractable or difficult-to-treat neurological deficits in children.[6,7] One of them is pharmacologically intractable epilepsy, composed of neuronal cells with an abnormal organization, responsible for seizures;[8] and mental retardation, as moderate to brief retardation was found in patients, as well as premature onset of conversions.[9] It is important to analyze the elements involved in NMD, and the general objective of the present essay is to describe the reasons why neuronal migration disorders occur, as well as the processes involved in the same.

Critical processes during CNS can lead to several changes in the brain organization, especially in the problems related to the neural tube and alteration in the different circuits, because the neurons did not reach the adequate position and growth for their development.[10] Over time NMD have become an important issue as neuronal activity can result in clinical manifestations such as ataxia, autism, schizophrenia and epilepsy.[11–13]

First, genetic studies have shown that one of the reasons for NMD is a genetic mutation in the DCX gene (also known as SCLH and XLIS), located on the long arm of the X chromosome (Xq23), which encodes a protein called doublecortin.[14] In addition, this anomaly is associated with subcortical band heterotopia, which also affect the abnormal development of the human neocortex, generating abnormal connections in neurons that cause epilepsy difficult to be treated.[15]

It has also been shown that NMD is caused by abnormal migration of cortical convolutions. In this case, lissencephaly occurs because the proteins encoding the LIS1 gene do not function properly, which causes neurons to be unable to have a proper displacement circuit and prevents the formation of synapses.[16] This is related to alterations in the RELN gene, since difficulty in producing the reelin protein has serious negative effects on brain development and can lead to psychiatric illnesses such as schizophrenia due to reduced levels of reelin in inhibitory neurons and other mental illnesses such as major depressive disorder, bipolar disorder and autism spectrum disorders.[17] It has recently been discovered that NMD is related to neurodevelopmental disorders such as autism. This is explained through a mutation in the CASPR2 or CNTNAP2 gene, which can cause an increase in suffering from epilepsies and autism; since the analysis of biopsies performed in humans evidenced that this gene is restricted to the temporal lobe, one of the most affected brain regions in autistic and epileptic patients.[18]

Acknowledgments

None.

Conflicts of interest

The authors declare no conflicts of interest.

References

1. Rahimi-Balaei M, Bergen H, Kong J, et al. Neuronal Migration During Development of the Cerebellum. *Frontiers in Cellular Neuroscience.* 2018;12(484):1–16.
2. Marín O, Valiente M, Ge X, et al. Guiding neuronal cell migrations. *Cold Spring Harb perspect Biol.* 2010;2(2):1–20.
3. Govek E, Hatten M, Van L. The role of Rho GTPase proteins in CNS neuronal migration. *Developmental neurobiology.* 2011;71(6):528–553.
4. Cooper J. Mechanisms of cell migration in the nervous system. *Journal of Cell Biology.* 2013;202(5):725–734.
5. Buchsbaum I, Cappello S. Neuronal migration in the CNS during development and disease: insights from *in vivo* and *in vitro* models. *Development.* 2019;146(1):1–17.

6. Barkovich A, Guerrini R, Kuzniecky R, et al. A developmental and genetic classification for malformations of cortical development: Update 2012. *Brain.* 2012;135(5):1348–1369.

7. LoTurco J, Booker A. *Neuronal Migration Disorders.* Connecticut: Academic Press. 2013.

8. Wirrell E, Wong-Kisiel L, Mandrekar J, et al. What predicts enduring intractability in children who appear medically intractable in the first 2 years after diagnosis?. *Epilepsia.* 2013;54(6):1056–1064.

9. Barkovich A, Dobyns W, Guerrini R. Malformations of cortical development and epilepsy. *Cold Spring Harb Perspect Med.* 2015;5(5):1–23.

10. Manto M, Gruol D, Schmahmann J, et al. *Handbook of the Cerebellum and Cerebellar Disorders.* Vol 4. Netherlands: Springer. 2013.

11. Demkow U, Ploski R. *Clinical Applications for Next-Generation Sequencing.* Cambridge, MA: Academic Press. 2015.

12. Marzban H, Del Bigio M, Alizadeh J, et al. Cellular commitment in the developing cerebellum. *Front Cell Neurosc.* 2015;8(450):1–26.

13. Qin R, Cao S, Lyu T, et al. CDYL deficiency disrupts neuronal migration and increases susceptibility to epilepsy. *Cell Rep.* 2017;18(2):380–390.

14. Gressens P, Passemard S, Sebag C, et al. *Encyclopedia of Neuroscience.* San Diego CA: Academic Press. 2009.

15. Guerrini R, Dobyns W, Barkovich A. Abnormal development of the human cerebral cortex: genetics, functional consequences and treatment options. *Trends Neurosci.* 2008;31(3):154–162.

16. Moon H, Hippenmeyer S, Luo L, et al. LIS1 determines cleavage plane positioning by regulating actomyosinmediated cell membrane contractility. *eLife.* 2020;9:1–31.

17. Moreau C, Urchs S, Kuldeep K, et al. Mutations associated with neuropsychiatric conditions delineate functional brain connectivity dimensions contributing to autism and schizophrenia. *Nature Communications.* 2020;11(5272):1–12.

18. van Sonderen A, Ariño H, Petit-Pedrol M, et al. The clinical spectrum of Caspr2 antibody-associated disease. *Neurology*. 2016;87(5):521–528.