Thyroid Hormones and Neuroplasticity

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"The term neuroplasticity rose to prominence in the second half of the 20th century when evidence suggested that the brain retains some adaptability during adulthood, as opposed to the earlier hypothesis that after a childhood critical period, the brain remains unchanged. Neuroplasticity is used as a broad term but is generally defined as the ability of the brain to institute long-term changes to its synaptic or cellular structure as a response to external stimuli. Whereas it is now clear that the brain is still adaptable during adulthood, neuroplasticity during development contains the majority of changes that will shape adult behavior. In this context neuroplasticity can be described as the component of general neurodevelopment that changes and settles depending on environmental influences."[1]

Neuroplasticity can also be defined as the ability of the nervous system to respond to intrinsic or extrinsic stimuli by reorganizing its structure, function and connections.[2] It is now generally accepted that even the mature brain can undergo neuroplastic changes.[3] Although the majority of studies are dedicated to the dynamic reorganization of the motor system after an acute event, such as stoke, these neuroplastic changes may also occur in a chronic disease such as multiple sclerosis.[4]

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Thyroid hormones play a critical role in normal physiology with effects on almost all tissues. They influence growth and development, maintain normal cognition, cardiovascular function, bone health, metabolism and energy balance. Thyroid hormones are regulated at the tissue level by various mechanisms. Cellular influx and efflux is controlled by the presence of several transmembrane proteins, which from an integrative medicine perspective, underscores the importance of a healthy phospholipid membrane, which allows for

normal receptor activity and normal transport across the cell membrane. The activity level of thyroid hormones in brain cells is controlled by activation of the T4, which predominately functions as a pre-hormone, to the more bioactive form T3. [5]Lastly, the signaling of T3 depends on the presence of nuclear receptors.

The thyroid hormones are a major factor in both developmental and adult neuroplasticity and it has become clear from multiple studies that thyroid hormone deprivation leads to defects in learning on all fronts.[6] "Considerable evidence links the formation and/or functioning of sensory systems to thyroid hormone signaling. In humans, adult-onset hypothyroidism causes defects in smell and taste, and deafness is a recognized symptom of prenatal thyroid hormone deficiency." [7]

Spatial memory is the ability to record information about one's environment and spatial orientation (i.e. remembering a position). For example, a person's spatial memory is required in order to navigate around a familiar city. The hippocampal volume is lower in both adults with hypothyroidism and children having suffered from congenital hypothyroidism during gestation, and adult-onset hypothyroidism is also known to impair hippocampal activity.[8] Thyroid dysfunction as also has been associated with anxiety, fear learning, vocal learning and motor learning all of which gives emphasis to the importance of normal thyroid function.

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[1] Raymaekers SR, Darras VM. Thyroid hormones and Learning-associated neuroplasticity. General and Comparative Endocrinology. Elsevier 247; 2017: 26-33.

[2] Flachenecker P. Clinical implications of neuroplasticity – the role of rehabilitation in multiple sclerosis. Frontiers in Neurology. March 2015. vol. 8; 38

[3] Ibid

[4] Ibid

[5] Ibid

[6] Raymaekers SR, Darras VM. Thyroid hormones and Learning-associated neuroplasticity. General and Comparative Endocrinology. Elsevier 247; 2017: 26-33.

[7] Ibid

[8] Ibid

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