Acute Regression in Young Adults with Down Syndrome

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Background

Young adults with Down Syndrome (DS) can experience acute regression as young as 11 years of age. This includes loss of life skills, speech and independence.[1] Current medical treatment focuses on anti-depressants and antipsychotic drugs which often lead to worsening of symptoms.[2] When recovery with drugs isn't forthcoming, electroconvulsive therapy is pursued, despite its potential to provoke tonic clonic seizures and side effects of confusion and memory loss.[3]

While the aetiology of regression in DS remains unknown,[4] catatonia is considered to be a likely cause.[5] Underlying catatonia are various psychiatric and somatic origins related to autoimmune, infective, metabolic and genetic disorders.[6]

Functional magnetic resonance imaging (MRI) studies indicate a decrease of gamma-aminobutyric acid (GABA) receptors, resulting in failure to regulate negative emotions such as depression; eleven times more prevalent in DS regression cases compared to controls.[7] Glutamate hyperactivity occurs with low GABA, and is thought to be a primary cause of catatonia in DS.[8] Also, when the brain is exposed to neurotoxic metals such as mercury, lead and aluminium, elevated glutamate levels enhance their ability to cause neuronal damage.[9,10,11]

Figure 1 below is a schematic view of a hypothesis of trehalose function in the brain.[12] Following the diagram; (1) Trehalose indirectly affects brain function through the regulation of gut microbes, which sends signals to the brain by dendritic immune activation or secretion of neurotransmitters and gut peptides that may be delivered through the vagus nerve to the brain. (2) Direct transport of trehalose to the brain, which passes through the blood-brain barrier and affects neuronal cells. (3) The brain sends signals to the enteric system to modulate trehalose function.

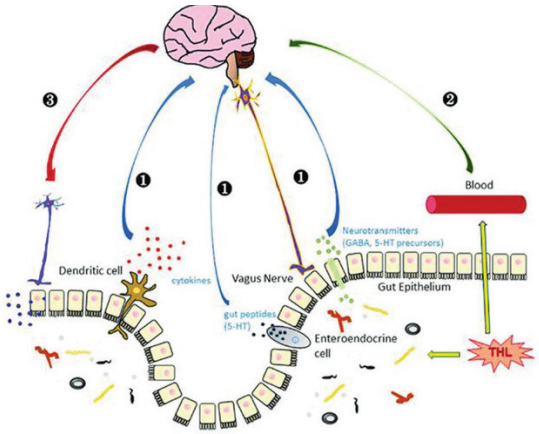

Figure 1: Schematic view of a hypothesis of trehalose function in the brain.[13]

Dixie Lawrence (US) has pioneered a new approach to treating regression based on reducing neuroinflammation, heavy metal detoxification, supporting methylation and addressing DS biochemistry. Patients have regained life skills, independence and speech, and are positive about the future. I was fortunate to be able to work with Dixie and her clients and here I present two case studies.

Treatment

Treatment was initiated with trehalose and heavy metal chelators; alpha lipoic acid, vitamin C and glutathione. Trehalose reduces neuroinflammation by clearing dead neurons,[14] as well as reducing glutamate accumulation.[15] Alpha lipoic acid, vitamin C and glutathione cross the blood brain barrier to chelate heavy metals, particularly mercury, lead and cadmium.[16,17] An anti-inflammatory diet is recommended at this time.

Once symptom improvement is noticed, LongVida® curcumin is introduced and is able to enter brain tissue to further reduce inflammation, recover memory and downregulate amyloid precurser protein (APP).[18] Following this, *Nutrigenomic Support for Down Syndrome* is introduced, along with specific polyphenols to support methylation, detoxification, neurotransmitter production and gene overexpression.

Case One: Heather

Heather was a vibrant girl who loved to read and write, and lead Zumba classes. At age 22 she was diagnosed with regression. She could barely walk, and couldn't understand written or spoken language. She avoided making eye contact, developed sensory processing disorder, became aggressive and was irritated by clothing. In July 2017, she started Dixie's protocol along with a gluten and dairy free diet.

Within twelve months, she went from 189 to 135 pounds (losing 24.5 kg equivalent), and could independently get up in the morning and dress herself. Heather now uses her smartphone to Facetime her brother and text and call family and friends. She is journaling her thoughts and plans and told her mother, "I need a job." Figures 2 and 3 below show Heather pre- and post-treatment, respectively.

Figure 2: Heather pre-treatment.

Figure 3: Heather post-treatment.

Case Two: Nathan

In June 2017, 18 year old Nathan lost verbal communication, lost interest in activities and stopped working with his dad, which he loved to do. He developed strange movements, facial grimaces and struggled to sleep, eat and wash himself. All actions were in slow motion. He couldn't fasten his seatbelt. Three months later he saw a neurologist, who diagnosed him with regression and prescribed an antidepressant.

In February 2018, Nathan's parents contacted Dixie and started the protocol. Within six months Nathan started conversing with his family and carers, Facetiming and joking around with them. He began playing sports again and when he returned to school, teachers started calling his parents to say he was talking and participating in class. He looks forward to going to school and with his father to work, grabbing his gloves, flashlight and tape measure on the way. Figures 4 and 5 below show Nathan pre- and post-treatment, respectively.

Figure 4: Nathan pre-treatment.

Figure 5: Nathan post-treatment.

Conclusion

Heather and Nathan are continuing to receive Naturopathic Support to achieve complete physical wellness. Correcting metabolic, nutritional, infective and genetic drivers of regression is crucial to maintaining optimum health and quality of life in these young people full of renewed zest for living.

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References

References available on request.