PRIMAL CPC 1: TOURETTE SYNDROME MEETS EVOLUTIONARY MEDICINE

READERS SUMMARY:

1. WHAT IS TOURETTE'S SYNDROME?

2. HOW MIGHT IT BE LINKED TO A LEAKY GUT, GRAINS, AND TIE TO OTHER DISEASE'S WE KNOW NADA ABOUT?

3. HOW DOES OUR HARDWIRING GIVE US NEW INSIGHTS?

4. HOW MIGHT NEUROSURGERY HELP CHRIS JOHNSON?

5. HOW MIGHT SOME PIONEERING DENTISTS TIED A NICE BOW ON THERAPY FOR CHRONIC TS PATIENTS?

Tourette's syndrome (TS) is defined as an inherited neurological disorder that is a chronic idiopathic syndrome that neurosurgeons consider in the family of movement disorders. Today's blog is called a CPC. That is known as a clinico-pathologic conference for my readers. Every so often I am going to post about a disease process and how evolutionary medicine may look at something that modern medicine is vexed by. We neurosurgeons generally do not treat these patients but neurologist however do. In my town of Nashville, we have a very famous running back, Chris Johnson, who is a superstar that has a variant of this condition. The disease is characterized by the presence of multiple muscle tics and occasionally vocal tics that have their beginning before adulthood. It usually occurs with an onset in childhood between ages 5-8. A diagnosis of TS was almost three times as likely for boys as girls, twice as likely for persons aged 12–17 years than for those aged 6–11 years, and twice as

likely for non-Hispanic white persons than for Hispanic and non-Hispanic black persons. Among persons ever diagnosed with TS, 79{a7b724a0454d92c70890dedf5ec22a026af4df067c7b55aa6009b4d34d5da3c6} also had been diagnosed with at least one co-occurring mental health or neurodevelopmental condition. I think the reasons for males is because they generally have larger area postrema in their fourth venticles of their brains than females allowing for more antibodies to pass into the CSF. This is a time where most brain growth is beginning to slow in the brainstem while the immune system is also becoming more mature. This implies that before these ages the developing brain and immune system are not ideally protected from inflammation, toxin, or antibodies. Modern medicine has no answer for the causes of this disease. I think evolutionary medicine might have an answer for some of the patients who suffer from it if they would just think about their disease from a new perspective. I think this syndrome and other movement disorders might be caused by a leaky gut due to gliadin antibodies that bind to synapsin on developing nerves and cause a short circuiting in the brain stem wiring that allows for nerve depolarization (action potential) to jump onto other damaged nerves that are close located to one another in the brainstem. I have long thought about these conditions as a neurosurgeon because I think the key insight is the wiring proximity in the brain stem that most in my profession have not realized are closely related. I think the key insight is that we now know that the gliadin protein has been modified over the last 50 years by man and the incidence of celiac disease in that same time frame has increase from 1{a7b724a0454d92c70890dedf5ec22a026af4df067c7b55aa6009b4d34d5da3c6} to 5{a7b724a0454d92c70890dedf5ec22a026af4df067c7b55aa6009b4d34d5da3c6} of the population now. The prevalence of Celiac Disease has increased five-fold overall since 1974. This increase was not due to increased sensitivity of testing either,but rather due to an increasing number of subjects that lost the

immunological tolerance to gluten in their adulthood. The proof is right here: Ann Med. 2010 Oct;42(7):530-8. It may also may surprise you is that functional movement disorders also have increased during the same time period.

Conventional medicine knows that gliadin antibodies cause a rare devasting syndrome called either idiopathic or sporadic cerebellar ataxia. This syndrome actually causes permanent ataxia because it causes destruction of the major nerves in the cerebellar hemispheres that coordinate our motor movements. It is a fully known and recognized cause of a movement disorder. Today it is one of the few movement disorders that conventional medicine has linked to gluten/gliadin antibodies. Even fewer people realize that Autism and other spectrum disorders all begin by altering hindbrain development in humans. Autism being the best known and essential tremor being the most common but rarely linked functional disorder of the cerebellum and thalamus.

Well today I am going to walk you down a path of maybe why TS maybe another disease that could be related to grain use too. The story will require you to learn some pretty complex neurosurgical wiring in the brainstem but I think the connections we make in this blog will help you really understand why the brain/gut axis is the fifth levee in my Quilt. A leaky gut can cause tremendous problems for our species whether conventional medicine believes it exists or not. The treatment I think is also too easy not to try…….just eat a paleolithic diet as outlined in the Paleo 1.0 diet books. I do think that this diet has to be started as soon as symptoms show up in children because the disease maybe reversible then. I think if the symptoms stay with the child they become hardwired into the brainstem because the shortcircuits act just as any neuron in the CNS. Neurons that fire together wire together. This is called Hebbian learning and any neuroscientists knows that this is how our brains are designed to work by evolution.

Associated diseases with Tourette Syndrome:

TS patients will sometimes exhibit co-morbid conditions that may occur with their symptoms. Some patients have shown the following associated conditions:

A. Attention Deficit Hyperactive Disorder (ADHD) The link is inflammation from the gut in my opinion.

B. Obsessive Compulsive Disorder (OCD) The link again is the leaky gut.

C. Sleep Disorder due to alterations in leptin and metabolic coupling

D. Increased Enuresis in children due to ADH release issues at the blood brain barrier

E. Bipolar Disorder Again we have another leaky gut link I believe.

Not all TS patients have these associated diseases, and it is not a certainty or requirement that these conditions exist with this disorder. The majority of TS patients ***have only*** the movement disorder without any other associated disorders. The CDC says otherwise in the 9th cite but that does not follow my clinical sensibilities.

Many pediatricians today know that there is a hypothesis out there that TS might because by streptococcal infections of early childhood in a syndrome known as PANDAS. In the April

2004 issue of Pediatrics, researchers Kurlan and Kaplan reviewed current scientific information and concluded “that PANDAS remains a yet unproven hypothesis.” TS and PANDAS provided me about 7 years to think about how this disease might happen from another mechanism. That mechanism that might cause this is gliadin antibodies that attack the brainstem wiring in the developing nervous system of children by entering the brain via the area postrema. The gliadin antibodies seem to bond irreversibly to a protein in the developing nervous system called synapsin. The area postrema is a circumventricular organ that is the seat of the vagus nerve. Damage to the area postrema can result in a syndrome called a central vagotomy. This is when the vagus nerve becomes completely disconnected to the gut. This is a huge link in eating disorder progression that I will be covering in the future. The area postrema is how the brain connects directly to the entire gut from the tips of your lips to the transverse mesocolon. The vagus nerve wiring is a critical link in understanding how this disease might be caused by gliadin antibodies.

Most of the motor tics in TS are linked to the head and neck. This is controlled by another cranial nerve called the trigeminal nerve. It has three divisions, called the opthalmic, maxillary, and the mandibular divisions. These fibers run very close to the fibers of the vagus nerve in the brainstem. I believe the source of TS is cross talk between these two cranial nerves due to damaged caused by gliadin and gluten antibodies. This is called ephaptic transmission. This type of abnormal wiring is known to happen in many biologic systems. In my 6th cite you can read about how it occurs in primates who are closely related to us. These findings suggest that nerve fibers of ***different types*** communicate with each other. This is especially true when sensory or motor neuron cells degenerate for any reason. The surviving neurons which have lost their connections to these nerve cells, may still send electrical signals to the many

brainstem nuclei through the synapses and ephapses of their neurites in the brainstem to cause the tic's that are classically associated with TS. I am going to hurt your head now…….with some deep seated neurosurgical anatomy to show the biologic plausibility of what I am proposing to you here in this blog.

THE WIRING DIAGRAM: You might need a neuroanatomy book to really get this down after you read it 1000 times.

Frequently one of the first clinical signs of TS is repetitive and uncontrollable eye blinking. If the pathway for the blink reflex is examined, we notice that CN V transmits tactile sensation from the cornea, which is perceived as irritation that evokes bilateral eyelid closure (an eye blink). Trigeminal opthalmic primary afferents send signals which end in the spinal trigeminal nucleus (subnucleus caudalis). From here, interneurons connect to the reticular formation. Within the reticular formation, interneurons send signals to the facial nucleus, cranial nerve VII. Facial nerve efferent neurons from the facial nucleus send their signal to the orbicularis oculi which closes the eyelid; blinking occurs. This is the simplified version of the corneal blink reflex neural circuit.

I believe the primary incoming afferent signals can come from other sensory branches of the trigeminal nerve itself. This is what may cause the reflex arc to occur. Very frequently and often unobserved, low order but constant nociceptive impulses transmitted by the auriculotemporal nerve, with its vast number of sympathetic fibers, come into the trigeminal ganglion and using the above described afferent neural pathway, and end in the subnucleus caudalis through ephaptic transmissons. The cross talk is allowed to occur because of

gliadin/gluten autoantibodies that cause myelinolysis and axonal electrical damage by binding to synapsin on the nerve. This affects aquaporin 4 function on the neuron cell membrane. This is now being studied today by Dr. Peter Green of Columbia University in NYC. He has written a book about gluten/gliaden related diseases and ironically I spoke to him yesterday on Doctor Radio on Sirius XM radio. Here is a link to his excellent book.

Motor efferent nerves from this nucleus may stimulate other muscles of facial expression such as frontalis, orbicularis oris, platysma, and zygomaticus major and minor, etc. These are some of the muscle groups which are implicated in facial motor tics. I believe that it is the afferent sensory fibers in the mandibular division of CN V that are causing oro-mandibular dyskinesia to be manifested into and through CN VII.

Another frequent finding in TS is throat clearing and sniffing. Examining the anatomy of the trigeminal nerve, one can see that within the spinal tract of V, which ends in the subnucleus caudalis, there are connections within the subnucleus caudalis to the glossopharyngeal nerve, CN IX, which contains general sensory fibers and provides sensation from the posterior 1/3 of the tongue, tonsil, skin of the external ear, internal surface of the tympanic membrane, and the pharynx. When this nerve is chronically stimulated, the cough/gag reflex becomes stimulated. If the primary chronic stimulus was not from CN IX, but was from chronic ephaptic stimulation within the subnucleus caudalis where CN IX and CN V decussate, would not the cough/gag reflex be stimulated possibly? I think this is precisely what happens in TS. I have not seen one neurological or neurosurgical discussion that can explain this phenomena as perfectly as this one does.

A well-documented finding in many movement disorder patients with TS is echolalia (the spontaneous utterance of sounds). Examining the anatomy within the subnucleus caudalis, one can

see that the vagus nerve, CN X decussates within the spinal trigeminal nucleus as does CN V. **It appears to me that the real cause of TS maybe the cross talk between the auriculotemporal nerve that is a branch of the trigeminal nerve with brainstem reticular formation that controls the many of the autonomic and complex reflexes that allow us to stand upright and turn our head and upper torso. These motor tics perfectly fit TS.**

OKAY DOC my head really hurts…….how does this tie into the LEAKY GUT? Its gonna hurt some more……..

The vagus nerve is also known as CN X in the neurosurgical literature. CN X is a general sensory afferent nerve providing sensation from the posterior meninges, concha (ear), and skin at the back of the ear and in the external acoustic meatus, part of the external surface of the tympanic membrane, the pharynx and the larynx (the vocal cords). As a result of its irritation, the voice feels hoarse and a clearing of the throat results. I believe that if the primary irritant was not from CN X itself but originated from CN V within the subnucleus caudalis' ephaptic connections, the vocal expressions of echolalia (throat clearing, grunting, or barking sounds) would occur

Another documented clinical sign with those who have TS is shoulder shrugging. We know that the muscles of the neck (sternomastoid) and shoulder (trapezius) are innervated by the spinal accessory nerve, CNXI. This nerve originates at the level of C1 through C5 as rootlets from the anteriolateral portion of the anterior horn of the spinal cord. The myelinated fibers of the spinal tract of V and the large spinal nucleus of V also extend to the level of C2. The

ventral horn of cranial nerve XI gives rise to the motor portion of the nerve, and it also goes to the level of C2. Second and most importantly, the trigeminal nerve also projects primary afferent nerves to the reticular formations raphe nuclei which stimulate the medial and lateral nucleus portions of the reticular formation(nuclei in the brainstem that allows you to wake up). These areas project both rostally (superior) and caudally (inferiorly) throughout the brainstem's tegmentum and influences both autonomic and voluntary muscles reflexes. According to the textbooks by Alf Brodal, The Central Nervous System (2004), and Charles Noback, The Human Nervous System (2005), the pontine medial reticular formation, when stimulated, mediates posture and orients head and neck movements and turns them to the ipsilateral side. **This is why people with TS have upper body tics that go along with their facial tics.** If you have not seen a person with this syndrome, google Tennessee Titans superstar RB Chris Johnson and watch his movements in interviews. It perfectly describes these mind bending neurological wiring diagrams I am laying out here for you. It also might help you visualize how a motor behavior could be influenced by our dietary choices.

WHAT MIGHT THE REAL CAUSE OF TS BE?

I think the a branch of the trigeminal nerve called the auriculotemporal nerve is "cross talking" with the reticular activating system in the brainstem due to gluten damage. This is the real cause of TS looking at it from an evolutionary medical perspective. These reticular cell groups are called premotor networks and they control activity of large groups of muscles such as in the neck, trapezius, sternocleidomastoid and axial muscles of the head and neck. This is the area when stimulated by CN V, implicates the shoulder shrugging and head turning classically seen in most TS patients.

OK……..now you are beginning to understand why neurosurgery residency is so damn long. It takes a while to learn all this complicated wiring might help explain these links that cross many disease processes. If you like this blog post you are going to love my book…….because it is filled with looking at diseases from this new perspective. It has fueled me to cure myself of diseases and along the way it made me realize that within us the answer to many diseases has always been right under our noses if we just look at the owners manual we came with…….That is how evolutionary medicine make you a healthier patient and a lot better healer for our species.

NOW WHAT MIGHT WE DO TO TREAT TS PATIENTS WHO ALREADY HAVE SUSTAINED THE PERMANENT DAMAGE TO THEIR BRAINSTEM FROM THE GRAINS?

The first thing to do would be to **eat a strict paleolithic diet that avoids all grains** that contain the gluten/gliadin proteins obviously. You might be shocked to learn……that this is a recommendation that most pediatricians and physicians do not make today because they have no clue about how TS might occur!

TS might just be a nutritional disorder that is easily treatable and possibly preventable if the parents/patients take action in spite of what modern medicine might tell you today. Doing an elimination diet is not damaging to any child or adult and it might just make a huge difference in their symptoms. I did tell you earlier, that once the cross talking of neurons is hardwired it might be hard to stop the classic tics. My concern is that for adults eliminating the grains might lessen the other medical co-morbidities that walk hand and hand with TS we mentioned above.

There is another more interesting possible cure for the older TS patients who have bad tics. Now I am going to draw on my previous dental education and introduce you to some dental ideas that may help people with this disorder after it becomes hardwired into their brainstems. This comes from work of some pioneering dentists (Anthony Sims, D.D.S.; Brendan Stack, D.D.S., M.S.) who believe that if they could "unload" the tempromandibular joint with a Neuro-cranial Vertical Distractor device they could possibly stop the constant pounding of the auriculotemporal nerve that is the source of the cross talk signals that are generated in TS. Personally, I think their idea makes a ton of neurological sense and again it requires no surgery to test this hypothesis. All one has to do is have an oral appliance made to unload the TMJ with a vertical distractor. I also have noticed clinically that many people with TS tend to be quiet and not very talkative. I think this is an adaptation that most use unconsciously to limit their tics. The more talking/eating one does the more activation of the TMJ and the auriculotemporal nerves occurs. This actually causes more tics to be present because it allows nerve depolarization to go from the nerve that innervates the TMJ and sends the signal into the brain stem to activate complex motor arcs that are generally automatic and not spontaneous. If you look at a TS patient you will see this in their behavior. This mechanism completely explains how this occurs. The fact that TS happens when the immune system and brainstem is still developing is no coincidence in my view. It is precisely why this syndrome occurs as it does in humans who have it. I have noticed that when Chris Johnson is interviewed here in Nashville that he suffers this same fate. I believe he is not aware of it, but I can say after observing him as a surgeon and fan of his for his 4 years here, I have picked up on this habit of his and it got me thinking about how this all occurs from a neurosurgical perspective. I think the wiring diagram above displays my feelings on how this might occur. I think gliadin antibodies are the likely trigger point for these disorders and I think

everyone with a movement disorder should be screened for autoimmunity and for the newer antibodies that we know are associated with the genetic modifications of wheat that have been outlined in the literature and recently made popular by Dr. William Davis's book called Wheat Belly.

The dental device holds great promise for those with long standing TS because it is so simple to fit and try. It fits perfectly with an evolutionary medicine perspective and that is likely why medicine won't discover it for another 100 years if they are lucky. It is the same reason why they still have their heads in the sand with the leaky gut.

I believe when medicine discovers how a leaky gut begins and what it does to the brain it will revolutionize gastroenterology disease like Crohn's, IBD, GERD, ALS, Guillian Barre syndrome, scleroderma, esophogeal cancer, pancreatic cancers, mental illness, movement disorders, and most autoimmune conditions. You would think that modern medicine would look under some "new stones" to gain insights to how one might cure a disease thought to be incurable today. Sadly, that is why dogmatic conventional wisdom creates a rut of thinking that makes the black box of these disease tighter and darker every passing year for patients with these disorders. The brain gut axis is the source of every autoimmune disease our species faces in my humble opinion. It is also why I think having a Leaky Gut Rx is a critical part of any physicians armamentarium to help people treat themselves at home to see if they improve by looking at their disease in a new way. It certainly is not something that is going to harm them long term. It is another option that just might cure them of a disease my profession has no answers for today.

TS is based on symptoms observed as described in the DSM and family history and ruling out secondary causes of the disorders. A PET scan, a CAT scan, and an MRI may be required to rule out brain abnormalities. Usually treatment is focused on relief of symptoms. Various medicines have been prescribed for treatment of TS. Such medicines are neuroleptics, (i.e., haloperidol) which has the only Food and Drug Administration (FDA) approval for the treatment of this disorder. Risperidone is used for tics. There are many side effects associated with these medicines. The antipsychotic medicines may cause fatigue, nausea, dystonia, Parkinsonism, deregulation of body temperature, increase in serum prolactin (hyperprolactinemia), and/or headaches. The benzodiazepines may cause or exacerbate preexisting states of confusion and hallucination or tardive dyskinesias. Also sedation, ataxia, and cognitive difficulties are associated with some of these medicines. The physician finds himself prescribing secondary medications to ameliorate the side effects of the initial medication.

One of the new treatments for TS movement disorders is the injection of the neurotoxin produced by the bacteria Clostridium botulinum, where the neurotransmitters that cause muscles to contract are inhibited. This treatment is repeated every three to four months. Patients may develop antibodies to the toxin rendering the treatment ineffective. Temporary weakness in the muscles that are being injected is usually one of the side effects that occur. Also flu-like symptoms may be evident. I am not a big advocate of any of these conventional treatments but that is what modern medicine peddles to patients these days.

The neurosurgical answers for these diseases are even more draconian. Remember that I am a neurosurgeon so when I tell you they are pretty dangerous and require specialized training and equipment the patient should do their due diligence and employ “caveat emptor” with those in my profession who do these surgeries. When there is a severe movement disorder and

the medicines mentioned don’t work or have become ineffective, surgery may be recommended. There are two differing types of surgery. One is called Deep Brain Stimulation (DBS) and the other is Ablative surgery. In DBS, a neurotransmitter is implanted to deliver an electrical stimulation to the area of the brain that controls bodily movement. The electrical impulse blocks the nerve signals that trigger abnormal movements. A small incision is made into the skull and an electrode is placed inside the brain extending to the area of abnormality within the brain. An insulated wire is passed through the skin of the neck, shoulder, and chest to the neurostimulator placed in the upper abdomen. These surgeries are about 75{a7b724a0454d92c70890dedf5ec22a026af4df067c7b55aa6009b4d34d5da3c6} effective in their treatment of movement disorders and may include multiple side effects. However, these surgeries have recently been questioned and have not been promoted for the treatment of TS that I know of today.

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