Autoimmune Encephalitis – A Treatable Form Of 'Autism'

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Autoimmune encephalitis is a class of inflammatory diseases of the brain that can present with a wide spectrum of neurological and psychiatric symptoms. The word autoimmune means that the body, or the

person's immune system, is attacking its own healthy tissues.

What has autoimmune encephalitis got to do with (regressive) autism?

Short answer: Possibly everything.

Long answer: Probably a lot, in some cases. Or in many cases. It is highly likely that

some, or even many, cases of regressive autism are misdiagnosed cases of autoimmune encephalitis. Keep reading...

A reminder that, by most estimates, regressive forms of autism account for about one in three of all autism cases. Other data indicate that up to 50% of all children with autism experienced some level of loss of skills and increase in severity of symptoms early in life.

Approximately one in 60 children has autism: this could mean thousands and thousands of these could actually be cases of misdiagnosed

autoimmune encephalitis.

Is this bad? And why?

This is very, very bad. Simply because Autoimmune Encephalitis (let's call it AE from now on) is in many cases treatable. Yes, that is correct. There are treatments out there that can reduce symptoms of AE, and often make it go away completely.

To state the obvious but crucial implication: this means that there could be tens of thousands, if not hundreds of

thousands, of children and adults out there who have been diagnosed with ‘autism’ whereas in fact they suffered/are still suffering from AE. Suffering needlessly.

This means that all those thousands of children and adults are spending their lives living with severe autism, and all the negative consequences it brings along, whereas they in fact could be receiving treatments for AE.

Getting better.

Functioning better.

“A diagnosis of pediatric AE should be

considered in previously healthy children who present with acute or subacute (less than 3 months) onset of new focal or diffuse neurologic deficits, cognitive difficulties, developmental regression, movement abnormalities, psychiatric symptoms, and/or seizures."

('Clinical approach to the diagnosis of autoimmune encephalitis in the pediatric patient' – Cellucci et al. 2020)

How similar are AE symptoms to autism

symptoms?

Before we talk about what AE is and how similar it is to autism let's first pin down what autism is. Or isn't. As well as what the main symptoms and defining characteristics of autism are.

We must direct our attention towards regressive autism.

We need to focus on the forms of autism where a previously bubbly, chatty, typically developing child stops using words. Stops communicating altogether. Stops reacting to people. Pointing at things. Waving goodbye.

Responding to his name.

Playing with his toys.

Recognising her surroundings.

Walking in a straight line.

And the child starts doing other strange and disturbing things. Like lining up toys. Running up and down. Walking on tiptoes. Screaming for long stretches. Biting. Vomiting. Banging his head against hard objects. Twisting her little body.

Losing balance and falling over.

Repeating a single sound. Sometimes for hours.

Rocking in the

corner. Sometimes for hours.

How autism is defined and diagnosed

The process of autism diagnosis can best be described as a scientifically unfortunate event.

Why call it scientifically or even medically unfortunate? Because we simply look at the surface symptoms and attach a label.

Lack of social communication? ✓

Restrictive and repetitive actions and/or interests? ✓

Yes, we have autism! Ta-da!!

Does anyone ask what caused that child to exhibit those symptoms? Well, no.

When a diagnosis of autism is given there is almost never investigation to rule out what possible reasons there might be for that child not being able to communicate. Not being able to learn with ease. Not being able to NOT bang his head. For not being able to NOT scream, sometimes for hours. For not being able to stop biting herself (or others).

The reasoning, such as it is, goes like this: at some point, parents and medical

professionals noticed children who suffered the kind of symptoms listed above – loss of skills, regressions, physical problems appearing – and that was called autism.

Now, each time a child presents with these complex, worrying, life-altering symptoms, we use this same shorthand: autism. A label, not a diagnosis.

Perhaps some will murmur something about it being genetic, more common than it once was, provide a list of resources, but essentially the label is provided and the health care system moves on.

Next, please.

Hypothetically, if thousands of forty year olds lost the ability to communicate, started banging their heads, withdrew to the extent they could not perform their jobs, do you believe their symptoms would warrant medical investigations?

Yes, we do too.

So why don't we treat each case of unexplained regression in the same way, regardless of the

age of the affected individual?

Especially since it is well known that genes themselves cannot account for all, or even MOST, of autism cases, and all levels of severity of autism.

Especially since we now know that identical twins with a history of early medical problems have much higher rates of autism compared to their genetically identical siblings who did not experience such negative health events.

Especially since it is well known that some infections can attack the human brain and cause symptoms and behaviours

that are identical to the very symptoms and behaviours we use to diagnose autism.

Especially since we know there is a significant association between autistic regression and family history of autoimmunity.

"...developmental regression, language loss or speech impairments may be presenting features of pediatric AE. Behavioral changes, such as repetitive or stereotypical behaviors, irritability, hyperactivity, hypersexuality, insomnia and anger outbursts, are common in pediatric AE"

('Clinical approach to the diagnosis of autoimmune encephalitis in the pediatric patient' – Cellucci et al. 2020)

Are you trying to say that we don't really know what autism is, below the surface?

Exactly.

We don't and it is alarming that most people don't seem to care. We can describe autism from the outside – we can list the surface symptoms and behaviours. This is what the official diagnosis consists of, a list of surface symptoms and behaviours.

But there has been an incomprehensible lack of curiosity and investigation into what causes these symptoms and behaviours, especially in cases of frank regression into those symptoms and behaviours after typical development.

In each case of autism, regressive and otherwise, there could be multiple underlying reasons driving symptoms and behaviours. We just don't know what they are in most cases because we so very, very rarely investigate.

You don't see things until you look. Refusal to

look doesn't mean something isn't there.

What do we know about AE – its surface symptoms and causes?

In stark contrast to autism, here we have a disorder with a set of common symptoms and behaviours, and pretty solid knowledge on what is driving them.

Pretty solid treatment options, too.

And here is an

important thing – many, if not all, symptoms and behaviours that are on the AE checklists also frequently pop up in autism. Especially in regressive forms of autism.

So how do we know that some cases of autism are not misdiagnosed AE?

We don't. We just don't know.

Not because the

answer is unknowable but because we do not look for answers.

What AE symptoms should (but never do) serve as red flags for ruling out AE in children with (misdiagnosed?) autism?

Myoclonus

Muscle twitches (but not tics). Officially described

as sudden, brief, shock-like, involuntary twitching or jerking of muscles.

Jerks or twitches can be variable in intensity and frequency. They can be localised to one muscle or a group of muscles in one part of the body, or spread all over the body. They can occur in a sequence or singly, many times each minute or infrequently.

How common are such sudden jerks and twitches in (severe and/or regressive) autism?

It would probably be easier to count children with regressive autism whose parents

report an absence of some degree of myoclonus, rather than its presence at some point in their child's history.

It is that common.

On the other hand we don't have precise numbers and statistics. No one has bothered to count.

Dystonia

Sustained or repetitious muscular contractions that cause twisting, unusual positioning or repetitive stereotyped movements.

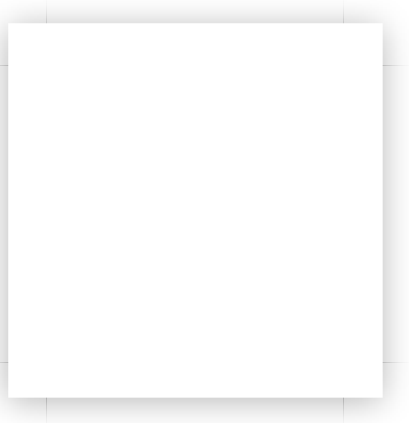
Read that again.

Unusual positioning. Abnormal limb postures. Twisting

of arms or legs. Repetitive muscle movements.

How common are those in (severe and/or regressive) autism?

Quite common, in fact. Most parents of children and adults with severe autism would have frequently observed their child in an abnormal posture, twisting their body, perhaps like this:



Dystonia may also manifest as:

- loss of muscle

coordination
- gait impairment
- dropping items
- muscle pain and/or cramping
- difficulty finding a comfortable position
- trembling/spasming in the diaphragm while breathing
- difficulty swallowing
- disturbed sleep
- exhaustion
- poor concentration
- blurred vision
- digestive problems
- short temper
- hyperventilation (fast breathing)
- blepharospasm (involuntary twitching, blinking or closure of eyelids)

Each of the above symptoms are common in severe, regressive autism.

And some of them – like loss of muscle coordination, disturbed sleep, exhaustion, poor concentration, digestive problems and short temper/irritability are extremely common.

Orofacial Dyskinesia (Tardive Dyskinesia)

Involuntary and abnormal movements of the jaw, lips and tongue. Typical symptoms include facial grimacing, sticking out the tongue, sucking or fish-like movements of the mouth.

While these odd movements are mostly limited to

the mouth, face, jaw, and tongue, symptoms of tardive dystonia also include slower, twisting movements of larger muscles of the neck and trunk. In some cases, the arms and/or legs may also be affected by involuntary rapid, jerking movements (chorea), or slow, writhing movements (athetosis).

It is not uncommon to observe children (and adults) with severe and/or regressive autism carry out movements like these:

Aphasia/mutism—also known as loss of language

Aphasia is a language disorder, affecting the production or comprehension of speech and the ability to read or write.

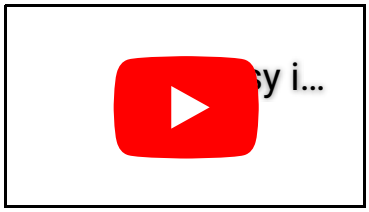
The significance of this is, of course, impossible to overstate. Loss of previously acquired language is the hallmark of regressive autism.

People have tried to untangle the relationship between autism and ‘pure’ aphasia as far back as the 1970’s, but could not get very far.

Seizures

(including ‘invisible’ Absence seizures)

Those can look like this:



Or this:

Also see:

Other symptoms of AE that could serve as ‘misdiagnosis red flags’ to initiate investigation in regressive

autism

- changes in behaviour and mood, especially irritability, anxiety, agitation
- cognitive impairment
- changes in speech patterns or (in)ability to form sounds
- insomnia
- fever
- loss of eye contact
- decreased appetite
- sensory disturbances: sensitivity to light, sound and touch.
- headache
- inconsolable crying
- urinary incontinence
- self-harming behaviour (e.g. self-biting)
- difficulty swallowing

All of these symptoms frequently appear during the onset of regressive autism.

"Parents described temper tantrums, behavioral change, agitation, aggression, and progressive speech deterioration as initial symptoms; however, these behaviors in children can initially be overlooked."

('NMDAR encephalitis in children and adolescents' – Florance et al. 2010)

Importance of distinguishing

features and symptoms of autoimmune encephalitis in children versus adults

AE manifests differently in children versus adults. In teenagers and adults the main symptoms are in almost all cases of psychiatric nature, such as psychosis, delusions and hallucinations, which are often absent in children.

AE in adults is also often associated with a tumour, which is a rare finding in children with AE.

In contrast, movement disorders and seizures are significantly more common in children, and so are irritability, anxiety and inconsolable crying, and loss of eye contact and interest in surroundings.

In very young children/toddlers one of the first red flags for suspicion of AE is movement and gait abnormalities – unsteady walking and loss of balance.

"This case series demonstrates a novel clinical phenotype of gait disturbance as an initial symptom in children <3 years old with anti-NMDAR encephalitis…Gait disturbance should raise the concern for anti-NMDAR encephalitis in young children.

('Gait disturbance as the presenting symptom in young children with anti-NMDA receptor encephalitis' – Yeshokumar et al. 2016)

"Paediatric patients with AE were more likely to present with psychiatric symptoms, sleep disturbances, focal seizures, and/or status epilepticus compared to adults ($p < 0.05$). Insomnia and hypersomnia are common sleep problems associated

with AE that should be screened early in the diagnostic evaluation."

('Clinical features of paediatric and adult autoimmune encephalitis: A multicenter sample' – Roliz et al. 2021)

"In conclusion, we believe that NMDAR encephalitis is probably underreported in infants and toddlers. This case, initially presenting with behavioral changes and autistic features, highlights the diagnostic challenges presenting in this age group.

('Anti-NMDA receptor encephalitis in a toddler: A diagnostic challenge' – Khundakji et al. 2018)

"Differences between paediatric and adult AE have become increasingly

recognized…While adults typically present with isolated, well-defined clinical syndromes, children often demonstrate multifocal neuropsychiatric symptoms that may overlap with other diseases. Young children may not be able to describe their symptoms, which can complicate diagnosis and delay management.

Additionally, paediatric AE is less likely to be associated with tumors than in adults, and the autoimmune profile between children and adults varies, with children more commonly testing positive for NMDAR antibody or negative for any known autoantibody."

('Clinical features of

paediatric and adult autoimmune encephalitis: A multicenter sample' – Roliz et al. 2021)

Autoimmune Psychosis vs Autoimmune Encephalitis

While most individuals with AE show neurological symptoms such as seizures, cognitive dysfunction, and movement disorders, in some AE patients the predominant symptoms are of behavioural and psychiatric or

psychotic nature.

On the other hand, psychosis and other psychiatric disorders frequently occur in patients with autoimmune diseases such as systemic lupus erythematosus and multiple sclerosis. This association is thought to be linked to inflammation and/or antibodies present in autoimmune diseases having an effect on brain function.

In other words, there is a clear overlap between psychosis and pathological processes associated with inflammation and (auto)immunologic

al dysregulation.

This clear overlap has prompted scientists to suggest that some cases of psychosis could be (auto)immune in origin.

Recent research has demonstrated that some individuals with psychotic disorders do have a specific autoantibody-mediated disease called autoimmune psychosis (AP). Accordingly, treatments aimed at AE—steroids, IVIG, plasmapheresis, rituximab—often lead to significant improvement or disappearance of psychotic symptoms in those patients.

But what has psychosis, or schizophrenia, got to do with autism?

The line between autism and schizophrenia, or autism and psychosis, is a very thin and blurry one.

The cluster of symptoms we call ‘autism’ today used to be termed ‘childhood-onset schizophrenia’. Even well into the 1970 researchers were still publishing papers

on ‘childhood schizoid disorder’, what we now call autism.

Psychosis is defined as a period of abnormal perceptions (hallucinations) and distortions of reality (delusions). The person can’t tell what’s real from what’s imagined. Such psychotic episodes are the hallmark of schizophrenia.

But it’s significant to note that schizophrenia also includes these symptoms:

- poor executive functioning (eg understanding and following instructions)
- focusing/paying attention
- forgetting or

losing things
- working memory (using new information immediately after learning it)
- communication difficulties (not speaking, or disorganised speech: uttering words and sentences that don’t make sense, jumping from one subject to another)
- lack of energy
- irritability and agitation
- lack of motivation and interest in surroundings
- repeating movements or gestures
- sensory processing problems
- avoidance of eye contact
- social withdrawal

All of the above

symptoms of schizophrenia are very frequent in autism, especially in individuals on the severe/profound end of the autism spectrum. In other words almost all of the common symptoms of schizophrenia overlap with autism.

All apart from hallucinations and delusions.

But, wait a minute! How would we know?

How would we be able to tell if a person affected with severe non-verbal autism is experiencing hallucinations? How could we know if they can

tell what is real, or not real, especially if they are very young and have experienced these hallucinations for years? Unless a person could communicate with some nuance, could they share any delusions they might have experienced, be experiencing?

When research looks at people with less severe autism, those able to communicate, we find much higher rates of schizophrenia. Like, three or four times higher.

If a person with severe autism who could not reliably communicate was experiencing altered perceptions

of reality could those around him or her know this?

If a person with autism tried to express their feelings and experiences through behaviours, would we simply call those ‘autistic behaviours’ and move on?

Sadly, very likely the answer is yes.

But what do we know about the causes of psychosis?

Short answer:
Mixed bag, same as

autism.

What we do know is that the family history of autoimmunity significantly increases the risk of psychoses, same as with autism (see above).

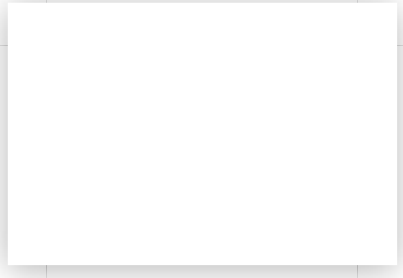
We also know that maternal/prenatal infections are associated with offspring psychosis. Same as autism.

We also know that the risk of psychotic disorders in maternal infections is several times higher for male children. Same as autism.

We also know that there is evidence of increased activation and density of microglia, the

immune cells in the brain. Same as autism.

As for autoimmune psychosis, it is thought to be triggered by central nervous system infections.



So how do we know that some cases of (regressive, severe) ‘autism’

are not in fact misdiagnosed cases of autoimmune psychosis?

We don't.

We never look.

Diagnosing AE and AP

> Investigations for diagnosing AE and AP can include brain MRI and EEG, as well as blood and cerebrospinal fluid tests for markers of

inflammation and presence of certain brain-specific antibodies.

However, negative test results for autoimmune antibodies do not rule out AE or AP. A significant percentage of AE cases, especially children, are suspected to be caused by antibodies for which a diagnostic test is not yet available, or by antibodies yet to be discovered. Because of this gap in knowledge, specific diagnostic criteria for "antibody negative" autoimmune encephalitis have also been defined.

Recent clinical guidelines for

diagnosing AE in children state that:

> *"A diagnosis of paediatric AE should be considered in previously healthy children who present with acute or subacute (less than three months) onset of neurologic deficits, cognitive difficulties, developmental regression, movement abnormalities, psychiatric symptoms, and/or seizures. A normal EEG is unusual in children with AE during active disease, although prolonged EEG may be needed for improved sensitivity.*

> *Therefore, focal or generalized seizures, epileptiform discharges, and encephalopathic changes, such as diffuse or focal slowing, may help to distinguish AE from primary psychiatric disorders …Adults with AE are more likely to have EEG changes predominantly involving the temporal lobes, whereas EEG findings in children may be more generalized. Specific EEG features, such as the "delta brush" pattern and extreme spindles, have been linked*

> *to anti-NMDAR encephalitis, but sensitivity is low. The most common autoantibodies identified in children target NMDAR, MOG, GAD65, and GABAAR…Given the rarity of other autoantibodies, further testing should be considered only if antibodies to these targets are negative and suspicion of AE persists… Antibody testing should be performed in both CSF and serum to avoid false-negative and false-positive results."*
> (Cellucci et al. 2020)

Treating AE and AP

Treatments include immunotherapies such as steroids, intravenous immunoglobulin (IVIG), plasmapheresis, rituximab.

Interestingly enough, some of those treatments have also been shown to reduce the core symptoms of autism.

A recent study of 40 children with autism, carried out by Rocha-Brito and colleagues, has shown corticosteroid prednisone to be an effective treatment for

improving social communication and language impairments in children with regressive autism.

Similarly, several small scale trials, like the one by Melamed and colleagues, have demonstrated that IVIG treatment can be effective at reducing core symptoms of autism in a subgroup of children.

“Follow-up during the next six months necessitated monthly IVIg. Several relapses occurred during treatment, manifesting as a reemergence of her irritability, insomnia and poor eye

contact. She received plasma exchange during her second and third relapses, in addition to rituximab. A dramatic improvement in her social skills and irritability appeared within hours following plasma exchange."

('Anti-NMDA receptor encephalitis in a toddler: A diagnostic challenge' – Khundakji et al. 2018)

So what needs to happen to make sure AE and AP are never misdiagnosed as

‘autism’?

More awareness. Less labelling and more diagnosing. More listening to parents. Less rejection of their observations.

More genuine concern for patients. And more professional curiosity.

According to the Hippocratic Oath, doctors should “*apply, for the benefit of the sick, all measures [that] are required, avoiding those twin traps of overtreatment and therapeutic nihilism.*”

One could argue that labelling everything as just ‘autism’, dismissing

the possibility those symptoms may point to a potentially treatable condition, and refusing to engage in differential diagnosis is extreme therapeutic nihilism.

Sentencing a patient to a lifetime of suffering and disability that results from a treatable condition, on the other hand, sounds like the opposite of "*do no harm*".

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Brain Glucose and Glycogen in Autism: Speech, Seizures, Sleep & Beyond

The following

Autism and metabolic diseases – more treatable ‘autisms’ hiding in plain sight?

Aggression In Autism – One Simple Cause

In addition to the core symptoms of autism, which include social

article is written by an autism parent and researcher, who has an interest in both autism and sports sciences. It lays out possible underlying reasons for some of the struggles and symptoms associated with autism. It is not possible to say who might…

Metabolism is the process through which our bodies convert nutrients from food and drink into energy. We need energy for the basics of life, such as breathing or blood circulation, and so countless metabolic processes take place in our cells and our organs all the…

communication difficulties, restricted interests, and sensory processing difficulties, both children and adults with autism often present with many other 'autism-related' symptoms and behaviours….

Depression in Autism – More Than Meets The Eye?

High rates of depression and suicide in autism Depression is a common and serious problem in autism, and one of

'From Bench to Biopharma' International Conference on Translational Research in Autism – Day 1 Recap

Synchrony symposia,

NEW UK Autism Research Hub: The Synapse Centre for Neurodevelopment ESNEFT

The Synapse Centre for Neurodevelopment The Synapse Centre, based within the ESNEFT (East Suffolk and

the main contributors to poor quality of life. Both children and adults with autism experience high rates of depression and other mood disorders. One...

Addressing Poor Health & High Death Rates in Autism

"We must first recognise ASD as a whole body disorder" Autism, or Autism Spectrum Disorder ASD, is traditionally seen as the result of behavioural and neuropsychiatric dysfunction. However there is a

organised by The BRAIN Foundation in partnership with UC Davis MIND Institute and CalTech, is the first and only international conference on translational research in autism that brings together academia, biotech, pharmaceutical companies and...

Synchrony 2019 – Translational Research in Autism symposia

The first Brain Foundation annual symposia, Pleasanton,

North Essex NHS Foundation Trust) is a new research centre based in the East of England looking to translate biomedical research into practical therapies for local...

Transcranial direct current stimulation tDCS – a novel treatment for autism?

One of the treatment modalities that has shown the greatest promise for reducing symptoms of autism in recent years is transcranial direct current stimulation (tDCS).

strong evidence that various physical, or biomedical, problems can...

Cannabis for Treating Core and Comorbid Autism Symptoms – Where are we at?

Several studies published in recent months investigated the effects of cannabis-based products for treating autism. Although the studies were open-label and relatively small in scale, the overall results were overwhelmingly positive, with

California 8-10 of Nov 2019, aimed to connect researchers with clinicians, donors & stake holders to help translate research efforts into evidence-based treatments for autism and its co-morbidities. It highlighted the need for multidisciplinary collaboration, detailed diagnostics and personalised treatment...

Century-old drug offers new hope for autism treatment

A small double-blind, placebo-

The most recent study confirmed and expanded on the findings of previous investigations, which strongly indicate that tDCS could have positive effects on cognition, behaviour and physical health, and improve quality of life and autonomy for a large percentage of individuals with autism.

Higher rates of autism in children with various congenital disorders

Children with

statistically significant improvements in social communication, language, restrictive/repetitive and challenging behaviours.

controlled trial shows dramatic effects of suramin as a treatment for autism. Improvements were seen in all three core features of autism: language, social interactions, and restricted or repetitive behaviours across multiple diagnostics in multiple tests in all who received the active treatments, absent in the placebo arm

Congenital Heart Disease, Tuberous Sclerosis Complex, Duchenne Muscular Dystrophy, Ehlers-Danlos Syndrome, Neurofibromatosis Type 1 suffer high rates of autism. Everolimus, an mTOR inhibitor with strong neuro-inflammation attenuating effects, reduces core autism symptoms in some children with TSC, epilepsy and autism...

4 Comments

Anthony ngugi on 13th April 2021 at 8:44 am

Reply

Hi..Anthony here with an autistic boy 13years .How is IViG administered ans how is the availability of the same

Pauline on 12th June 2022 at 2:05 pm

Reply

Hi, thank you for your broad information. I have a 13 year old non verbal child. How can he be assisted with the treatment of Immuno therapies, JVIG to reduce the core symptoms of autism eg social and language impairment, ability to read and write, loss of eye contact etc

Frazer on 26th June 2022 at 12:17 pm

Reply

Hey, I am one of the many out there who have regressed into a fully-blown Autistic state. I've known since day one that 'Autism' is the END RESULT of PATHOLOGY. Me, as an individual organism with my unique neurochemistry has ZERO pathology to show for my Autism, despite my subjective experience and my affect.

What brought me to your website was encephalitis/encephalopathy. I KNOW my immune system isn't right. WHY? Because every time I use CBD and activate those CB2 receptors in the periphery, I am bed

bound with vomiting, sickness behaviour and vomiting. What else have I noticed? GABA and GLUTAMATE! THC activates the CB1 receptors and inhibits intracellular calcium which gives me INSTANT (WITHIN SECONDS) relief of SEVERE sensory issues. I was house-bound after my brain was ruined by Venlafaxine (an SNRI) which led to my brain unable to tolerate a humming sound, let alone use a phone or watch TV or read. Pure silence just left to rot until I started using cannabis. I can laugh, enjoy music, and I don't shut up, whereas I am not far from mute. My brain literally has nothing to say.

I regressed at age 21 after a period of INTENSE exercise (boxing) and now I'm 28. Misdiagnosed with ME/CFS, but on paper all my symptoms match. According to time and dates, it's wrong. I've been punched which broke my jaw at 14 and I had whiplash in a car crash a year before.

I genuinely believe my body is going through a state of 'immuno-excito-toxicity' or something similar. These are my symptoms now at age 28:

. Can't tolerate much eye contact (wear sunglasses in the rain and in public)

. Limbic system activation (fear) when I accidentally make eye contact with people.
. Decrease in executive functioning (frontal issues)
. Constant cortical pressure at the front of my brain (feels bruised)
. Unable to make decisions (especially on the spot)
. Hand deformity (x-ray says it's normal, but it isn't)
. ADULT-ONSET paranoia and delusions (I genuinely believe these assholes want people to be unwell and they don't give a fuck about anyone UNLESS there's profit.
. Severe muscle fatigue (ATP issues triggered by intense exercise)
. Anhedonia

These are a few symptoms that came to me. Parents, love your children and KNOW that THEY are right, not you. ALWAYS listen. Our brains aren't like yours, so stop projecting your desires on your child. COMPLETELY DIFFERENT WIRING. This isn't a MIND thing. It's not a WILL thing either. The 'mind' and the 'will' are an END PRODUCT of neurochemistry, not the beginning.

Leah on 29th November 2023 at 6:07 am

Where can I find a clinic and or Doctor to guide me in testing my 5yr old son for regressive ASD? He was a perfectly normal child until age 3.5. He lost all communication, stopped responding to his name. Became agitated and aggressive, lost eye contact. He had a seized around age 2.5.

Reply