



The science behind the lion program.

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Area of study:

Breakdown of the effects of stress hormones in behaviour; on the development of the Corpus Callous, amygdala and the hippocampus; in child abuse/ neglect and high intakes of trauma. Cell division in the NG2 Glial cells. In fault pattern broken and default as cells damage and have to find way back to its pathway before fault occurred.

and neurons in stress response and the growth peaks in brain pathways

The effects the hormones play in this role.

Time frame studied; up to 21 years of age

Aims of the science paper:

To prove childhood trauma is mimicking Dual Autism and ADHD behavioural pathways in brain development. Also looking for the opening of the chemical balance in puberty. The opening of pathways in bipolar in teenager and later life. Also then triggering the schizophrenia pathways in reality separation, starting off as paranoid schizophrenia due to the high levels of paranoia in childhood trauma when the child does not feel safe, is looking for threat and is survival mode.

Pathways opening in later puberty... and developing into the 20s.

To see how the cognitive and sensory pathways are developing within this time frame and the part hormones play in this.

Three pathways for ADHD and Autism are;

Pathway one: Inherited.

We know this as the forebrain development; changes occurred during the very start of its development. That only happen when the conditions are inherited. The forebrain is the first part of the brain to develop in the womb. If you like it's a seed that's grows... in seed mode... we see fault in the forebrain which must be genetic. The baby was not foamed enough to feel emotional responses etc... it is early in pregnancy. It must be genetic.

Add in the genetic work done in autism which already prove it 80 per cent inherited. Add in forebrain development and pathway of inherited is established.

Pathway Two: stress response in the womb.

The change starts and its not as big of affect as the first one. Its a slight change that slowly affects the right side of the basal forebrain. The baby is foamed and able to feel emotions and stress..

Pathway three: Childhood trauma over prolong time.

The forebrain has no changes. The baby has been born healthy but went on to have a childhood of trauma

Genetic factors are a significant part of fetal development in the womb because they provide the blueprint for the development of the fetus. Inherited traits

The first part of the brain to foam in the fetus is the forebrain, which develops into the cerebrum. This area is responsible for higher functions such as thinking and problem-solving. The forebrain begins to take shape at the top of the neural tube, which forms during the early weeks of pregnancy

The forebrain, a critical area for cognitive and social functions, shows distinct changes in individuals with autism spectrum disorder (ASD). Research indicates an **imbalance of excitatory cortical neurons** in the forebrains of ASD patients, which may indicate the onset of the disorder. This imbalance is linked to specific genes or transcription factors essential for early brain development.

The first neuron in the fetal brain becomes active around the **sixth week of gestation**. This marks the beginning of the fetal brain's functioning, with initial neural activity detected shortly thereafter. The first synapses in the spinal cord form around the **fifth week after conception**, indicating the start of detectable brain activity in the fetus. These early steps are crucial for forming the neural pathways that will support movement and behavior, and by the time of birth, the fetus can exhibit simple reflexive movements driven by these early neural circuits

The development of excitatory cortical neurons in fetal brain is a complex process that involves the differentiation of neural progenitor cells (NPC) into excitatory neurons (EN) and inhibitory neurons (IN). This process is influenced by various factors, including the presence of FGF2 and the lineage specificity of neural progenitors. The culture of fetal prefrontal cortex tissues at different gestational weeks has shown that excitatory neurons are predominantly present in the culture of fetal prefrontal cortex

tissues obtained at gestation weeks 11 and 20. This transition in cell proportions is primarily driven by the differential lineage specificity of neural progenitors in the fetal cortical tissues at distinct stages of fetal brain development.

The study also highlights the importance of PAX6 and SOX2 protein markers in the ventricular zone as early as at gestational week 8, indicating the early differentiation of NPCs into neurons. The sequential differentiation of NPCs into neurons in the subventricular zone by gestational week 12 and into oligodendrocytes and astrocytes in outer sVZ by gestational week 23 further underscores the intricate developmental process

Introduction:

Autism, ADHD is down to the inherited genetically defect in the **forebrain** then leading to reduction in the amygdala and hippocampus and all areas of the brain during foetal development.. having a knock. affecting the basal forebrain patterns, accumbens nucleus, cerebellar hemispheres, and perisylvian regions, including insula and putamen. Frontal lobe.

(Yes, the forebrain development is influenced by genetically factors.)on effect on other areas of the brain.in pre-birth of the **1GF1 which in term causing a reduction in the proteins that are needed for the grey matter and so on.** **However, a core feature of autism is repetitive or restricted behaviour, which may arise from dysfunction in a brain region called the basal ganglia. This region controls goal-directed movements and habit learning.**

The amygdala's development and function are influenced by genetic factors.**The amygdala's grey matter plays a crucial role in emotional processing and behavior. It is involved in the processing of fear, anxiety, and aggression, and is part of the limbic system, which is essential for survival. The amygdala's grey matter density can be affected by various factors, including genetic predispositions and environmental influences.**

One must remember if a child is born early before the due date, Autism and ADHD pathways can occur (**in pre-birth 1GF1 stress response**). As we do see both conditions on the family tree, brothers, mothers, grandparent have one or the other, which is rather interesting. One must remember the foundations of anything in the human body requires so aspect of genetic blueprint.

The amygdala's development in children is a complex process that involves both structural and functional changes. During fetal development, the medial amygdala (MeA) plays a crucial role in regulating social and emotional behaviors. Malformation of the amygdala is a hallmark feature of disorders of social cognition such as autism spectrum disorders (ASD)

The forebrain is the first part of the brain to develop, starting from the third gestational week. It includes the forebrain, midbrain, and hindbrain, with further differentiation resulting in secondary regions like the telencephalon, diencephalon, and metencephalon in subsequent weeks. The forebrain is crucial for higher brain functions, including thinking, reasoning, and planning. Reduction **Basel forebrain= reduced volume**.

After birth What we are seeing in trauma is that dual autism and ADHD pathways are being created through the stress response of trauma (**1GF1 + GH+ stress hormones**) in child brain development. Which depending on use of inner head depression, drugs, medication, drug to change the brain chemicals is coke and crack. Enhancing paranoid and concept of reality and what is real. Could in punning stage open other pathways main one being bipolar. Due to reduces in sizes of parts of the brain and the white mattering. Depending on use of safe space in childhood during abuse, highest levels of safe space use being in extreme childhood abuse and neglect. Children built their safe place in their own minds; some will invent imaginery friends if they feel isolated. Most common is safe place we go to in our heads during the trauma, as it allows us to block out pain and seek safety in a place where there is none at home.

Summary of paper.

In a prolong stress response in the developing child brain we see cell, Neurons and axonal fibres patterns go into a default. During peak times of growth hormones and (1GF1=1).

In stress response in peak Growth hormones in 2 years and puberty. We see a fault in the pattern.

Neurons in stress response do into a default mode and become damaged or weakened. A redirection is then seeker by the synaptic connection as the pattern tries to restore itself. In pruning both at say 2 and puberty... The pruning eliminate.

The weakened neurons, to allow learning and development to continue.

Default-damage-redirect-pruning=eliminate to allow for more room.

The prefrontal cortex in prolong stress... neurons follow the default pattern.

Resulting in a reduction in the prefrontal cortex.

Hippocampus both sides left and right all together have 16 million neurons, in prolong stress default pattern followed in peak developments, and reduction occurs. One must remember that an abused child is in survival mode and is in fear and in a stress state for long periods of time. The longer the time, the more effect on the size and changes in the behaviour. How the child process emotions and anger, memory and so on. Again, the neurons in stress response will become damaged due to peaks in growth hormones and 1GF1-1.

The cerebral cortex, 16 Millon neurons. All following this principle in neuron pattern in stress response again damaged neurons and knock on effect on the grey matter. The Amygdale becomes overactive as the stress response reacts with the growth hormones etc.

Neurons or neuron cells are the only cell that cannot achieve division..... instead use the pruning stage to elimainate the weakened neurons.

The Amyloid beta protein (AB) in the stress response becomes overactive.... and decreased the grey matter in prolong trauma. The primary protein for the grey matter.

(NG2) glial cells, supporting cell in stress response again the cell division pattern is broken. The cell goes into default and reduces and then try to fix the cell division pattern.

Frontal lobe again in the response grey matter volume is reduced.

The corpus callosum contains 200 to 300 Millon axonal fibres connecting both sides, again in stress weakened and then redirect and pruning again eliminate the (AF). Allowing for better and stronger connections. Myelination coating reduces and synaptic connections again in the neurons ... again go into a

default, redirect and the weakened connections are pruned. The brain is the most amazing part of the body. In the abused child's brain, we see changes that will last a lifetime.

Changes to the amygdala due to stress response over long period of time due to the child being in survival mode, will trigger openings of increased problem solving as the child seeks to find solutions to the hell they are living in and this change in the development of the amygdala due to stress response opening up a pathway of dyslexia, as the child's creativity levels are enhanced due to the child using their imaginary world inside their mind. The stress response in prolonged child abuse causing overactivity of the amygdala. (Foaming of memories related to Fear and emotional events.)

Breakdown of the science work:

It is important to state that during peaks of growth hormones in stress response... ok so we see in the 0-to-3-time frame... if too much =adhd pathways to form... This is why you see it starting around this age. Add in environment and attachment into mix. If stress responses is triggered over long time during this time frame both dual pathways will form... Then as we go through childhood... reduction occurs in several areas of the brain. And reduction of white matter and grey. Then we add in puberty and again pruning and growth hormones... add in stress response over a long period of time and opening can occur of new

disorders as over production is achieved. Due to growth hormones etc.. we see it clearer in soldiers who have suffered PTSD.. The over production. One must consider females live inside their head and males outwards.. Which is why women have greatest levels of depression and self harm.. Males having greatest levels of ADHD.. Although in pressure point mode.. Both enter the hyper stage when unable to process their thoughts and feelings in a stress response.. Also we learn how to cope in stress through our attachment to the world and our social connection (learnt crisis responses in PCM) adhd hyper reducing as we enter later life in adulthood, as all disorders begin to reduce due to their relationship with hormones and our devolving of coping skills and reactions to crisis enols.

We have two peaks of growth hormones that effect our brain development in childhood. 2 year old peak and puberty..

Hormonal fluctuations that trigger growth spurts

Toddlers experience **hyperactivity at growth hormone peaks** due to hormonal fluctuations that trigger growth spurts. During these periods, **growth hormone** and **insulin-like growth factor-1 (IGF-1)** levels increase, promoting rapid growth and development. This surge in hormones can lead to increased energy levels, restlessness, and changes in behavior, as toddlers may become more energetic and test boundaries during growth spurts

IGF-1 in ADHD

IGF-1 has been studied for its role in ADHD, a neurodevelopmental disorder that affects attention, behavior, and sleep. Research indicates that children with ADHD exhibit lower levels of IGF-1 and TSH, with elevated free T3 and free T4 levels compared to those with normal sleep patterns. This hormonal change is associated with sleep disturbances observed in children with ADHD. The study highlights the importance of sleep for daily functioning, health, and well-being, emphasizing the need for adequate sleep duration and quality to maintain good physical and mental health, as well as enhanced behavior, attention span, and cognitive abilities.

IGF-1 plays a crucial role in the growth and development of adolescents during puberty. Its levels peak during puberty, driven by growth hormone (GH) secretion, and are

influenced by factors such as sexual dimorphism, nutritional status, and the timing of puberty onset. These interactions highlight IGF-1's critical role in skeletal growth, sexual maturation, and overall development.

IGF-1 levels differ by sex, influenced by androgen and estrogen modulation, with higher levels noted during central precocious puberty in girls. Malnutrition and chronic illness blunt IGF-1 levels, limiting growth, while well-nourished adolescents maintain higher IGF-1 levels.

IGF-1 is a crucial hormone in the body's response to stress. It plays a significant role in promoting growth during childhood and maintaining tissues and organs throughout life. IGF-1 is synthesized in the liver and enters nerve tissues through the blood-brain barrier or cerebrospinal fluid in the choroid plexus. It is involved in cell growth, division, and regeneration in various tissues, making it essential for normal growth and development. Chronic stress can disrupt IGF-1 production

The basal ganglia consist of several interconnected nuclei that are primarily involved in the control of movement and coordination. They are located at the base of the forebrain and are composed of the following main structures:

Caudate Nucleus: This C-shaped structure is involved in various functions, including motor control and learning. It is located medially and is part of the striatum, which also includes the putamen.

Putamen: Situated laterally to the caudate nucleus, the putamen works closely with the caudate to process information related to movement and motor skills.

Globus Pallidus: This structure is divided into two parts: the external (GPe) and internal (GPi) segments. It plays a key role in regulating voluntary movement by sending inhibitory signals to the thalamus.

Subthalamic Nucleus: Located below the thalamus, this nucleus is involved in the regulation of motor control and is part of the indirect pathway of the basal ganglia circuitry.

Substantia Nigra: This structure is crucial for movement regulation and is known for its production of dopamine, a neurotransmitter that plays a significant role in reward and movement. It is divided into two parts: the pars compacta (which produces dopamine) and the pars reticulata (which has inhibitory functions).

Synaptic pruning is a natural process that occurs in the brain, primarily during early childhood and adolescence. It involves the elimination of unnecessary synapses, allowing the brain to maintain efficient communication and optimize its neural connections.

Early Childhood: The brain experiences a surge in synapse formation, known as synaptic exuberance, followed by pruning that begins around 2 years of age, with up to 50% of synaptic connections potentially pruned.

The growth hormones peak in the pre puberty pruning

The peak of neuronal development occurs around the age of three. At this stage, the brain has formed about 100 trillion connections, which are crucial for learning and cognitive functions. The brain continues to grow and develop beyond this point, but the rapid growth and connection formation during the early years are particularly significant for a child's overall development

Adolescence: Another phase of pruning occurs during adolescence, particularly affecting the prefrontal cortex, which is crucial for decision-making and social behavior.

The **peak of neuronal development during puberty** is influenced by several factors:

Puberty onset is crucial for neural development, with changes in both white and grey matter occurring during this period.

1

Gonadotropin-Releasing Hormone (GnRH) neurons play a vital role in ensuring the onset of puberty, which is essential for successful reproduction.

1

The molecular and neuroendocrine networks governing puberty are complex and involve genetic and environmental factors.

1

Overall, puberty is a significant maturational event that affects various aspects of brain development, including resilience and immune function

Add in stress response of trauma and we will see reductions... and then go into over drive as puberty everything speeds up in terms of brain development.

The brain's influence on neurons is profound and multifaceted. Neurons are the building blocks of the nervous system, and their activity is crucial for various functions, including mood regulation, memory, and emotional expression. The brain's complex structure and the intricate network of neurons enable it to process and transmit information, forming the basis of thought, memory, and emotion. Neurons

communicate through synapses, where neurotransmitters are released and bind to receptors on neighboring neurons, passing the signal along. This process, known as neurotransmission, is essential for controlling countless functions in the brain and body, including mood, muscle movement, and overall behavior.

Hormones play a crucial role in the nervous system by influencing various physiological processes. They are involved in the regulation of temperature, metabolism, and sleep, among other functions. Key hormones such as Cortisol, Estrogen, Progesterone, and Testosterone have specific effects on the nervous system. For instance, cortisol is associated with the stress response, while estrogen and progesterone can modulate brain function. Testosterone, particularly in males, is linked to spatial cognition. These hormones contribute to the body's overall functioning and can affect mood, cognitive function, and **behavior**.

Growth hormones, particularly growth hormone (GH), have a significant impact on neurons in the body. GH is secreted by the anterior pituitary gland and plays a crucial role in growth, cell repair, and metabolism. It affects various cognitive functions such as memory, learning, attention, and problem-solving.

The reason why growth hormones are so important in this science work... is when we cross examine against the work in cognitive stages of brain development. (see research paper) One can see with ever growth spur comes a cognitive development spur. Which must mean that growth hormones are the super food of the brain during development stages...We see this better when we look at how trauma effects puberty.

Childhood trauma has been shown to alter the default mode network (DMN) in several ways. Research indicates that trauma can lead to **reduced capacity for self-referential processing, impaired social cognition, and increased mind-wandering**. These changes in DMN function can have significant implications for an individual's ability to process thoughts and emotions, which may contribute to the development of PTSD and other mental health conditions. The DMN is one of the first networks affected after a traumatic event, making it a valuable tool for understanding the impact of trauma on brain function

Childhood trauma can significantly impact the control executive network, leading to difficulties in cognitive control, emotional regulation, and decision-

making. The brain's development is highly sensitive to early environmental influences, and prolonged exposure to stress can alter its structure and function. Trauma can disrupt brain development, particularly in areas related to cognitive control, emotional regulation, and decision-making. Adults who experienced trauma during their formative years often struggle with essential executive functioning skills, such as self-regulation, planning, and organization. These difficulties can manifest in various ways, from impaired self-regulation to challenges in planning and organization. With the right interventions and strategies, trauma survivors can work to rebuild their cognitive strengths and lead more organized, productive, and emotionally balanced lives.

The **corpus callosum** contains approximately **200–300 million axonal projections**, making it one of the largest white matter structures in the human brain

Reduction of axonal fibres.. Purnning of the damage fibres.

The **corpus callosum** is a crucial structure in the brain that connects the cerebral hemispheres and plays a significant role in communication between them. During **stress**, particularly from traumatic brain injury (TBI), axonal degeneration can occur, leading to disruptions in axonal transport and subsequent axonal swelling and disconnection.

Autism or ADHD causes would then be:

DNA Passed through inherited root.

DNA is the key to this process, carrying the genetic instructions that determine how we look, grow, and function

Enviromental and socially. Trauma.

Damage to the brain. Which would create deeper pathways.

Too much growth hormones

The dual pathways of autism and ADHD are open by the pituitary gland in trauma responses during childhood development stages.

The gland:

Producing and releasing hormones that regulate growth, metabolism, reproduction, and response to stress or trauma. Controlling the function of most other endocrine glands in the body. Maintaining homeostasis by secreting hormones that communicate with various organ systems. Sensing the body's needs and regulating different processes to maintain an appropriate environment.

Anxiety and the pituitary gland are connected through the following processes:

When the amygdala perceives a threat, it sends signals to the hypothalamus. The hypothalamus activates the pituitary gland. The pituitary gland then activates the adrenal gland. The hypothalamus also releases corticotropin-releasing hormone (CRH), which triggers the release of adrenocorticotrophic hormone (ACTH) from the pituitary gland. ACTH prompts the adrenal glands to release cortisol.

In autism we see:

One of the most significant findings related to hypothalamic function in autism is the **alteration in the hypothalamic-pituitary-adrenal (HPA) axis**. The HPA axis is responsible for the body's stress response and plays a crucial role in emotional regulation.

In ADHD we see:

Attention-Deficit/Hyperactivity Disorder, Its ...

This study aims to examine the co-occurrence rate of attention deficit hyperactivity disorder (ADHD) and adrenal gland disorders, as well as whether pharmacotherapy may affect ADHD patients' risk of developing adrenal gland ...

The pituitary gland is found at the base of the brain and is 'pea-sized'. The pituitary gland is an overall controller of a number of other glands in the body, overseeing the function of these organs through hormones.

Your pituitary gland (also known as hypophysis) is a small, pea-sized gland located at the base of your brain below your [hypothalamus](#). It sits in its own little chamber under your brain known as the sella turcica. It's a part of your [endocrine system](#) and is in charge of making several essential [hormones](#). Your pituitary gland also tells other endocrine system glands to release hormones.

A gland is an organ that makes one or more substances, such as hormones, digestive juices, sweat or tears. Endocrine glands release hormones directly into your bloodstream.

Hormones are chemicals that coordinate different functions in your body by carrying messages through your blood to various organs, skin, [muscles](#) and other tissues. These signals tell your body what to do and when to do it.

Your pituitary gland is divided into two main sections: the [anterior pituitary](#) (front lobe) and the posterior pituitary (back lobe). Your pituitary is connected to your hypothalamus through a stalk of [blood vessels](#) and nerves called the pituitary stalk (also known as infundibulum).

The anterior lobe of your pituitary gland makes and releases the following hormones:

- **[Adrenocorticotrophic hormone \(ACTH or corticotrophin\)](#)**. ACTH plays a role in how your body responds to stress. It stimulates your adrenal glands to produce [cortisol](#) (the “stress hormone”), which has many functions, including regulating metabolism, maintaining [blood pressure](#), regulating [blood glucose \(blood sugar\) levels](#) and reducing [inflammation](#), among others.
- **[Follicle-stimulating hormone \(FSH\)](#)**. If you’re [male](#), FSH stimulates sperm production. If you’re female, FSH stimulates the ovaries to produce estrogen and plays a role in egg development. This is known as a gonadotrophic hormone.
- **Growth hormone (GH)**. In children, growth hormone stimulates growth. In other words, it helps children grow taller. In adults, growth hormone helps maintain healthy muscles and bones and impacts fat distribution. GH also impacts your [metabolism](#) (how your body turns the food you eat into energy).
- **[Luteinizing hormone \(LH\)](#)**. LH stimulates ovulation if you have ovaries and testosterone production if you have testicles. LH is also known as a gonadotrophic hormone because of the role it plays in controlling the function of the ovaries and testes, known as the gonads.
- **[Prolactin](#)**. Prolactin stimulates breast milk production ([lactation](#)) after giving birth. It can affect fertility and sexual functions in adults.
- **Thyroid-stimulating hormone (TSH)**. TSH stimulates your thyroid to produce thyroid hormones that manage your metabolism, energy levels and your [nervous system](#).

The posterior lobe of your pituitary gland stores and releases the following hormones, but your hypothalamus makes them:

- **Antidiuretic hormone (ADH, or vasopressin)**. This hormone regulates the water balance and sodium levels in your body.
- **[Oxytocin](#)**. Your hypothalamus makes oxytocin, and your pituitary gland stores and releases it. Oxytocin helps [labor](#) to progress during childbirth by sending signals to the

uterus to contract. It also causes breast milk to flow and influences the bonding between parent and baby. Oxytocin also plays a role in moving sperm.

all children going through abuse and neglect or trauma under the age of 12 (as aged 12 is when the C/C is full developed for life.) Will copy ADHD and Autism behavioural traits. The younger the child and the earlier the stage of the development of the C.C will then affect the levels of traits in their behaviour.

in boys and girls that have gone through abuse is quite simple... hormones... neurons require **blood** to function properly. [When a brain region becomes active, blood flow increases to supply local neurons with oxygen and glucose for energy¹. Additionally, neurons are energy-demanding units, and the circulatory system provides the necessary blood to fuel their activity². Glial cells, which support neurons, also play a role in maintaining the environment around them³.](#) So, blood supply is crucial for neuronal health and function!

Hormones need blood which carries to the neurons ... enable activity

The CNS is crucial to the operation of the body, and any compromise in the brain and spinal cord can lead to severe difficulties. The CNS has a **privileged blood supply**, as suggested by the blood-brain barrier.

[The **cerebrum** is the primary part of the brain responsible for cognitive functions. It is divided into two hemispheres, each controlling the opposite side of the body, and is further divided into four lobes: frontal, temporal, parietal, and occipital. Each lobe has distinct functions, such as decision-making, problem-solving, and sensory processing. The **frontal lobe** is particularly important for reasoning, judgment, planning, and impulse control. The **temporal lobe** houses the auditory cortex and memory centers, aiding in hearing and language comprehension. The **parietal lobe** processes sensory input, while the **occipital lobe** is dedicated to vision. These lobes work together to form the command center for thought, creativity, and behavior](#)

Growth hormones playing a big part at of the two major pruning we will be looking at

nervous system pruning (before ten)

Brain pruning (puberty)

We need to be looking close at the blood supply and growth hormones.

Darwin's work being the main focus point in our stress response. The link between growth hormones and developmental stages in childhood trauma (his work being in the orchard which we changed over to the female cannabis plant)

Structural maturation of neural pathways in children and adolescents: in vivo study.

[Paus T1](#), [Zijdenbos A](#), [Worsley K](#), [Collins DL](#), [Blumenthal J](#), [Giedd JN](#), [Rapoport JL](#), [Evans AC](#).

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it is causing these adhd and autism pathways. what i found somewhat interesting is in john Bowlby work on attachment... he sees the behavioral traits and does not link them to the adhd, etc but has elements of inwards and outwards behavior because they weren't class as disorders in his time frame....and does not

acknowledge the link between the **child's instinctive behavior**...and the enhancement of the **problem solving** capabilities..

main ingredients to a healthy fully developed corpus collasum is **hormones**... it feeds off it to hit its development stages. i mean its common sense the hormones are going to be there... as its basic knowledge that.

brain cells **will** die if the **supply** of **blood** which carries oxygen **is** stopped, the **brain** has top priority for the **blood**. Even if other organs **need blood**, the body attempts to **supply** the **brain** with a constant **flow** of **blood**. The **blood** brings many materials necessary for the **brain** to function properly

To develop the **corpus collsaum** needs the supply of **blood** and **oxygen** it carries, which also contains the hormones. It would not be able to reach peak developmental targets without it. The only reason it has never been properly proven in humans and has with animals, is due to the lack of opportunities to really study the area without breaking fundamental morals of our society. The cognitive circuit board needs the hormones for the responses and pathways it creates and has jet to create.

The blood carries the hormones to the corpus collausm, for it to develop, it needs the hormones for the ability to perform a natural emotional response pathway.

Nerves are incredibly blood thirsty and consume 20% of the bodies' entire **oxygen supply** even though they comprise only 2% of the body's weight. **Nerves need** a continuous **supply** of **blood** and begin to lose function rather quickly with **oxygen** deprivation

As well as the cornea, other areas of the **body** that don't have **blood vessels** include hair, nails, tooth enamel and the outer skin layers. Blood vessels **feed** the corpus callosum through every stage of development, traveling in the blood during a stress reaction are the stress hormones.

The **corpus callosum** gets its **blood** supply from a branch of the anterior communicating artery (median **callosal** or subcallosal artery), from the pericallosal artery (distal part of the anterior cerebral artery) and from the posterior pericallosal artery (usually a branch of the posterior cerebral artery

Base behavioral pattern=

An **emotional response** has a behavioral component, an autonomic component, and a hormonal component. The hormonal component includes the release of adrenaline.

The behavioral **component** consists of muscular movements that are appropriate to the situation that elicits them. **Autonomic responses** facilitate the behaviors and provide quick mobilization of energy for vigorous movement

(see summary for breakdown of a cognitive response behavioral pattern)

Sensory nervous system

The sensory system is the portion of the nervous system responsible for **processing input from the environment**.

sensory nervous system is a part of the **nervous system** responsible for processing **sensory** information. A **sensory system** consists of **sensory neurons** (including the sensory receptor cells), **neural pathways**, and parts of the **brain** involved in **sensory perception** and **interoception**. Commonly recognized sensory systems are those for **vision, hearing, touch, taste, smell, balance** and visceral sensation. Sense organs are **transducers** that convert data from the outer physical world to the realm of the mind where people interpret the information, creating their **perception** of the world around them. We are seeing dual adhd and autism being created.

Which one has to look at the high levels of dyslexia being created within the development. High levels of creativity and problem solving being created.

ADHD (outward) and Autism (inwards); mimicking of behaviour pathways.

So in boys we see a smaller reduction in the c/c and boys following ADHD coped behavioral traits (**outwards**)... this is down to the male hormones and adrenaline hormone ... the boys become more aggressive in behavior...releasing an outlet for

the damage the trauma has done... and with testosterone levels and aggression and adrenaline all being linked in

In girls we see them copying the autism behavioral traits (**inwards**) more... and the reduction more in size.... this is due to the female hormones

the hormones acting as a **divider**...putting the boys and females in different behavioral groups... boys being more dominantly ADHD and girls more dominantly autism.

why does the pathways not follow the classic ADHD and autism ...? and the production stages of the c/c.... why does it mimic only but have same reduction.... cause it's a different social interaction and environment abuse and neglect kids have than a child with classic ADHD and autism... the hormones when released are causing a different affect as the child walks on egg shells... fear produces enhance levels of adrenaline in the development stages of the c/c... the hormones are being released differently but in almost exact pathways... as the 200 million fibers connecting to form the c/c and the white matt decreases due to increase in adrenaline hormones

One must remember the growth hormones having two areas of peak, at 2 years of age and then puberty. These peaks allow our brain to develop.

The hippocampus undergoes significant development from the third trimester of pregnancy through early childhood, with rapid growth occurring in the first year of life and continuing into adolescence.

Developmental Stages

Fetal Development: The hippocampus begins to develop in the third trimester (around 27-38 weeks gestational age), where it starts to

resemble the adult structure. This period is crucial for establishing the foundational architecture of the hippocampus.

2

Infancy: After birth, the hippocampus continues to grow rapidly. Studies indicate that hippocampal volumes increase exponentially during the first year of life, supporting cognitive and affective functions such as memory and spatial processing.

1

Early Childhood: The growth rate of the hippocampus begins to slow around the age of 2 years, but it continues to develop structurally and functionally throughout early childhood. This period is essential for the development of episodic memory and other cognitive skills.

2

Adolescence: The hippocampus undergoes further changes during adolescence, with ongoing maturation that influences cognitive functions. Research suggests that the integration of different hippocampal subfields continues to evolve, impacting memory and learning processes.

The **hippocampus** is a crucial part of the brain involved in several key functions:

It plays a significant role in **forming new memories** and recalling past experiences.

1

The hippocampus is essential for **learning**, helping to process both short- and long-term memories.

1

It is involved in spatial awareness and navigation, aiding in understanding and mapping environments.

2

Additionally, the hippocampus contributes to emotional processing, influencing how we respond to emotions and social connections.

2

Overall, the hippocampus is vital for memory, learning, and emotional regulation.

With CBT new Neron in the hippocampus would be able to develop till late adulthood, as proven in latest science work.

Growth hormones play a significant role in the hippocampus, influencing various cognitive functions. They are locally produced and help maintain cellular populations, regulating neuroplasticity necessary for normal cognitive performance. Growth hormone also alters synaptic plasticity, which is crucial for learning and memory. Additionally, it is produced within the hippocampal formation, contributing to emotional experiences and cognitive functions. Overall, growth hormone's effects on the hippocampus are essential for normal brain function and cognitive health

The origin of a fantastic variety of living things could be explained by the contribution which novel features, possible of random provenance, made to survival.

The primary cells in the hormones and supporting cells are following the pathways of basic evolution.... changing to their interaction with the stress hormones response within the trauma. As the child hits the developmental stages of the foaming of the corpus callosum.

I'm getting a different result in classic ADHD, autism through the sensory system is creating a weighting scale. On one side we have adhd and the other autism. The female and male hormones then tipping in either ADHD (male) outwards behaviour and then Autism (female) inwards behaviour.

when I've stripped it down the male hormone is the one linking in with the ADHD behavior traits...running, impulsivity... in girls they aren't got it... they are displaying autism behavior traits in ADHD...that's why boys are 3 times more likely to get classic ADHD (according to the center for disease control) girls behavior traits in classic ADHD are autism behavior traits... ADHD is produced by the over production of growth hormone and hormones... its linked in with the set of behavior that the male and female hormone releases aggression, running, impulse in boys (**outwards**) and being withdrawn, anxiety and verbal aggression in girls (**inwards**)...the gender difference in ADHD is huge in terms of behavior... two totally different sets of behavior...

HGH (**growth hormones**), produced by the pituitary gland, spurs growth in children and adolescents. It also helps to regulate body composition, body fluids, muscle and bone growth, sugar and fat [metabolism](#), and possibly [heart](#) function.

High dose GH treatment before **puberty** accelerates bone age and induces an **earlier** onset of **puberty**; **what would happen if the stress response kicked off a enhance response from the growth hormone's one would then ask?**

Puberty:

Pre puberty: the growth hormones and hormones increasing to get ready for the big developmental stage of puberty. In girls.

Research is showing that the symptoms of **ADHD** often intensify for girls during **puberty** when estrogen increases in their bodies. ... These hormonal changes may cause mood and behavioral difficulties for girls with **ADHD** especially

Estrogen regulation of **growth hormone** action. ... GH plays a pivotal role in regulating body **growth** and development, which is modulated by sex steroids. A close interplay between **estrogen** and GH leads to attainment of gender-specific body composition during puberty.

In boys;

Changing testosterone levels during **puberty** is associated with greater risk-taking behavior among **boys**. Testosterone also interacts in complex ways with dopamine and other hormones that are relevant to **ADHD**, **Testosterone** increases levels of **growth hormone**.

somehow the girls and boys who have been abused or neglect and are presenting the behavioral traits of the three gateway disorders and d and d knitting together... somehow, they vee ended up being classed as ADHD, that's due to link in c/c and white matter decrease.

Since girls with ADHD usually don't display "typical" ADHD behavior, the symptoms may not be as obvious as they are in boys. The symptoms include:

- being withdrawn
- low self-esteem
- anxiety
- intellectual impairment
- difficulty with academic achievement
- inattentiveness or a tendency to "daydream"
- trouble focusing
- appearing not to listen
- verbal aggression, such as teasing, taunting, or name-calling

this is the gateway disorders... and as the girl walks on eggshells the Adaline levels will raise and create a mincing of false ADHD... from fear... these kids are getting put in wrong behavior groups.

Sugar/energy creating the same adrenaline release as false ADHD combined with poor parenting will produce false ADHD

ADHD;

in fact, being over production of female or male hormones and growth hormones if left alone will even out itself. (due to growth spurs in developmental terms)

Many more boys than girls are diagnosed on the [autism spectrum](#): more than four boys for every autistic girl, according to the latest numbers from the Centers for Disease Control. Researchers point to genetic differences. But clinicians and researchers have also come to realize that many “higher functioning” autistic girls are simply missed.

Female ADHD being produced from the stress response from the female hormone's reacting with the growth hormone's producing mimicking of autism behavioral traits in girls.

Male ADHD being produced from the stress response from the male hormones reacting with the growth hormones producing the mimicking of (what was classic) ADHD behavioral traits.

its coping the bipolar pathways too... bipolar elements are made up of classic ADHD take both the girls' and boys' traits of classic ADHD and you got bipolar... it's the 3 gateway disorders mixing with the d n d.... before the corpus callosum hits the completion stage in development at 12...it is copying the bipolar pathways too. any trauma in development... kicks off a reaction... in behavior... the three gateway disorders are causing mayhem in the development of the c/c

the reduction in the corpus callousm and decrease in white matt... then cause a unique behavioral pool. Putting them in a family behavioral pool... that's why ADHD, autism and bipolar share the same family pool and now kids who have been suffered trauma are in same pool... the reaction to the trauma creates the reduction and decrease in white mas though the stress hormones reactions to its social interaction and environment it connects to within the c/c... in its development stages... creating a family pool linking them all together...rather interesting pool.

Developmental stages of the Corpus Callosum;

Ref; Megumi M. Tanaka-Arakawa, Mie Matsui, [...], and Kyo Noguchi

Developmental Changes in the Corpus Callosum from Infancy to Early Adulthood: A Structural Magnetic Resonance Imaging Study

We are seeing men ADHD with childhood trauma have 3 in 1 chance of developing schizophrenia in teens. As they are more outwards in behaviour and impulsive. We also see more women peaking in teens bipolar and depression due to the inward living inside their own heads. The growth hormones changing the behaviour to men outwards, women in our own head so to speak. One must remember that age and hormones decline can open or close a pathway for disorder.

Histological and MRI studies have verified that myelination of the corpus callosum begins at around 4 months of age and continues throughout adolescence [14]. Studies of children and adults suggest that myelination processes might affect the size of the corpus callosum.

The corpus callosum was segmented into seven subareas of the rostrum, genu, rostral body, anterior midbody, posterior midbody, isthmus and splenium

The structure of the corpus callosum is eventually formed at approximately 20 weeks in the womb.

gonadal hormones have been found in corpus callosum development, everything needs hormones to advance, growth hormones enable the body to reach the targeted growth peaks.

The **corpus callosum** changes structurally **throughout** life, but most dramatically **during** childhood and **adolescence**. ... Alternating phases of **callosal** growth and shrinkage may reflect a permanent adjustment and fine-tuning of fibers connecting homologous cortical areas **during** childhood and **adolescence**.

Worked used see above ref work:

So, what was very interesting.

Table 1. Frequency, height and weight of Participants; Development of Corpus Callosum from Infants to Adults; doi:10.1371/journal.pone.0118760.t00;

the increase on the table, we want to be looking at.

boys the leap we are looking at on the table is 8/10, 10/12 and 12/14

Females the leap we are looking at on the table is 10/12 and 12 /14

Time frames the **callous growth** and **Pruning**; younger children showed the most pronounced growth in the anterior CC, while the splenium began to overtake the anterior parts of the CC starting from the age of 9-10 years in girls and of 11-12 years in boys.

the jump due to the enhanced developmental period of puberty; the c/c following the body's natural developmental stages. As the male and female growth hormones and hormone's peak to reach their developmental goals set by nature.

Myelinated **neurons** are typically **found** in the peripheral nerves (sensory and motor **neurons**) The **neuron** is the basic working unit of the **brain**, a specialized cell designed to transmit information to other **nerve cells**, muscle, or gland cells. **Neurons** are cells within the nervous system that transmit information to other **nerve cells**, muscle, or gland cells. Most **neurons** have a cell body, an axon, and dendrites.

*myelination changes in response to **chronic stress** can be seen in research work with rats and mice. The stress hormones must be present for the change to occur.*

Puberty occurs when a part **of** the brain called the hypothalamus begins to produce a **hormone** (gonadotropin) that influences the testes and ovaries causing an increase sex **hormone** — **estrogen** in girls (inwards) and testosterone in boys (outwards)

Pruning means that the abundance of [neural](#) connections achieved during the sponge-like soaking in of knowledge during the childhood period will be whittled down, shaped like a garden. What was surprising to many was that such a pruning process would be so robust, a process that can be [intensified with stress](#).

And it is this pruning process that may explain the finding that most of the major [psychiatric](#) disorders—of thought, mood, and [anxiety](#)—have their major onset during this vulnerable period.

For adolescents, this means that the **pruning** down of existing neurons and the laying down of *myelin* sheaths connecting the remaining linked neurons will continue years after we stop referring to them as “teenagers”.

Callosal growth; Increases at the isthmus in boys (although less pronounced) became apparent at age 11–12 years. In both boys and girls, parts of the **callosal** isthmus continue to grow until the age of 15–16 years before the whole isthmus starts decreasing at age 17–18 years

What we are seeing on the pre puberty is the present of the growth hormones that help spur the enhancement growth stage. Boys being earlier than girls, which is why we see a natural size reduction. The presents of dyslexia and facial expression processing deficits in cognitive pathways. Would indicter this growth spur is due to the c/c building the foundations, so to speak. To allow the child to move from the **amygdala** to the prefrontal cortex at a later stage of development.

The enzyme **aromatase** can be found in many tissues including gonads (granulosa cells), brain, adipose tissue, placenta, blood vessels, skin, and bone, as well as in tissue of endometriosis, uterine fibroids, breast cancer, and endometrial cancer. It is an important factor in sexual development

Seeing the peak areas of development being the **critical period** of attachment and **pre puberty**. In pre puberty the girl and boy hormones react with the growth hormones to reach their targets. The pre puberty start of the increase being matched with the start of natural behavioral changes within the boy or girl child at the time. Boys being more boyish **outwards** in behavior and running about, girls being more **inwards** and emotional based.

Teen **hormones affect** teenagers' moods, **emotions**, and impulses as well as their body. The **mood** swings that teens experience are **caused** by fluctuations in **estrogen**, progesterone, and testosterone—the sex **hormones**

Classic ADHD normally only targeting the **pre puberty** and **puberty** and then disappearing in most cases. Which would be an indicator it was a reaction to the hormone's and growth hormone's levels increasing due to the developmental stage.

data suggest that estrogens, and aromatase, are not only present, but also actively regulated during key phases of the *developing cerebral cortex*

Aromatase: An enzyme involved in the production of estrogen that acts by catalyzing the conversion of **testosterone** (an androgen) to **estradiol** (an estrogen). The hormones are active in the development of the c/c as previously thought. The **corpus callosum** is a band of nerve fibers that divides the **cerebral cortex** lobes into left and right hemispheres, *connecting* the sides of the brain.

The limbic system is composed of numerous structures, including the amygdala, thalamus, hypothalamus, hippocampus, **corpus callosum** (callus), and several other brain segments. Aromatase having been stated as in being found in more of the areas of the system and neurons that connect the areas together. Hormones have been detected within this system; that connects; hormones must be present

Data collected by researchers have found hormones in corpus callosum of rats and monkeys. gonadal hormones have also been found in human c/c. We are still learning how hormones are affecting the c/c growth peaks.

Relationship Between Corpus

Callosum Abnormalities and Schneiderian First Rank Symptoms in Antipsychotic-Naïve

Schizophrenia Patients Naren P. Rao, MBBS, M.D. Ganesan Venkatasubramanian, MBBS, M.D. Rashmi Arasappa, MBBS, D.P.M. Bangalore N. Gangadhar, MBBS, M.D. The corpus callosum (CC),

has been hypothesized to be implicated in the pathogenesis of schizophrenia; however, the findings from magnetic resonance imaging studies are conflicting. Moreover, the relationship between first-rank-symptoms (FRS) and CC abnormalities in schizophrenia is yet to be examined. The authors examined CC morphometry, based on Witelson's method, in antipsychotic-naïve schizophrenia patients in comparison with matched healthy-control subjects. Patients had significantly smaller CC, splenium, and isthmus areas than control subjects. A novel finding of the study is that only those without FRS differed from control subjects, but not those with FRS. Study findings support a neurodevelopmental hypothesis and possible connectivity abnormalities in

symptom-genesis. (The Journal of Neuropsychiatry and Clinical Neurosciences 2011; 23:155–162)

we cross exam behavior traits of agenesis of the c/c... against autism and we get same behavior in different strength... both caused by same thing which is;

It occurs when the development of the *corpus callosum*, the band of white matter connecting the two hemispheres in the brain, in the embryo is disrupted

any deficits to the process... **will result in a stress response and the stress hormones will create damage to the neurons.** leading to similar but different watered-down behaviors... that we are seeing. whether its deficits that occur in the womb before birth or during the enhancement periods of development the corpus callosum .

The creation on womb is a huge development target... The formation of the corpus callosum begins with the first midline crossing of pioneer axons around week 12 in the [prenatal development](#) of the human **In the womb by week 8 we see the;**

The neural tube (brain, spinal cord and other neural tissue of the central nervous system) is well formed. The digestive tract and sensory organs begin to develop.

By week 26;

We are seeing a behavioral reaction to things; Baby responds to sounds by moving or increasing the pulse

Which indicates the cognitive circuit board is beginning to start to form and attachment is **preprogrammed unconditionally** before birth as Bowlby stated in his work.

(Cell division stress response occurring due to the C/C being in the growth stage and working towards completion.)

However; in primary **gender differences** in the c/c, advanced analytical techniques of computational neuroanatomy developed in the 1990s showed that sex differences were clear but confined to certain parts of the corpus callosum, and that they correlated with cognitive performance in certain tests

The adrenal medulla secretes the **hormone** adrenaline. This **hormone** gets the body ready for a fight or flight **response**. Physiological **reaction** includes increased heart rate. Adrenaline leads to the arousal of the sympathetic nervous system and reduced activity in the parasympathetic nervous system...the reason the behavior is different is the DNA make up of chromosomes in male and female. and their responses to the stress reaction hormones... There is a **difference** in the **stress response** exhibited by men and **women**. It is characterized by 'fight-or-flight' in men and 'tend-and-befriend' in **women**. ... [61] The observed limbic activation to **stress** in **female** subjects is more consistent with a 'tend-and-befriend' rather than a 'fight-or-flight' model... and cause A **hormone** is a chemical that is **made** by specialist **cells**...

The **endocrine system** is a complex network of glands and organs. It uses hormones to control and coordinate your body's metabolism, energy level, reproduction, growth and development, and response to injury, stress, and mood;

Adrenal gland. An adrenal gland is located on top of each kidney. Like many glands, the adrenal glands work together with the hypothalamus and pituitary gland. The adrenal glands make and release corticosteroid hormones and epinephrine that maintain blood pressure and regulate metabolism. Adrenal glands produce **hormones** that help regulate your metabolism, immune system, blood pressure, response to stress and other essential functions.

Norepinephrine is a naturally occurring chemical in the body that acts as both a stress hormone and **neurotransmitter** (a substance that sends signals between nerve cells). It's released into the blood as a stress hormone when the brain perceives that a stressful event has occurred.

The **hypothalamus** controls body temperature, hunger, important aspects of parenting and attachment behaviours, thirst, fatigue, sleep, and circadian rhythms.

The **pituitary gland** is a part of your endocrine system. Its main function is to secrete **hormones** into your bloodstream.

All reaction to the stress response cause by the release of the stress hormones.

Myelination is the process of coating neurons with myelin, which helps the transfer of information between neurons. The process is believed to occur until an individual's thirties with peak growth in the first decade of one's life. **Thinner, lightly myelinated fibers are slower conducting and they connect the association and prefrontal areas. Thicker and fast-conducting fibers connect the visual and motor areas.**

Myelin enables nerve cells to transmit information faster and allows for more complex **brain** processes. The **myelination** process is vitally **important** to healthy central nervous system functioning.

Myelin is **made** by two different types of support cells. In the central nervous system (CNS) — the brain and spinal cord — cells called **oligodendrocytes** wrap their branchlike extensions around axons to create a **myelin** sheath. In the nerves outside of the spinal cord, **Schwann** cells produce **myelin**

The **central nervous system** (CNS) controls most functions of the body and mind. It consists of two parts: the brain and the spinal cord. The brain is the center of our thoughts, the interpreter of our external environment, and the origin of control over body movement.

myelin is the white matter... i mean its basic science... The **white color** is from the myelin sheaths...were the reaction to the stress responses and causes an under production of the white matter, which then leads on to the under coating of the myelin. Then leading to a reduction in the c/c. Which can clearly be seen as the damaged neurons create the reduction after long period of time die stress response.

Vagus nerve longest in the body. Made me wonder what was going on in the sensory nervous system to cause such a reaction to the behaviour in childhood trauma.

The myelin sheaths response;

The **electrical impulses** (nerve impulses) during the stress response and the release of the stress hormones;... seeks to find a solution and under coats the fibers in white matter **myelin** its response to the reaction. =damaged and weak neurons...

A nerve **impulse** is the way nerve cells (**neurons**) communicate with one another. Nerve **impulses are** mostly **electrical signals** along the dendrites to produce a nerve **impulse** or action potential

Myelination also occurs in the peripheral nervous system;

Peripheral Nervous System. The **peripheral nervous system** is divided into **two** major **parts**: the somatic **nervous system** and the autonomic **nervous system**;

The somatic system is the part of the peripheral nervous system that is responsible for carrying motor and **sensory** information both to and from the central nervous system (CNS). This system is made up of nerves that connect to the skin, **sensory** organs, and all skeletal **muscles**.

The autonomic nervous system is a control system that acts largely unconsciously and regulates bodily functions such as the heart **rate**, **digestion**, respiratory **rate**, pupillary

response, urination, and sexual arousal. This system is the primary mechanism in control of the fight-or-flight response

Damaged neurons due to stress response. Resulting in the reduction of white matter and reduction. Creating the behavior that will soon become a cognitive pathway. Hundreds of cognitive pathways foaming, as we learn to do simple tasks and interact with the world around us.

any reaction to the **myelination** process being disruption and the primary cells that make up the hormones subtracting to the reaction and multiplying to fix it... after birth... any abuse, trauma again creates the stress reaction in the neurons in the hormones, stress hormones Creating the reduction in the c/c as same in the womb....and the result is an reduction **of white matter (myelin)** which then affects the processing of information through the fibers. As the child engages with the world around them and absorbs the learning that it provides.

creating different affects due to **different growth hormones** and hormones make up in male and female DNA. That's why we see different behavior and different sizes in the c/c and reduction sizes and white matter.

in trauma it affects the c/c development process if the child is under 12 and the c/c is going through its development... which then creates the 3 gateway disorders mixed in with d n d ... otherwise known as bipolar. and if left untreated will develop on to schizophrenia during a manic. (decrease seen in grey matter in bipolar.) defaults also seen in the grey matter in schizophrenia.)

So; then the stress hormones react with the brain transmitters... we have 7 major neurotransmitters.... the one we want to be looking at it the Glutamate is the primary excitatory **transmitter** in the central nervous system.... **Neurotransmitters** are stored in synaptic vesicles, clustered close to the cell membrane at the axon terminal of the presynaptic neuron. **Neurotransmitters** are released into and diffuse across the synaptic

cleft, where they bind to specific receptors on the membrane of the postsynaptic neuron.... There are both small and large **vesicles**. Small **vesicles** are produced by the Golgi apparatus **located** in the soma and transported down the axon via current flow in the cytoplasm (fluid inside the cell). Small **vesicles** store neurotransmitters. In some **neurons**, larger **vesicles** are also **found** in smaller quantity.

Glutamate;

is a small, amino acid **neurotransmitter**, and is the **primary excitatory neurotransmitter** at almost all synapses in the central nervous system. This molecule binds multiple postsynaptic receptors including the NMDA receptor, AMPA receptor, and kainate receptors.

It is activated when **glutamate** and glycine (or D-serine) bind to it, and when activated it allows positively charged ions to flow through the cell membrane. The NMDA receptor is very important for **controlling speed** synaptic plasticity and **memory function**

The α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptor (also known as AMPA receptor, AMPAR, or quisqualate receptor) is an ionotropic transmembrane receptor for **glutamate** that mediates **fast (the speed)** synaptic transmission in the central **nervous system** (CNS).

Kainate receptors, or **kainic acid receptors** (KARs), are ionotropic **receptors** that respond to the neurotransmitter glutamate. ... Presynaptic **kainate receptors** have been implicated in inhibitory neurotransmission by modulating release of the inhibitory neurotransmitter GABA through a presynaptic mechanism

One of the larger connections and roles that kainate receptors have been shown to have is to several neurological diseases and conditions. KAR expression and distribution has shown a linkage to [schizophrenia](#), [depression](#), [autism](#), [Huntington's](#), [bipolar disorder](#), and epilepsy among others

as you can see the reaction the stress hormones cause to deficit then reacts to the brain transmitters and onto the fibers which every neurons when confronted by a deficit to its process will lead to a weak or damaged neurons through stress response to the trauma

whilst in the growth stage.... causing over production of each cell ... resulting in reduction of c/c and decrease in white matter and effecting behavior and personality development in the prefrontal cortex during punning in puberty

Neonatal Abstience Syndrome...As the embryo develops in the womb and the corpus callusom starts to form and enter its development stages... any trauma or foreign bodies in the development stage. will react the stress response of the hormone cells to react and the response to occur; subtract **(react)** and multiple **(repair/change to the new environment)**. Neround weak or damage and replacement ..Again, in this we see a reduction of the c/c and increased white matt from children who are affected by this syndrome. What is somewhat interesting is it then joins the re formatted behavior pool. as behavior regarding cognitive, social and behavior traits... just again different strengths of it. so, to speak. Purning in 2 years will again change the structure

Cell division in plants;

So rather interesting; Darwin's work in this area with stress response but his choice of plants, wasn't correct. All I did was move his work over to cannabis plants. Put the plant into stress state and look for cell division to occur; as the plants were young below 6 weeks... So; what was interesting is the cell division in the plant during stress, would then lead the plants to find solution to making its growth stages and in fact enhancing its crop ability and size. Therefore; people who grow cannabis put the plant in temporary shock during its young growth stage to encourage it to produce more yield, so to speak.

Darwin in his search of a flower to prove this cell division occurred in stress, orchids being his main love. Darwin did not take in to account the flowers being limited in what they were able to achieve. I changed his work over to cannabis, as it is not a flower but a weed-based plant; cell division from stress response could then clearly be seen; as the plants looked for a solution to achieve life and its growth targets.

By definition, a weed is known as a **plant in the wrong place** and has the built-in ability to define nature at any given chance.

Finishing Darwin's work in this area;

darwin had proved that a stress response in plants and growth hormones were connected to cell division that would then lead to cell mutation, he could not locate the source and cause.

Phytoestrogens are **estrogens** that occur in some **plants**. Foods containing phytoestrogens include beans, soy products, peas, lentils, and whole grains and seeds, especially flaxseed, rye and millet. Lignans, isoflavones and coumestans have been the most carefully studied

What we see with cannabis plants is when the female plant is in shock at a young age... when growth hormones are added to the plant... the plant comes back to life and finds a way to meet its development targets

le female plant goes into stress... **clonex** (hormone product in high levels will bring the plant back to life and enable it to reach its targets and produce more bud. So to speak. Here we can clearly see that the **estrogen + growth hormones** having a clear effect for us to see. **Cell division** is clearly occurring.

Cell division is not limited to creation as one would think.

shock to the process= stress hormones to be released (causing a reaction) and damage to the the primary neurons in the hormones react with the growth hormones.

and that's why in dissociation in behavior in girls we see inwards behavior than in boys... we see more extreme adverse effects; semi or fully locked in; in girls...

because inward behavior is produced as the c/c nervous during the myelination process go into shock due to stress or trauma and release the stress hormones. the girl's neurons in the hormones subtract and multiple to fix the fault and have a knock-on effect to the neurons, transmitters and reduces the corpus callosum and increases the **under producing** of the white matter and damaged neurons.. Then pruning removing the damage nerouns creating a reduction over time

In trauma through abuse and neglect.

Girls;

Inward behavior= female growth hormone/ hormone's being over produced. (girls) mild autism traits. plus, reaction to adrenaline hormones.

Boys;

Outward behavior= male growth hormones/hormones being over produced (boys) ADHD. plus, reaction to adrenaline hormone's

When the hormone imbalance is created in the c/c development both inwards and outwards behavior starts to be displayed. (before age of 12)

Any genetic **fault** in chromosomes leading to a stress response.

The human genome is the set of genetic information encoded in 46 chromosomes found in the nucleus of each cell. The chromosomes are organized into 23 pairs — one chromosome of each pair is inherited from the mother and one from the father. One pair of chromosomes — X and Y — determine sex; the other 22 pairs are called autosomes.

Estrogen;

the adrenal gland releases the stress hormone **adrenaline** and **Cortisol** ... the growth hormones and cells will subtract and multiple to fix the fault and [Estrogen](#) is then amplified to the stress response. which then has a knock-on effect in the prefrontal cortex. Causing an decrease in white matter.... white matter abnormality predominated in the anterior corpus callosum

Estrogen receptors located on the cell membrane can interact with a variety of cellular processes to induce rapid neuronal and behavioral change.

Estrogens can regulate behavior through rapid nongenomic mechanisms or through slower genomic mechanisms

Environmental cues such as photoperiod can modulate whether estrogens activate nongenomic or genomic pathways

This flexibility allows a single hormone molecule, such as estradiol, to have diverse effects on a range of social behaviors

the presence of estrogen-like compounds (both naturally occurring and man-made) in diet can have a dramatic impact on the behavioral effects of estrogen signaling

Stress **Hormones** (Adrenaline, Cortisol)

Stress **hormones** are released in situations where you feel out of control, overwhelmed, or severe **anxiety** (such as during the fight-or-flight response), which can increase your social **anxiety**

The three sub-regions – the dentate gyrus, the cornu ammonis and the subiculum – are all known to be vulnerable to the effects of **stress hormones**, which probably interfere with the formation of cells and new tissue as the immature brain develops. All areas of the brain and supporting cells responding to the reaction cause by the stress hormones

Read more: <https://www.newscientist.com/article/dn21469-child-abuse-shrinks-keyhttps://www.newscientist.com/article/dn21469-child-abuse-shrinks-key-brain-memory-centre/brain-memory-centre/#ixzz6EpEOojpC>

The prefrontal cortex is a part of the brain located at the front of the frontal lobe. It is implicated in a variety of complex behaviors, including planning, and greatly contributes to personality development.

The brain's **prefrontal cortex** is thought to be the **seat of cognitive control**, working as a kind of filter that keeps irrelevant thoughts, perceptions and memories from interfering with a task at hand.

Cortisol calls the body into action to combat stress. **Cortisol** also regulates the HPA axis. When high amounts of **cortisol** interact with the hypothalamus, the HPA axis **will** slow down its activity. The amygdala detects stress in the environment, while the **prefrontal cortex** regulates our reactions to stress.

High levels of **cortisol** can wear down the **brain's** ability to function properly. ... **Stress** can kill **brain** cells and even reduce the size of the **brain**. Chronic **stress** has a shrinking effect on the prefrontal cortex, the area of the **brain** responsible for memory and learning.

Basic research has found that high levels of **catecholamine** release during **stress** rapidly impair the topdown cognitive functions of the **prefrontal cortex** (PFC), while strengthening the emotional and

habitual responses of the amygdala and basal ganglia. ... Patients with PTSD have signs of PFC dysfunction.

The catecholamine; These **hormones** are released in response to physical or emotional stress. **Catecholamines** are **hormones** produced by the adrenal glands, which sit on top of the kidneys. Dopamine, epinephrine (adrenaline), and norepinephrine are the main **catecholamines**

After an emotional response; the **amygdala** sends a distress signal, the hypothalamus activates the sympathetic nervous system by sending signals through the autonomic nerves to the **adrenal glands**. These glands respond by pumping the **hormone epinephrine** (also known as **adrenaline**) into the bloodstream

The **amygdala** is responsible for the perception of emotions such as anger, fear, and sadness, as well as the controlling of aggression. The **amygdala** helps to store memories of events and emotions so that an individual may be able to recognize similar events in the future

The **amygdala** is responsible for processing fear which includes the modulation of attention and memory for fear-related stimuli, fear recognition, the induction of fear-related behaviours, and fear conditioning. The **amygdala** also plays an **important** role within the fight or flight response.

Hippocampus plays a vital role in flexible and goal-directed **behavior**. An intact **hippocampal** activity is required for forming and reconstructing relational memory (required for remembering arbitrary associations between objects or events) associated with flexible cognition and social **behavior**

The **amygdala** is specialized for input and processing of emotion, while the **hippocampus** is essential for declarative or episodic memory. During emotional reactions, these two brain regions interact to translate the emotion into particular outcomes

Hippocampal shrinkage also **damages** cognitive functions and interferes with the process of creating memories, which has a profound impact on both **behavior** and the ability **to** form a stable, realistic, and cohesive sense of self

Childhood maltreatment or abuse is a major risk factor for mood, anxiety, substance abuse, psychotic, and personality disorders, and it is associated with reduced adult **hippocampal volume**, particularly on the left side.

studies have reported that patients diagnosed with posttraumatic stress disorder (PTSD) resulting from traumatic experiences such as military combat, sexual assault, or child abuse have a smaller hippocampus (up to 8% reduction); Taekwan Lee, Tim Jarome, [...], and Fred J. Helmstetter

Norepinephrine is a naturally occurring chemical in the body that acts as both a stress hormone and neurotransmitter (a substance that sends signals between nerve cells). It's released into the blood as a stress hormone when the brain perceives that a stressful event has occurred.

In abuse and neglect;

***males more likely to show more stronger traits of outward behavior**

***girls showing stronger traits of inwards behavior**

The pathways following the copying of bipolar are in fact the start of Reactive Attachment Disorder... the three gateway disorders knitting together with the dissociation and desensitization in extreme child abuse and neglect cases.

and like goes without saying that reactive attachment disorder at the early stages of the disorder presents the mixed behavior of ADHD and autism.

ADHD linked to schizophrenia (7 times more like to develop during childhood trauma response.)

The frontal lobe is the largest part of the brain affected by ADHD. This part of the brain may mature at a slower pace or show disrupted activity and connectivity in people with ADHD

Research suggests that schizophrenia affects brain areas including the **frontal and temporal lobes, white matter, and thalamus regions.**

Childhood trauma has been shown to alter thalamic functional connectivity, particularly in OCD patients. A study found that OCD patients with high levels of childhood trauma exhibited increased thalamic functional connectivity with the prefrontal cortex, while those with low levels showed decreased connectivity. This suggests that childhood trauma may predispose individuals to certain mental health conditions, including OCD. The research highlights the complex relationship between childhood trauma and brain connectivity, emphasizing the need for further investigation into the impact of trauma on thalamic regions

answer

during the Myelination process

shock to the process= stress hormones then released causing an imbalance and the primary neurons, male or female growth hormones, hormone's seek to fix the fault = leading to a reduction in c/c and decrease in white matter foaming through over production and damaged and weakened neurons.

Damage to the neurons **occurring due to the stress response; leading to the reduction in myelin. Myelin being white of color; from the reaction that's occurring at the time in the myelin sheaths and the electrical impulses creating the under coating of the fibers in myelin (white matter)** and then pruning and removal of damaged axonal fibres and creating reduction in size.. In c/c

(although male and female growth hormones would primarily trigger a different size reduction naturally)

Foaming of the reduction in the c/c and the decrease in white matter then leading to the malnutrition of the prefrontal cortex (smaller) neurons in stress response...

Glia cell supporting cell is creating a fault in the cell pattern when the stress occurs in pruning..and enhanced brain development

the higher levels of abuse and longer time frame = the more stress hormones released during the abuse.

the **inherited DNA** from parents will have a **primary** determination on how the child handles the abuse (the child's resistance to the abuse)

One must also consider Neurons cannot achieve cell division. This is why we see the under coating of the white matter... it is the reaction caused by the cell division around it. A default one would say... a reaction to the cells trying to fix the fault. Around it. Resulting in under coating of the fibres in myelin.

Neurons cannot divide because they lack centrioles. Each nerve cell has a specific place in our nervous system.

Chronic stress can lead to the damage of neurons in the brain, particularly in the amygdala, hippocampus, and prefrontal cortex. These areas are crucial for processing emotions, memory, and decision-making. The amygdala, which is involved in emotional regulation, can become hyperactive, leading to heightened anxiety and emotional responses. The hippocampus, responsible for memory formation, may experience reduced neurogenesis and dendritic atrophy, impairing memory formation and retrieval. The prefrontal cortex, which helps regulate emotions and make decisions, can weaken, resulting in emotional control issues and difficulty in decision-making. These changes can have lasting effects on an individual's emotional well-being and ability to cope with stress.

All areas reduced due to the damaged neurons... which over time would cause a reduction in sizes of hippocampus amygdala as pruning removes all the damaged connections and neurons.. same as we see in axonal fibres in C/C during pruning..weakened ones eliminated.

Challenging John Bowlby work in Attachment Theory regarding problem solving abilities; in child abuse and neglect only.

Foaming of the reduction of the amygdala and then Foaming of the reduction in the c/c and the decrease in white matter then leading to the malnutrition of the prefrontal cortex. **Then the malnutrition of the prefrontal cortex then; Smaller**

(otherwise thought to be the seat of the cognitive control) and **frontal lobe and cerebral cortex and reduction in grey matter.**

Reduce volumes in the Hippocampus. DAMAGED NEURONS axonal fibres in c/c weaken like neurons and follow same pathway in pruning. And then supporting glial cell... cell division occurs due to stress response. All working together as the brain goes through extreme enhanced growth during this period

The amygdala's reduction in size due to childhood trauma is a significant consequence of prolonged exposure to stress

Childhood trauma can lead to significant changes in the cerebrum, particularly in the default mode network (DMN) and central executive network (CEN). These regions are crucial for emotional regulation, memory processing, and stress response. Traumatic experiences can disrupt the development of these brain areas, leading to lifelong changes in brain function

Causes the release of stress response hormones , growth hormones during the stress/ shock in the process. (stress response) in the prefrontal cortex... on the left side of the p/c causing an increase in the creativity levels due to inner head. We know dyslexia and creativity are closely linked.

So what we see is reduction in brain areas as the stress response kicks in.. And then in puberty.. Everything speeds up and over production is then achieved.. During the peak of growth hormones in puberty.

Creativity being the foundation required for intelligence.

Everything is reducing in size and volume.. Due to pre long trauma response before puberty..

Ref: Evidence cross examined from latest work available on; Science Daily.com

so Bowlby was right till he hit 2 and a half... the critical period it would have all the effects of attachment.... but then if it was after this period it would then be a different effect... cause the cognitive circuit board would already begin to develop...it would then in a child thats abused become dissociation attachment... and the effects would be different.... the older the child was... and thats why we see creativity levels higher and problem solving..thats kind of cool... so then as the c/c develops... the effects to it change with the age of the child... and thats why we see these kids having good problem solving skills... the foundations are already there.. and thats why we see the deficits in facial expression reading and dyslexia. Cause of the stress hormones and the response

Summary

We define intelligence by;

Ability to reason; a child in danger will reason with they're abusers due to fear of being hurt. And to be safe and have food. They will reason with the parent

Plan: a child who is abused will always plan ways to avoid the abuse and to keep safe or even feed.

Solve problems; A child will always have to solve problems; over and over again the child will try to find a way to stop being mistreated. They will always solve how they will eat, sleep.... what to do if they're parent comes home drunk... how to explain the bruises if any teacher were to ask, how to make the social worker believe everything is ok when your parent is watching.

Learn quickly; any abused child will learn quickly what triggers the abuse to try to avoid it.

Learn from experience; if a child if leathered black and blue every night, that child will learn very fast what not to do to trigger a leathering. They will learn from every leathering he or she may have.

As you can see the highlighted words are the words we need for intelligence and as you can see abused kids will use these skills daily in order to survive the abuse they are enduring; as the fear triggers the stress hormones to be released.

What was Classic ADHD then becoming defined in the following four groups;

***Trauma ADHD:** Created by the stress response. (Decrease In white matter

***Food ADHD:** High sugar diet combined with bad parenting.

***Hormonal ADHD;** cause by over production in puberty of hormone; enhanced by the over production of growth hormones. (Decrease in the whit matt present.)

***Lifestyle:** is the child in a stress response and high levels of anxiety are present, ie parents with anxiety, social media effect on the child's life, the environment, does it contain high levels of anxiety and why

Bipolar in child abuse and neglect then becomes defined as two groups;

Reactive Attachment Disorder type one (+); Outwards behaviour + Dual ADHD and Autism; in **males**.

Reactive Attachment Disorder type two (-); Inwards behaviour + Dual ADHD and autism; in **females**.

Reactive Attachment Disorder now being defined as to be made up of the following; The three gateway disorders knitting together with the dissociation and desensitization in child abuse and neglect cases.

Pathways then opening bipolar in late teens and young adults and manic pathways during early adulthood.

Bipolar disorder is associated with significant structural and functional changes in the brain, including alterations in neurotransmitter levels and specific brain regions that affect mood regulation and cognitive functions.

Structural Changes

Gray Matter Volume: Research indicates that individuals with bipolar disorder often exhibit reduced gray matter volume, particularly in areas responsible for emotion regulation and cognitive functions. A meta-analysis found significant reductions in gray matter in regions such as the prefrontal cortex and the hippocampus, which are crucial for mood and memory.

2

Hippocampal Shrinkage: The hippocampus, essential for memory and emotional processing, may be smaller in those with bipolar disorder.

MRI studies have shown that this region can experience significant volume loss compared to individuals without mood disorders.

1

Frontal Cortex Thinning: Longitudinal studies have revealed that individuals with bipolar disorder may experience accelerated thinning of

the frontal cortex, particularly in those with a higher frequency of manic episodes. This thinning is associated with impaired impulse control and emotional regulation.

The brain changes in psychosis are primarily due to the malfunction of two key brain systems: the "filter" and the "predictor." These systems are responsible for directing attention and predicting rewards, respectively. When these systems are impaired, individuals with psychosis experience difficulties in filtering relevant information and predicting what is likely to happen. This dysfunction leads to symptoms such as hallucinations and delusions, where patients may hear voices or hold false beliefs. The research indicates that these malfunctions are central to the development and progression of schizophrenia, a serious mental illness. Understanding these brain changes can help in developing targeted treatments and prevention strategies for psychosis

The brain's "filter" system is responsible for directing attention to important external events and internal thoughts, while the "predictor" system anticipates rewards. These systems malfunction in psychosis, leading to hallucinations and delusions. The "filter" directs attention, and the "predictor" pathways anticipate rewards. Dysfunction in these systems makes it difficult to know what's real, manifesting as hallucinations and delusions.

The **filter** and **predictor** systems in the brain are crucial for processing information and making predictions.

The filter system is responsible for screening out irrelevant information and focusing on important aspects of the environment, helping individuals distinguish between real and imagined experiences.

2

The predictor system, on the other hand, helps anticipate and make predictions about events based on past experiences, which can be disrupted in individuals with psychosis, leading to difficulties in interpreting and predicting events accurately.

2

These systems are essential for maintaining reality and understanding the world around us, and their dysfunction can contribute to conditions like psychosis

The thalamus plays a crucial role in processing sensory information and sending signals to the brain's fire alarm, the amygdala, when danger is perceived. In the context of childhood trauma, the thalamus's response to danger can be oversensitive, leading to the release of stress hormones like cortisol, which can affect the hippocampus and memory formation. This oversensitivity can result in the brain's survival responses taking over, bypassing the pre-frontal cortex and leading to difficulties in thinking clearly or thinking at all during traumatic experiences.

The thalamus is a large mass of gray matter located on the lateral wall of the third ventricle, forming the dorsal part of the diencephalon. It plays a crucial role in relaying sensory and motor signals to the cerebral cortex, facilitating communication between the brain's sensory and motor systems. The thalamus is connected to the cerebral cortex through nerve fibers known as the thalamocortical radiations, allowing for the exchange of information in all

directions. Additionally, it regulates various functions such as consciousness, sleep, and alertness

Dissociation attachment.

Under 2 and a half;

Attachment before this time frame in the critical period would be Bowlby attachment. And follow all his work.

Over 2 and a half;

As the cognitive board is already established, and attachment is there. Any abuse over this time frame would then be dissociation attachment. What's interesting in dissociation attachment; is we see this new disorder so to speak, in anyone that has suffer a high level of trauma; I.e. ex-service men.

(One must consider ex-service man join at 16 years of age... and PTSD levels being at their highest. Will see a small **decrease**, then **increase** of white matt)

As the c/c is affected during the abuse by the stress hormone's; what we see then which I have to say was somewhat interesting; is the affect this has on how we process information. We then see the mixing up of the processing of facial expression; cause by the deficit of the cognitive pathways. Which then leads me on to dyslexia in child abuse and neglect. Again; another rather interesting reaction to the deficits, unlike classic Dyslexia; this water down concept of it, producing the odd words mixed up inside the words. Ie... water would then look like wetar; but

not all the time maybe once in a sentence, depending to the level time frame of the abuse and the child's resilience to the abuse.

What we see when we look at ex soldiers aged 16 to 26. Is a **over coating** and reduction in c/c... this is down to the late stage they hit the trauma. We also see very light dyslexia pathways and mixing up of facial reading... all depending on how long they served and the age they were and the stage they were at. In brain development.

What we are seeing in ex-soldiers is that as the **myelin process** has already been established... a **increase** occurs instead.

Scientists have shown in both anxious rats and military veterans with PTSD that acute stress is associated with increased myelination of axons in areas of the brain associated with memory and emotions. These areas in the brain's gray matter are normally only lightly myelinated. Since myelin speeds communication in the brain, the increased myelination may be making some neural circuits hyperresponsive to memories of trauma.

(See science daily reports in ref listing on PTSD and anxiety)

Link to advanced puberty and childhood trauma.

Link between pruning stage + drug taking ie crack (adding to the chemical response the brain has already created with the hormones.)= psychosis.

Also; might I add that it is not only abused kids that can develop **Reactive Attachment Disorder**; what I found somewhat interesting when I was studying behaviour in the ex-service men's hostels. These ex-Service men, were displaying all the signs of the gateway disorders, mixed with d and d, just a different affect; due to the different social and environmental connection the different groups had.

Also; in child soldiers we see stronger behavioural traits than one would see in an ex-service man... this is down to the child soldier being at the

under 12-time frame of development; making the traits stronger and service men... (being 18 plus of age) ... having watered down behavioural traits. This is due to the development of the c/c being more advanced and reached completion... and the **Myelination process ends in around our thirties.**

Child soldiers due to their age being under 16= under coating and reduction of the c/c.

Child abuse and neglect then having very similar strengths of behaviour traits of the R.A.D to child soldiers; due to the stress hormone's reaction in the development of the c/c stages.

Autism and trauma.

Rather interesting pathway so this is what happens... so it stills follows the hormones divider... so boys being **outwards** in behavior.... girls **inwards**... so this pathway..... its un notice able... so what happens in the womb... if the mother goes into a stress response... or stress response in the womb through foreign body.. then the stress hormones amplify it in the womb... which then leads the embryo to go into stress... and release the embryos stress hormones... creating what would be Mimicking autism pathways being created... which then cell division tries to fix the fault leading to deficits in the embryo ...

pathway

Glucocorticoids are naturally produced **hormones** and they are also known as **stress hormones** because of their role in the **stress** response. ... The enzyme 11 β -HSD2 is present in the placenta and the developing **fetal** brain where it is thought to act as a shield to protect against the harmful actions of cortisol.

Cross examining work from; Rohan M. Lewis, ... Keith M. Godfrey, in [Reproductive and Developmental Toxicology \(Second Edition\)](#), 2017

The stress response then affecting the enzyme 11b hsd2 in the fetal brain development we see.

Fetal brain 11 β -hydroxysteroid dehydrogenase type 2 selectively determines programming of adult depressive-like behaviors and cognitive function, but not anxiety behaviors in male mice

Caitlin Wyrwoll, Marianne Keith, [...], and Megan C. Holmes

Then we see the maturation of the corpus callosum at development stages, due to cell division response created by the amplifying of the stress hormones response. As every cell fight to reach the Crucial fetal Deveolpment stages that are the natural body clock targets nature creates.

11 β -Hydroxysteroid dehydrogenases and the brain: From zero to hero, a decade of progress

Caitlin S. Wyrwoll, Megan C. Holmes, and Jonathan R. Seckl

So, what we are seeing is the reaction the fetal brain's hormone has with the mother's stress hormones when the corpus callosum is beginning to structure develop. Any stress will result in the process will bring into play the stress hormones that will trigger an over production of the hormones due to cell

division being trigger in the primary cells of the supporting areas. Any form of trauma will trigger a stress response from both the mother and then passed on through the blood to the fetal brain. What is interesting is the fact development continues after birth as the structural build of the corpus collaosum is not jet in completion stage.

Now we have seen in research that the corpus collaousm;

Histological and MRI studies have verified that myelination of the corpus callosum begins at around 4 months of age....which is very interesting...

is we have seen The changes in these *white matter* tracts in the *brain* are visible at *6 months on the autism brain scans*. Ref; y [Anil Ananthaswamy](#) news daily (science)

so stress hormone's in the mother or any foreign body in the process so to speak will lead to the reaction from the stress hormones in the c/c.

But this is like the best bit... the c/c aint like normal things.... after birth it continue its development out of the womb. It has not started the myelination stage... that starts at 4 months.. Now we then look at the jabs baby's have and look at the time frame in developmental terms.; Jabs at eight, twelve and sixteen weeks of **age**.

In the time frame the myelination begins to start; this being the cognitive processing of information to all areas of the brain. Like in fetal development, up until the 4 months start time frame. Any foreign bodies in the process will trigger a stress response of the hormones as they try to reach their natural targets.

Decrease in the production of enzyme 11 β -HSD2 will result in cognitive and behavioral pathway deficits in autism. The corpus callousum needs the hormone to build its gateway to what will be the cognitive circuit. the hormones is present in the gonads hormones already proven in the c/c.

The syndrome of AME is a rare form of juvenile **hypertension** in which **11-HSD** is defective. This **deficiency** allows mineralocorticoid receptors to be occupied by cortisol, leading to **hypertension**, because plasma concentrations of cortisol are much higher than those of aldosterone. ... There are two known isozymes of **11HSD**. Following same pathways but then affecting the myelination process. Adding the hypertension into the behavior.

11 beta HSD type 2 protects the mineralocorticoid receptor from being activated by cortisol. Thus, specificity of this receptor in vivo is enzyme and not receptor mediated. The syndrome of apparent mineralocorticoid excess is caused by a congenital deficiency of 11 beta HSD type 2. Liquorice-induced hypertension is an example of an acquired defect in dehydrogenase activity of 11 beta HSD, caused by glycyrrhetic acid. 11 beta HSD may play a role in the pathogenesis of 'essential' hypertension, obesity and type 1 diabetes mellitus. Angiotensin converting enzyme inhibitors enhance dehydrogenase activity of 11 beta HSD, which may contribute to their natriuretic effect

[11 beta-hydroxysteroid-dehydrogenase: characteristics and the clinical significance of a key enzyme in cortisol metabolism].

[Article in Dutch]

[Kerstens MN](#)¹, [Dullaart RP](#).

Cortisol and epinephrine facilitate the movement of immune cells from the bloodstream and storage organs, such as the spleen, into tissue where they are needed to defend against infection. **Glucocorticoids** do more than help the body respond to **stress**. They also help the body respond to environmental change

Mineralocorticoid receptors (MR) bind both **mineralocorticoids** and glucocorticoids with high affinity (deoxycorticosterone = corticosterone >/= aldosterone = cortisol), and are **found** in both Na(+) transporting epithelia (e.g. kidney, colon) and nonepithelial tissues (e.g. heart, brain)

roids to act at *mineralocorticoid receptors* (MRs) in lung and brain. ... of neuroendocrine *development* of *fetal* brain-pituitary-adrenal axis in late gestatio

We then add in kainate receptors; GluK4 for almost exclusively in the hippocampus ([Darstein et al, 2003](#))

Every part of the stress response reaches every part of the brain structure, as the hormones feed the development stages.

The mother's hormone's having a the dominantly effect on the fetal stress hormone's response.

Cortisol reacting with 11 β -hydroxysteroid dehydrogenase type 2 during trauma or stress response. Causing deficits in the 11 HSD production and then leading to a knock-on effect in all other areas of Development.

[Mune et al. \(1995\)](#) raised the possibility that mild 11-beta-HSD deficiency may predispose to low birth weight and increased risk for developing hypertension.

Medical comorbidities are also commonly seen in autism spectrum disorder including PANS/PANDAS, ADD/ADHD, seizures, dental issues, sleep disturbances and gastrointestinal symptoms. The conditions listed below all exhibit similar behavioral symptoms to autism spectrum disorder.

People with autism spectrum disorder have a higher risk of developing cardiometabolic diseases, including diabetes and heart disease, according to a review of studies published today in JAMA Pediatrics

We are seeing the effect of 11-beta-HSD in the autism pathways... hypertension

Adults with autism are at increased risk for hypertension, cardiovascular disease, and death compared with the general population

What is somewhat interest is the link with low birth rate in autism... which then added with hypertension would, one would say would have to of been an inherited factor and a mimicking trauma factor. In autism.

Pruning and high intelligence.

now another rather interesting pathway... so the child when coming away from the soaking in stage and going into the prune stage... this is very interesting... so the child if they have high IQ... will then become prone to bipolar or schizophrenia. social anxiety... self-harming.. if any stress response... so like say you got a 15 year old... gifted. and they have the manic that being gifted can create in the education sense when working something out.. it can create a mis labeling of bipolar... and if the child is unaware it is happening ... will go into stress... hormones are all over place... so behavior is affected... and mixed up kids... who don't understand why they act and feel the way they do

Amygdala: emotions, memory and social behaviour

Corpus callosum Motor function communication between left and right of brain

Cognitive function: problem solving etc.

Sensory integration..

In all of the three neurons are actively connecting and forming to be able to do their job...

cognitive response behavioral pattern=

An emotional response has a behavioral component, an autonomic component, and a hormonal component. The hormonal component includes the release of adrenaline.

The behavioral component consists of muscular movements that are appropriate to the situation that elicits them. Autonomic responses facilitate the behaviors and provide quick mobilization of energy for vigorous movement

+

absorbing the learning through the social interaction that the world around them provides and development in how they feel, think and what thoughts they have with the world around them.

=

Hormones and behavior go together like salt and pepper. You can't have one without the other.

Sexual abuse pathways.

One would always de layer the behaviour. One would look at the critical time frame (0-5years of age) in the client's life and then the pruning stage (puberty). As a guide. One would see that ADHD which can be caused by drugs, trauma or extreme hormone levels, diet made.

(Trauma ADHD, ptsd, etc) Will open at pruning stage, in sexual abuse an opening for schizophrenia. Due to high levels of dissociation within the behaviour.

One must remember that during puberty the brain moves over sides, which could then create a chemical imbalance. If you add say crack cocaine. Extreme imbalance would occur. Or if trauma occurs within the time frame. One must look carefully at the behaviour to tackle the negative effects.

Bipolar being the opening for clients that had Prev been on the autism scale and ADHD (ADHD Autism) scale before the abuse would then see a change in pathways and the bi poler gateway open. Which then could go on to psychosis

.

As behaviour is built up over the course of our life through biology and environmental and our social interact with the world around us. One would ask this question:

Why is it that we are still treating clients with one stamp disorders. When in fact the clients' disorders change and lay up (so to speak) as we enter different times in our lives.

Our hormones also changing throughout our lives. Which in return has a knock-on effect on our behaviour. I.e the menopause in women. One can be also diagnosed with the bipolar stamp., even if never had any mental health disorders at all within their lives.

We must remember that our mental health changes with our age and not treat it as the same.

End sums:

Fetal and up to 4 months (4 months being the start of myelination process)

Disorders to include the following:

***Agenesis**

***Autism**

(In trauma pathways only.)

(enzyme 11 β -HSD2 (subtract by two) on stress reaction)

+

(Cortisol (**x 4**) to the reaction to repair and adapt to the new environment.)
Cortisol being the stress hormones more dominantly in fetal or in under 4 months of childhood etc.... result of the reaction.)

= the decrease then leading to the HSD11B2 (type 2) to >cell mutation = due to the need to seek to find a solution to the change in its environment the mothers stress hormone's has triggered.

The mutation of HSD11B2 type 2 being in the fetal brain and developing brain ages 4 months after live birth.

(Progesterone plays a role in maintaining pregnancy. The **hormone** is produced in the ovaries, the placenta (when a woman gets pregnant) and the adrenal glands)

Can be passed on though the mother's stress hormone's after birth in breast milk.
> mothers stress hormone's always having a dominantly effect. Mothers hormones after birth being readjusting to the rush of required hormones to create the foetal life.

This is trauma pathways not actual autism pathway, which is clearly an inherited condition, passed on through the family tree.

Cell division and mutation of the HSD 11 B2 being caused by Estrogen

>Environmental cues such as photoperiod can modulate whether estrogens activate **nongenomic** or genomic pathways.

Through **non genomic** pathways>

Effects on other hormones that stimulate **cell** division: **estrogen** can indirectly stimulate **cell** division by instructing a target **cell** to make receptors for other hormones that stimulate **cells** to divide.

> **kainate** receptors> causing GluK4 =to lower hippocampus (up to 8% reduction);

Darwin's evidence of his theory.

Marijuana: interaction with the estrogen receptor.

by MA Sauer - 1983 -

Crude *marijuana* extract competed with estradiol for binding to the estrogen receptor of ... Apigenin, the aglycone of a flavinoid *phytoestrogen* found in *cannabis*, .

[Marijuana and Medicine: The Endocrine Effects of Cannabis ...](#)

[www.reliasmedia.com › articles › 41368-marijuana-and-medicine-the-...](#)

1 May 1999 - In addition to cannabinoids and *phytoestrogens*, *marijuana* contains ... true of acetylcholinesterase inhibitors intrinsic to *cannabis plants*.²¹
The .

(PDF) The effects of Cannabis sativa L. seed (hempseed) in ... [www.researchgate.net](#)
› [publication › 47743299 The effects of Canna...](#)

25 Oct 2016 - PDF | *Cannabis sativa* L. has been used for the treatment of various gynecological ... such as cannabinoids, *phytoestrogens* and n-3- or n-6polyunsatu- ... in hempseed depends on the type of *plant* (fiber or drug hemp), as.

(PDF) A Phytoestrogens and Their Health Effect - ResearchGate [www.researchgate.net
› publication › 331127374 A Phytoestrogens an...](http://www.researchgate.net/publication/331127374_A_Phytoestrogens_an...)

24 Apr 2019 - PDF | *Phytoestrogens* have structures that are very similar to endogen estradiol ... derived from *plants*, which are structurally similar to ... prevent skin from oxidative *stress* and apoptosis. ... *response*, and these effects were reversible. ... Rietjens IM, Louisse J, Beekmann K. The *potential* health effects o

[Effect of Long-Term Strontium Exposure on the Content of ... www.ncbi.nlm.nih.gov
› pmc › articles › PMC6321324](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC6321324)

by S Dresler - 2018 -

4 Dec 2018 - Abiotic *stress*, including metal excess, can modify *plant* metabolism. ... of *isoflavones* increased in *plants* in *response* to various stresses, e.g., ... were transferred into pots filled with Hoagland's nutrient (five *plants* per *pot*)

Cannabis on human hormones and behavior affect;

[The Effects of Cannabis on your Hormones | ZRT Laboratory www.zrtlab.com ›
blog › archive › the-effects-of-cannabis-on-your-h...](http://www.zrtlab.com/blog/archive/the-effects-of-cannabis-on-your-h...)

by Z Hauck -

20 Jun 2019 - *Cannabis*'s effects on the hypothalamus-pituitary-adrenal (HPA) axis. The HPA axis controls the *stress response* ultimately through the release

Evidence supporting mutation of the HSD11B2 type two is possible although rare;

[analysis of the 11β-Hydroxysteroid Dehydrogenase Type 2 ...](#)

[academic.oup.com › ajh › article](#) by

B Mariniello - 2005 -

Mutations in the HSD11B2 gene cause the syndrome of apparent mineralocorticoid excess (AME), characterized by severe hypertension, hypokalemia and low

[Genetic Association of 11 \$\beta\$ -Hydroxysteroid Dehydrogenase ...](#)

[www.ahajournals.org › doi › full › 01.HYP.28.3.478](#)

by B Watson - 1996 -

1 Sep 1996 - 11 β -Hydroxysteroid dehydrogenase *type 2* (11 β -HSD2) specifically ...
Mutations in the HSD11B2 gene (both exonic and intronic) have been

Added references and notes:

[Anxiety and PTSD linked to increased myelin in brain's ...](#)

SD

[Science Daily](#)

<https://www.sciencedaily.com › releases › 2022/01>

7 Jan 2022 — Scientists have shown in both anxious rats and military veterans with PTSD that **acute stress is associated with** increased myelination of axons ...

[mother or the fetus? 11 \$\beta\$ -Hydroxysteroid dehydrogenase type 2 null mice provide evidence for direct fetal programming of behavior by endogenous glucocorticoids](#)

[MC Holmes](#), CT Abrahamsen, KL French... - Journal of ..., 2006 - Soc Neuroscience

[Inhibition of 11 \$\beta\$ -hydroxysteroid dehydrogenase, the foeto-placental barrier to maternal glucocorticoids, permanently programs amygdala GR mRNA expression and ...](#)

[LAM Welberg](#), [JR Seckl](#)... - European Journal of ..., 2000 - Wiley Online Library

... Normally, **11 β -hydroxysteroid dehydrogenase** type 2 (11 β -HSD2) rapidly inactivates

glucocorticoids in placenta and ... permanent alterations of the HPA axis and anxiety-like **behaviour**

in aversive ... The behavioural and HPA effects may reflect GR gene programming in amygdala ...

[11 \$\beta\$ -hydroxysteroid dehydrogenase type 1 expression is increased in the aged mouse hippocampus and parietal cortex and causes memory impairments](#)

[MC Holmes](#), RN Carter, J Noble, S Chitnis... - Journal of ..., 2010 - Soc Neuroscience

[The role of 11 \$\beta\$ -hydroxysteroid dehydrogenases in the brain](#)

[MC Holmes](#), [JR Seckl](#) - Molecular and cellular endocrinology, 2006 - Elsevier

... 1. Interconversion of corticosterone to 11-dehydrocorticosterone by **11 β -hydroxysteroid**

dehydrogenases (11 β -HSDs ... producing the syndrome of apparent mineralocorticoid **excess** (SAME) ...

and production of antisera to human placental **11 β -hydroxysteroid dehydrogenase** type 2 ...

[Corticosteroids, receptors, and the organ-specific functions of 11 \$\beta\$ -hydroxysteroid dehydrogenase](#)

C Monder - The FASEB journal, 1991 - Wiley Online Library

... and laboratory studies of the properties and **behavior** of ...

2Abbreviations: Mr, mineralocorticoid

receptor; GR, glucocorticoid receptor; 11 β HSD, 11/3-hydroxysteroid **dehydrogenase**; AME,

apparent mineralocorticoid **excess**; GE, glycyrrhetic acid. Page 2. C H2O H H 3048 Vol ...

[Prenatal stress reduces postnatal neurogenesis in rats selectively bred for high, but not low, anxiety: possible key role of placental 11 \$\beta\$ -hydroxysteroid dehydrogenase ...](#)

[PJ Lucassen](#), [OJ Bosch](#), E Jousma... - European journal of ..., 2009 - Wiley Online Library

... C) or prenatally stressed (PS) high anxiety-related **behavior** (HAB) and low anxiety-related

behavior (LAB) rats ... D) Bioactivity of **11 β -hydroxysteroid dehydrogenase** type 2 ... only in LAB rats

implies that LAB offspring are protected from maternal glucocorticoid **excess**, whereas the ...

[HTML] [Epigenetic effects of prenatal stress on 11 \$\beta\$ -hydroxysteroid dehydrogenase-2 in the placenta and fetal brain](#)

[CJ Peña](#), [C Monk](#), [FA Champagne](#) - PloS one, 2012 - ncbi.nlm.nih.gov

... mutation of the HSD11B2 gene leads to hypertension, **excess** mineralocorticoid activity ... of

11 β -hydroxysteroid dehydrogenase type 2 (HSD11B2),
11 β hydroxysteroid dehydrogenase

-1 (HSD11B1 ... Hydroxysteroid dehydrogenase (HSD11B1),
{ "type": "entrez-nucleotide", "attrs": { " ...

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[CS Wyrwoll](#), [MC Holmes](#), [JR Seckl](#) - Frontiers in neuroendocrinology, 2011 - Elsevier

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HL Bradlow, MI NewA syndrome of apparent mineralocorticoid **excess** associated with

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[https://doi.org/10.1016/0022-3999\(82\)90073-](https://doi.org/10.1016/0022-3999(82)90073-)

Differences in Personality Between Sustained Hypertension, Isolated Clinic Hypertension and Normotension

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Department of Clinical Psychology, Universidad Complutense de Madrid, Spain

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Personality characteristics such as anxiety and anger have long been associated with essential *hypertension*, but the results of past studies have often been confounded by inadequate diagnosis of *hypertension*, antihypertensive medications, and use of unvalidated measures of *personality*

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IA Kretchy - 2019 -

2 Jan 2019 - In some patients, the etiology and prognosis of *hypertension* have been linked to psychological factors including *personality traits*. One primary .

Male behavior and HSD11B2:

[11 \$\beta\$ -Hydroxysteroid dehydrogenase type 2 in zebrafish brain ...](#)

joe.bioscientifica.com/view/journals/joe by L Foppiani -

1999 -

The type 2, 11 β -hydroxysteroid dehydrogenase (*Hsd11b2*) converts active glucocorticoids ... To assess the tissue distribution of *hsd11b2*, one adult *male* zebrafish was ... for direct fetal programming of *behavior* by endogenous glucocorticoids

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T Mune - 2013 -

28 Feb 2013 - Thus, *HSD11B2* CA-repeat genotype is not associated with ... A total of 174 normal *male* subjects from the Japan Self-Defense ... or sodium depletion activated these neurons to affect *behavior* and to drive sodium appetit

[Patterning in Placental 11-B Hydroxysteroid Dehydrogenase ...](#)
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5 Sep 2013 - This study demonstrated that prenatal socioeconomic adversity was associated with lower levels of placental *HSD11B2* methylation, particularly for *males*. ... Less methylation of placental *HSD11B2* may therefore be adaptive and promote the effective management of stress associated with social adversit

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L Li - 2015 -

Zearalenone showed potent inhibition of *HSD11B2* with the half-maximal inhibitory ... *Male* Sprague-Dawley rats (250–300 g) were purchased from Shanghai ... stress: role of glucocorticoid metabolism in *behavioral* dominance,

[The Role of 11 \$\beta\$ -Hydroxy Steroid Dehydrogenase Type 2 in ...](#)
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FJG Shearer - 2019 -

One mechanism underpinning the downregulation of HSD2 in response to social adversity is increased methylation of placental *HSD11B2*, particularly in *male* placentas [60]. In a rodent model of prenatal stress, placental *Hsd11b2* mRNA was decreased and methylation of the promoter increased

[The Role of Placental 11-Beta Hydroxysteroid ... - NCBI - NIH](#)
[www.ncbi.nlm.nih.gov › pmc › articles › PMC4652612](#)

by BB Green - 2015 -

18 Mar 2015 - In addition, placental *HSD11B2* expression and activity has been associated with ... *Males* displayed no correlation between *HSD11B2* mean ... antenatal anxiety and children's *behavioural*/emotional problems at 4 year

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... *behaviors* and cognitive function, but not anxiety *behaviors* in *male* mice ... Expression of *Hsd11b1* and *Hsd11b2* in fetal brain, and *Nr3c1* and *Nr3c2* in adult

Female behavior and HSD11BS

[Epigenetic regulation of the placental HSD11B2 barrier and its ...](#)
[www.ncbi.nlm.nih.gov › pmc › articles › PMC4065178](#)

by KL Togher - 2014 -

9 Apr 2014 - Furthermore, *women* experiencing high levels of stress during ... following prenatal anxiety in fact leads to the *behavioral* changes observed in

[Prenatal Major Depressive Disorder, Placenta Glucocorticoid ...](#)
[europepmc.org › articles › pmc6541396](#)

Keywords: *depression*, cortisol, *HSD11B2*, pregnancy, serotonin, infant ... and their healthy infants (48% *female*, mean age=32 days) from the *Behavior* and

[Maternal Prenatal Mental Health and Placental 11 \$\beta\$ -HSD2 ...](#)
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by S Seth - 2015 -

17 Nov 2015 - Expression levels of 11 β -HSD2 (encoded by the gene, *HSD11B2*) rise ... for high anxiety-related *behaviour* or low anxiety-related *behaviour* and ... *women* with untreated *depressive* or anxiety symptoms, and a control group

[Prenatal major depressive disorder, placenta ... - NCBI](#)
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by LR Stroud - 2016 -

29 May 2019 - Exposure to maternal *depression* is one of the most common forms of early adversity, with known links to long-term health and behavioral outcomes. ... As hypothesized, we found that both placental *HSD11B2* methylation and SLC6A4 gene expression moderated the influence of prenatal MDD on infant cortisol regulation.

[Fetal brain 11 \$\beta\$ -hydroxysteroid dehydrogenase type 2 ...](#)
[www.sciencedirect.com › science › article › pii](#)

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As adults, HSD2BKO show *depressive-like behavior* and have cognitive impairments. ... *Female* offspring were not assessed in this study due to the potential ... Expression of *Hsd11b1* and *Hsd11b2* in fetal brain, and *Nr3c1* and *Nr3c2* in adult

Sum one supporting evidence.

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by S Seth - 2015 -

17 Nov 2015 - Expression levels of 11 β -HSD2 (encoded by the gene, *HSD11B2*) rise in ... Kinney D.K., Miller A.M., Crowley D.J., Huang E., Gerber E. *Autism*

[Multidisciplinary and Comparative Investigations of ... - Frontiers](#)
[www.frontiersin.org › articles › fcimb.2019.00269 › full](#)

by BR Beck - 2019 -

25 Jul 2019 - The *increase* in the expression of *Hsd11b2* may contribute to stress ... The *increase* in serotonin level observed in *autism* spectrum disorder

Sum two supporting evidence.

[Prenatal Major Depressive Disorder, Placenta Glucocorticoid ...](#)
[europepmc.org › articles › pmc6541396](#)

Keywords: depression, cortisol, *HSD11B2*, pregnancy, serotonin, infant. Go to: ... steroid medications during pregnancy, psychotic or *bipolar disorder*, medical .

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1 May 2012 - The frequency of mutations in the *HSD11B2* gene is rare and thus alone ... the patients.^{15,16} To date, no study of polymorphisms in the *HSD11B2* gene ... Antidepressant-Associated Mania in *Bipolar Disord*

[Altered systemic cortisol metabolism in bipolar disorder and ...](#) [www.researchgate.net](#)
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WEB9 May 2023 : The five major neurotransmitters and four types of **brain** chemistry play a vital role in regulating brain function and mood. Genetics, stress, diet, physical activity, and

Cardiovascular, Respiratory, and Gastrointestinal Disorders Reed E. Pyeritz, Bruce R. ... 2 Congenital adrenal hyperplasia (21-hydroxylase deficiency) [69] *HSD11B2* ... The project analyzed seven common diseases: *bipolar disorder*; coronar

[Changes in the Maternal Hypothalamic-Pituitary-Adrenal Axis ...](#)
[www.karger.com › Article › Fulltext](#) by

L Duthie - 2013 -

19 Sep 2013 - Maternal HPA axis activity and activity of *HSD11B2* may be modified by ... to stress and susceptibility to physical and mental illness in later life. ... cortisol-dehydroepiandrosterone ratios in schizophrenia and *bipolar disorde*

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[DNA methylation profiles at birth and child ADHD symptoms ...](#)
[www.researchgate.net › publication › 259108557_DNA_methylation_pr...](#)

16 Dec 2019 - Attention deficit/hyperactivity disorder (*ADHD*) is a common and ... Alterations to NR3C1 and *HSD11B2* and the Developmental Origins of

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by KL Togher - 2014 -

9 Apr 2014 - Consistent with preclinical data, chronic prenatal anxiety and *depression* were found to be associated with *reduced* activity and expression of placental *HSD11B2*. ... These studies indicate that there are multiple mechanisms through which prenatal stress may affect placental *HSD11B2* expression and/or activity

[Maternal Prenatal Mental Health and Placental 11 \$\beta\$ -HSD2 ... \[www.ncbi.nlm.nih.gov
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by S Seth - 2015 -

17 Nov 2015 - (2012) [32], which found a 30% *reduction* in placental *HSD11B2* expression in anxious pregnant women and a negative (although non-significant) association between maternal *depression* and *HSD11B2*. ... In contrast, animal research has indicated that chronic stress lowers *HSD11B2* expression in the placenta

[Prenatal major depressive disorder, placenta glucocorticoid and serotonergic signaling, and infant cortisol response](#)

Laura R. Stroud, Ph.D., George D. Papandonatos, Ph.D., [...], and Carmen J. Marsit, Ph.D

