

Developmental changes in Stress Response in the Brain: Childhood Trauma

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Area of study:

Effect of Stress response pathways and hormones; on the development of the brain.

Key Findings in the paper.

*Classic Autism has inherited traits. The **SHANK3 gene** is located on **chromosome 22** and is one of the three members of the Shank gene family. It encodes a protein that plays a crucial role in the postsynaptic density, connecting neurotransmitter receptors, ion channels, and other membrane proteins to the actin cytoskeleton and G-protein-coupled signaling pathways.

*Prolonged stress response in childhood causes autism and ADHD pathways to foam. The younger the child the more profound the dual pathways will be.

Through abuse during critical brain developmental stages in childhood, changing the brain wiring and mimicking the autism and adhd pathways in behaviour. 0-6 years of age seeing biggest changes to the brain. The autism and adhd are mirroring each other and at times. Both will be present, as well as influencing personality development.

* Chemical imbalance now deemed a hormone imbalance due to 80 per cent is hormones and hormones are a chemical.

*estrogen masking adhd and autism in girls, due to producing inward behaviour and often will go unnoticed till dips or raises in estrogen levels.

* **Genetic ADHD** caused by growth hormones.

Trauma and stress response mimicking ADHD is caused by adrenaline over drive due to constant triggering of stress response.

*Neuroinflammation in childhood trauma is a critical factor that can lead to long-term cognitive, behavioral, and psychosocial disruptions. Chronic neuroinflammation caused by early adversity can reprogram immune responses, increasing the risk of psychiatric disorders later in life.

*Childhood trauma can significantly impact gut bacteria, leading to various health issues. Research indicates that children exposed to adversity early in life may experience gastrointestinal symptoms that could affect their emotional health as they grow.

*The effects of sensory processing in childhood trauma can significantly impact a child's ability to navigate daily tasks and participate in various aspects of life. Trauma can alter the processing of sensory

input, leading to difficulties in regulating the sensory system. This can manifest as over-reacting to sensory stimuli, lack of coordination, clumsiness, poor motor planning, and balance issues.

*Early-life trauma, such as abuse, neglect, or household dysfunction, introduces **toxic stress** during critical periods of brain development, typically between ages 0–6, when the brain is highly plastic and forming neural connections (synaptogenesis)

***The autism and ADHD pathways then increasing the opening of bipolar, schizophrenia, etc in puberty. All the disorders start off on the pathways in childhood trauma background.**

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.

To see how the cognitive and sensory pathways are developing within this time frame and the part hormones play in this. The level of trauma and the younger the age it started and length of time, will see profound changes in the brain structure.

Two pathways for Dual ADHD and Autism are;

Pathway one: Inherited.

Profound autism and Aspergers.

Inherited factors.

Chromosome 22q13.3 refers to a specific location on chromosome 22..

chromosome 22, specifically a region known as 22q11.2,

About Chromosome 22:

Chromosome 22 is one of the 23 pairs of **chromosomes** in human **cells**. Humans normally have two copies of chromosome 22 in each cell. Chromosome 22 is the second smallest human chromosome, spanning about 51 million **DNA base pairs** and representing between 1.5 and 2% of the total DNA in **cells**.

In 1999, researchers working on the **Human Genome Project** announced they had determined the sequence of base pairs that make up this chromosome. Chromosome 22 was the first human chromosome to be fully sequenced.^[4]

Human chromosomes are numbered by their apparent size in the **karyotype**, with **chromosome 1** being the largest and chromosome 22 having originally been identified as the smallest. However, genome sequencing has revealed that **chromosome 21** is actually smaller than chromosome

The **22q11.2 deletion syndrome**, also known as DiGeorge syndrome, is associated with a higher prevalence of **autism spectrum disorder (ASD)** in some individuals. Studies indicate that individuals with deletions involving the LCR-A to B region of 22q11.2 show a **39–44% rate of ASD**, compared to **0%** in those without this deletion. Additionally, children with 22q11.2 deletion syndrome often exhibit psychiatric disorders, including autism-like symptoms, but may not meet full criteria for ASD. This suggests a complex relationship where some individuals with this deletion may present with autism-like behaviors, but not all will be diagnosed with ASD.

The 22q11.2 deletion syndrome is also associated with IGF-1, an insulin-like growth factor that plays a crucial role in regulating growth and development. IGF-1 levels can be influenced by genetic variations, and in the context of the 22q11.2 deletion syndrome, it may contribute to the cognitive and behavioral challenges observed in individuals with autism. Understanding the relationship between IGF-1 and the 22q11.2 deletion syndrome can provide insights into the genetic basis of autism and inform treatment strategies.

SHANK3 gene deficiency syndrome is associated with mutations in the SHANK3 gene, which is located on chromosome 22. This gene encodes a protein that plays a crucial role in the development and function of the nervous system. SHANK3 is involved in synapse formation and dendritic spine maturation, which are essential for proper brain function. Deficiencies in this gene can lead to various neurodevelopmental conditions, including autism spectrum disorder and Phelan-McDermid syndrome. The SHANK3 protein acts as a structural scaffold that helps maintain strong, efficient connections between nerve cells and supports communication between neurons. Mutations in the SHANK3 gene can result in the loss of one copy of the gene (haploinsufficiency) or changes within the gene itself, which can lead to impaired synapse formation and other neurological symptoms. **(In childhood trauma in abuse from very young age, under 6 years of age. We see no changes to shanks 3 protein.)**

IGF-1 may rescue function in Rett syndrome and ASD caused by changes of the *SCHANK3* gene.

Phelan–McDermid syndrome (PMDS) is a complex neurodevelopmental disorder characterized by global developmental delay, severely impaired speech, intellectual disability, and an increased risk of autism spectrum disorders (ASDs)¹. PMDS is caused by heterozygous deletions of chromosome 22q13.3. Among the genes in the deleted region is *SHANK3*, which encodes a protein in the postsynaptic density (PSD)^{2,3}. Rare mutations in *SHANK3* have been associated with idiopathic ASDs^{4,5,6,7}, non-syndromic intellectual disability⁸, and schizophrenia⁹. Although *SHANK3* is considered to be the most likely candidate gene for the neurological abnormalities in PMDS patients¹⁰, the cellular and molecular phenotypes associated with this syndrome in human neurons are unknown. We generated induced pluripotent stem (iPS) cells from individuals with PMDS and autism and used them to produce functional neurons. We show that PMDS neurons have reduced SHANK3 expression and major defects in excitatory, but not inhibitory, synaptic transmission. Excitatory synaptic transmission in PMDS neurons can be corrected by restoring SHANK3 expression or by treating neurons with insulin-like growth factor 1 (IGF1). IGF1 treatment promotes formation of mature excitatory synapses that lack SHANK3 but contain PSD95 and *N*-methyl-D-aspartate (NMDA) receptors with fast deactivation kinetics. Our findings provide direct evidence for a disruption in the ratio of cellular excitation and inhibition in PMDS neurons, and point to a molecular pathway that can be recruited to restore it.

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.

Autism spectrum disorders (ASD) is a neurodevelopmental disorder with multifaceted etiology; with the hormonal imbalance playing an important role. Recent evidence suggests that plasma levels of Growth Hormone (GH) and ghrelin are altered in autistic children

The umbilical cord contains stem cells that are multipotent, meaning they can develop into various cell types. These stem cells have been studied for their potential impact on growth hormones and other biological processes. Research indicates that umbilical cord blood hormones and growth factors, such as insulin-like growth factor-1 (IGF-1), are strongly correlated with hematopoietic stem cell concentrations, which are surrogates for overall stem cell potential. Changes in stem cell concentration associated with mitogens have been analyzed, showing that IGF-1 levels are significantly correlated with stem/progenitor concentrations.

Low IGF-1 levels can result from various factors, including inadequate growth hormone production, liver dysfunction, or nutritional deficiencies.

The role of IGF-1 in gut bacteria is complex and bidirectional. IGF-1 can influence the gut microbiota, and the gut microbiota can also impact IGF-1 levels.

Low levels of blood cholesterol have been found in some children with **autism** spectrum disorders (ASD). Cholesterol is a **waxy, fat-like substance** found in your blood that your body needs to build healthy cells, produce hormones, and make vitamin D. It is produced naturally by the liver and is also obtained from certain foods..

Emerging research suggests a potential link between growth hormone deficiency and ADHD, indicating that hormonal imbalances may influence cognitive and behavioral symptoms.

The genetic traits associated with IGF-1 are primarily linked to its role in growth and development.

Pathway two:

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 form 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

One must also consider when looking at trauma in childhood or ADHD, the blunt reward pathways. And the relationship with sugar... The relationship between sugar and ADHD is complex and multifaceted. While sugar does not directly cause ADHD, it can exacerbate symptoms by disrupting

dopamine balance, attention, and emotional regulation. The ADHD brain's reward system is particularly sensitive to glucose, leading to a cycle of craving, consumption, and crash. This can result in a desensitization of dopamine receptors, requiring more sugar to achieve the same effect.. Add in the reward pathways and stress pathways .. The concept of "blunt dopamine" in ADHD refers to the idea that ADHD symptoms are not caused by a deficiency of dopamine itself but rather by the way dopamine interacts with its receptors in the brain. ADHDers often have variations in dopamine receptor genes, which can lead to lower dopamine signal strength. This means that while they may not have a lower level of dopamine, they may require more stimulation to feel engaged and settled.

Research has shown that ADHDers are more likely to have specific genetic variations in dopamine receptors, which can affect how dopamine is detected and processed in the brain. These variations are part of what makes ADHDers neurodiverse compared to the majority of individuals, who are considered neurotypical.

It's important to note that the "low dopamine" myth oversimplifies the role of dopamine in ADHD. ADHD is associated with disruptions in pathways related to dopamine and other neurotransmitters, such as norepinephrine, which impacts learning, memory, mood, and emotions.

Seen most clear, when you have someone coming off drugs etc, will go straight to sugar addiction. Unknow aware of the relationship between recovery of addiction and blunt pathways in reward system. One must remember addiction over long time will create a mincing of dual pathways due to stress response and reward pathways yo going.

When we look at mixing up of facial expressions.. The fusiform face area (FFA) is a critical region in the brain responsible for processing facial features and spatial relations among them. Individuals with autism spectrum disorder (ASD) often exhibit abnormalities in the FFA, which can lead to difficulties in recognizing and processing faces. Research has shown that the FFA in individuals with ASD may have reduced activation during face processing tasks.

Individuals with autism often exhibit atypical cortisol regulation, showing heightened or prolonged stress responses that can influence behavior and adaptation.

Cortisol plays a crucial role in regulating the inflammatory response, acting as both an anti-inflammatory agent and a potential contributor to chronic inflammation under prolonged stress. When looking at inflammatory in autism and adhd. Which would then cause neuroinflammation during childhood in both trauma response mimicking autism and adhd and classic adhd and autism. This neuroinflammation **disrupts serotonin synthesis and BDNF production**, contributing to mood instability. Specific genetic variants in the serotonin, dopamine, oxytocin, and cortisol pathways affect BPD risk during childhood developmental stages in childhood trauma.

Neuroinflammation is a significant factor in the pathophysiology of schizoaffective disorder. It involves the activation of the immune system in the brain, leading to inflammation and damage to neurons. This process is associated with oxidative stress and neuroinflammatory responses, which can exacerbate neuronal damage and contribute to the psychiatric symptoms of the disorder. The interplay between neuroinflammation and oxidative stress is modulated by various pathways, including the WNT/ β -catenin pathway, which is involved in the inflammatory response.

Chronic stress activates the immune response in the brain, leading to the excessive activation of microglia (the brain's resident immune cells) and the release of pro-inflammatory cytokines. This neuroinflammatory response can disrupt normal brain function and is associated with the development of mental health disorders, particularly major depressive disorder. In 0 to 6 if stress response is triggered

constantly due to abuse or neglect..during attachment, personality development, the cortisol imbalance would change the structure of the brain etc.

When looking at cells and energy source. Cells in the body obtain their energy primarily through **cellular respiration**, a process that occurs in the mitochondria. This process involves breaking down nutrients, especially glucose, into smaller molecules that are then converted into ATP, the universal energy currency. The relationship between growth hormones and mitochondrial respiration is significant, as GH and IGF-1 play crucial roles in regulating mitochondrial mass and function. These hormones peak during puberty and decline during adulthood. **mitochondrial dysfunction may play a significant role in the development and symptoms of autism spectrum disorder.** Mitochondrial dysfunction is a significant factor in the pathophysiology of ADHD.

ADHD primary causes of it to foam are down to genetic fault and trauma response.

Adrenaline, also known as epinephrine, is a hormone secreted by the adrenal glands during stress, preparing the body for rapid action. It triggers the body's fight-or-flight response, causing heart rate and blood pressure to increase, blood vessels to constrict, and muscles to release energy reserves. This hormone is crucial for immediate physical responses during stressful situations.

Growth hormones, also known as somatotropes, are secreted by the pituitary gland and play a role in growth and development. They stimulate the growth of tissues and organs, including bones, muscles, and skin. Growth hormones are essential for normal growth and development during childhood and adolescence.

Both adrenaline and growth hormones are part of the endocrine system, which uses hormones to control and coordinate the body's internal metabolism, reproduction, growth and development, and response to injury, stress, and environmental factors.

Genetic ADHD caused by growth hormones.

Trauma and stress response mimicking ADHD is caused by adrenaline over drive due to constant triggering of stress response.

Introduction.

Autism, ADHD is down to the inherited genetically defect which 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 prediluvian 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 **IGF1 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.**

IGF-1 deficiency, particularly Primary Insulin IGF-1 deficiency, particularly Primary Insulin-like Growth Factor Deficiency (PIGFD), occurs when the body does not produce sufficient levels of IGF-1, a hormone crucial for growth and development. This deficiency can result from genetic conditions, such as Laron syndrome, where the growth hormone receptor is dysfunctional, leading to low IGF-1 levels despite normal or high growth hormone-like Growth Factor Deficiency (PIGFD), occurs when the body does not produce sufficient levels of IGF-1, a hormone crucial for growth and development. over production would then point then to a genetic, passed through the family in classic autism.

Increased IGF-1 levels: Studies have found that serum IGF-1 levels are higher in individuals with ASD compared to non-autistic controls,

Research indicates that **high peripheral IGF-1 levels are associated with bipolar disorder.**

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 IGF1 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 **Basal 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 (**IGF1 + 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 imaginary 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.

The relationship between autism and blindness is complex and multifaceted. Research indicates that **autism is more than 30 times as common among blind people as among sighted people.** This prevalence is attributed to the fact that vision is crucial for brain development, and without it, social and communication skills may be disrupted.

Insulin-like growth factor-1 (IGF-1) plays a significant role in vision and eye health. It is involved in the development and maintenance of retinal structures, influencing processes such as retinal angiogenesis and neuroprotection.

individuals on the autism spectrum may experience elevated rates of both type 1 and type 2 diabetes, influenced by genetic pathways, gut microbiome differences, and the stress of managing a chronic condition

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 (IGF1=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 prolonged 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 processes emotions and anger, memory and so on. Again, the neurons in stress response will become damaged due to peaks in growth hormones and IGF1-1.

The cerebral cortex, 16 Million neurons. All following this principle in neuron pattern in stress response again damaged neurons and knock on effect on the grey matter. The Amygdala 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 eliminate the weakened neurons.

The Amyloid beta protein (AB) in the stress response becomes overactive.... and decreased the grey matter in prolonged 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 Million 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.)

Supporting proteins and Glial cell being the main primary pattern in achieving this. Glial cell in stress response creating cell division.

Yes, glial cells can achieve cell division. NG2 cells, a type of glial cell, have been shown to retain the ability to proliferate throughout life, even after completing myelination. This ability to divide and maintain differentiated properties is a significant aspect of their role in the adult CNS. In childhood trauma cell density is lower, due to the stress response in the growth peaks of not only growth hormones and IGF1-1 which are required to achieve pruning. Peaks of growth hormones occurring in peaks of pruning. Which is trauma having a knock-on effect on the protein and cells required to produce the supporting white matter and myelin.

One must remember the cognitive growth the brain does through in those first 3 years of life.

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 are 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 developing 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.

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 and internal 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 behaviour.

then go into overactivity 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. Pruning of the damaged 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.

In prolonged stress response ... the reward pathway and stress response pathway bang against each other and have a knock on effect.

Cortisol and adrenaline are both hormones produced by the adrenal glands, but they serve different functions in the body. **Cortisol** is primarily released in response to stress and is involved in the body's fight-or-flight response. It helps to increase heart rate and blood pressure, suppress digestion, and alter the immune system to prepare the body for immediate action. Cortisol also plays a role in regulating metabolism, blood pressure, and immune function.

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Adrenaline, also known as epinephrine, is produced in the medulla of the adrenal glands and is involved in the body's immediate response to stress. It works alongside cortisol to prepare the body for action, with adrenaline increasing heart rate and blood pressure, and cortisol also increasing heart rate and blood pressure. Both hormones are essential for survival during acute stress but can lead to health problems if they are produced in excess.

Cortisol is a key hormone released by the adrenal glands that helps the body respond to stress by regulating energy, metabolism, and immune function.

Role in Stress

Cortisol, often called the “stress hormone,” is produced by the adrenal glands and plays a central role in the body’s response to stress clevelandclinic.org+1. When a person encounters a stressor, the **amygdala** in the brain signals the **hypothalamus**, which activates the **hypothalamic-pituitary-adrenal (HPA) axis**. This triggers the adrenal glands to release cortisol into the bloodstream [Science Times](https://www.sciencetimes.com)+1. Cortisol works alongside adrenaline to prepare the body for a **fight-or-flight response**, providing a burst of energy and heightened alertness.

Cortisol, a steroid hormone produced in the adrenal glands, has a complex relationship with neurons. While cortisol is not a neurotransmitter, it can have significant effects on neuronal activity. It binds to receptors in the brain, including glucocorticoid receptors, which are found throughout the brain and cell types. These receptors are involved in stress response, memory

consolidation, and behavioral adaptation. The two types of receptors have opposite effects on the same neuron, with one type increasing excitability and the other dampening it. This dual action explains cortisol's broad influence on the brain, affecting memory retrieval, emotional processing, and decision-making.

A cortisol imbalance being created in the stress response. Most seen better in soldiers.. Due to their in war or on camp and the effects of flight or fight..

Cortisol imbalance in soldiers can lead to various health issues due to the prolonged stress and physical demands of military training.

Enhancing the cptsd... as the imbalance or both cortisol and aderline yo yo up and down to cater for the response that is going to be produced... also the reward pathway as the person seeks escapism then yo yoining... Having knock on effect after stress reponse with reward pathway..

Mesocorticolimbic circuit

Reward hormone pathway

The reward hormone pathway, also known as the mesocorticolimbic circuit, is a group of neural structures responsible for incentive salience, associative learning, and positively-valenced emotions. It plays a crucial role in motivating behavior and driving the pursuit of rewards. The pathway begins with the ventral tegmental area (VTA), which releases dopamine in response to rewarding activities. This dopamine then travels to the nucleus accumbens (NAc) and the prefrontal cortex (PFC), where it influences decision-making and planning. The pathway is involved in both intrinsic and extrinsic rewards, influencing behavior through positive reinforcement and classical conditioning

The mesocorticolimbic circuit is the brain's primary reward system, integrating motivation, pleasure, and goal-directed behavior through dopaminergic pathways originating in the ventral tegmental area (VTA).

Overview

The **mesocorticolimbic system** is a complex neural network responsible for reward-related cognition, motivation, and positively-valenced emotions such as pleasure and euphoria ^{Wikipedia}+1. It plays a central role in guiding behavior toward survival-enhancing and rewarding stimuli, including food, social interaction, and reproductive activities ^{Wikipedia}.

Dysregulation of this circuit is implicated in addiction, where substances or behaviors overstimulate dopamine release, leading to compulsive seeking and neuroadaptations

Autism or ADHD causes would then be: genetic or prolonged stress response.

DNA Passed through inherited root.

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

Environmental and socially. Trauma.

Damage to the brain. Which would create deeper pathways.

Too much growth hormones

The human amygdala is critical for emotional learning, valence coding, and complex social interactions, all of which mature throughout childhood, puberty, and adolescence. Across these ages, the amygdala paralaminar nucleus (PL) undergoes significant structural changes including increased numbers of mature neurons. The PL contains a large population of immature excitatory neurons at birth, some of which may continue to be born from local progenitors. These progenitors disappear rapidly in infancy, but the immature neurons persist throughout childhood and adolescent ages, indicating that they develop on a protracted timeline. Many of these late-maturing neurons settle locally within the PL, though a small subset appear to migrate into neighboring amygdala subnuclei. Despite its prominent growth during postnatal life and possible contributions to multiple amygdala circuits, the function of the PL remains unknown. PL maturation occurs predominately during late childhood and into puberty when sex hormone levels change. Sex hormones can promote developmental processes such as neuron migration, dendritic outgrowth, and synaptic plasticity, which appear to be ongoing in late-maturing PL neurons. Collectively, we describe how the growth of late-maturing neurons occurs in the right time and place to be relevant for amygdala functions and neuropsychiatric conditions.

The amygdala undergoes significant changes during early puberty, particularly in the paralaminar nucleus (PL), which is crucial for emotional learning and valence coding. During this period, the PL experiences increased numbers of mature neurons, which are essential for processing emotions like fear. The maturation of these neurons is influenced by sex hormones, which promote developmental processes such as neuron migration, dendritic outgrowth, and synaptic plasticity. These changes are part of the broader development of the brain's structure, chemistry, and synapses, which are critical for social interactions and emotional responses during adolescence.

The paralaminar nucleus (PL) of the amygdala is a subregion that exhibits delayed maturation and contributes to the increase in size and cell number of the amygdala between birth and adulthood. In individuals with autism spectrum disorder (ASD), the PL has been found to harbor a large population of immature neurons, which may contribute to the altered trajectory of amygdala growth observed in ASD. This suggests that the PL may play a significant role in the development and function of the amygdala in ASD, potentially influencing emotional and social learning processes.

The amygdala, particularly the paralaminar nucleus (PL), plays a significant role in the neurobiology of ADHD. It is involved in emotional regulation, learning through reward and punishment, attention, impulsivity, memory, and decision-making.

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.

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.

Early puberty in girls (as girls are easier to trace) ADHD and Autism triggered at times a common rate of changes to the puberty. But they also have late puberty. The puberty is imbalanced.. Which means there is a issues with the growth hormones.

Both share a close bond with hormones from the start and changes are seen in the puberty time frame.

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 ...

Women having more chance of having bipolar than men, due to estrogen..

Estrogen's role in bipolar disorder is significant, as it interacts with brain chemicals that regulate mood.

As estrogen levels fluctuate, it can trigger depressive dips, manic surges, or mixed episodes in women with bipolar disorder.

The pituitary gland is found at the base of the brain and is 'pea-sized'. The pituitary gland is an overall controller of several 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 the 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 oversees 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...nerve fibres in the C/C and supporting neurons that support the c/c 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 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)

it is causing these ADHD and autism pathways. what i found somewhat interesting is in John Bowlby's work on attachment... he sees the behavioural traits and does not link them to the ADHD, etc but has elements of inwards and outwards behaviour because they weren't classed as disorders in his time frame....and does not acknowledge the link between the child's instinctive behaviour...and the enhancement of the problem solving capabilities.. One must look at the smaller prefrontal cortex and see the changes occurring.

Schizophrenia is more frequently diagnosed in men due to a combination of hormonal protection in women, earlier onset in men, genetic factors, and sociocultural influences.

Hormones influence brain function by interacting with neurotransmitters such as **dopamine, serotonin, glutamate, and GABA**, which are central to psychotic symptoms like hallucinations and delusions. Dysregulation in the **hypothalamus-pituitary-adrenal (HPA) axis** or **hypothalamus-pituitary-thyroid (HPT) axis** can destabilize these neurotransmitter systems, potentially triggering or exacerbating psychotic episodes. For example, **cortisol excess** in Cushing's syndrome or deficiency in Addison's disease can lead to psychotic symptoms, while thyroid dysfunction can cause "myxedema madness" or hyperthyroid-related psychosis.

Estrogen appears to have a protective effect in women, with lower levels linked to increased risk of psychosis, particularly during **menopause, postpartum, or premenstrual periods**. Women with schizophrenia often show milder symptoms and respond to lower doses of medication, partly due to estrogen's modulation of neurotransmitter activity and neuroprotection.

Men are diagnosed with schizophrenia roughly 1.4 times more often than women, particularly in younger age groups, though lifetime prevalence is similar between sexes (risk ratio: 1.42) [S scienceinsights.org](https://www.scienceinsights.org).

One key biological explanation involves **estrogen**, which appears to protect women by modulating the dopamine system, the neurotransmitter pathway most closely linked to psychotic symptoms. Estradiol influences dopamine transporters, receptors, and synthesis, reducing vulnerability to early psychotic episodes [S scienceinsights.org](https://www.scienceinsights.org). Men, lacking this hormonal protection, often experience earlier onset and more severe negative symptoms, such as emotional flattening, social withdrawal, and cognitive deficits.

Often children have high levels of ADHD.

Women tend to have more bipolar rates

The higher prevalence of bipolar disorder in women is attributed to several factors, including hormonal influences, biological differences, and societal factors. Hormonal fluctuations, particularly those associated with the menstrual cycle, pregnancy, and menopause, can significantly impact mood regulation and exacerbate bipolar symptoms. Women with bipolar disorder are more likely to experience rapid cycling, defined as having four or more mood episodes within a year, and mixed features, where

symptoms of both mania and depression occur simultaneously. These differences in symptom presentation can lead to delays or misdiagnoses, especially for women whose symptoms might initially be mistaken for unipolar depression.

main ingredients to a healthy fully developed corpus callosum are 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 callosum 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 yet to create.

The blood carries the hormones to the corpus, 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 behavioural pattern=

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

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

(see summary for breakdown of a cognitive response behavioural 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 must 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 behavioural traits (outwards)... this is down to the male hormones and adrenaline hormone ... the boys become more aggressive in behaviour...releasing an outlet for the damage the trauma has done... and with testosterone levels and aggression and adrenaline all being linked in, plus stress hormones

In girls we see them copying the autism behavioural 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 behavioural 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 fibres 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 behaviour traits...running, impulsivity... in girls they aren't got it... they are displaying autism behaviour traits in ADHD...that's why boys are 3 times more likely to get classic ADHD (according to the centre for disease control) girls behaviour traits in classic ADHD are autism behaviour traits... ADHD is produced by the over production of growth hormone and hormones... its linked in with the set of behaviour 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 behaviour... two totally different sets of behaviour...

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 estrogenic increases in their bodies. ... These hormonal changes may cause mood and behavioural difficulties for girls with ADHD especially

Estrogenic 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 estrogenic 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 behaviour 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 behavioural 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 and reductions in other parts of the brain

Since girls with ADHD usually don't display "typical" ADHD behaviour, 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 behaviour 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 Centres 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 behavioural 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 behavioural 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 behaviour... the three gateway disorders are causing mayhem in the development of the c/c

the reduction in the corpus callouses and decrease in white matt... then cause a unique behavioural pool. Putting them in a family behavioural 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.

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. 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 fibres connecting homologous cortical areas **during** childhood and **adolescence**.

Time frames the **callosal 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** — **estrogenic** 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 behavioural changes within the boy or girl child at the time. Boys being more boyish **outwards** in behaviour 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 is **caused** by fluctuations in **estrogenic**, 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 oestrogens, 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 estrogenic that acts by catalysing the conversion of **testosterone** (an androgen) to **oestradiol** (an estrogenic). The hormones are active in the development of the c/c as previously thought. The ***corpus callosum*** is a band of nerve fibres 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.

we cross exam behaviour traits of agenesis of the c/c... against autism and we get same behaviour 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 fibres and supporting cells and neurons that support the C/C.** leading to similar but different watered-down behaviours...that we are seeing. whether its deficits that occur in the womb before birth or during the enhancement periods of development the corpus.

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 behavioural 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 led to the arousal of the sympathetic nervous system and reduced activity in the parasympathetic nervous system...the reason the behaviour is different is the DHA 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;

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 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 fibres are slower conducting, and they connect the association and prefrontal areas. Thicker and fast conducting**

fibres 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 centre 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 colour** 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 fibres create the reduction after long period of time die stress response.

Vagas 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 fibres 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 and fibres and supporting cells due to stress response. Resulting in the reduction of white matter and reduction. Creating the behaviour 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 fibres 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 fibres. 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 behaviour 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. Reduction 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 kainite 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 Quisqualis receptor) is an ionotropic transmembrane receptor for **glutamate** that mediates **fast (the speed)** synaptic transmission in the central **nervous system** (CNS).

Kainite receptors, or **kainic acid receptors** (KARs), are ionotropic **receptors** that respond to the neurotransmitter glutamate. ... Presynaptic **kainite 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 kainite receptors have been shown to have is to several neurological diseases and conditions. KAR expression and distribution have 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 fibres 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 behaviours and personality development in the prefrontal cortex during punning in puberty.

Neonatal Abstinence Syndrome...As the embryo develops in the womb and

the corpus callus 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**). Fibers weaken and damage and element. 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 behaviour pool. as behaviour regarding cognitive, social and behaviour traits... just again different strengths of it. so, to speak. Pruning 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.

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

Phytoestrogens are **oestrogen's** 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 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... **clone** (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 **estrogenic + 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 primary neurons in the hormones react with the growth hormones.

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

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

In trauma through abuse and neglect.

Girls;

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

Boys;

Outward behaviour= 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 behaviour 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.

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 a decrease in white matter.... white matter abnormality predominated in the anterior corpus callosum

Estrogenic receptors located on the cell membrane can interact with a variety of cellular processes to induce rapid neuronal and behavioural change. can regulate behaviour through rapid nongenomic mechanisms or through slower genomic mechanisms

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

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

the presence of estrogenic-like compounds (both naturally occurring and man-made) in diet can have a dramatic impact on the behavioural effects of estrogenic signalling

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**, **women tending to be friend and tend, instead of working on themselves.**

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 caused by the stress hormones

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 behaviours, 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 top down 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 **behaviour**. 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 **behaviour**

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 outcomes

Hippocampal shrinkage also **damages** cognitive functions and interferes with the process of creating memories, which has a profound impact on both **behaviour** 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)

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 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. Dual autism and ADHD then going into other disorders in puberty and late teens/20s

and like goes without saying that reactive attachment disorder at the early stages of the disorder presents the mixed behaviour 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

The Maths: during the Myelination process

shock to the process= stress hormones then released causing an imbalance and the primary male or female growth hormones and IGF1-1 both seek to fix the fault in pattern= leading to a reduction to c/c and decrease in white matter foaming through under production and damaged and weakened axonal fibres. And fault in synaptic connections and supporting neurons patterns in other supporting areas of the brain.

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

**Undercoating of myelin = (due to reduction in cell density in glial cell in myelin sheaths.)
reduction in Myelin basic protein (MBP)**

(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) weak or damaged neurons and synaptic connections in stress response... then affecting the neurons in the prefrontal cortex

Glial cell supporting cell is creating a fault in the cell pattern when the stress occurs in pruning. And enhanced brain development (cell density reduction due to the fault)

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)

Damage occurring over a prolonged period of stress. The greater the reduction, the longer and more extreme the stress response was.

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 elated.

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 and stress hormones. All working together as the brain goes through extreme enhanced growth during this period +GH +IGF1-1

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 dyslexic and creativity are closely linked.

what we see is reduction in brain areas as the stress response (stress hormones) 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.

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 that's abused become dissociation attachment... and the effects would be different.... the older the child was... and that's why we see creativity levels higher and problem solving. That's kind of cool... so then as the c/c develops... the effects to it change with the age of the child... and that's why we see these kids having good problem-solving skills... the foundations are already there. and that's why we see the deficits in facial expression reading and dyslexia. Cause of the stress hormones and the response.

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 is 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 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;** caused by over production in puberty of hormone; enhanced by the over production of growth hormones. (Decrease in the white matter 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 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. 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 grey 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

Link to advanced puberty and childhood trauma.

Link between pruning stage + drug taking i.e. 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 effect; 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 behaviour.... 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 **foetal** brain where it is thought to act as a shield to protect against the harmful actions of cortisol.

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

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

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 foetal Development stages that are the natural body clock targets nature creates.

So, what we are seeing is the reaction the foetal 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 foetal brain. What is interesting is the fact development continues after birth as the structural build of the corpus is not yet in completion stage.

Now we have seen in research that the corpus.

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)

stress hormones 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 isn't 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 jab's babies 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 foetal 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 behavioural pathway deficits in autism. The corpus needs the hormone to build its gateway to what will be the cognitive circuit. the hormones are 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 behaviour.

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 glycyrrhizin 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

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 $\geq$ 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 *foetal* brain-pituitary-adrenal axis in late gestation

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 foetal 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 behavioural symptoms to autism spectrum disorder.

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 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 labelling of bipolar... and if the child is unaware, it is happening ... will go into stress... hormones are all over place... so behaviour 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.

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=

Hormones and behaviour 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.

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 hormone 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. Ie the menopause in women. One can be also diagnosed with the bi poler 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.

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

Disorders to include the following:

***Autism**

(In trauma pathways only.)

(enzyme 11β-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 foetal 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 hormones has triggered.

The mutation of HSD11B2 type 2 being in the foetal 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 through the mother's stress hormones after birth in breast milk. > mother's stress hormone's always having a dominant effect. Mothers' hormones after birth being readjusting to the rush of required hormones to create the foetal life.

Cell division and mutation of the HSD 11 B2 being caused by Estrogenic >Environmental cues such as photoperiod can modulate whether oestrogens activate **nongenomic** or genomic pathways.

Through **non genomic** pathways>

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

> **kainite** 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-6 polyunsaturated ... 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... 24 Apr 2019 - PDF | Phytoestrogens have structures that are very similar to endogenous 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

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Genetic Association of 11 β -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