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Archaeal Digoxin and Regulation of Speech

Global warming induces a genomic change in humans. Global warming induces endosymbiotic archaeal and RNA viroidal growth. The porphyrins form a template for the formation of RNA viroids, DNA viroids, prions, isoprenoids and polysaccharides. They can symbiose together to form primitive archaea. The archaea can further induce HIF alpha, aldose reductose and fructolysis resulting in further porphyrinogenesis and archaeal self replication. The primitive archaeal DNA is integrated along with RNA viroids which are converted to their corresponding DNA by the action of redox stress induced HERV reverse transcriptase into the human genome by the redox stress induced HERV integrase. The archaeal DNA sequences that are integrated into the human genome forms endogenous archaeal human genomic sequences akin to HERV sequences and can function as jumping genes regulating genomic DNA flexibility. The integrated endogenous genomic archaeal sequences can get expressed in the presence of redox stress forming endosymbiotic archaeal particles which can function as a new organelle called the archaeaons. The archaeaon can express the fructolytic pathway constituting an organelle called the fructosome, cholesterol catabolic pathway and digoxin synthetic forming an organelle called the steroidelle, the shikimic acid pathway forming an organelle called the neurotransminoid, antioxidant vitamin E and vitamin C synthetic organelle called the vitaminocyte as well as the glycosaminoglycan synthetic organelle called glycosaminoglycoid. The archaeaon secreting RNA viroids is called the viroidelle. The archaeal symbiosis and digoxin secretion modulates the evolution of human speech. Speech evolves in its primitive form in the Neanderthal population and it reached its fully flowered form in the homo sapiens. Speech disorders are thus related to neanderthalisation of the species.

The increase in endogenous EDLF, a potent inhibitor of membrane Na^+-K^+ ATPase, can decrease this enzyme activity. The results showed increased endogenous EDLF synthesis as evidenced by increased HMG CoA reductase

activity, which functions as the rate limiting step of the isoprenoid pathway. Studies in our laboratory have demonstrated that EDLF is synthesized by the isoprenoid pathway. The endosymbiotic archaeal sequences in the human genome get expressed by redox stress and osmotic stress of global warming. This results in induction of HIF alpha which will upregulate fructolysis and glycolysis. In the setting of redox stress all glucose gets converted to fructose by the induction of enzymes aldose reductase and sorbitol dehydrogenase. Aldose reductase converts glucose to sorbitol and sorbitol dehydrogenase converts sorbitol to fructose. Since fructose is preferentially phosphorylated by ketohexokinases the cell is depleted of ATP and glucose phosphorylation comes to a halt. Fructose becomes the dominant sugar that is metabolized by fructolysis in expressed archaeal particles in the cell functioning as organelle called fructosoids. The fructose is phosphorylated to fructose 1-phosphate which is acted upon by aldolase B which converts it into glyceraldehyde 3-phosphate and dihydroxy acetone phosphate. Glyceraldehyde 3-phosphate is converted to D1,3-biphosphoglycerate which is then converted to 3-phosphoglycerate. The 3-phosphglycerate is converted to 2-phosphoglycerate. 2-phosphoglycerate is converted to phosphoenol pyruvate by the enzyme enolase. Phosphoenol pyruvate is converted to pyruvate by the enzyme pyruvic kinase. The archaeaon induces HIF alpha which upregulates fructolysis and glycolysis but inhibits pyruvate dehydrogenase. The forward metabolism of pyruvate is stopped. The dephosphorylation of phosphoenol pyruvate is inhibited in the setting of pyruvic kinase inhibition. Phosphoenol pyruvate enters the shikimic acid pathway where it is converted to chorismate. The shikimic acid is synthesized by a pathway starting from glyceraldehyde 3-phosphate. Glyceraldehyde 3-phosphate combines with the pentose phosphate pathway metabolite sedoheptulose 7-phosphate which is converted to erythrose 4-phosphate. The pentose phosphate pathway is upregulated in the presence of

the suppression of glycolytic pathway. Erythrose 4-phosphate combines with phosphoenol pyruvate to generate shikimic acid. Shikimic acid combines with another molecule of phosphoenol pyruvate to generate chorismate. The chorismate is converted to prephenic acid and then to parahydroxy phenyl pyruvic acid. Parahydroxy phenyl pyruvic acid is converted to tyrosine and tryptophan as well as neuroactive alkaloids. The shikimic acid pathway is structured in expressed archaeaon organelle called the neurotransminoid. The fructolytic intermediates glyceraldehydes 3-phosphate and pyruvate are the starting points of the DXP pathway of cholesterol synthesis. Glyceraldehyde 3-phosphate combines with pyruvate to form 1-deoxy D-xylulose phosphate (DOXP) which is then converted to 2C methyl erythritol phosphate. 2C methyl erythritol phosphate can be synthesized from erythrose 4-phosphate a metabolite of the shikimic acid pathway. DXP combines with MEP to form isopentenyl pyrophosphate which is converted to cholesterol. Cholesterol is catabolised by archaeal cholesterol oxidases to generate digoxin. The digoxin sugars digitoxose and rhamnose are synthesized by the upregulated pentose phosphate pathway. Glycolytic suppression leads to upregulation of the pentose phosphate pathway. The expressed archaeaon organelle concerned with cholesterol catabolism and digoxin synthesis is called the steroidelle. The suppression of glycolysis and stimulation of fructolysis results in upregulation of the hexosamine pathway. Fructose is converted to fructose 6-phosphate by ketohexokinases. The fructose 6-phosphate is converted to glucosamine 6-phosphate by the action of glutamine fructose 6-phosphate amidotransferase (GFAT). Glucosamine 6-phosphate is converted to UDP N-acetyl glucosamine which is then converted to N-acetyl glucosamine and various amino sugars. UDP glucose is converted to UDP D-glucuronic acid. UDP D-glucuronic acid is converted to glucuronic acid. This forms the uronic acid synthetic pathway. Uronic acids and hexosamines form repeating units of glycosaminoglycans. In

the setting of glycolytic suppression and fructolytic metabolism fructolysis leads to increase synthesis of hexosamines and GAG synthesis. The GAG synthesizing archaeaon particles are called the glycosaminoglycoids. The expressed archaeaon particles are capable of synthesizing antioxidant vitamin C and E. The UDP D-glucose is converted to UDP D-glucuronic acid. UDP D-glucuronic acid is converted to D-glucuronic acid. D-glucuronic acid is converted to L-gulonate by enzyme aldoketoreductases. L-gulonate is converted to L-gulonolactone by lactonase. L-gulonolactone is converted to ascorbic acid by the action of archaeal L-Gulo oxidase. The vitamin E is synthesized from shikimate which is converted to tyrosine and then to parahydroxy phenyl pyruvic acid. Parahydroxy phenyl pyruvic acid is converted to homogentisate. Homogentisate is converted to 2-methyl 6-phytyl benzoquinone which is converted to alpha tocopherol. 2-methyl 6-phytyl benzoquinone is converted to 2,3-methyl 6-phytyl benzoquinone and gamma tocopherol. Vitamin E can also be synthesized by the DXP pathway. Glyceraldehyde 3-phosphate and pyruvate combined to form 1-deoxy D-xylulose 5-phosphate which is converted to 3-isopentenyl pyrophosphate. 3-isopentenyl pyrophosphate and dimethyl allyl pyrophosphate combined to form 2-methyl 6-phytyl benzoquinone which is converted to tocopherols. The ubiquinone another important membrane antioxidant and part of the mitochondrial electron transport chain is synthesized by the shikimic acid pathway and DXP pathway. The isoprenoid moiety of ubiquinone is contributed from the DXP pathway and the rest of it by tyrosine catabolism. The tyrosine is generated by the shikimic acid pathway. The archaeaon particles concerned with the synthesis of vitamin C, vitamin E and ubiquinone which are all antioxidants are called the vitaminocyte.

Dyslexia and delay in development of the speech motor milestone is common in children. The left brain is a verbal and logical controlling half while the right is the non-verbal, practical and intuitive side. In most people's brain, language

is processed mainly in the left hemisphere. People also have a language area in their right hemisphere which is smaller and much less efficient in processing and organizing language based skills. 95% of right handers have their language centre in the left or dominant brain. Many now believe that dyslexic difficulties could arise when language areas are split more evenly between the two halves of the brain. A dyslexic brain is postulated to have more messages to be passed from one hemisphere to the other. It is as if a confusing traffic jam of signals builds up in the corpus callosum between the language areas in the opposite sides of the brain complicating a dyslexics understanding of verbal or written speech.

The neurotransmitter concerned with speech is more likely to be dopamine as dopamine agonist has been used in the treatment of aphasias. Clinical trial using bromocryptine have demonstrated significant effects in aphasia patients. It was therefore considered pertinent to assess the neurotransmitter patterns in: (1) dyslexic, (2) children with delayed and early onset speech milestone, (3) patients with early and delayed recovery from aphasias. The hypothalamus produces an endogenous membrane Na^+-K^+ ATPase inhibitor digoxin which can regulate neurotransmitter especially dopamine transport. It was therefore considered pertinent to study the digoxin status in these groups of patients. Digoxin, being a steroidal glycoside is synthesized by the isoprenoid pathway. Therefore the isoprenoid pathway was also be assessed in these individuals.

The results of the study were as follows:

(1) The results showed that HMG CoA reductase activity, serum digoxin and dolichol were increased and serum ubiquinone, RBC membrane Na^+-K^+ ATPase activity and serum magnesium reduced in dyslexia, late onset of speech milestone and delayed recovery from global aphasia. The results also showed that HMG CoA reductase activity, serum digoxin and dolichol were decreased and serum ubiquinone, RBC membrane Na^+-K^+

ATPase and serum magnesium increased in early onset of the speech milestone and early recovery from global aphasia.

(2) The results showed that the concentration of tryptophan, quinolinic acid, serotonin, strychnine and nicotine was found to be higher in the plasma of patients with dyslexia, late onset of speech milestone and delayed recovery from global aphasia while that of tyrosine, dopamine, norepinephrine and morphine was lower. The results showed that the concentration of tryptophan, quinolinic acid, serotonin, strychnine and nicotine was found to be lower in the plasma of patients with early onset of speech milestone and early recovery from global aphasia while that of tyrosine, dopamine, norepinephrine and morphine was higher.

(3) The results show an increase in the concentration of serum total GAG, carbohydrate components of glycoproteins (hexose, fucose and sialic acid) and serum glycolipids (cerebroside, ganglioside and glycosyl-diglyceride) in delayed onset of speech milestone, dyslexia and delayed recovery from global aphasia. The increase in the carbohydrate components - total hexose, fucose and sialic acid - in delayed onset of speech milestone, dyslexia and delayed recovery from global aphasia was not to the same extent in all cases suggesting qualitative change in glycoprotein structure. The pattern of change in individual GAG in the serum was different, however heparan sulphate (HS) and chondroitin sulphates (ChS) increased in delayed onset of speech milestone, dyslexia and delayed recovery from global aphasia. The activity of GAG degrading enzymes (beta glucuronidase, beta N-acetyl hexosaminidase, hyaluronidase and cathepsin-D) and that of glycohydrolases (beta galactosidase, beta fucosidase and beta glucosidase) showed significant increase in the serum in delayed onset of speech milestone, dyslexia and delayed recovery from global aphasia. The results show a decrease in the concentration of serum total GAG, carbohydrate

components of glycoproteins (hexose, fucose and sialic acid) and serum glycolipids in early onset speech milestone and early recovery from global aphasia. The decrease in the carbohydrate components - total hexose, fucose and sialic acid - was not to the same extent in early onset speech milestone and early recovery from global aphasia suggesting qualitative change in glycoprotein structure. The pattern of change in individual GAG in the serum was different, however heparan sulphate (HS) chondroitin sulphates (ChS) decreased in early onset speech milestone and early recovery from global aphasia. The activity of GAG degrading enzymes (beta glucuronidase, beta N-acetyl hexosaminidase, hyaluronidase and cathepsin-D) and that of glycohydrolases (beta galactosidase, beta fucosidase and beta glucosidase) showed significant decrease in the serum early onset speech milestone and early recovery from global aphasia.

(4) The cholesterol: phospholipid ratio of the REC membrane is increased in dyslexia, late onset of speech milestone and delayed recovery from global aphasia. The concentration of total GAG, hexose and fucose content of glycoproteins increased in the serum and decreased in the RBC membrane in dyslexia, late onset of speech milestone and delayed recovery from global aphasia. The cholesterol: phospholipid ratio of the RBC membrane decreased in early onset speech milestone and early recovery from global aphasia. The concentration of total GAG, hexose and fucose content of glycoproteins decreased in the serum and increased in the RBC membrane in early onset speech milestone and early recovery from global aphasia.

(5) The results showed that HMG CoA reductase activity serum digoxin and dolichol were increased and ubiquinone reduced in left-handed/right hemispheric dominant individuals. The results also showed that HMG CoA reductase activity serum digoxin and dolichol were decreased and ubiquinone increased in right-handed/left hemispheric dominant

individuals. The results showed that the concentration of tryptophan, quinolinic acid serotonin, strychnine and nicotine was found to be higher in the plasma of left handed / right hemispheric dominant individuals while that of tyrosine, dopamine, morphine and norepinephrine was lower. The results also showed that the concentration of tryptophan, quinolinic acid serotonin, strychnine and nicotine was found to be lower in the plasma of right handed / left hemispheric dominant individuals while that of tyrosine, dopamine, morphine and norepinephrine was higher.

Discussion

Archaeal Digoxin and Membrane Na^+-K^+ ATPase Inhibition in Relation to Disorders of Speech

The archaeaon steroidelle contributes to lipid synthesis and metabolism. The archaeaon steroidelle DXP pathway and the upregulated pentose phosphate pathway contributes to digoxin synthesis. The increase in endogenous digoxin, a potent inhibitor or membrane Na^+-K^+ ATPase, can decrease this enzyme activity in delayed recovery from aphasia, delayed onset of speech milestones and dyslexia. In all the disorders studied, there was significant inhibition of the RBC membrane Na^+-K^+ ATPase and this inhibition appears to be a common feature. There was also increased digoxin synthesis as evidenced by increased HMG CoA reductase activity. Studies in our laboratory have demonstrated that digoxin is synthesized by the isoprenoid pathway. The inhibition of Na^+-K^+ ATPase by digoxin is known to cause an increase in intracellular calcium resulting from increased Na^+-K^+ exchange, increased entry of Ca^{++} via the voltage gated calcium channel and increased release of Ca^{++} from intracellular endoplasmic reticulum Ca^{++} stores. This increase in intracellular Ca^{++} by displacing Mg^{++} from its binding sites causes a decrease in the functional availability of Mg^{++}. This decrease in the availability of Mg^{++} can cause

decreased mitochondrial ATP formation, which along with low Mg^{++} can cause further inhibition of Na^{+}-K^{+} ATPase, since the ATP-Mg^{++} complex is the actual substrate for this reaction. Cytosolic free calcium is normally buffered by two mechanisms, ATP dependent calcium extrusion from cell and ATP dependent sequestration of calcium within the endoplasmic reticulum. The Mg^{++} related mitochondrial dysfunction results in defective calcium extrusion from the cell. There is thus a progressive inhibition of Na^{+}-K^{+} ATPase activity first triggered by digoxin. Low intracellular Mg^{++} and high intracellular Ca^{++} consequent to Na^{+}-K^{+} ATPase inhibition appear to be crucial to the pathophysiology of these disorders. The intracellular positive Ca^{++} signal and negative Mg^{++} signal can regulate diverse cellular process. Ca^{++} on entry into the cell is used to charge up the internal endoplasmic reticulum stores which then release a burst of signal calcium responsible for activating a large variety of calcium dependent cellular processes. The information processing capability of the calcium signalling system is enhanced by amplitude and frequency modulation. The Ca^{++} is released from channels on internal ER individually or in small groups (blip/quark and puffs/sparks). Further diversity of calcium signalling is produced by compartmentalization as cytosolic calcium signal and nuclear calcium signal. Serum Mg^{++} was assessed in all these disorders and was found to be reduced.

Archaeal Digoxin and Regulation of Neurotransmitter Synthesis and Function in Relation to Disorders of Speech

The archaeaon neurotransminoid shikimic acid pathway contributes to tryptophan and tyrosine synthesis and catabolism generating neurotransmitters and neuroactive alkaloids. The isoprenoid pathway was assessed in patients recovering from global aphasia, dyslexic individuals and in children with delayed and early attainment of speech milestones. Dopamine is the neurotransmitter most commonly associated with the neuronal networks related

to speech. Dopamine agonist especially bromocryptine has been used in the treatment of aphasias. There is an increase in tryptophan and its catabolites and a reduction in tyrosine and its catabolites in the serum of patients with delayed onset of speech milestone, dyslexia and delayed recovery from global aphasia. This could be due to the fact that digoxin can regulate the neutral amino acid transport system with preferential promotion of tryptophan transport over tyrosine. The decrease in membrane Na^+-K^+ ATPase activity in all the disorders studied could be due to the fact that the hyperpolarising neurotransmitters (dopamine, morphine and noradrenaline) are reduced and the depolarising neuroactive compounds (serotonin, strychnine, nicotine and quinolinic acid) are increased. Dopamine is the neurotransmitter concerned with speech. Low dopamine synthesis consequent to the low tyrosine levels can lead on to delayed onset of speech milestone and delayed recovery from global aphasia. It could also play a part in the genesis of dyslexia.

In the right hemisphere chemically dominant hyperdigoxinemic state there is upregulated serotoninergic, cholinergic, strychninergic, nicotinergic and glutamatergic transmission and downregulated dopaminergic, glycinergic, morphinergic and noradrenergic transmission. Those individuals with right hemispheric chemical dominance develop dyslexia and have delayed onset of the speech milestone as well as delayed recovery from global aphasia.

Archaeal Digoxin and Regulation of Golgi Body / Lysosomal Function in Relation to Disorders of Speech - The Glycosaminoglycoid

The archaeaon glycosaminoglycoid and fructosoid contributes to glycoconjugate synthesis and catabolism by the process of fructolysis. The pattern of glycoconjugate metabolism in dyslexia, delayed recovery from global aphasia and delayed onset of the speech milestone is the same. The elevation in the level

of dolichol may suggest its increased availability of N-glycosylation of proteins. Magnesium deficiency can lead on to defective metabolism of sphinganine, producing its accumulation, which may lead to increased cerebroside and ganglioside synthesis. In Mg^{++} deficiency the glycolysis, citric acid cycle and oxidative phosphorylation are blocked and more glucose 6-phosphate is channelled for the synthesis of glycosaminoglycans (GAG). Intracellular Mg^{++} deficiency also results in defective ubiquitin dependent proteolytic processing of glycoconjugates as it requires Mg^{++} for its function. The increase in the activity of glycohydrolases and GAG degrading enzymes could be due to reduced lysosomal stability and consequent leakage of lysosomal enzymes into the serum. The increase in the concentration of carbohydrate components of glycoproteins and GAG inspite of increased activity of many glycohydrolases may be due to their possible resistance to cleavage by glycohydrolases consequent to qualitative change in their structure. Proteoglycan complexes formed in the presence of altered Ca^{++}-Mg^{++} ratios intracellularly may be structurally abnormal and resistant to lysosomal enzymes and may accumulate.

The alteration in the sulphated proteoglycan matrix of the synaptic vesicles can alter neurotransmitter release into the synapse and produce a defect in dopaminergic transmission contributing to a speech defect in dyslexia, delayed onset of the speech milestone and delayed recovery from global aphasia. Membrane Na^{+}-K^{+} ATPase inhibition can lead to defective notch signalling. Notch is a transmembrane protein that acts as a signal receptor and is important in neurogenesis. Neuronal growth by extending neurites and forming connections is regulated by the notch signalling pathway. The notch signalling inhibits extension of neurites and keeps them stable in the mature brain. A notch ligand known as delta regulates neurogenesis by binding to notch in membranes of embryonal cells and prevents them from developing along the neuronal pathway. Notch activation by the ligand causes notch to be cleaved releasing the

notch intracellular domain. This then passes in to the nucleus and activates transcription as part of the DNA binding complex with CSL protein. Intracellular cleavage of the notch is regulated by presenilin and also depends upon the lysosomal protease. In the presence of a lysosomal instability consequent to defective lysosomal membranes notch cleavage by protease is defective contributing to defective synaptic connectivity in dyslexias as well as delayed recovery from global aphasia and delayed onset of the speech milestone. Altered glycoproteins, glycolipids and GAG of the neuronal membrane can also contribute to disordered synaptic connectivity in these disorders.

The protein processing defect can result in defective glycosylation of endogenous neuronal glycoprotein antigens with consequent defective formation of the MHC class-1 neuronal glycoprotein antigen complex. The MHC linked peptide transporter, a P-glycoprotein which transports the MHC-antigen complex to the antigen presenting cell surface, has an ATP binding site. The peptide transporter is dysfunctional in the presence of Mg^{++} deficiency. This results in defective transport of the MHC class-1 neuronal glycoprotein antigen complex to the antigen presenting cell surface for recognition by the CD_4 or CD_8 cell. The MHC glycoproteins are involved in formation of synaptic connectivity during neuronal development. Defective formation and presentation of the MHC class-1 neuronal glycoprotein complex can lead on to disordered synaptic connectivity and contribute to dyslexia as well as delayed onset of the speech milestone.

Archaeal Digoxin and Alteration in Membrane Structure and Membrane Formation in Relation to Disorders of Speech

The archaeaon steroidelle, glycosaminoglycoid and fructosoid contribute to cell membrane formation synthesizing cholesterol by the DXP pathway and glycosaminoglycans by fructolysis. The alteration in the isoprenoid pathway

specifically, cholesterol as well as changes in glycoproteins and GAG can affect cellular membranes. The upregulation of the isoprenoid pathway can lead to increased cholesterol synthesis and Mg^{++} deficiency can inhibit phospholipid synthesis. Phospholipid degradation is increased owing to increase in intracellular calcium activating phosplipase A_2 and D. The cholesterol phospholipid ratio of the RBC membrane was increased in patients with dyslexia, delayed onset of speech milestone and delayed recovery from global aphasia. The concentration of total GAG, hexose and fucose of glycoprotein decreased in the RBC membrane and increased in the serum suggesting their reduced incorporation into the membrane and defective membrane formation. The glycoproteins, GAG and glycolipids of the cellular membrane are formed in the endoplasmic reticulum, which is then budded of as a vesicle which fuses with the golgi complex. The glycoconjugates are then transported via the golgi channel and the golgi vesicle fuses with the cell membrane. This trafficking depends upon GTPases and lipid kinases which are crucially dependent on magnesium and are defective in Mg^{++} deficiency. The change in membrane structure produced by alteration in glycoconjugates and the cholesterol: phospholipid ratio can produce changes in the conformation of Na^{+}-K^{+} ATPase resulting j further membrane Na^{+}-K^{+} ATPase inhibition. The same changes can affect the structure of the organelle membrane. This results in defective lysosomal stability and leakage of glycohydrolases and GAG degrading enzymes into the serum. Altered neuronal membranes can lead on to defective synaptic connectivity contributing to delayed onset of the speech milestone, dyslexia and delayed recovery from global aphasia.

Archaeal Digoxin and Immunoregulation in Relation to Disorders of Speech - The Fructosoid, Steroidelle and Viroidelle

The archaeaon fructosoid contributes to fructolysis and immune activation. Fructose can contribute to induction of NFkB and immune activation. The

archaeaon steroidelle synthesized digoxin induces NFkB producing immune activation. Membrane Na^+-K^+ ATPase inhibition can produce immune activation and is reported to increase CD4/CD8 ratios as exemplified by the action of lithium. Increased intracellular calcium activates the calcium dependent calcineurin signal transduction pathway, which can produce T-cell activation and secretion of interleukin-3, 4, 5, 6 and TNF alpha. TNF alpha binds to its receptor TNFR1 and activates the transcription factors HFkB and AP-1 leading to the induction of proinflammatory and immunomodulatory genes. This can also explain the immune activation in dyslexia. TNF alpha can also bring about apoptosis of the cell. It binds to its receptor and activates caspase-9, an ICE protease which converts IL-1 beta precursor to IL-1 beta. IL-1 beta produces apoptosis of the neurons. The increased intracellular calcium and ceramide related opening of the mitochondrial PT pore also leads to volume dysregulation of the mitochondria causing hyperosmolality of the matrix and expansion of the matrix space. The Outer membrane of the mitochondria ruptures and releases apoptosis inducing factor and cytochrome C into the cytoplasm. This results in activation of caspase-9. Caspase-9 can produce apoptosis of the cell. Apoptosis has been implicated in neuronal degeneration. Apoptosis can produce defective synaptogenesis and synaptic connectivity contributing to dyslexia, delayed onset of the speech milestone and delayed recovery from global aphasia.

Archaeal Induced Hyperdigoxinemic State and Hemispheric Dominance in Relation to Disorders of Speech

The archaeaon related organelle - steroidelle, neurotransminoid and vitaminocyte contribute to hemispheric dominance. In children with early onset of the speech milestone, and in patients with early recovery from global aphasia the patterns were reversed. The patterns were similar to those obtained in left

hemispheric chemical dominance with a downregulated isoprenoid pathway, reduced digoxin and dolichol levels, increased RBC membrane Na^+-K^+ ATPase activity, increased serum magnesium, reduced serum glycoconjugates and reduced membrane cholesterol: phospholipid ratios. The tryptophan and its catabolites were reduced while tyrosine and its catabolites principally dopamine were increased. All these parameters contribute to early onset of the speech milestone and early recovery from global aphasia.

Thus hemispheric chemical dominance plays a crucial role the genesis of dyslexia onset of the speech milestone and recovery from global aphasia. Hemispheric chemical dominance has no strict correlation with handedness. The pattern of right hemispheric chemical dominance leads to dyslexia, delayed onset of the speech milestone and delayed recovery from global aphasia. On the other hand the pattern of left hemispheric chemical dominance leads to early onset of the speech milestone and early recovery from global aphasia.

References

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