

The Influence of Pathologies upon Sensory Perception and Sensory Coordination in Children with Developmental Dyslexia and Learning Disorders: A Unified Theory of Developmental Dyslexia

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Abstract

This case is presented to explain that developmental dyslexia and related autistic spectrum disorders have solely pathological origins. There is a general consensus of opinion which supports the phonological theory. However, this largely ignores the biological basis for all aspects of the brain's development and function, and hence, for its dysfunction. A unified explanation must take into account all salient features including cognitive dysfunction, encephalograph (EEG) frequencies, neural networks, physiological systems, autonomic nervous system and the function of the cerebellum. It must explain the significance of the brain waves and neurons and their normally synchronized or coherent function. This article builds upon an earlier article by the authors, which incorporates a review and discussion of the prevailing theories or models for developmental dyslexia. It looks at the issues from a top-down 'systems biology' perspective. It concludes that it may be only the body's biochemistry and, in particular, the onset of pathologies that explain the phenomena which we recognize as developmental dyslexia. Pathologies experienced in the early prepubescent years influence neural development. They influence the speed and coherent transmission of data between the senses and neural centers. It is proposed that this explains the nature and occurrence of what we recognize as developmental dyslexia.

Keywords: Autistic spectrum disorder, Autonomic nervous system, Cognition, Developmental dyslexia, Encephalograph frequencies, Physiological systems

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Introduction

The difficulties diagnosing the existence of autistic spectrum disorders (ASDs)^[1] is increasingly recognized.^[2-4] The absence of a precise understanding of the condition and of reliable diagnostic tests, prevents a proper understanding of the condition. Current dyslexia research fails to consider the influence of pathology, yet every organ is solely biochemical.

There is no evidence to support any claims for improved educational standards.^[5] There is no mechanism which distinguishes between the ability of teachers to teach and children to learn. The prevalence of dyslexia has increased significantly since the mid-1970s. Arguably, the cognitive abilities of the developed world are in decline. Learning problems are increasingly prevalent. In the US 16% of children are diagnosed with learning dysfunction and circa 2M children are being prescribed Methyl Phenidate to aid their concentration; 25% of grade 12 adolescents cannot read at a proficient level^[6] and in the UK circa 20% of the adult population has significant problems with literacy and numeracy. The occurrences of ASDs are no longer exceptional.

Any explanation for developmental dyslexia (DD) should seek to explain, for example:

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DOI:

10.4103/1947-2714.93878

1. DD develops in the pre-pubescent child. It becomes increasingly difficult to treat or manage dyslexia when the child has progressed beyond puberty; therefore, its occurrence is associated with pre-pubescent physiology and is fixed by post-pubescent physiologies. The pathologies which develop during early childhood influence the development and/or function of the neural structures including the transmission of data through the magnocellular and parvocellular pathways. DD cannot be due to any single factor.^[3]
2. Sense perception and coordination can be altered by pathologies, by drugs and experiences such as low gravity during space flight. ASDs which influence learning abilities are influenced by the brain's biology, neural development and associated processes which regulate its function, rather than any significant failure of its structures.
3. Many dyslexic children may have excellent speech, hearing, visual perception, IQ, memory and/or balance. DD affects children differently, and exists in different levels of severity and influences different aspects of cognitive functions and is not necessarily an impediment to success. Some very successful people have Dyslexia. They may be good with numbers, be creative, motivated, have an active life, be in good health and/or they might simply have a high IQ. This indicates that, for many, the condition is more associated with the brain's coordinated function rather than its structures.^[7]
4. In some autistics the symptoms regress whilst the child has a fever. This indicates that ASDs are influenced by the processes which regulate the body's function.
5. The existence of prosopagnosia and/or synaesthesia illustrate, how the sense perception and sensory coordination are disrupted by genetic factors which influence protein expression.^[8]
6. Genetic changes have been linked to abnormalities in the brain development and of the associated cognitive deficits.^[9]
7. The body's physiology and regulation of its function is linked to different encephalograph (EEG) frequencies^[10] and how this influences the system function and/or the regulation or release of biochemicals in the ANS.
8. Neurons interact in functionally coherent groups or patterns which are involved in the fixation and recall of memories.

The Genetic/Epigenetic Component

In general, developmental dyslexia is not an inheritable genetic condition,^[11] though some dyslexic children may inherit the condition because their parents also have a similar condition.^[12] This may not

be genetic, for example, if the parents lead a sedentary lifestyle then the child is also likely to have the same lifestyle. It will influence their genetic and epigenetic profile (in ways which differ from child to child) but, in many, it may be reversible if addressed in the pre-pubescent years.

The gene is a dynamic entity which influences its environment through its expression of proteins. It also responds to environmental influences,^[13,14] for example, someone with type 2 diabetes will have an altered gene expression profile. However, if the same person was to undertake an exercise program their gene expression profile will return to its pre-morbid state. Nutrition and exercise alter the gene expression profile; stress alters the gene expression profile and suppresses immune function; and viruses and vaccines alter the gene expression profile, suppress immune function and introduce foreign proteins. Any alteration to gene expression (genotype) and the ability of such proteins to react (phenotype) will ultimately be manifested as pathology.

The Significance of EEG Frequency and Rhythm

An encephalograph monitors the electrical activity of the brain. Although the EEG is a widely used tool in medical research, its link to underlying neural activity during the processing of sensory data from the external and internal environments, remains poorly understood. It measures the electrical fluctuations of ionic current which flows within the neuronal structures, which are influenced by the prevailing biochemistries (proteins, pH and levels of minerals, etc.), and which also influence the functions of neurons and neuronal networks. These electrical fluctuations are expressed in terms of frequency and amplitude. There are four significant frequency bands: Beta, alpha, theta and delta. Each is perceived to have a cognitive or biological significance. An EEG measures the total synchronized activity of many thousands of neurons. Variations known as sleep spindles and spikes reflect underlying neuronal and related biochemical processes. The main clinical applications are the diagnosis of epilepsy, coma, encephalopathies, and confirmation of brain death.

Whilst there is a perceived link between EEG frequencies and the neuronal networks, it is also recognized that there is a poor level of understanding of this relationship. Some networks appear to be better understood than others, for example, the resonant frequency involving the thalamus and cerebral cortex which is associated with sleep spindles.^[15] Furthermore, the delta frequency plays a role in the consolidation of memories.^[16] There is, therefore, an established link

between EEG frequencies and neural networks which plays a role in the consolidation of memories and the learning process(es).

The body's biochemistry and the prevailing EEG frequencies exist in a dynamic equilibrium.^[10] Exposure to flashing lights can cause photosensitive migraine whilst selected flashing lights are used to treat migraine. Biochemical extremes influence the prevailing EEG frequency(s), and hence, the ability to fix and recall memories, concentrate and ultimately influence the ability of the body to function. Omega-3 fatty acid supplements lessen the symptoms of dyslexia and improve the behavior of children^[17] whilst stimulants raise EEG frequencies, energy levels and lower concentration. By contrast, elevated levels of ethyl alcohol or blood glucose are associated with lowered EEG frequencies, concentration and increased predisposition to sleep.

The body's multi-level function is linked to the prevailing EEG frequencies.^[10] In the case of DD the following associations with EEG frequencies have been recorded: Delta activity,^[18] delta and theta,^[19] alpha,^[20] beta^[21] and gamma EEG activity.^[22] The brain records and reacts to the physiological significance of an event by using frequency i.e. in the case of most severe trauma and to recuperate (delta); when experiencing pain or stress (theta); violence, stress and/or significant achievements (alpha). That the delta and theta frequencies act at the physiological level is proven by a number of precedents e.g. For example:

1. Delta and theta are active throughout the 24 h period whereas gamma, beta and alpha are mainly active when awake;
2. Growth and repair are significantly enhanced during the periods of sleep (predominantly delta);
3. Lack of sleep (predominantly delta) increases morbidity and mortality;
4. Sleep is a physiological system.^[23]

The development of pathologies, the introduction of foreign proteins and the consequence of exposure to viruses and/or through vaccination, alters the delicate equilibrium which exists between biochemistry and EEG frequency(s) and also alters the function and coordination of the senses, for example, influencing visual perception. In the most extreme case(s) this will manifest as a pathological functional system where the brain seeks out the best-fit solution in order to most effectively maintain the body's physiological stability.^[24]

The EEG reflects the speed of neuronal processing i.e. the regulation of a physiological significant system is structured to make slow adjustments, for example, at the delta (1-4 hz) or theta frequencies (4-8 hz), by comparison with the rapid adjustments which are required to handle

cognitive input (30-60 hz) and subsequent behavioral responses.

The effects of nicotine in smokers, decreases delta and theta frequencies and increases high alpha and beta frequencies. It increases the speed of the information being processed.^[25,26]

1. Both hearing and visual systems handle sensory input through magnocellular and parvocellular pathways^[27] which influence color perception, visual contrast, sound frequency and loudness.
2. There is a relationship between color perception and the autonomic nervous system.^[28]
3. Magnocellular pathways process information more quickly than the parvocellular pathways.^[29,30]
4. Magnocellular processing deficits have been found throughout the neuro-visual system.^[31]
5. Magnocellular and parvocellular processing is linked to color perception and visual contrast but must also be linked to the function of the brain.^[32]
6. Alterations of blood glucose levels before and after meals are associated with increased predisposition to sleep and lowered levels of concentration. It alters the speed and significance of the information being processed.

This indicates that visual perception, in particular those deficits associated with DD, is associated with the speed of neural processing, the ANS and prevailing pathologies.

That speech is rhythmic is significant because the ears do not work in a continuous mode. Rhythm helps the brain to identify the nature of the word (e.g., whether a word comprises of two, three, four or more sounds). The perception of words is not formed from a combination of sounds. It is based upon the first and last sounds in each word.^[33] This is the way that a child forms language i.e. of ever increasing complexity but from the simplest origins. A young child will often shorten a word, for example, 'Cin-der-ella' may be shortened to 'Cin-ella'. Moreover, sounds can be in and out of phase. If such phasing, or speed of processing is distorted, a dyslexic child may miss part of a word or have difficulty discerning the spoken word from background noise.^[34] It explains why some children with dyslexia have difficulty keeping the beat in music.^[35]

Instability in the dynamic relationship involving frequency and biochemistry manifests in different ways, for example, the difficulty keeping the beat in music, altered vocal spectrum, poor sleep, poor fixation and recall of memories, etc.

There are various light and sound-based therapies which are used to treat ASDs.^[36] In the treatment of DD flashing

light biofeedback therapies are used although they are experiential, controversial and often lack evidence of effectiveness.^[37]

The EEG frequencies are linked to the regulation of the physiological systems.^[10] This explains the apparently synchronized function of the brain and neural networks. The idea that the learning process requires synchronized neural activity is not new,^[38,39] however, this has excluded consideration of a link between the brain, sensory organs and visceral matrix. It illustrates how flashing lights can induce photosensitivity and also that the frequency and colors used in flashing light therapies can be selected with therapeutic effect, for example, in the treatment of hyperactivity,^[40] dyslexia,^[41] ASDs,^[42-44] tinnitus, hypertension, asthma, diabetes mellitus, migraine and sleep apnoea, etc. There is a principle, which contemporary medical research and/or educational psychology has yet to explain.^[10,41]

Neurons interact in coherent groups.^[45,46] However, the nature and significance of their function, coordinating interactions between the prefrontal cortex and hippocampus,^[47] has only recently been recognized. This is significant because the pre-frontal cortex regulates or controls the body's "executive functions."^[48,49] The orthodox opinion considers that this area is associated with 'the coordination and synthesis of emotions, thinking, memory and body or physical movement'. In addition the brain uses frequency to organize the activity of these "functionally coherent groups of neurons."^[50]

Motor neurons receive signals from the neural centers. These signals initiate the contractions which alter the function of the muscles and glands. There is a significant connection between neuronal function and the ANS. What we consider as "executive functions" resembles "the functional or physiological systems." It is the neural regulation of the physiological systems and ANS which influence emotions, thought processes, the establishment and recall of memories and the function of the body and/or physical movement.

The nature of this relationship i.e. between cognition, the neural networks, physiological systems, EEG frequencies and ultimately with cellular and molecular biochemistry,^[51,52] has been incorporated into a mathematical model and technology.^[10,41]

Pathological Precedents

Regulating physiological systems is necessary to ensure the supply of oxygenated blood to the body and brain, to regulate the immune system and to maintain the health and function of the inner and middle ear. Antibiotics and other medications^[53] reduce or eliminate ear infections

such as Otitis Media,^[54] although, the condition often appears to re-occur.

There is an unresolved link between cognition and cellular and molecular biology.^[55] Cognitive dysfunction may be the inevitable consequence of extremes of multi-sensory input which manifests as pathologies.

A lack of exercise or poor diet degrades the condition of the muscle(s) in the heart and other organs and influences the levels of minerals which are necessary for proper metabolic function and the metabolism of blood glucose. This leads to problems with neural blood flow in later life, often manifested as pathologies, for example, diabetes, cardiovascular disease, hypertension, atherosclerosis, etc.

Exercise is associated with educational achievement.^[56] It improves neural blood flow and stimulates the biochemistries which support neuron and synapse formation.^[57] Exercise^[58] and a balanced diet^[59] in children reduce the occurrence of DD. Diet influences the supply of nutrients to the brain. It influences the condition of the neural structures, and the ability to memorize and concentrate. For example:

1. Acidic (and/or alcoholic) drinks alter the natural metabolic processes. Different enzymes (isozymes) metabolize alcohol at lower pH. The metabolites alter lipid metabolism and increase weight.^[60]
2. The prevailing pH influences the levels and bioavailability of minerals for example, the levels of sodium, potassium, magnesium and calcium,^[61] which are necessary to facilitate the flow of ions across neuronal membranes. This influences the action potentials, rates of neuronal firing, and ultimately the fixation of memories.
3. Low levels of iron influences the delivery of oxygen to the neural tissues^[62] and affects attention and concentration.
4. Low levels of magnesium influences the metabolism of blood glucose.^[63] This alters EEG frequencies, predisposition to sleep and concentration.
5. Sensorineural hearing loss has been associated with hypothyroidism.^[64] This is significant because the endocrine glands are integral aspects of the body's function.
6. Omega-3-fatty acids^[59,65] are implicated in a range of neurodevelopmental disorders including ADD, developmental dyslexia and ASDs. Such substrates are sources of unsaturated fatty acids and are essential for neural development and function. The use of omega-3 fatty acid supplements is associated with health improvements:
7. The use of Methyl Phenidate to improve concentration indicates that developmental dyslexia has biochemical origins.

8. Higher levels of blood glucose increases blood viscosity and lowers immune function, and hence influences the function of the sensory organs.
9. Dietary Protein influences the fixation of memories.^[66]
10. Cognitive dysfunction is a commonly observed feature of (i) drugs,(ii) diseases and (iii) viral infections, for example, in herpes simplex, hepatitis C, enteroviruses and Epstein-Barr virus, etc. Visual and Phonological deficits are a common feature of diabetes mellitus,^[67] heart disease(s), migraine, alzheimer's disease, multiple sclerosis, DD, regressive autism, tetanus, ADD^[68] and viral infections, for example, mumps, measles, rubella, pneumococcal meningitis.

Such observations indicate that the understanding of DD must include the structure of the brain and its biological function, because they are mutually dependent. Poor diet,^[69] lack of exercise and/or the influence of stress (ors), particularly in early childhood, influences the development and function of the neural structures which process sensory input, the fixation and recovery of memories and concentration etc.

The Relationship between Cognition and Pathology

The body glows with energy^[70] which is emitted as heat and light. Light^[71] is a fundamental requirement for many biochemical processes. Many proteins and enzymes are visually active. Light is absorbed and/or emitted in many protein reactions.^[72] Pathologies influence the spectrum of light emitted. This bioluminescence influences visual perception and can be measured.

Proteins exist in different multi-level conformations. However, they need to be activated in order to react with their substrates. If the temperature, pH or their associated biochemistries are not sustained at an optimum level this will influence protein conformation. Many diseases are linked to the degree of coiling or uncoiling of proteins. Biophotons of light deliver the energy which activates proteins and specific biochemistries; regulates the function of the physiological systems;^[10,71] and synchronizes the activity of groups of neurons^[73] and their electrical impulses.^[74]

The prevailing understanding of color perception is linked to mutations in specific genes. Any epigenetic factors which, directly or indirectly, influence the structure of chromosome and genes will influence protein expression. However, it is the reaction of proteins and substrates which influence color perception. Proteins have the necessary structures and energetic characteristics to release light as they react. Accordingly the shifts in color perception which are often associated with DD are associated with the onset of pathologies.

It is also plausible that sound can be used to deliver a therapeutic effect although this may be limited by the phasic way in which the ears function. The delivery of sound and music, at appropriate frequencies, have a coherent effect upon the brain waves and our sensory perception.^[10] Indeed, the steady deep rhythms now employed in contemporary music such as Shamanic music or aboriginal chanting and dancing, have an unexplained physiological effect. By contrast, the experiential selection of frequencies can be expected to have a hit-and-miss, and/or negative effect.

Problems with the inner ear inevitably influence balance and the auditory spectrum i.e. the loudness level and also the ability to discern the spoken word. This has consequences for memory and learning. If the child cannot discern the spoken word, or cannot relate the spoken word to the written word, it will be difficult for them to engage in spoken or written dialogue with their peers. Similar outcomes will be observed in the cases of children who have the symptoms of 'moving words' when reading (a symptom occasionally noted in patients with trigeminal neuralgia) and/or who have untypically low levels of concentration. Problems with balance are associated with the function of the cerebellum in which the Purkinje cells receive information from all parts of the body. The cerebellum is a significant organ, assimilating multi-sensory information from the senses and visceral organs. It contains more neurons than the rest of the brain. Accordingly, balance will be affected without the coherent feed-forward of sensory and visceral data to the cerebrum by the cerebellum i.e. to transfer sensory coordinates into motor coordinates. This enables the cerebrum to activate the muscles required to stabilize balance and coordination.^[75]

Memories are stored as the biochemical correlate of sensory input.^[76] The body uses the significance and importance of memories as a template against which it can gauge its current or future predisposition and abilities. There is a link between physiology and memory. It is the prevailing physiology which determines the continuous reconstruction of the neural structures and fixation of memories – from the simplest memories borne before, during and after birth; and of ever more complex interneuronal structures which build up throughout life. If the brain recorded only the memory of every event, the physiological correlate would not have any meaning. It is only by correlating the new memory to past memory(s) that it becomes significant.^[77]

The body's function is ultimately attributable to the rate at which proteins are expressed by genes, the rate at which proteins react with their substrates (and their outcomes), and the factors which influence the rates of their reaction. There is no part of the body's structure or

function which does not have a biochemical nature. This includes its sensory and cognitive abilities. The receipt of sensory input and the subsequent fixation of memories are biochemical processes.^[78]

Cognitive deficits are the consequence of underlying pathology(s). The fixation of memories is associated with the prevailing biochemistries. Memories are laid down from birth as a biochemical data matrix of ever increasing levels of complexity, linked to the different neural centres which store and process memories, until the body's metabolism is no longer able to sustain the creation and function of neurons, axons and synapses and of associated memories; and/or the body's metabolism is no longer able to sustain the function of existing neural structures. Every memory has a biochemical context. Consequently, the stimulation of past memories, for example, of musical memories, may have a therapeutic effect by its association with the memory of the body's internal health.^[79]

The body's function and the regulation of the physiological systems is significant and is frequency dependent. It is only by understanding the multi-level significance of the EEG frequencies that dyslexia and ASDs can be managed.^[10,80,81]

Conclusions

Current dyslexia research is focussed upon the qualitative and quantitative determination of the extent of cognitive dysfunction in those with impaired learning and DD. However, there is little research which examines the pathological origins. In order to diagnose a condition i.e. of cognitive or medical dysfunction, it is necessary to understand the fundamental nature of the condition. Without such understanding, any methods used for its diagnosis or treatment will have inherent limitations. This is the case with DD. There can be little doubt that pathologies have a significant influence upon cognition. Moreover pathologies encountered in the pre-pubescent stage are likely to alter physiological development. This article illustrates that pathologies are associated with visual and auditory perception and coordination, and the ability to memorize and concentrate. It supports the earlier article(s) by Ewing, Ewing and Parvez which indicates that the brain's coordinated function, in particular the activity of neurons or the synchronized activity of groups of neurons, are required to facilitate and coordinate the sensory processing and memorizing of information, which is required as part of the learning process. It indicates that this process requires synchronized activity between the brain, sensory organs, ANS (and visceral organs) and limbs.

Pathologies influence the nature and speed of transmission of data from the senses to the brain. It illustrates how,

in the pre-pubescent child, the coordinated effect of the factors which influence genotype and phenotype (such as stress, lack of outdoor exercise, poor diet, exposure to viruses, and the insidious effect of vaccines i.e. factors which contribute to lowered immune function) alter the body's physiological stability and contribute to varying degrees of sensory dysfunction, in particular, a lack of coordination between the senses arising from the influence of pathologies upon the speed of processing, involving the magnocellular and parvocellular pathways. The brain looks to establish physiological stability. If this is not achievable it looks for a 'best-fit' and selects the most appropriate set of circumstances, involving frequency, which enables it to stabilize the body's function. This explains why dyslexia becomes a chronic condition - although relatively benign from the physiological perspective. It also explains how frequency can be adapted with therapeutic effect.

It indicates that the range of genetic and phenotypic influences which manifest as DD are too complex to be attributed to any single genetic cause i.e. that epistasis influences the complexity of our gene expression profile. Multiple gene-gene interactions influence the expression of proteins and ultimately their manifestation as pathologies.^[82,83]

This paper is based upon a developed technology which is based upon a mathematical model of the relationship between visual perception, and the ANS.^[3,52] This model incorporates a revised understanding of the nature and significance of the physiological systems. The argument presented as a unified explanation for developmental dyslexia appears consistent with the theme of various reference papers-published by Galaburda, Rosen, Stein, Baron-Cohen, Fields RD, Richardson, Habib, and others -many of which appear in the references section of this article.

Acknowledgement

We thank the many researchers who through their work have contributed to this article.

References

1. Skuse DH, Mandy W, Steer C, Miller LL, Goodman R, Lawrence K, *et al.* Social communication competence and functional adaptation in a general population of children: Preliminary evidence for sex-by-verbal IQ differential risk. *J Am Acad Child Adolesc Psychiatry* 2009;48:128-37.
2. Elliott JG, Gibbs S. Does dyslexia exist? *J Philos Educ* 2008;42:475-91.
3. Ewing GW, Ewing EN, Parvez SH. Developmental dyslexia: The link with the autonomic nervous system and the physiological systems. *Biogenic Amines* 2009;23:115-90.
4. Breteler MH, Arns M, Peters S, Giepmaqns I, Verhoeven L. Improvements in spelling after QEEG-based neurofeedback

- in dyslexia: A randomized controlled treatment study. *Appl Psychophysiol Biofeedback* 2010;35:5-11.
5. Green D, de Waal A, Cackett B. Education: Better results and declining standards? Online Briefing, 2002. Available from: <http://www.civitas.org.uk/pdf/educationBriefingDec05.pdf>. [Last accessed on 2011 Oct 18].
 6. National Assessment of Educational Progress 2009. Available from: http://nationsreportcard.gov/math_2009/index_alt.asp. [Last accessed on 2011 Oct 18].
 7. Fletcher JM. Dyslexia: The evolution of a scientific concept. *J Int Neuropsychol Soc* 2009;15:501-8.
 8. Asher JE, Lamb JA, Brocklebank D, Cazier JB, Maestrini E, Addis L, *et al*. A whole-genome scan and fine-mapping linkage study of auditory-visual synesthesia reveals evidence of linkage to chromosomes 2q24, 5q33, 6p12, and 12p12. *Am J Hum Genet* 2009;84:279-85.
 9. Galaburda AM, LoTurco J, Ramus F, Fitch RH, Rosen GD. From genes to behavior in developmental dyslexia. *Nat Neurosci* 2006;9:1213-7.
 10. Ewing GW. A theoretical framework for photosensitivity: Evidence of systemic regulation. *J Comput Sci Syst Biol* 2009;2:287-97.
 11. Schulte-Körne G, Warnke A, Remschmidt H. Genetics of dyslexia. *Z Kinder Jugendpsychiatr Psychother* 2006;34:435-44.
 12. Berninger V, Richards T. Inter-relationships among behavioral markers, genes, brain and treatment in dyslexia and dysgraphia. *Future Neurol* 2010;5:597-617.
 13. Strohmman RC. Genetic determinism as a failing paradigm in biology and medicine: Implications for health and wellness. *J Soc Work Educ* 2003;39:169-91.
 14. Booth FW, Lees SJ. Fundamental questions about genes, inactivity, and chronic diseases. *Physiol Genomics* 2007;28:146-57.
 15. Ferrarelli F, Huber R, Peterson MJ, Massimini M, Murphy M, Riedner BA, *et al*. Reduced sleep spindle activity in schizophrenia patients. *Am J Psychiatry* 2007;164:483-92.
 16. Tamminen J, Payne JD, Stickgold R, Wamsley EJ, Gareth Gaskell M. Sleep spindle activity is associated with the integration of new memories and existing knowledge. *J Neurosci* 2010;30:14356-60.
 17. Schuchardt JP, Huss M, Stauss-Grabo M, Hahn A. Significance of long-chain polyunsaturated fatty acids (PUFAs) for the development and behaviour of children. *Eur J Pediatr* 2010;169:149-64.
 18. Penolazzi B, Spironelli C, Angrilla A. Delta EEG activity as a marker of dysfunctional linguistic processing in developmental dyslexia. *Psychophysiology* 2008;45:1025-33.
 19. Arns M, Peters S, Breteler M, Verhoeven L. Different brain activation patterns in dyslexic children: Evidence from EEG power and coherence patterns for the double-deficit theory of dyslexia. *J Integr Neurosci* 2007;6:175-90.
 20. Rothenberger A, Moll GH. Standard EEG and dyslexia in children-new evidence for specific correlates? *Acta Paedopsychiatr* 1994;56:209-18.
 21. Ackerman PT, Dykman RA. Reading-disabled students with and without comorbid arithmetic disability. *Dev Neuropsychol* 1995;11:351-71.
 22. Fell J, Fernandez G, Klaver P, Elger CE, Fries P. Is synchronized neuronal gamma activity relevant for selective attention? *Brain Res Rev* 2003;42:265-72.
 23. Ewing GW. Mathematical modeling the neuroregulation of blood pressure using a cognitive top-down approach. *North Am J Med Sci* 2010;2:341-52.
 24. Sudakov KV. The basic principles of the general theory of functional systems. *Medicine* 1987;S:26-49.
 25. Sahakian B, Jones G, Levy R, Gray J, Warburton D. The effects of nicotine on attention, information processing, and short-term memory in patients with dementia of the Alzheimer type. *Br J Psychiatry* 1989;154:797-800.
 26. Stough C. Nicotine and information processing: Recent studies. In: Koga V, Nagata K, Hirata K, editors. *Brain topography today*. Amsterdam: Elsevier; 1998. p. 799-802.
 27. Livingstone MS, Hubel DH. Psychophysical evidence for separate channels for the perception of form, color, movement, and depth. *J Neurosci* 1987;7:3416-68.
 28. Kravkov SV. Color vision and the autonomic nervous system. *J Opt Soc Am* 1941;31:335-42.
 29. Maunsell J, Gibson J. Visual response latencies in striate cortex of the macaque monkey. *J Neurophysiol* 1992;68:1332-44.
 30. Schiller P, Malpeli J. Functional specificity of lateral geniculate nucleus laminae of the rhesus monkey. *J Neurophysiol* 1978;41:788-97.
 31. Livingstone MS, Rosen GD, Drislane FW, Galaburda AM. Physiological and anatomical evidence for a magnocellular defect in developmental dyslexia. *Proc Natl Acad Sci U S A* 1991;88:7943-7.
 32. Hofer H, Carroll J, Neitz J, Neitz M, Williams DR. Organization of the human trichromatic cone mosaic. *J Neurosci* 2005;25:9669-79.
 33. Ucles P, Mendez M, Garay J. Low-level defective processing of non-verbal sounds in dyslexic children. *Dyslexia* 2009;15:72-85.
 34. Wagner RK, Torgesen JK, Rashotte CA. *Comprehensive Test of Phonological Processing (CTOPP)*. Pub: PRO-ED, Austin; 1999.
 35. Thomson JM, Goswami U. Rhythmic processing in children with developmental dyslexia: Auditory and motor rhythms link to reading and spelling. *J Physiol Paris* 2008;102:120-9.
 36. Chung JC, Lai CK. Snoezelen for dementia. *Cochrane Database Syst Rev* 2002;CD003152.
 37. Liddle E, Jackson G, Jackson S. An evaluation of a visual biofeedback intervention in dyslexic adults. *J Dyslexia* 2005;11:61-77.
 38. Petsche H. Brainwave coherence. *Music Percept* 1993;11:117-51.
 39. Baron-Cohen S, Harrison J, Goldstein L, Wyke M. Coloured speech perception: Is synaesthesia what happens when modularity breaks down? *Perception* 1993;22:419-26.
 40. Tansey MA, Bruner RL. EMG and EEG biofeedback training in the treatment of a 10-year-old hyperactive boy with a developmental reading disorder. *Biofeedback Self Regul* 1983;8:25-37.
 41. Vysochin Yu V, Lukoyanov VV, Yaichnikov IK, Tkachuk MI, Chyev VA, Yemelyanenko VV, *et al*. The research of the universal computer medical-diagnostic and prophylactic program "Virtual Scanner" efficiency (1) for the establishment of the Russian Federation "Health of the Nation" program and (2) for the invigoration of different population groups. 2003. English translation available from: <http://www.montague-diagnostics.co.uk>. [Last accessed on 2011 Oct 18].
 42. Coben R. Connectivity-guided neurofeedback for autistic spectrum disorder. *Biofeedback* 2007;35:131-5.
 43. Jarusiewicz B. Efficacy of neurofeedback for children in the autistic spectrum: A pilot study. *J Neurother* 2002;6:39-49.
 44. Scolnick B. Effects of electroencephalogram biofeedback with Asperger's syndrome. *Int J Rehabil Res* 2005;28:159-63.
 45. Hebb D. *The Organization of Behavior: A Neuropsychological Theory*. New York: Pub. Wiley; 1949.

46. Fields RD. Beyond the neuron doctrine. *Sci Am Mind* 2006;17:20-7.
47. Jones MW, Wilson MA. Theta rhythms coordinate hippocampal-prefrontal interactions in a spatial memory task. *PLoS Biol* 2005;3:e402.
48. Caine G, Caine RN. Meaningful learning and the executive functions of the human brain. In: Johnson S, Taylor K, editors. *The Neuroscience of Adult Learning*. Josssey-Bass; 2006. p. 53-62.
49. Hoeft F, McCandliss BD, Black JM, Gantman A, Zakerani N, Hulme C, *et al.* Neural systems predicting long-term outcome in dyslexia. *Proc Natl Acad Sci U S A* 2011;108:361-6.
50. Canolty RT, Ganguly K, Kennerley SW, Cadieu CF, Koepsell K, Wallis JD, *et al.* Oscillatory phase coupling coordinates anatomically dispersed functional cell assemblies. *Proc Natl Acad Sci U S A* 2010;107:17356-61.
51. Uhlhaas PJ, Singer W. Neural synchrony in brain disorders: relevance for cognitive dysfunctions and pathophysiology. *Neuron* 2006;52:155-68.
52. Ewing GW, Ewing EN. Cognition, the autonomic nervous system and the physiological systems. *J Biogenic Amines* 2008;22:140-63.
53. Narula AA, Bradley PJ. Glue ear: The new dyslexia? *Br Med J (Clin Res Ed)* 1985;291:411-2.
54. Hartley DE, Moore DR. Effects of otitis media with effusion on auditory temporal resolution. *Int J Pediatr Otorhinolaryngol* 2005;69:757-69.
55. Kandel E. The new science of mind. *Sci Am Mind* 2006;17:62-7.
56. Hillman CH, Erickson KL, Kramer AF. Science and society: Be smart, exercise your heart: Exercise effects on brain and cognition. *Nat Rev Neurosci* 2008;9:58-65.
57. Pereira AC, Huddleston DE, Brickman AM, Sosunov AA, Hen R, McKhann GM, *et al.* An *in vivo* correlate of exercise-induced neurogenesis in the adult dentate gyrus. *Proc Natl Acad Sci U S A* 2007;104:5638-43.
58. Hillman CH, Pontifex MB, Raine LB, Castelli DM, Hall EE, Kramer AF. The effect of acute treadmill walking on cognitive control and academic achievement in pre-adolescent children. *Neuroscience* 2009;159:1044-54.
59. Richardson AJ. Omega-3 fatty acids in ADHD and related neurodevelopmental disorders. *Int Rev Psychiatry* 2006;18:155-72.
60. Zakhari S. Overview: How is alcohol metabolized by the body? *Alcohol Res Health* 2006;29:245-53.
61. Tucker KL, Morita K, Qiao N, Hannan MT, Cupples LA, Kiel DP. Colas, but not other carbonated beverages, are associated with low bone mineral density in older women: The Framingham Osteoporosis Study. *Am J Clin Nutr* 2006;84:936-42.
62. Petranovic D, Batinac T, Petranovic D, Ruzic A, Ruzic T. Iron deficiency anaemia influences cognitive functions. *Med Hypotheses* 2008;70:70-2.
63. Rodriguez-Moran M, Guerrero-Romero F. Oral magnesium supplementation improves insulin sensitivity and metabolic control in type 2 diabetic subjects. *Diabetes Care* 2003;26:1147-52.
64. Thornton AR, Jarvis SJ. Auditory brainstem response findings in hypothyroid and hyperthyroid disease. *Clin Neurophysiol* 2008;119:786-90.
65. Carpentier YA, Peltier S, Portois L, Sebedio JL, Leverve X, Malaisse WJ. Rapid reduction of liver steatosis in omega3-depleted rats injected with a novel lipid emulsion. *Horm Metab Res* 2008;40:875-9.
66. Scharf MT, Woo NH, Lattal KM, Young JZ, Nguyen PV, Abel T. Protein synthesis is required for the enhancement of long-term potentiation and long-term memory by spaced training. *J Neurophysiol* 2002;87:2770-7.
67. Kurtenbacha A, Schiefera U, Neub A, Zrennera E. Preretinopic changes in the colour vision of juvenile diabetics. *Br J Ophthalmol* 1999;83:43-6.
68. Roessner V, Banaschewski T, Fillmer-Otte A, Becker A, Albrecht B, Uebel H, *et al.* Color perception deficits in co-existing attention-deficit/hyperactivity disorder and chronic tic disorders. *J Neural Transm* 2008;115:235-9.
69. Jenkins DJ, Chiavaroli L, Wong JM, Kendall C, Lewis GF, Vidgen E, *et al.* Adding monounsaturated fatty acids to a dietary portfolio of cholesterol-lowering foods in hypercholesterolemia. *CMAJ* 2010;182:1961-7.
70. Kobayashi M, Kikuchi D, Okamura H. Imaging of ultraweak spontaneous photon emission from human body displaying diurnal rhythm. *PLoS One* 2009;4:e6256.
71. Ewing GW. There is a need for an alternative or modified medical paradigm incorporating an understanding of the nature and significance of the physiological systems. *North Am J Med Sci* 2010;2:1-6.
72. Shimomura O, Chalfie M, Tsien R. The Nobel Prize in Chemistry 2008. Available from: http://nobelprize.org/nobel_prizes/chemistry/laureates/2008/chalfie-lecture.html. [Last Accessed on 2010 May 26].
73. Cox RH, Shealy CN, Cady RK, Liss S. Pain Reduction and Relaxation with Brain Wave Synchronization (Photo-Stimulation). *J Neurol Orthop Med Surg* 1996;17:32-4.
74. Bower B. Perception may dance to the beat of collective neuronal rhythms. *Sci News* 1998;153:120.
75. Nicolson RI, Fawcett AJ, Berry EL, Jenkins IH, Dean P, Brooks DJ. Association of abnormal cerebellar activation with motor learning difficulties in dyslexic adults. *Lancet* 1999;353:1662-7.
76. Pellionisz A, Llinas R. Space-time representation in the brain. The cerebellum as a predictive space-time metric tensor. *Neuroscience* 1982;7:2949-70.
77. Oberman LM, Pineda JA, Ramachandran VS. The human mirror neuron system: A link between action observation and social skills. *Soc Cogn Affect Neurosci* 2007;2:62-6.
78. Lynch G, Baudry M. The biochemistry of memory: A new and specific hypothesis. *Science* 1984;224:1057-63.
79. Bennet D, Bennet A. Engaging tacit knowledge in support of organizational learning. *VINE* 2008;38:72-94.
80. Coben R, Clarke AR, Hudspeth W, Barry RJ. EEG power and coherence in autistic spectrum disorder. *Clin Neurophysiol* 2008;119:1002-9.
81. Levesque J, Beauregard M, Mensour B. The effect of neurofeedback training on the neural substrates of selective attention in children with attention deficit/hyperactivity disorder: A functional MRI study. *Neurosci Lett* 2006;394:216-21.
82. Berninger VW, Raskind W, Richards T, Abbott R, Stock P. A multidisciplinary approach to understanding developmental dyslexia within working-memory architecture: Genotypes, phenotypes, brain, and instruction. *Dev Neuropsychol* 2008;33:707-44.
83. Moore JH. The ubiquitous nature of epistasis in determining susceptibility to common human diseases. *Hum Hered* 2003;56:73-82.

How to cite this article: Ewing GW, Parvez SH. The influence of pathologies upon sensory perception and sensory coordination in children with developmental dyslexia and learning disorders: A unified theory of developmental dyslexia. *North Am J Med Sci* 2012;4:109-16.

Source of Support: Nil. **Conflict of Interest:** Graham Ewing is a Director of Montague Healthcare, a company devoted to the future commercialisation of Virtual Scanning.