

BioInfoPhysics Models of Neuronal Signal Processes Based on Theories of Electromagnetic Fields

Kang Cheng and Chang Hua Zou

Department of BioMedical InfoPhysics, Science Research Inst., 205 Hana Road, Edison NJ 08817, USA

Abstract: Problem statement: The aphasia is one of human language and action related brain associative diseases. The mechanisms of the diseases and the brain association are still unclear. In this study, we proposed our models of the neuronal signal processes, in a view of BioInforPhysics, to understand the mechanisms. **Approach:** Our models are based on today's solidest Electromagnetic Fields (EMF) theoretic fundamentals: Maxwell EMF equations, Poynting theorem and vector, Lorentz law and other well known EMF principles, as well as published biomedical data. Methods cover the signal collections and analysis, correlations and synthesis; the correlations include functions derivatives as well as the functions. **Results:** (a) The signals have three attributes (or elements): the information, the energies and the matters; (b) the fields intensities are the Information Intensities (II), products of the II are the Information Response Intensities (IRI) of energies expressions, products of the II and the matters (charges) are the IRI of forces expressions; (c) the information can produce the new information; (d) the energies can carry or (and) transmit the information; (e) the matters (charges) can store and produce the information. The EMF information is not conservative in biological fluids because of the charges or the attenuation of the II. Our models in this study are the signals oriented and combine the information, the energies and the matters. **Conclusion:** Approximately, neurons work like microcomputers; the synapses work like signal input interfaces and perform the signal collections and analysis; the neuronal bodies work like microprocessors and execute the signal correlations and synthesis in parallel; DNA, RNA, proteins and other cellular components work like memories or circuits; the axons work like signal output interfaces and segregate the signal stream to other neurons. The all processes in the neurons and the nervous system are automatically completed by the natural laws. We intended to approach the natural laws with our models.

Key words: Maxwell, poynting, lorentz, information, energy, matter, correlation, association, analysis, synthesis

INTRODUCTION

The aphasia is one of human language and action related brain associative diseases, similar diseases include dyslexia, anomia, agraphia and dysgraphia (Purves, 2007; Crosson, 2008; Pulvermüller and Berthier, 2008; Conway *et al.*, 2008; Henry *et al.*, 2008; Rapcsak *et al.*, 2009; Timothy *et al.*, 2008). For more than 150 years neurologists, psychologists and linguists have been investigating language disorders caused by strokes and other diseases of the brain (Pulvermüller and Berthier, 2008). To study the mechanisms of the diseases and the brain association, many significant models have been proposed in views of neuroscience (Pineda, 2008), mathematics (computation) or physics (Deco *et al.*, 2008; Brunel and Wang, 2001; Corchs and Deco, 2004; Chizhov and Graham, 2007; Harrison *et al.*,

2005). However, to our knowledge, the mechanisms are still unclear.

Maxwell EMF functions, Poynting theorem and vector, Lorentz law and other well known EMF theories have been successfully applied in various technologies and instrumentations, such as EEG, ECG. However, to our knowledge, all of the theories have not been used to develop models for neuronal signal processes with a combination of the information, the energies and the matters in a view of BioInfoPhysics.

Traditionally and currently, a correlation is described only with an integration of a product of two functions. As far as we know, function's derivatives have not been considered for any correlation.

In one (Cheng and Zou, 2007) of our previous studies, we modeled the memories, the transmission and the recognition of neuronal signals. In another one

(Cheng and Zou, 2006) of our previous studies, we modeled DNA recognition of DNA synthesis and proposed concepts of Information Intensity (II), Information Response Intensity (IRI) and Information Flux (IF).

In this study, we develop, propose and summarize our models of neuronal signal processes with a combination of the information, the energies and the matters in a view of BioInforPhysics. We believe our models will be helpful to understand the feeling, the associating, the thinking and the learning as well as the mechanisms of the human brain associative diseases.

MATERIALS AND METHODS

Based on published biomedical data (Purves, 2007; Timothy *et al.*, 2008), we believe most behaviors of brains and neurons are dependent on distributions and activities of the EMF and charges. We think Maxwell EMF equations, Poynting theorem and vector, Lorentz law and other well known EMF theories are today's solidest fundamentals to develop our models for the distributions and the activities. In this study, we study the distributions and the activities at levels of a neuron and a cortex. We assume: A brain association cortex has totally I neurons, the ith neuron has distributions of the Electric Field (EF) intensity E_i , the Magnetic Field (MF) intensity H_i , the Charge Density (CD) ρ_i and the velocity v_i of ρ_i ; E_i , H_i , ρ_i and v_i are functions of spatial coordinates x, y, z and temporal coordinate t; E_i , H_i and v_i are vectors and ρ_i is a scalar. ϵ , μ and σ are respectively permittivity, permeability and conductivity of the neuronal fluid and they are assumed to be quasi homogenous constants to simplify our models. Therefore, one of Maxwell EMF equations about the EF, the permittivity and the CD in a point form is:

$$\epsilon \nabla \bullet E_i = \rho_i \quad (1)$$

Where:

∇ = Nabla operator

\bullet = Scalar product

Equation 1 means the CD is a flux source of the EF and the EF is not conservative in biological fluids because $\rho_i \neq 0$ strictly. An equation (derived from Maxwell EMF equations) about the conductive EF, the conductivity and the CD in a point form is:

$$\sigma \nabla \bullet E_i = -\frac{\partial}{\partial t} \rho_i \quad (2)$$

Equation 2 means that the temporal changing CD is a flux source of the conductive EF and the charges are

conservative. One of Maxwell EMF equations about the MF and the permeability in a point form is:

$$\mu \nabla \bullet H_i = 0 \quad (3)$$

Equation 3 means the MF doesn't have any magnetic flux source.

Lorentz law describes the EMF Forces (EMFF) and its point form is:

$$\text{EMFF} = \rho_i (E_i + v_i \times \mu H_i) \quad (4)$$

where, \times denotes a vector product. The EF Energy Density (EFED) and the MF Energy Density (MFED) are respectively:

$$\text{EFED} = \epsilon E_i \bullet E_i / 2 \quad (5)$$

and:

$$\text{MFED} = \mu H_i \bullet H_i / 2 \quad (6)$$

Poynting vector represents the EMF Energy Flow Density (EMFEFD) and it is:

$$\text{EMFEFD} = E_i \times H_i \quad (7)$$

For any volume V with a surface S in the neuron, the energy flow in or out of the volume is:

$$\oint_S (E_i \times H_i) \cdot ds = \int_V \nabla \cdot (E_i \times H_i) \cdot dv \quad (8)$$

Equation 8 represents a Poynting theorem in a spatial integral form. Using a vectoring equation (Ramo *et al.*, 1994), we obtain:

$$\nabla \bullet (E_i \times H_i) = H_i \bullet (\nabla \times E_i) - E_i \bullet (\nabla \times H_i) \quad (9)$$

Equation 9 represents a divergence or a flux source of the EMFEFD. One of Maxwell EMF equations is:

$$\nabla \times E_i = -\mu \frac{\partial H_i}{\partial t} \quad (10)$$

Equation 10 means a temporal variable MF is a spatial curl source of an EF, i.e., a temporal changing MF produces a spatial curl EF. The complement of the Eq. 10 is another Maxwell EMF equation:

$$\nabla \times H_i = \left(\sigma E_i + \epsilon \frac{\partial E_i}{\partial t} \right) \quad (11)$$

Equation 17 means a temporal changing CD is a flux source of the conductive EF information. The both Eq. 16 and 17 imply the EF information is not conservative in biological fluids because of the charges. But, the charge matters are conservative and they store and produce the EF information. Using Eq. 3 and 15b, we obtain:

$$\nabla \bullet [H_{i,c} + \sum_m^{iM} (H_{i,a,m} + H_{i,b,m})] = 0 \quad (18)$$

Equation 18 means there is not any magnetic flux source for the MF information. Using Eq. 4 (Lorentz law), 15a and b, we obtain EMF forces of the cellular $\rho_{i,c}$:

$$\begin{aligned} \rho_{i,c}(E_i + v_{i,c} \times \mu H_i) &= \rho_{i,c}(E_{i,c} + v_{i,c} \times \mu H_{i,c}) \\ &+ \rho_{i,c} [\sum_m^{iM} (E_{i,a,m} + E_{i,b,m}) \\ &+ v_{i,c} \times \mu \sum_m^{iM} (H_{i,a,m} + H_{i,b,m})] \end{aligned} \quad (19)$$

Equation 19 means interactions between the charges matters and the fields (EF and MF) information. At right side of the equation, the first term denotes interactions between the neuronal charges and the neuronal fields. The second term denotes interactions between the neuronal charges and the externally produced fields. These interactions are IRI of forces expressions (Cheng and Zou, 2006).

Using equations 5 and 6, 15a and b, we obtain respectively:

$$\begin{aligned} \frac{\epsilon}{2} E_i \bullet E_i &= \frac{\epsilon}{2} [E_{i,c} \bullet E_{i,c} + 2 \sum_m^{iM} E_{i,c} \bullet E_{i,a,m} \\ &+ 2 \sum_m^{iM} E_{i,c} \bullet E_{i,b,m} + \sum_m^{iM} \sum_n^{iM} E_{i,a,m} \bullet E_{i,a,n} \\ &+ 2 \sum_m^{iM} \sum_n^{iM} E_{i,a,m} \bullet E_{i,b,n} + \sum_m^{iM} \sum_n^{iM} E_{i,b,m} \bullet E_{i,b,n}] \end{aligned} \quad (20)$$

$$\begin{aligned} \frac{\mu}{2} H_i \bullet H_i &= \frac{\mu}{2} [H_{i,c} \bullet H_{i,c} + 2 \sum_m^{iM} H_{i,c} \bullet H_{i,a,m} \\ &+ 2 \sum_m^{iM} H_{i,c} \bullet H_{i,b,m} + \sum_m^{iM} \sum_n^{iM} H_{i,a,m} \bullet H_{i,a,n} \\ &+ 2 \sum_m^{iM} \sum_n^{iM} H_{i,a,m} \bullet H_{i,b,n} + \sum_m^{iM} \sum_n^{iM} H_{i,b,m} \bullet H_{i,b,n}] \end{aligned} \quad (21)$$

where, n is an index to denote nth synapse. The both equations represent the information correlations as well as the IRI of the static energy expressions (Cheng and

Zou, 2006) in a point form and mean energies carry the information. We define all terms at right side of Eq. 20 as the auto or cross correlations of the EF information; the cross correlations are interactions of the fields and the auto correlations are auto actions of the fields; e.g., the first term is the neuronal auto correlation; the second and the third terms are the auto or function cross correlations between the neuron and the externals if they have the same motifs or not; the fourth and the last terms are the external auto correlations, the fifth term is the auto or the function cross correlation between the two externals if they have the same motifs or not. We also define the first, the fourth and the last terms as the isogenous because the information comes from the same source, other terms as the heterogenous because the information comes from different sources, an iso-synapse product for the expanded items with $m = n$ because the information comes from the same synapses.

In the same way to define the terms in Eq. 20, we define the terms in Eq. 21, but they are magnetic and involve the permeability. Using Eq. 10, 15a and b, we obtain:

$$\begin{aligned} \nabla \times [E_{i,c} + \sum_m^{iM} (E_{i,a,m} + E_{i,b,m})] &= \\ -\mu \frac{\partial}{\partial t} [H_{i,c} + \sum_m^{iM} (H_{i,a,m} + H_{i,b,m})] \end{aligned} \quad (22)$$

Equation 22 means, the temporal variable MF information is a spatial curl source of the EF information, i.e., the temporal variable MF information produces the new spatial curl EF information. Using Eq. 11, 15a and b, we obtain:

$$\begin{aligned} \nabla \times [H_{i,c} + \sum_m^{iM} (H_{i,a,m} + H_{i,b,m})] &= \sigma \sum_m^{iM} [E_{i,c} + (E_{i,a,m} \\ &+ E_{i,b,m})] + \epsilon \frac{\partial}{\partial t} [E_{i,c} + \sum_m^{iM} (E_{i,a,m} + E_{i,b,m})] \end{aligned} \quad (23)$$

Equation 23 means the conductive or the temporal variable EF information is a spatial curl source of the MF information, i.e., the conductive or the temporal variable EF information produces the new spatial curl MF information. Equation 23 also implies the EMF II is not conservative because of the attenuation of the EMF II in biological fluids. Using Eq. 7, 15a and b, we obtain Eq. 24. We define all terms at right side of Eq. 24 as the fields cross correlations of information between the EF and the MF. The equation represents the IRI of the dynamic energies expressions and means the EMFEFD is equivalent to a sum of the information correlations and the energies carry and transmit the information:

$$\begin{aligned}
 E_i X H_i &= E_{i,c} X H_{i,c} + \sum_m^{iM} E_{i,c} X H_{i,a,m} + \sum_m^{iM} E_{i,c} X H_{i,b,m} \\
 &+ \sum_m^{iM} E_{i,a,m} X H_{i,c} + \sum_m^{iM} E_{i,b,m} X H_{i,c} \\
 &+ \sum_m^{iM} \sum_n^{iM} (E_{i,a,m} X H_{i,a,n}) + \sum_m^{iM} \sum_n^{iM} (E_{i,a,m} X H_{i,b,n}) \\
 &+ \sum_m^{iM} \sum_n^{iM} (E_{i,b,m} X H_{i,a,n}) + \sum_m^{iM} \sum_n^{iM} (E_{i,b,m} X H_{i,b,n})
 \end{aligned} \tag{24}$$

Using Eq. 12, 15a and b, we obtain:

$$\begin{aligned}
 \nabla \bullet (E_i X H_i) &= -\epsilon [E_{i,c} + \sum_m^{iM} (E_{i,a,m} + E_{i,b,m})] \\
 &\bullet \frac{\partial}{\partial t} [E_{i,c} + \sum_m^{iM} (E_{i,a,m} + E_{i,b,m})] \\
 &- \sigma [E_{i,c} + \sum_m^{iM} (E_{i,a,m} + E_{i,b,m})] \\
 &\bullet [E_{i,c} + \sum_m^{iM} (E_{i,a,m} + E_{i,b,m})] \\
 &- \mu [H_{i,c} + \sum_m^{iM} (H_{i,a,m} + H_{i,b,m})] \\
 &\bullet \frac{\partial}{\partial t} [H_{i,c} + \sum_m^{iM} (H_{i,a,m} + H_{i,b,m})]
 \end{aligned} \tag{25}$$

The Eq. 25 represents the IRI of the dynamic energies expressions in a point form too and means to exam the divergence or the flux source of the EMFEFD is equivalent to sum the correlations of the EMF information. The first and the third terms at the right of Eq. 25 respectively represent the EF and the MF information correlations and the energy transformations. We define all of the expanded terms from the first and the third terms in Eq. 25 as temporal derivatives cross correlations between the 0 order of the derivative and the first order of the derivative, of the information function.

The second term at the right of Eq. 25 represents the information correlations as well as the conductive energy dissipation. For the expanded terms from the second term, the definitions of correlations are the same or similar to that for Eq. 20. The definitions and meanings of the isogenous, the heterogenetic and the iso-synapse product for the Eq. 24 and 25 are the same as that of the Eq. 20.

Using Eq. 8 and 25, we can obtain an integral form of information correlations in a spatial domain. In a similar way, we can obtain that in a temporal domain (Table 1). To completely summarize our models, two more neuronal signal processes based on other two Maxwell EMF equations are listed at the end of the Table 1.

Table 1: Neuronal EMF signal processes with a combination of the information, the energies and the matters. The matters are photos. Meanings of m and n are the same as that in the text

Equation of signal process	Information characteristic	Energy characteristic
$E = [E_1, E_2, \dots, E_m, \dots, E_M]$	EF information analysis	
$H = [H_1, H_2, \dots, H_m, \dots, H_M]$	MF information analysis	
$E = \sum E_m$	EF information synthesis	
$H = \sum H_m$	MF information synthesis	
$\epsilon (\sum E_m) \bullet (\sum E_n) / 2$	function correlations of EF and EF	Energy volume density
$\mu (\sum H_m) \bullet (\sum H_n) / 2$	Function correlations of MF and MF.	Energy volume density
$\nabla X \sum E_m = -\mu \frac{\partial}{\partial t} \sum H_m$	Temporal changing MF information produces new spatial curl EF information.	
$\nabla X \sum H_m = \sigma \sum E_m + \epsilon \frac{\partial}{\partial t} \sum E_m$	Conductive or temporal variable EF information produces new spatial curl MF information.	
$(\sum E_m) X (\sum H_m)$	Fields cross correlations of EF and MF.	Energy flow density
$\nabla \bullet [(\sum E_m) X (\sum H_m)]$	Temporal derivative cross correlations of 0 and 1, or 0 and 0 orders and EF and EF or MF and MF.	Energy conservation, point form
$\int \nabla \bullet (\sum E_m) X (\sum H_m) dt$	The same as the above.	Energy conservation, temporal integral form
$\int \nabla \bullet (\sum E_m) X (\sum H_m) dv$	The same as the above.	Energy conservation, spatial integral form
$\nabla \bullet \sum E_m = \rho / \epsilon$	Divergence or flux source of EF information.	
$\nabla \bullet \sum H_m = 0$	For MF information, flush in = flush out	

For an association cortex area with I neurons, there are I neuronal bodies to process the signals in parallel and there are sum (iM) of synapses, where i is from 1 to I, to input the signals.

Another important result is to output the signals. The output ports are axons usually (Purves, 2007). The forces or the energies of the EMF open channels at an axon hillock and segregate the signal streams to other neurons if the forces or the energies are beyond the threshold values. We define an effective output of the EMFEFD as $E_{i,j}XH_{i,j}$ for output port j of neuron i, the fields intensities $E_{i,j}$ and $H_{i,j}$ are the correspondent II of EF and MF respectively. We assume there are totally iJ output ports for the neuron i and define the matrices of the global output II as:

$$[E_i] = [E_{i,1} \dots E_{i,j} \dots E_{i,iJ}] \quad (26)$$

$$[H_i] = [H_{i,1} \dots H_{i,j} \dots H_{i,iJ}] \quad (27)$$

A matrix of a global effective output of EMFEFD is defined as:

$$[E_iXH_i] = [E_{i,1}XH_{i,1} \dots E_{i,j}XH_{i,j} \dots E_{i,iJ}XH_{i,iJ}] \quad (28)$$

The matrices of II output for the cortex are:

$$[E] = [[E_i] \dots [E_i] \dots [E_i]] \quad (29)$$

$$[H] = [[H_i] \dots [H_i] \dots [H_i]] \quad (30)$$

The matrix of the cortical effective output of EMFEFD is defined as:

$$[EXH] = [[E_iXH_i] \dots [E_iXH_i] \dots [E_iXH_i]] \quad (31)$$

Equations 28 and 31 are the IRI of dynamic energies expressions.

Maxwell EMF equations are the fields sources oriented and illustrate the fields' variation. Poynting theorem and vector are the fields energies oriented and elucidate the energies conservation. Lorentz law is the fields forces oriented and determines the moving path. Our models in this study are the signals oriented and combine the three attributes (or elements) of the information, the energies and the matters.

DISCUSSION

The EMF signal attributes (or elements) and relationships of the information, the energies and the matters could be suitable to the Gravitational Field (GF) too. But, the matter is the mass, the II and the IRI are about the GF. To estimate the Gravitational Field (GF) role, we define a Gravitational Field Flux Density (GFFD):

$$GFFD = GFI/G \quad (32)$$

where, GFI and G are the well known GF Intensity (GFI) and gravitation constant respectively. A GF Flux (GFF) is (Cheng and Zou, 2006):

$$GFF = \int GFFD \bullet ds \quad (33)$$

Based on Gauss law, a vectoring equation and a derivation method of EFED (Ramo *et al.*, 1994), we propose a formula of Gravitational Field Energy Density (GFED), $GFED = GFI \bullet GFFD$. Using the formula and published data (Purves, 2007; Ramo *et al.*, 1994), our estimation shows the GFED produced by a neuron is about 10^{34} times weaker than the EFED around the membrane. Therefore, the GF role is ignored compared with that of the EF. Usually and naturally, the EF plays a major role, the MF plays a minor, in neurons (Cheng and Zou, 2007; Cheng and Zou, 2006).

Considering Newtonian mechanics, Einstein's equation of matters and energies in the special relativity and the uncertainty principle in quantum mechanics, we generalize relationships of the information (intensities), the energies, the matters and other important concepts in physics, (Fig. 2), where we define an uncertain product as a product of the uncertain principle.

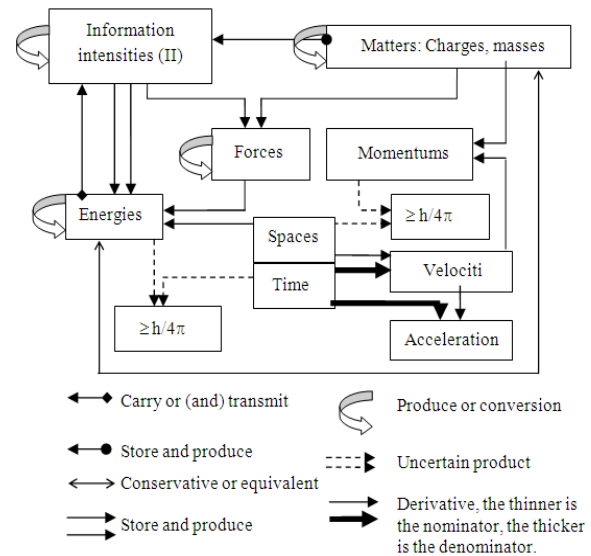


Fig. 2: Relationships of the information (intensities), the energies and the matters of the signals. h is Planck constant. The product means the vector, the scalar or the multiplication product. The uncertain product means the product of the uncertainty principle

The information, the energies and the matters are self producible. The matters store and produce the information. The energies carry or (and) transmit the information. The matters and the energies are conservative and equivalent. The products of the information produce energies. The products of the information and the matters produce the forces.

Though our models are developed with two external signals and for multipolar neurons, we believe the principles of our models are applicable to multiple signals and for the bipolar and the unipolar neurons too and it could be also suitable to the signal processes for other cells, where the roles of the synapses and axons could be replaced by that of other interfaces, such as membrane proteins. If the neurons in an association cortex are ill or damaged, or the signals can not be normally transmitted (input or output), the correlations can not be performed or completed. These problems could cause aphasia disease or other brain associative diseases.

We think, after a neuron receive the external signals, the cellular inherited or inner information, energies or matters interact with the acquired signals (Cheng and Zou, 2007); if the external signals are strong enough, repeat many times, or induce resonances, the interactions could produce new cellular components. The new components could build new (association) memories and (electric) circuits that could play roles in the new neuronal signal processes. The new components could also change the distributions and the activities of the cellular $E_{i,c}$, $H_{i,c}$ and $\rho_{i,c}$ significantly. The new distributions and activities could relate the neuronal learning.

The second term in Eq. 19, the interaction between the neuronal charges and the externally produced fields, could involve the neuronal feeling and sensing, or the simple signal recognition when the external information is from the sensors (Cheng and Zou, 2007). The correlations in Eq. 20-25 could involve the neuronal association, thinking or complex signal cognition and recognition.

Our models imply, the isogenous auto correlations could relate to isogeneous associations, e.g., analysis and synthesis of an event (or object) self; the heterogenetic auto correlations could relate to heterogenetic auto associations, e.g., comparing the same or similar events (or objects) and the heterogenetic cross correlation could relate to heterogenetic cross associations, e.g., conditional responses (reflexes), complex events (or objects) associations. We believe all of the correlations could relate to the thinking. The cortical association area activities could involve multiple signals and multiple

levels of neuronal correlations. The more neurons and the more synapses, the more complicated the associations. Our models also imply:

- The released biological signals could help rehabilitation of patents
- The embryonic and children development of neurons' synapses could influence the development of the ability of association thinking or learning
- EEG δ or α waves could relate to the inner signals and EEG β waves could relate to the external signals
- Both low and high levels of association cortices could involve the working memories. The highest level of association cortices could make decisions by all of the neurons, (Eq. 31)

CONCLUSION

Approximately, neurons work like microcomputers; the synapses work like signal input interfaces and perform the signal collections and analysis; the neuronal bodies work like microprocessors and execute the signal correlations and synthesis in parallel; DNA, RNA, proteins and other cellular components work like memories or circuits; the axons work like signal output interfaces and segregate the signal stream to other neurons. The all processes in the neurons and the nervous system are automatically completed by the natural laws. We intended to approach the natural laws with our models.

ACKNOWLEDGEMENT

The authors thank Miss Vivien Cheng for helpful suggestions and comments for this publication.

REFERENCES

- Brunel, N. and X.J. Wang, 2001. Effects of neuromodulation in a cortical network model of object working memory dominated by recurrent inhibition. *J. Comput. Neurosci.*, 11: 63-85.
- Cheng, K. and C.H. Zou, 2006. Informatics and physics models of recognitions of DNA replication and their biomedical applications. *Am. J. Applied Sci.*, 3: 2059-2062.
- Cheng, K. and C.H. Zou, 2007. Biomedicine and Informatics Model of Alzheimer's disease. *Am. J. Biochem. Biotechnol.*, 3: 145-149.

- Chizhov, A.V. and L.J. Graham, 2007. Population model of hippocampal pyramidal neurons, linking a refractory density approach to conductance-based neurons. *Phys. Rev. E. Stat Nonlinear Soft Matter Phys.*, 75: 011924.
- Conway, T., K.M. Heilman, K. Gopinath, K. Peck and R. Bauer *et al.*, 2008. Neural substrates related to auditory working memory comparisons in dyslexia: An fMRI study. *J. Int. Neuropsychol. Soc.*, 14: 629-639.
- Corchs, S. and G. Deco, 2004. Feature-based attention in human visual cortex: Simulation of fMRI data. *Neuroimage*, 21: 36-45.
- Crosson, B., 2008. An intention manipulation to change lateralization of word production in nonfluent aphasia: Current status. *Semin. Speech Lang.*, 29: 188-200.
- Purves, D., 2007. *Neuroscience*. 4th Edn., Oxford University Press, New York. ISBN: 10: 0878936971, pp: 810.
- Deco, G., V.K. Jirsa, P.A. Robinson, M. Breakspear and K. Friston, 2008. The dynamic brain: From spiking neurons to neural masses and cortical fields. *PLOS. Comput. Biol.*, 4: e1000092.
- Harrison, L.M., O. David and K.J. Friston, 2005. Stochastic models of neuronal dynamics. *Philos. Trans. R. Soc. Lond. B. Biol. Sci.*, 360: 1075-1091.
- Henry, M.L., P.M. Beeson and S.Z. Rapcsak, 2008. Treatment for anomia in semantic dementia. *Semin. Speech Lang.*, 29: 60-70.
- Pineda, J.A., 2008. Sensorimotor cortex as a critical component of an 'extended' mirror neuron system: Does it solve the development, correspondence and control problems in mirroring? *Behav. Brain Funct.*, 4: 47.
- Pulvermüller, F. and M.L. Berthier, 2008. Aphasia therapy on a neuroscience basis. *Aphasiology*, 22: 563-599.
- Ramo, S., J.R. Whinnery and T.V. Duzer, 1994. *Fields and Waves in Communication Electronics*. John Wiley and Sons, New York, ISBN: 10: 0471585513, pp: 864.
- Rapcsak, S.Z., M. Beeson, M.L. Henry, A. Leyden and E. Kim *et al.*, 2009. Phonological dyslexia and dysgraphia: Cognitive mechanisms and neural substrates. *Cortex*, 45: 575-591.
- Timothy, L.H., D.L. Daniel, C.B. Dodrill, G.A., Ojemann *et al.*, 2008. Language reorganization in aphasics: An electrical stimulation mapping investigation. *Neurosurgery*, 63: 487-497.